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NORDISKE ARBEJDSPAPIRER

N O R D I C W O R K I N G P A P E R S

NNR 2012: Responses from hearing process

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NNR 2012: Public consultation – Chapter 4 (Breastfeeding)

Organisation/ Company	General & detailed comments	WG response and actions
Lifelong European learning programme "Eating Together"	<p>I write on behalf of the Lifelong learning program n° 2011-1-BE3-GRU06-03638 (http://www.dietethics.eu/en/nutrition/national-guidelines.php). The Danish Animal Alliance takes part in this European group, collecting official dietary guidelines for vegan diets around the world to help to protect people who choose to live on vegan diets. The Nordic Nutrition Recommendations are very interesting. We have requested an in depth study of your very good drafts and previous editions by Stephen Walsh (coordinator of the International Vegetarian Union's science committee). His suggestions on the breastfeeding section are relevant to breastfeeding vegan and vegetarian mothers whose vitamin B12 intakes should be emphasized.</p> <p>Detailed comments "Breastfeeding and vitamin D and iron status" (starting Line 269) seems an appropriate section to add a useful comment on vitamin B12 intake for vegan and vegetarian mothers. Vegan and vegetarian mothers who are breastfeeding should ensure an adequate current intake of vitamin B12 in order to avoid risk of deficiency in the infant (http://ajcn.nutrition.org/content/52/6/1073.long http://www.biomedcentral.com/content/pdf/1471-2431-12-85.pdf).</p>	<p>Thank you for reminding us. A sentence about this has been added in the introductory text.</p>
LRF Mjöl, Sweden	<p>Detailed comments Line 210-211: Infant formula with intact cow's milk protein is not relevant for Sweden. Is it relevant for the other Nordic countries? Otherwise we suggest it should be clarified to prevent unnecessary worrying among parents.</p>	<p>Infant formula (for the age group discussed in the text you refer to) with intact cow's milk protein is not recommended or produced in any of the Nordic countries. However, the reference is relevant. The treatment in formula preparation may vary and as far as we know it is legal to import and sell any kind of formula in the Nordic countries as long as they agree with EU-regulations.</p>

Organisation/ Company	General & detailed comments	WG response and actions
Norwegian Directorate of Health	<p>General comments</p> <p>It is nice that you have done a SLR and that the results are thoroughly covered in this chapter. However, this information is extensive for the reader. You may consider making a table that summarizes the conclusions of the SLR. E.g. list outcome and grade of evidence.</p> <p>Detailed comments</p> <p>L 30-32 versus line 34-47. We appreciate that you use the stringent classification (L 30-32), but in the next section (L 34-47) you start to use expressions as “weaker” and “seems”. This is a bit confusing. The use of terminology is more stringent the abstract of the SLR, and the section regarding results may be repeated here.</p> <p>L 53-57 seems to be a repetition of the section L 34-47.</p> <p>L 61-67. The heading and the text in this section does not match. Consider to include the section on line 314-318 under this heading. L 62 Why just refer to energy yielding nutrients and not to other nutrients and dietary components? L 65-66 The sentence “Increasinglong-term beneficial effects .. health” seems rather general and misplaced.</p> <p>L 112 - 20% - in which group? General; the whole population; among adults; children?</p> <p>L 111-119 - is one long sentence. Consider to split it up.</p> <p>L 149-151 – this sentence is a bit difficult to understand. Regarding the protective effect in relation to type 1 diabetes both probable and suggestive evidence is mentioned in the same sentence.</p> <p>L 271-272 – you just spend one sentence on vitamin D and more space on iron. You may add some information about vitamin D. L 293 – vitamin D supplements. It would be nice if you can add information about at what age one should start to give vitamin D supplements and for how long.</p> <p>L 335-345 – the inclusion of the Cochrane review seems misplaced here. It would fit better under the corresponding sections about obesity and cognitive ability earlier in the chapter.</p> <p>L 358 table 1. This table is difficult to read. You may consider replacing it with a figure.</p>	<p>A table has been added as suggested (Table 1 in the end of the Introductory text).</p> <p>Thank you for the observation. This has now been changed.</p> <p>Agree and changed. The text has been combined and rephrased, and partly placed in the introductory text and partly in “Health benefits of breastfeeding”, which makes the text clearer. It regards the general population and this has now been clarified. Agree and has now been changed.</p> <p>We agree and have now changed this.</p> <p>Text about vitamin D is now added giving more information and in line with the chapter on vitamin D in the NNR 2012. The question about age, is a hard one, but after the first weeks is the best answer we have, in some of the Nordic countries it is after the first week in other it is about the age of one month.</p> <p>Agree and has now been changed.</p> <p>We do not find it right to replace the table</p>

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	<p>L 368-389 – this section is heavy to read. Consider to rephrase it.</p> <p>L 372-385 – It seems not proportionate that you spend so much space on Swedish data here.</p>	<p>with a figure as the accurate values referred to will then be lost. They are not published in detail anywhere else so we find this information very useful. Agree and has now been changed.</p> <p>Agree and has now been changed.</p>
National Food Agency, Sweden	<p>General comments The chapter needs revision of the language to improve clarity.</p> <p>Methodological issues with assessing outcomes of breastfeeding (for example lack of RCTs, many potential confounders, different definitions of exclusive breastfeeding etcetra) could receive more attention, perhaps even a separate heading.</p> <p>Detailed comments 43 Suggestion “..following a duration of breastfeeding longer than 6 months..”</p> <p>57 define later</p> <p>58-59 please revise to improve clarity. 76 Please clarify that the review for NNR5 led to the conclusion that the evidence was suggestive. 78-92 Could these hypotheses be summarized more briefly as to not take the focus from the conclusion that there may be several different mechanisms, where one is the simple issue of what foods are replacing breastmilk? 95-97 please revise sentences about reference 10. 111 Suggestion: “...breastfeeding may reduce overweight and obesity by 20%. 113-119 Please make this very long sentence at least two. Remove duplication of wording. 139-143 “The physiological explanation...” please revise sentence 165 Suggestion: replace “There seems also to be” with “There is”</p> <p>231 and onwards where differences in results from different trials are discussed: The difficulties in methodology and the potential strong confounding effect of other factors associated with breastfeeding</p>	<p>Agree and the language will be checked and corrected before publication. A separate heading in this chapter is not relevant. More text about methodological issues is proper and this is now in several places i.e. “Health benefits of breastfeeding” in the last paragraph; “Breastfeeding and growth, overweight and obesity” in the 3rd and last paragraphs; “Breastfeeding and atopy and asthma” in the 3rd paragraph; “Breastfeeding and neurological development” in the 1st and last paragraphs; and a sentence in “Breastfeeding and irritable bowel disease”.</p> <p>Agree and has now been changed.</p> <p>The sentence has been deleted.</p> <p>The text has now been changed in accordance with these and other comments.</p> <p>Have now changed this and e.g. added words</p>

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	<p>3. Generally the language within the overall paper needs editing, below I have tried to give some examples below but a thorough editing is recommended to improve understanding e.g. many sentences are too long.</p> <p>4. Made edits on a paper copy that could be scanned and sent if this is useful.</p> <p>5. Missing a conclusion</p> <p>Detailed comments</p> <p>Lines 34-40 There is convincing evidence that breastfeeding protects infants against the development of overweight and against infections (2). The evidence was weaker for other health outcomes: breastfeeding seems to: have a role in reducing serum cholesterol levels and blood pressure in childhood;; have beneficial effects on children’s IQ and developmental scores; protect against irritable bowel syndrome (IBS) and coeliac disease; when comparing any breastfeeding with none it seems to help prevent diabetes mellitus type 1 (T1DM) and type 2 (T2DM) (2).</p> <p>Lines 51-59 Exclusively breastfed means given no food or liquids (e.g. teas, water) other than breast milk. Vitamin D supplements are recommended for all infants being breastfed in the Nordic countries. More information is needed especially about links between exclusive breastfeeding and allergy, where results vary between studies. For example there is no evidence for an association between decreased risk of allergy and later introduction of food. If breastfeeding is for some reason not possible or not chosen, infant formula formulated according to Codex rules is recommended.</p> <p>Lines 97-106 – needs rephrasing with shorter sentences because hard to understand In this Randomised Control Trial (RCT) all infants were exclusively breastfed at 4 months and then randomized into the two different groups while continuing breastfeeding and similar growth and body composition were seen in both groups (10). Selective reporting or publication bias in this area cannot be totally excluded and well-performed prospective studies with longer duration of breastfeeding and follow-up data are still needed. (11) found that smaller size was strongly associated with increased risks of subsequent weaning and of discontinuing exclusive breastfeeding especially between 2 and 6 months which may interfere with the results showing longer duration of exclusive breastfeeding in (12) associated with larger size at 6.5 105 years or a confounder.</p>	<p>criteria, and there we write “Health effects in the mother, as well as other potential effects, such as the bonding between mother and child, have not been reviewed.”</p> <p>Thank you. Your suggested changes have now been used as far as they do not overlap with some other valid suggestion by other reviewers. The manuscript of the chapter will get a thorough English review before publication.</p> <p>Thank you so much. The English will be improved before final publication.</p> <p>Hope this is clear now.</p> <p>This is now changed in the final version.</p> <p>This is now changed in the final version.</p> <p>This is now changed in the final version.</p> <p>This is now changed in the final version.</p>

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	<p>Lines 108-119 Nordic collaboration with data from prospective longitudinal infant cohorts is urgently required as they are likely to provide the best possibilities for improved studies on longer duration of both exclusive and any breastfeeding relevant to the Nordic populations. International reports have concluded that breastfeeding may diminish overweight and obesity by about 20%. WHO has developed new growth charts which are more relevant for breastfed infants and these may help to take the pressure of breastfeeding women to give their babies formula or other food too early, and also decrease the risk of overfeeding. These new growth charts give a more realistic view of normal growth rates (13, 14 15, 16), (17)</p> <p>Lines 122-132 There is convincing evidence of a protective effect against the overall incidence of infections including acute otitis media, gastrointestinal and respiratory tract infections (2). The immunological protection against infections appears to last for some years after cessation of breastfeeding. Thus, breast milk contains many protective factors that may exert long-term health benefits. The magnitude of the effect varies depending on the specific outcome and the exclusiveness of breastfeeding. A protective dose/duration-response effect on gastrointestinal or respiratory tract infections was found in the ??? (SLRs) of Dujits et al and Kramer (18, 19), as well as in the prospective studies by Fisk et al and Ladomenou et al (20, 21). In other reports the lowered risk of about 20% for otitis media, 50% for gastroenteritis and about 30% for lower respiratory tract infections have also been cited(13, 14).</p> <p>LINES 135-162 The influence of breastfeeding on the risk of CVD is unclear but there is probable evidence of significant small effects on the reduction of blood pressure levels and blood cholesterol levels later in life i.e. in adulthood (2). Breastfeeding has also been associated with lower blood pressure levels in childhood and adolescence (22), but more evidence is needed. The physiological explanation might be through lower salt intake and higher intake of n-3 fatty acids in breastfed infants resulting in more elasticity of the blood vessels. Similarly the lower serum cholesterol levels in adulthood might be as a result of constituents in breast milk such as cholesterol and n-3 fatty acids. Even though the physiology is unclear reports do agree on a small but significant effect of breastfeeding on reducing blood pressure (13, 14, 15) as well as blood cholesterol levels (13, 15) in adulthood.</p> <p>Breastfeeding and diabetes mellitus. There is probable evidence that breastfeeding is protective against diabetes mellitus (type 1 and 2). The evidence of a large protective effect of duration of breastfeeding is limited though suggestive against evidence of diabetes type 1 (2). This protective effect of breast milk may depend on the different proteins in breast milk compared with infant formula. However but other routes of association between short duration of breastfeeding or the introduction of food or drinks and diabetes have been suggested. A systematic literature review supports the evidence that longer duration of breastfeeding compared with short-term may contribute to reduced risk of development of DM type 1. Reports from EFSA (2009) and a joint statement from the UK's COT/SACN (2011) recommend the introduction of gluten containing foods while still breastfeeding gives some protection against diabetes type 1 (23, 24). Both SACN (15) and WHO (13) state that infants who are not breastfed are at greater risk of type 2 diabetes.</p>	<p>This is now changed in the final version.</p> <p>This is now changed in the final version.</p> <p>This is now changed in the final version.</p> <p>This has now been deleted.</p> <p>This is now changed in the final version.</p>

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	<p>Lines 327-388 Knowledge of this difference and that the results do not conflict with each other is important. Do not understand this sentence?</p> <p>Lines 335- 345 WHOLE paragraph NEEDS REPHRASING</p> <p>Lines 350-351 – recommendation should be added to recommend disaggregating data into different social strata in order to reduce inequalities in Nordic countries.</p> <p>Lines 368-377 Could these data (comparing NNR4 with NNR5) be better displayed as a table?</p>	<p>Do not think so. There is another table including a lot of data and the text content has now been made smaller.</p>
Amningshjälpen, Sweden	<p>General comments</p> <p>Although we are pleased to see this work to have been done we have some comments about the text. General considerations: Lack of comments on breastfeeding for more than 6 months and for over 12 months. If there is total lack of research in this area this should be mentioned. We also want to question whether formula feeding is considered the same as bottle feeding or if there are studies in different ways to give formula. In Sweden we have noticed a rise in feeding formula from a nursing system, or probe attached to the breast. We want to remind that formula feeding can be done in various ways. And overall, formula can be substituted with breastmilk from other lactating women.</p> <p>Detailed comments</p> <p>58 We wish to point out that breastmilk too is a very nice substitute instead of breastfeeding. Specially for newborns and premature and low weight for gestation age children. There is a lack of comments on evidence showing even more benefit in promoting breastfeeding and supplement feed with breastmilk for premature and low weight for gestation age children.</p>	<p><u>WG observation and/or action:</u> In general the chapter focuses on all issues where we might have evidence at this moment and that is the strength of NNR. This is the reason why we do not touch upon all interesting theories, where the research needs more time. And to your comments: There are too few studies on exclusive breastfeeding for more than 6 months and on breastfeeding for more than 12 months to be mentioned here, even though interesting. The most general time periods were used in the systematic literature review this chapter is based on and that is the reason for this scarceness – sorry. Formula feeding is not the same as bottle feeding – too few studies existed which differentiated between formula given in a bottle or given in another way (or breast milk given in a bottle) for a review on that question. We have now added a text in the introductory section “In addition, too few studies existed to enable reviewing the importance of the product vs. the mode of feeding i.e. differentiating between breastfeeding and breast milk given in a bottle, and formula given in a bottle or given in another way.”</p> <p>Agree, but premature and sick children were not included in the Systematic literature review. Text is now added in the beginning</p>

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	<p>214-241 There is a lack of evidence about neurological conditions as a barrier for breastfeeding. It should be mentioned in the text. There is some evidence for example that babies, that later in life is diagnosed with ADHD, exhibit special features while breastfeeding. And we don't yet know if those features hinder breastfeeding or not. But if those features do hinder breastfeeding it would be yet one explanation for the difference in development between these two groups.</p> <p>269-283 There is research now showing that cutting the umbilical cord too early is the greatest impact on the iron status for the small child. This should be mentioned in the text because many advisors keep referring to breastmilk as a possible risk for develop low iron levels in the blood of the small child and therefore recommending mothers to cease exclusive breastfeeding against the wishes of the mother and child. Also, there are research suggesting a negative impact on the health of grown ups when ingesting too much iron for example by taking food supplements. Therefore it is a dangerous path highlighting the low iron content in breastmilk as a possible health hazard. We found no evidence supporting the old theory of exclusive breastfeeding causing iron deficiency as long as the mother is not badly nourished. We look forward to the end of the iron debate when it come to breastfeeding and breastmilk.</p> <p>301-306 and 358-366 Table 1. We wish to highlight the fact that there is a group of young children that won't eat almost anything until they are about a year old. From what we know from the tales of these mothers their children are doing fine. Though, we need more research about exclusive, or almost exclusive, breastfeeding in children from 6 to 12 months of age. "From the field" we have learned that these mothers either lie to their health profession or cease breastfeeding too early trying to force their babies to start eating solid food instead of breastmilk. Research show that parents tend to tell their health profession what they think they want to hear. It is very unfortunate that we are not able to support these mothers and babies to continue breastfeeding due to lack of evidence. And specially since the NNR5 in line 358 Table 1 shows how low the frequency of breastfeeding beyond 6 months is in Sweden. Therefore we find this matter to be of great importance. We also want to question the belief that babies need to develop the skill of eating solid food at a certain age while skills like walking and talking have a much longer expected time interval. Many mothers now use the baby-led-weaning method when introducing solid food to their babies and children. Therefore there is a need for the authorities to follow up with a review of research done in this matter.</p>	<p>about inclusion and exclusion criteria, and there it says now: "Studies including sick or preterm infants have also been excluded but it is likely that health effects of breastfeeding are more pronounced in vulnerable infants such as these groups "</p> <p>This sounds very interesting but as we know so little it is out of the scope of this chapter this time.</p> <p>Text about this is now added.</p> <p><u>WG observation and/or action:</u> Agree and hope to be able to deal with this later on, however it was not part of the present literature review.</p>

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Norwegian Resource Centre for Breastfeeding	<p>General comments</p> <p>The Norwegian Resource Centre for Breastfeeding will take the opportunity to comment the draft chapter on breastfeeding in the Nordic Nutrition Recommendation. We support the concluding recommendations for exclusive breastfeeding and breastfeeding duration. We would like to congratulate the working group for a good systematic review, and have only minor comments, suggestions and questions.</p> <p>Detailed comments</p> <p>55 Proposed specification: More information is needed especially about exclusive breastfeeding BEYOND 4 MONTHS and allergy.</p> <p>59 For how long is infant formula recommended? When can ordinary cow's milk be introduced?</p> <p>66 Reference to other systematic reviews and meta-analysis.</p> <p>69 The protective effect of breastfeeding against obesity is assessed as convincing. The study by Kramer et al. JAMA 2013; 309:1005-13 questions this conclusion, and we recommend to substitute "convincing" with "probable".</p>	<p>Thank you so much.</p> <p>The whole issue on allergy and breastfeeding is quite contradictive now and research is scarce on longer duration of breastfeeding, so we prefer to not include any time periods. Ordinary cow's milk can probably be introduced in small amounts in the last months of the first year without any detected negative effects. However, as this amount is not defined and people do not necessarily measure the intake of usual cow's milk, it is preferred to recommend avoidance of ordinary cow's milk during the first year. After one to two years of age would be the most correct answer, as both 1 and 2-year-olds have been shown to develop iron deficiency with intake of cow's milk. This is dealt with in the iron chapter.</p> <p>The references follow in each of the sub-chapters on each of the outcomes in the text that follow.</p> <p>Yes we know. The reference you mention compares groups with less difference in breastfeeding duration than the Nordic systematic review referred to in the chapter. The smaller difference in intake i.e. breastfeeding might not be large enough to indicate the relevant difference. This is explained in the chapter now i.e. last</p>

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	<p>69- Lower levels of protein in human milk compared to formula has also been hypothesized as a mechanism for the protective effect of breastfeeding (Escribano et al, 2012)</p> <p>112 The WHO Child Growth Charts are relevant for both breastfed and formula fed infants, as breastfed babies constitutes the standard for growth / optimal growth. We would recommend to exchange the word “realistic” with “optimal”.</p> <p>174 – A study by Strandl et al. could be of interest to understand the conflicting evidence of breastfeeding and asthma (see below).</p> <p>215 Regarding the effect of breastfeeding on neurological development, we assess the effect as convincing. The consistency in results, even in populations where breastfeeding is inversely correlated to socio-economic status (Brion, 2011, reviewed in Agostini, 2009) and in the PROBIT trial with small differences in breastfeeding rates between intervention groups and control groups, lead us to this conclusion.</p> <p>314 There is considerable knowledge on mechanisms which can explain the positive effects of breastfeeding; i.e. the enteromammaric link. The paragraph should mirror this to a larger degree, although this is not the place for explaining in detail. The paper by Lucas and Cole, Lancet 1990 could be a reference. This randomised controlled trial demonstrated that premature babies given human milk by bottle, had an IQ 8.3 higher than formula fed babies at the age of 7 years, demonstrating that constituents in human milk were active irrespective of feeding mode.</p> <p>320- We recommend that the following information is included in this section, to document that there is an effect of EBF for 6 months: The Kramer & Kakuma Cochrane Database Syst Rev. 2012 on Optimal duration of exclusive breastfeeding concludes that there is less gastrointestinal infections in infants exclusively breastfed for 6 months compared to those EBF for 4 months. Proposed addition on Sudden Infant Death Syndrome: A meta-analysis by Hauck et al (2011) including 18 good quality studies, concluded that “Breastfeeding is protective against SIDS, and this effect is stronger when breastfeeding is exclusive”. Relevant papers Effect of protein intake and weight gain velocity on body fat mass at 6 months of age: the</p>	<p>paragraph in “Breastfeeding and growth, overweight and obesity”.</p> <p>This is now described under the heading “Breastfeeding and growth, overweight and obesity”</p> <p>Agree and this is now changed.</p> <p>A sentence about this is now added.</p> <p>Thank you for this interesting information. We explain the findings in the systematic literature review (SLR) used as a base for NNR and the chapter. The SLR came to the conclusion that this association was of a 2nd grade of evidence, and this is mirrored in the chapter. The references you mention are taken into account in the SLR. We found as you that there are no contradicting results in the literature (no negative effects), but several findings become insignificant when adjusted for socioeconomic factors. More research might reveal that your conclusion is right, but at this moment we could not go any further.</p> <p>This has now been taken into consideration in the chapter.</p>

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	<p>EU Childhood Obesity Programme. Escribano J, Luque V, Ferre N, Mendez-Riera G, Koletzko B, Grote V, Demmelmair H, Bluck L, Wright A, Closa-Monasterolo R; European Childhood Obesity Trial Study Group. Int J Obes (Lond). 2012 Apr;36(4):548-53. Pediatrics Unit, Universitat Rovira i Virgili, IISPV, Reus, Spain.</p> <p>Abstract INTRODUCTION: Higher protein intake during the first year of life is associated with increased weight gain velocity and body mass index (BMI). However, the relationship of protein intake and weight gain velocity with body composition is unclear. OBJECTIVE: To assess if the increases in weight gain velocity and BMI induced by protein intake early in life are related to an increase in fat or fat-free mass. MATERIALS AND METHODS: In all, 41 infants randomized at birth to a higher or lower protein content formula (HP=17 and LP=24, respectively) and 25 breastfed infants were included. Anthropometric measures were assessed at baseline, 6, 12 and 24 months, and fat-free mass (FFM) and fat mass (FM) were assessed by isotope dilution at 6 months. RESULTS: Weight gain velocity (g per month) during the first 6 months of life was significantly higher among HP infants (807.8 (±93.8) vs 724.2 (±110.0) (P=0.015)). Weight gain velocity strongly correlated with FM z-score (r=0.564, P<0.001) but showed no association with FFM z-scores. FFM showed no association with BMI. Nevertheless, FM strongly correlated with BMI at 6, 12 and 24 months (r=0.475, P<0.001; r=0.332, P=0.007 and r=0.247, P=0.051, respectively). FFM and FM z-scores did not differ significantly between HP and LP infants (0.32±1.75 vs -0.31±1.17 and 0.54±2.81 vs -0.02±1.65, respectively). CONCLUSION: Our findings support the hypothesis that higher protein intakes early in life are associated with faster weight gain and in turn to higher adiposity. This mechanism could be a determinant factor for later obesity risk. FADS gene cluster modulates the effect of breastfeeding on asthma. Results from the GINIplus and LISAplus studies. Standl M, Sausenthaler S, Lattka E, Koletzko S, Bauer CP, Wichmann HE, von Berg A, Berdel D, Krämer U, Schaaf B, Lehmann I, Herbarth O, Klopp N, Koletzko B, Heinrich J; GINIplus and LISAplus Study Group. Allergy. 2012 Jan;67(1):83-90. Helmholtz Zentrum München, German Research Centre for Environmental Health, Institute of Epidemiology I, Ingolstädter Landstrasse 1, Neuherberg, Germany. Abstract BACKGROUND: The protective effect of breastfeeding (BF) on the development of asthma has been widely recognized, even if not all results have been consistent. Gene variants of the FADS gene cluster have a major impact on fatty acid composition in blood and in breast milk. Therefore, we evaluated the influence of the FADS1 FADS2 gene cluster polymorphisms on the association between BF and asthma. METHODS: The analysis was based on data (N=2245) from two German prospective birth cohort studies. Information on asthma and BF during the first 6 months was collected using questionnaires completed by the parents. Logistic regression modelling was used to analyse the association between exclusive BF and ever having asthma stratified by genotype. RESULTS: In the stratified analyses, BF for 3 or 4 months after birth had a protective effect for heterozygous and homozygous carriers of the minor allele (adjusted odds ratio between 0.37 (95% CI: 0.18-0.80) and 0.42 (95% CI: 0.20-0.88). Interaction terms of BF with genotype were significant and ranged from -1.17 (P-value: 0.015) to -1.33 (0.0066). Moreover, heterozygous and homozygous carriers of the minor allele who were exclusively breastfed for 5 or 6 months after birth had a reduced risk of asthma [0.32 (0.18-0.57) to 0.47 (0.27-0.81)] in the stratified analyses. For individuals carrying the homozygous major allele, BF showed no</p>	

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	significant effect on the development of asthma. CONCLUSIONS: The association between exclusive BF and asthma is modified by the genetic variants of FADS genotypes in children.	
National Institute for Health and Welfare, Finland	<p>General comments only</p> <p>p. 4, paragraph on Breastfeeding and diabetes mellitus</p> <p>o The statement that “there is probable evidence that breastfeeding is protective against diabetes mellitus (type 1 and 2)” is an overstatement especially regarding type 1 diabetes. The review paper by Hörnell et al. (done for NNR5) does take into account only one prospective cohort study, where pre-type 1 diabetes was used as an endpoint for type 1 diabetes (Couper et al. 2009), whereas three other major ones were forgotten (Norris et al. JAMA 2003, Ziegler et al. JAMA 2003, Virtanen et al. Diabetologia 2006). These authors did not find any association between breastfeeding and development of early or advanced pre-type 1 diabetes. As there are very few cohort studies available with clinical type 1 diabetes as an endpoint these cohorts should be considered. Case-control studies give much worse evidence for causality as compared to prospective cohorts.</p> <p>o The statement that “introduction of gluten containing foods while still breastfeeding gives some protection against type 1” is an overstatement. This evidence is really very weak and based on too small numbers.</p>	<p><u>WG observation and/or action:</u> Thank you. The papers you suggest included mainly infants/children with especially high risk for diabetes and were therefore not included in the 5th ed. NNR, as explained in the section on exclusion criteria. However, some of the papers you mention were parts of literature reviews included in the systematic literature review (NNR 5 SLR), e.g. Norris et al 2003 and Ziegler et al 2003. When looking at the papers we have found that some of them support the conclusion of the SLR, i.e., an association between any breastfeeding and lower risk of diabetes type 1. Norris et al JAMA 2003 saw a reduced risk for islet autoantibody in breastfed when the first introduction to cereals was while still breastfed (Table 3 (hazard ratio 0.50 (CI: 0.25-0.99)). Ziegler et al. JAMA 2003 saw an increased risk if gluten was given before the age of 3 months, but those who got gluten so early were not breastfed, also they found shorter breastfeeding among mothers with type 1 diabetes. In the study on high risk population Virtanen et al 2006 found that the overall or exclusive duration of breastfeeding was not associated with the risk of beta cell autoimmunity (which could be a sign for later diabetes type 1) but all participants got some breastfeeding. We have added this reference to the chapter. Please note that in the chapter we only</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>This sentence should be removed. p. 4, paragraph on Breastfeeding and atopy and asthma o The review paper by Hörnell et al. is missing many major prospective cohort studies about infant feeding and the risk of asthma and atopy (Table 8 and 9). E.g., Swedish study by Hesselmar et al. (Acta Paediatr 2010), Finnish studies by Nwaru et al (Pediatrics 2010), Virtanen et al. (BJN 2010) were not included. o This paragraph could be written in a more concise and balanced way, taking all the evidence into account. The evidence regarding infant feeding and development of asthma and atopic diseases is conflicting and findings of prospective studies more and more suggest that actually early age at introduction of new foods is associated with decreased risk of atopic asthma and other allergic diseases. There may not yet be time for reconsideration of infant feeding recommendations, but one should not either forget those studies which give “not nice results”.</p> <p>p.9; the last paragraph should read as: In Finland breastfeeding has been measured every 5-year period starting from 1995 up to 2010. Exclusive breastfeeding at four months has increased from 10 % to 15% and further to 34% and is in 2010 going down again to 23%. Total breastfeeding at six months was respectively 40%, 49%, 55% and 58% during years 1995, 2000, 2005 and 2010.</p>	<p>found the association probable for decreased risk of diabetes type 1 and any breastfeeding (as compare to none). All these papers include children with increased risk for diabetes, and one of them support the results of the NNR 5 SLR, but two do not really test the same question. We have clarified and adjusted the text about the introduction of gluten containing foods while still breastfeeding, thanks for pointing this out.</p> <p>The papers included infants/children with high risk for allergy and as said above and as stated in the NNR 5 systematic literature review one of the exclusion criteria was if the mother or child was sick at start or at increased risk for disease. This is a very important area of research and text is now included in the chapter referring to the studies you mention.</p> <p>The text has been adjusted a little to decrease the amount of numerical information, but this information is now added.</p>
University of Bergen, Norway	<p>In general the chapter on breastfeeding in the NNR 2012 gives a good background for the given recommendations. Here are some few comments and suggested changes:</p> <p>It could be worthy to also mention the immune related factors and breast milk as a source of biochemical messages, to make it clear that breast milk has more beneficial factors than its macronutrients, vitamins and minerals. I suggest that the following sentence could be included between the sentences in line 22: “In addition to the macronutrients, vitamins and minerals in breast milk, breast milk also contains immune related factors and hormonal factors important for infant health and growth (Wagner et al. and Bernt et al.)”</p> <p>Wagner CL, Anderson DM, Pittard WB, 3rd: Special properties of human milk. Clin Paediatr (Phila) 1996, 35(6):283-293.</p>	<p>Agree and text about this is now included in the chapter.</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>Bernt KM, Walker WA: Human milk as a carrier of biochemical messages. Acta Paediatrica 1999, 88:27-41. In the sentence before, it could be nice to include references: "Breast milk gives the newborn essential nutrients in efficiently absorbable combination, and has positive health effects on the child as well as later in life." I suggest the reference by Ogra et al. before the comma (Ogra PL, Greene HL: Human milk and breast feeding: an update on the state of the art. Pediatr Res 1982, 16(4 Pt 1):266-271.) and the reference 14 by Ip et al. (which is already included in the reference list) before the dot.</p> <p>It could also be useful to add that the content of human milk differs from e.g. cow's milk. I suggest the following sentence to be included after the above mentioned sentences: "Both the macronutrient content, the content of different vitamins and minerals, -immune related factors and hormonal factors differ between human milk and other animal milks such as e.g. cow's milk (Sellen, Cuthbertson)."</p> <p>Sellen DW: Evolution of infant and young child feeding: implications for contemporary public health. Annu Rev Nutr 2007, 27:123-148.</p> <p>Cuthbertson WF: Evolution of infant nutrition. Br J Nutr 1999, 81(5):359-371.</p> <p>It is written that "There is convincing evidence for that breastfeeding protects against the development of overweight and against infections (2)." I completely agree with this and would have also added "autoimmune diseases" (as inflammatory bowel disease, atopic eczema/dermatitis, coeliac disease, diabetes mellitus type 1, asthma and allergy all are pathologically related.) The following references could here be useful:</p> <ol style="list-style-type: none"> 1. Barclay AR, Russell RK, Wilson ML, Gilmour WH, Satsangi J, Wilson DC: Systematic review: the role of breastfeeding in the development of pediatric inflammatory bowel disease. J Pediatr 2009, 155(3):421-426. 2. Oddy WH, Holt PG, Sly PD, Read AW, Landau LI, Stanley FJ, Kendall GE, Burton PR: Association between breast feeding and asthma in 6 year old children: findings of a prospective birth cohort study. Brit Med J 1999, 319(7213):815-819. 3. Ip S, Chung M, Raman G, Chew P, Magula N, DeVine D, Trikalinos T, Lau J, Chelmow D, Dewey KG et al: Breastfeeding and Maternal and Infant Health Outcomes in Developed Countries. Evidence Report/Technology Assessment No. 153. Edited by Tufts-New England Medical Center Evidence-Based Practice Center (under Contract No. 290-02-0022). B, Massachusetts, Agency for Healthcare Research and Quality (AHRQ) R: Tufts-New England Medical Center Evidence-Based Practice Center, Boston, Massachusetts; 2007. (reference already in the reference list of breastfeeding, NNR 2012) 4. Hanson LA: Human milk and host defence: immediate and long-term effects. Acta Paediatr Suppl 1999, 88(430):42-46. 5. Kramer MS, Chalmers B, Hodnett ED, Sevkovskaya Z, Dzidkovich I, Shapiro S, Collet JP, Vanilovich I, Mezen I, Ducruet T et al: Promotion of Breastfeeding Intervention Trial (PROBIT): a randomized trial in the Republic of Belarus. Jama 2001, 285(4):413-420. (reference already in the reference list of breastfeeding, NNR 2012) 6. Akobeng AK, Ramanan AV, Buchan I, Heller RF: Effect of breast feeding on risk of coeliac disease: a systematic review and meta-analysis of observational studies. Arch Dis Child 2006, 91(1):39-43. (reference already in the reference list of breastfeeding, NNR 2012) 	<p>Agree and text about this is now included in the chapter.</p> <p>Agree and have now added text and the references you mention.</p> <p>These references are useful and most of them already included in the NNR 5 SLR the breastfeeding chapter is based on as part of a included SLR or as such, as you point out. The evidence, however, is not convincing for all of these outcomes (grading varies between grades 2-4). However, the text is now rephrased to make the discussion clearer.</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>7. Hoppu U, Kalliomaki M, Laiho K, Isolauri E: Breast milk--immunomodulatory signals against allergic diseases. Allergy 2001, 56 Suppl 67:23-26.</p> <p>Similarly, I suggest that the sections/paragraphs on "Breastfeeding and diabetes mellitus", "Breastfeeding and IBD", "Breastfeeding and coeliac disease" and "Breastfeeding and atopy and asthma" are discussed under the same title "Breastfeeding and auto-immune diseases". It is worthy to note that reference 25 by Yang et al. concludes as written that "There is no strong evidence of a protective effect of exclusive breastfeeding for at least 3 months against AD". However, it also concludes that "Breastfeeding was associated with a decreased risk of AD". Thus, breastfeeding compared to no breastfeeding can be protect against atopic dermatitis although no significant differences between exclusive breastfeeding and non-exclusive breastfeeding has been seen in terms of atopic dermatitis. (Yang, W., et al. (2009). "Exclusive breastfeeding and incident atopic dermatitis in childhood: a systematic review and meta-analysis of prospective cohort studies." The British journal of dermatology 161(2): 373-383.)</p> <p>In line 49, the sentence is written in passive "It is recommended". I agree that it is a reasonable recommendation and think it will be useful to add the source of the recommendation (e.g. the WHO reference number 55).</p>	<p>Understand your point, but the NNR 5 SLR was made with the outcomes the chapter includes as sub-headings, so it was decided not to change the order of sections. The text is now rephrased under the sub-heading of "Breastfeeding and atopy and asthma".</p> <p><u>WG observation and/or action:</u> Agree and this has now been added.</p>
NIFES, Norway	<p>General comments</p> <p>Vi savner et avsnitt som omhandler mors kostholds påvirkning på næringsstoffsammensetningen i brystmelken. Litteraturen tyder på at mors kosthold påvirker nivåene av DHA i morsmelk, og at nivåene er høyere hos mødre som for eksempel spiser sjømat eller tar supplement. Det er også mulig at et høyt inntak av omega-6-fettsyrer reduserer egensyntese av marine omega-3-fettsyrer. Vi ber dere vurdere teksten i anbefalingene i lys av at tilsetning av marine omega-3 fettsyrer i morsmelkerstatning er relativt nytt i nordisk sammenheng, og at morsmelkerstatning i dag i mange tilfeller vil ha et høyre innhold av marine omega-3 fettsyrer enn brystmelk i dag.</p> <p>Detailed comments</p> <p>Når det gjelder de marine omega-3-fettsyrene virker det som man har fastslått at morsmelk inneholder høye nivåer av for eksempel docosahexaensyre (DHA) og at nivåene er høyere i brystmelk enn i morsmelkerstatning. For eksempel linje 139-143 "The physiological explanation might possibly be on blood pressure through lower salt intake of breastfed infants and and the higher intake of n-3 fatty acids of breastfed infants might be associated to...." og linje 217-219 "The favorable effects of breastfeeding on healthy neurological development of the infant may be caused by the high content of docosahexaenic acid (22:6, n-3) in breast milk, ..." Har dere godt nok belegg til å slå fast at brystmelk inneholder mer marine n-3 fettsyrer enn morsmelkerstatning? Referanser: Gibson RA, Muhlhausler B, Makrides M (2011). Conversion of linoleic acid and alpha-linolenic acid to long-chain polyunsaturated fatty acids (LCPUFAs), with a focus on</p>	<p>Thanks for pointing this out. We have now added some text and three references considering this in „Breastfeeding and development of chronic disease" and subheading "Breastfeeding and risk factors for cardiovascular disease"</p> <p>Thanks for pointing this out. We have now added text about this.</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>pregnancy, lactation and the first 2 years of life. Maternal & Child Nutrition 7 (Suppl 2), 17-26. Innis SM (2007). Human milk: maternal dietary lipids and infant development. Proceedings of the Nutrition Society 66, 397-404. Koletzko B et al. (2008). The role of long-chain polyunsaturated fatty acids in pregnancy, lactation and infancy: review of current knowledge and consensus recommendations. J Perinata Med 36, 5-14.</p>	
National Breastfeeding Committee, Sweden	<p>General comments</p> <p>The Swedish National Breast Feeding Committee welcomes this overview of the benefits of breastfeeding and has the following comments: 1. For the sake of clarity it would be appropriate to state in the beginning of the chapter that the overview only covers the health effects in the child and that health effects in the mother, as well as other potential effects, such as the bonding between mother and child, have not been reviewed. It could also be mentioned that the chapter does not cover health conditions in which breast feeding is contra-indicated.</p> <p>2. It would be very helpful for the reader to have an overview presented in a table at the end of the chapter with all mentioned health effects and their respective level of evidence.</p> <p>3. In the chapter some issues are formulated in a subjective or defensive way, rather than in an objective manner. Examples of this are found at lines 235 and 338.</p> <p>4. The advantages of breast milk/breastfeeding for premature infants of low birth weight for gestational age could be mentioned. This is a group for which exclusive breastfeeding is particularly beneficial.</p>	<p>Agree and has now added text about this in the introduction</p> <p>Agree and has now added a table as suggested.</p> <p>Agree and has now changed this.</p> <p>Has now added text in the beginning about inclusion and exclusion criteria, and there it says "Studies including sick or preterm infants have also been excluded but it is likely that health effects of breastfeeding are more pronounced in vulnerable infants such</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>5. The chapter focuses on breastfeeding during the first six months. Comments on research on breastfeeding on partial breastfeeding for 12 months or longer are lacking. If there is no evidence for continued breastfeeding after six months this should be mentioned. If there is a need of further research in the area this could as well be mentioned.</p> <p>6. There is an inconsistent use of numbers (e.g. six and 6), and per cent and % and the text needs proofreading.</p> <p>Detailed comments</p> <p>49: It could be clarified who is recommending 6 months exclusive breastfeeding.</p> <p>82-84: It might be relevant to mention that formula has previously been much higher in protein than breast milk, but that this has now been changed.</p> <p>113-119: The following wording has been doubled: "which might growth".</p> <p>102-103: Sentence starts with reference number, name of author is missing. The sentence is in general unclear. Is it low-weight babies that are likely to be given other foods earlier?</p> <p>121: It would be more relevant to write breast milk than breastfeeding. Also consider replacing the word anti-infective. Alternative headings could be considered, e.g. "Breastfeeding and infections" (in line with other headings)</p> <p>123-125: Minor re-wording could bring greater clarity to the text, e.g. "There is convincing evidence of a protective effect against overall infections, acute otitis media, gastrointestinal and respiratory tract infections (2). Breast milk contains many protective factors that may have long-term effect on health. The immunological protection against infections lasts for some years after cessation of breastfeeding."</p> <p>139-145: Could benefit from reformulation for improved understanding.</p> <p>145: This sentence could be moved to line 143 to improve understanding of the text.</p> <p>157: "diabetes" is missing after "type 1".</p> <p>168: Consider replacing of "evaluation" by "review".</p> <p>171: Consider replacing "applicable to judge" by "assessing".</p> <p>175-176: Clarification on the studies reviewed is needed. "Of two.." is a confusing beginning of the sentence.</p> <p>182: Clarification needed. Did the prospective studies come to the same conclusions as the studies referred to earlier in the section?</p> <p>215-241: Could be relevant to mention the difficulties of excluding confounding factors in this area.</p> <p>223: Consider whether keeping the phrase about the studies that are not supportive or the statement that they can be "explained".</p> <p>228: "Children breastfed..." is a smoother formulation than only "Breastfed...".</p> <p>254-255: The mentioning of "not later than 6 months" and "too late (>7 months)" might be confusing. 255: Unclear with what COT/SACN agrees.</p>	<p>as these groups"</p> <p>Text is now added in the beginning about this "The amount of studies on health effects of partial breastfeeding for 12 months or longer was also to scarce for any conclusions"</p> <p>Agree and has now changed this.</p> <p>Agree and text is now added about this.</p> <p>Agree and text is now added about this.</p> <p>This has now been changed.</p> <p>Agree and has now added text about this.</p> <p>This has now been changed.</p> <p>This has now been changed</p> <p>Has been improved.</p> <p>Has been changed</p> <p>Has been changed</p> <p>Has been changed</p> <p>Has been changed</p> <p>Has been changed</p> <p>This has now been clarified.</p> <p>Some text has now been added.</p> <p>This has now been clarified.</p> <p>Agree and this has now been changed.</p> <p>This has now been clarified.</p> <p>A review of the English will be made</p>

Organisation/ Company	General & detailed comments	WG response and actions
	<p>273-274: The sentence needs clarification.</p> <p>286-345: Recommendation for breastfeeding would preferably be the last section of the chapter.</p> <p>290: Consider replacing “the addition” by “supplements”.</p> <p>294-296: It could be mentioned that, if available, expressed breast milk, own or from others, could be given in the place of infant formula.</p> <p>We would also suggest deletion of some sentences in this section: a. Line 302: “under the supervision of the national health care system”. This gives the impression that the health care system is controlling what parents give to their children. b. Line 308-312: “Special guidance effects on health.” This part is out of the area of responsibility of the NNR expert group and it contains assumptions that are not confirmed. c. Line 324-331: “This should not..... Breastfeeding after 6 months”. Unclear what this means.</p> <p>315-316: Consider the wording “action of breastfeeding”. Alternatives could be “actual breastfeeding”, “feeding practice” or similar.</p> <p>331-333: The following sentence could preferably be moved to the introduction of the chapter: “The definition of breast feeding in the literature.”</p> <p>335-345: This section feels slightly misplaced. It does not concern the actual recommendation, but the importance paid to a particular review.</p> <p>379: Specify that rates are increasing in all NORDIC countries except Sweden.</p>	<p>Has now been changed.</p> <p>Agree and has now been changed.</p> <p>Agree and has now added this.</p> <p>Thank you – done.</p> <p>Agree and has now changed this to “the act of breastfeeding”</p> <p>Agree and has now changed this.</p> <p>Agree and this has now been moved.</p> <p>Agree and has now changed this</p>
Confederation of the Danish Food Industry	<p>General comments</p> <p>There is much emphasis the Nordic Nutrition Recommendations are based on solid scientific evidence. Thus we suggest the rationale for the recommendation on breastfeeding is made in line with the scientific evidence. Although we agree to the recommendation on breastfeeding, and agree breastfeeding decreases the risk of developing allergies, we question whether there is sufficient evidence to suggest it prevents obesity later in life.</p>	<p>Thank you for your comment. Based on the systematic literature review it concludes as stated in the chapter “There is convincing evidence of a protective effect of breastfeeding, exclusive or any, against overweight and obesity in childhood and adolescence. The evidence is probable that exclusive breastfeeding for longer than 4 months is associated with slower weight gain during the second half of the first year, compared with shorter duration”. We have noted that different observations may come to different conclusions depending on which groups are compared. If e.g. one is comparing groups which are very similar e.g. in breastfeeding you see no significant difference in the outcome tested, but if</p>

Organisation/ Company	General & detailed comments	WG response and actions
		comparing groups which really differ e.g. in being breastfed or not, or in the duration of breastfeeding a difference is seen in the outcome.

Public consultation NNR 2012: Chapter 5 (Foods, food patterns and health outcomes: Guidelines for a healthy diet)

Organisation Company	General and detailed comments	WG response & actions
Måbrahälsa, Sweden	<p>Only detailed comments:</p> <p>Line 330 "Suggestive evidence was found for whole grains, cereal fibre, dairy products and prudent dietary patterns to protect against weight increase" This is from the source. "Suggestive evidence was found for a protective role against increasing weight from whole grains, cereal fibre, high-fat dairy products and high scores in an index describing a prudent dietary pattern." Why did "High-fat" get deleted? There wasn't even any correlation when they looked at low-fat or all dairy fat. Please consider this and remove the "high-fat to low-fat" recommendation.</p> <p>A couple of studies and reviews to look at http://www.ncbi.nlm.nih.gov/pubmed/19682399 http://www.ncbi.nlm.nih.gov/pubmed/23026077 http://www.ncbi.nlm.nih.gov/pubmed/19259609 http://www.ncbi.nlm.nih.gov/pubmed/21348924</p>	<p>Thank you for pointing this out. The citation has been corrected.</p> <p>The low-fat dairy recommendation rests on a broad scientific background. A switch of dairy consumption from high fat to low fat is generally needed, in order to sustain intakes of calcium and other essential nutrients while reducing SFA. However, healthy diets probably could include some high-fat dairy products, if the balance between unsaturated and saturated fats can be maintained; more than 2/3 of all fat should be unsaturated and poly-unsaturated fat.</p>

Organisation Company	General and detailed comments	WG response & actions
	http://www.ncbi.nlm.nih.gov/pubmed/22810464 http://advances.nutrition.org/content/3/3/266.short Thank you	
Svenskt Kött	<p>Only general comments:</p> <p>Comments on line numbers 251-261 Organisationen Svenskt Kött is asking for a definition of "processed meat" in the proposal for NNR5 that is better adjusted to the Nordic countries and the products that are available here.</p> <p>In a press release concerning the NNR5 from Livsmedelsverket (2012 06 05) "processed meat" is translated to the Swedish word "chark" and that is not satisfactory since the definition of processed meats in the statement from WCRF is: "Processed meat refers to meat preserved by smoking, curing or salting, or addition of chemical preservatives, including that contained in processed foods." In Sweden we have a rich tradition of manufacturing sausages and other processed meats, so called charcuteries (charkuterier/chark). Some of these are ok with the definition from WCRF and some are not. Liver pate, black pudding and cooked ham (lightly salted) are some examples of charcuteries that do not fit in to the WCRF's definition of processed meat.</p> <p>Svenskt Kött requests more knowledge about what components in the processed meats that should be limited before recommending a population to decrease the consumption of this group of foods. Is it for example the salt, the nitrites or the saturated fatty acids? This knowledge is essential for the general public to be able to compare the different food items within the group and make healthier choices. Is for example liver pate a better choice for the sandwich than salami with high proportions of fat and salt? Svenskt Kött also finds this knowledge very important to be able to motivate the meat industry to develop healthier charcuteries for the future.</p>	<p>The text uses the definition of "processed meat" as indicated by the WCRF/AICR, and the exact citation of that report is now included.</p> <p>Unfortunately, most studies that have examined processed meat use different definitions.</p> <p>We agree that more research in this field is needed; currently there are very few studies that specifically examine Swedish or Nordic meat products. In fact, research projects in this area initiated by "Svenskt Kött" would be most welcome.</p> <p>We agree that here are many aspects of modern diets that still require thorough examination.</p>

Organisation Company	General and detailed comments		WG response & actions
Valio Ltd, Finland	<p>Line 137-138: "Milk and milk products are important sources of fat, protein, vitamin A, riboflavin, vitamin B12, calcium and iodine". As milk protein has been evaluated to be of high quality compared to other dietary proteins, I think this should be mentioned. I would suggest rewording the sentence as follows: Milk and milk products are important sources of fat, high-quality protein, vitamin A, riboflavin, vitamin B12, calcium and iodine. Reference: Dietary protein quality evaluation in human nutrition. Report of an FAO Expert Consultation. FAO Food and Nutrition Paper 92. Food and Agriculture Organization of the United Nations, Rome, 2013</p>	<p>Lines 234-236: "However, a recent prospective study in Finland indicates that heavy use of butter is associated with an increased risk of acute myocardial infarction (Voutilainen, Nurmi et al. 2013)." Is this statement based on only one study? Several other studies have shown either no association or even an inverse association between dairy fat consumption and myocardial infarction: Warensjö et al. Am J Clin Nutr. 2010 Jul;92(1):194-202. doi: 10.3945/ajcn.2009.29054 Biong et al. 2006 Eur J Clin Nutr. 2006 Feb;60(2):236-44 Aslibekyan et al. Nutr Metab Cardiovasc Dis. 2012 Dec;22(12):1039-45. doi: 10.1016/j.numecd.2011.02.003 I would suggest rewording the sentence to reflect that evidence is not consistent.</p>	<p>Warensjö et al. used an unspecific biomarker (i.e. C15:0 and C17:0) that also exist in fish not only in milk-fat.</p> <p>Voutilainen et al. used a detailed dietary assessment method (i.e., food records) in a prospective cohort study with 20 years of follow up and with a "hard" end point.</p> <p>However, the text has now been edited (systematically and substantially) in line with your suggestions and with those made by other professionals in the field.</p>
LRF Mjöl/LRF Dairy Sweden	<p>The comments from LRF Dairy Sweden (LRF Mjöl, tidigare Svensk Mjöl) are given by Helena Lindmark-Månsson, adjunct professor and nutrition expert, Annika Smedman, nutritionist and PhD, and Maja Nordström, M Sc Agr. General comments: It is positive that the Nordic Nutrient Recommendations promote a food pattern including natural foods, such as vegetables, fruits and berries, nuts and seeds, wholegrain, fish and sea food and low-fat dairy products. In the recommendations, however, all of these food groups are mentioned</p>	<p>Detailed comments:</p> <p>Page 3, line 73. Anti-nutrients in wholegrain should be discussed.</p> <p>Page 3, line 99. Include the processing conditions in the manufacture of oils, margarine and spreads.</p> <p>Page 4, line 111. Please include that the trans fatty acids occur naturally in ruminant fat. Butter and ruminant fat naturally contain 3-5 % trans-fatty acids (TFA).</p> <p>Page 4, 139. The fat composition is equal in all dairy products, regardless of total fat content.</p> <p>Page 4, 143. Please extend the discussion regarding nutrient content of cheese. The nutrient composition is much more than calcium.</p> <p>Page 5, line 145. What is the reason of focusing on cholesterol? Cholesterol is not a part of the nutritional recommendations.</p>	<p>Thank you for your valuable comments. We have systematically edited the text in line with your suggestions and those given by several other professionals in the field.</p> <p>We agree that the wording when citing published reports should be correct (i.e., have edited the text regarding citations of the WCRF/AICR reports). However, Åkesson et al 2013 systematically reviewed studies examining total dairy consumption. The wording of the conclusions from that report was correctly cited in the draft chapter, and should not be confused with the wording of the conclusions from the WCRF/AICR report.</p> <p>We agree that the objective of the Sonestedt et al SR was to assess the effect of sugar intake from beverages, and that the findings should be cited correctly.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>in the green part of Table 1. except the low-fat dairy. It is misleading to exclude low fat dairy from the green box since it is included in the conclusion. Therefore we wish to include low fat dairy in the green box.</p> <p>In the red part of table 1 milk products and cereals has been pointed out together with sodas, sweets and bakery items for example, as drinks and food products with added sugar. It is misleading and confusing or the consumer to evaluate snacks and sweets in the same category as breakfast products like milk and cereals, even if they contain some added sugar. Milk-products with added sugar contains in addition to the sugar also many vitamins and minerals and contributes therefore to the overall nutrition.</p> <p>In Riksmaten vuxna 2010-11 the percentage contribution of carbohydrates from milk and fermented milks was 7 % in the diet, whereas food items with a low nutrient content such as sodas, sweets, soups among others, contributed with 14 % of the carbohydrate intake.</p> <p>In Riksmaten barn 2003 the contribution was 11-13 % from milk and fermented milk whereas the contribution of carbohydrates from sweets, sodas and snacks</p>	<p>Page 5, line 178. It is misleading and confusing or the consumer to evaluate snacks and sweets in the same category as breakfast products like milk and cereals, even if they contain some added sugar. Milk-products with added sugar contains in addition to the sugar also many vitamins and minerals and contributes therefore to the overall nutrition.</p> <p>Page 6, line 184. The EPIC studies were conducted during the 1990s. That shouldn't be regarded as currently.</p> <p>Page 7, line 240. "...foods high in calcium probably increases the risk..." This is not correct. In the report from WCRF/AICR 2007, page 129 it says "Diets high in calcium". The sentence should be corrected.</p> <p>Page 7, line 236. The introduction states that "the chapter mainly summarizes information from recent reports relevant for the Nordic situation: the extensive and systematic reviews of..." A paper in press in Finska Läkartidningen, "Finnish Medical Journal", has been included on butter and its relation to increased risk of myocardial infarction. Why was this paper selected from a journal lacking a peer-reviewing system? Other recent publications are available, for example, Kratz, M et al. The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. EUROPEAN JOURNAL OF NUTRITION,,, type 2013, 52 1, p1-p24.</p> <p>Page 7, line 243. It is confusing to refer to the consumption of total fat included in the paragraph on milk and dairy. Milk and dairy product contribute only to approximately 8 % of total intake according to Riksmaten 2010-11. Page 8, line 278. Why was not food items such as sweets, bakery items and sodas included in the study? These food</p> <p>Table 1.1 represents the interpretation of findings from <u>all</u> gathered evidence. The table attempts to describe an overall optimal diet including many aspects of diet-disease risks (e.g., energy density, nutrient requirements and micro-nutrient density, fat quality (SFA vs. MUFA+PUFA), carbohydrate quality, specific food choices as well as the overall dietary pattern). There is a need to change within food groups to achieve this optimal diet, but apart from plant foods there is no recognized need to increase any other food group. Table 1.1 has been edited in response to many comments received in the public consultation.</p> <p>Currently there is no well-defined and commonly agreed upon definition of the Nordic diet. Although, similar and common foods are examined in these studies, definitions may vary across studies.</p> <p>We agree that the health effect of a whole range of different foods and food groups needs to be evaluated.</p> <p>In the project by Åkesson et al (2013) that systematically reviewed health effects of food groups and chronic disease it was absolutely necessary to limit the research questions due to the limited resources and the limited number of persons involved (6 researchers and 2 librarians).</p> <p>The NNR 2012 was a complex project that required a great amount of time and resources. The coordination of all tasks was a challenge, which may not have been obvious to persons not directly involved. Unfortunately the publication timeline was seriously delayed and therefore less than perfect for everyone involved.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>contributed to 23 to 25%. In our opinion milk-products should be excluded from the list of food items to limit, since the contribution of carbohydrates from these products is low compared to other sugar sweetened food items. The sugar sweetened milk products contribute with 18 out of 22 of the recommended nutrients we need every day despite the added sugar. It's surprising that the chapter on food patterns is available for public consultation before all nutrient chapters (for example macronutrients) have been reviewed.</p> <p>When citing the report from WCRF/AICR 2007 the correct wording is crucial. There are currently several incorrect citings which is misleading and confusing the target group of the NNR 2012. Regarding papers included in the back ground material it would be appropriate that the studies are published in Food and Nutrition Research before their inclusion in the chapter to be valued in the process of public consultations.</p>	<p>items contribute significantly to the intake of added sugar and fat. Also cereals and fish are missing in the evaluation.</p> <p>Page 9, line 292. To be correct the report WCRF/AICR 2007 concludes on page 129 that the strength of evidence for the relationship between milk and dairy products and prostate cancer is limited-suggestive. It is very important to cite the report correctly and not saying only "suggestive" as in this chapter. Change the sentence to the correct wording and also include the importance of intake of calcium from other sources such as supplements.</p> <p>Page 9, line 306. The title of the paragraph should be changed. The objectives of the paper of Sonestedt et al. was to assess the effect of sugar intake from beverages, not foods. Based on the conclusions drawn in line 307-317 there is no evidence for decreasing the consumption of sweetened dairy products. Page 10, line 343. It is not correct that total dairy products increases the risk of prostate cancer. Use the correct wording from the report from the report WCRF/AICR 2007 that on page 129 concludes that the strength of evidence for the relationship between milk and dairy products and prostate cancer is limited-suggestive. It is very important to cite the report correctly. Change the wording of the sentence.</p> <p>Page 11, line 379-404. An explanation of the expression Nordic diet is necessary. Are the studies mentioned on traditional Nordic diets or Nordic versions of Mediterranean diets? Table 1.1, page 20. Include in the text, a reference substantiating that milk-products with added sugars should be limited. If there is no such studies remove milk-products from the column. See also the comment above page 9, line 306. In the red part of table 1 milk products has been pointed out together with</p>

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	<p>sodas, sweets and bakery items for example, as drinks and food products with added sugar. Milk-products with added sugar contains in addition to the sugar also many vitamins and minerals and contributes therefore to the overall nutrition.</p> <p>In Riksmaten vuxna 2010-11 the percentage contribution of carbohydrates from milk and fermented milks was 7 % in the diet, whereas food items with a low nutrient content such as sodas, sweets, soups among others, contributed with 14 % of the carbohydrate intake.</p> <p>In Riksmaten barn 2003 the contribution was 11-13 % from milk and fermented milk whereas the contribution of carbohydrates from sweets, sodas and snacks contributed to 23 to 25%. In our opinion milk-products should be excluded from the list of food items to limit, since the contribution of carbohydrates from these products is low compared to other sugar sweetened food items.</p> <p>The sugar sweetened milk products contribute with 18 of 22 of the recommended nutrients we need every day despite the added sugar. Table 1.1, page 20. What is the rational for the recommendation to reduce high-fat dairy to low fat dairy? References to be considered are:</p> <ul style="list-style-type: none"> • Kratz, M et al. The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. European Journal of Nutrition, 2013, 52 1, p1-p24. • Elwood PC et al. The consumption of milk and dairy foods and the incidence of vascular disease and diabetes: an overview of the evidence. Lipids. 2010, 45, :925-39. 	

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SIK - The Swedish Institute for Food and Biotechnology and Chalmers University of Technology	<p>Only general comments:</p> <p>Thank you very much for giving me the opportunity to read and comment the NNR 2012 document. It is well written and contains a lot of interesting recommendations, however I would like to make some comments regarding my area of expertise – Food Processing. I would like also to advice a consultation at EUFIC (European Food Information Council homepage : http://www.eufic.org/article/en/food-technology/food-processing/expid/benefits-processed-food-review/ http://www.eufic.org/page/en/page/FAQ/faqid/do-processed-foods-offer-any-benefits/ as well as a document prepared by the International Food Information Council Foundation http://www.foodinsight.org/LinkClick.aspx?fileticket=wtg018sd8qk%3D&tabid=1398 to support some of my comments.</p> <p>Lines 170-185: The terminology ultra-processed foods is not a scientific terminology and “ Soft drinks, confectionary, candy bars.....” are for sure not ultra-processed foods. The use of “refined and highly processed food products” is also unclear. I am personally not familiar with the concept “refined”. Most foods that we eat are processed in some way. Without food processing we would not have the large variety of food products available and not all processed foods are unhealthy. In fact, processing operations like freezing and thermal processing can make foods more safe and keep their most of their nutritional value. The terminology “ultra” is used for example in UHT (Ultra high temperature) that is a thermal processing that have been demonstrated to preserve the most of the nutrients in milk. No milk is found in the shops that has not been processed. I can however agree, that the foods mentioned (with exception to bakery goods and powdered sauces and soups) are not healthy anyway, however the main reason is not processing but the high content of sugar or fat. One terminology that could be used is “indulgence foods”, or simply foods with high sugar or fat content. Bakery goods includes also bread that has more nutritional advantages than disadvantages – better to use “sweet bakery goods”, and powdered soups are not always unhealthy – depends on the formulation and product quality.</p> <p>It is correct that some thermal processes can develop toxic substances, however I believe that is the responsibility of the Swedish Food Agency (Livsmedelsverket) to control and give recommendation on this area.</p> <p>See for example http://www.slv.se/sv/grupp1/Risker-med-mat/Kemiska-amnen/Akrylamid/</p> <p>I am afraid that recommendations to avoid processed foods in the context of the document presented or needs to be extended or it will just create confusion and</p>	<p>Thank you for your valuable comments and advice. The text has been updated in line with these and other comments.</p> <p>The section now titled “Characteristics of dietary patterns” discusses some issues related Westernized diets and cites published scientific papers that have suggested food group definitions according to the degree of processing.</p>

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	<p>misunderstanding. I hope my comments will contribute to improve the document, and do not hesitate to contact me if you have any question. Lilia Ahrné Professor in Food Technology at Chalmers University of Technology</p>	
Unilever	<p>General comments</p> <p>INTRODUCTION Helping consumers to make better choices and improving their health and nutrition requires consumer friendly, clear and evidence based information. We believe this is essential if consumers are to make informed healthier choices. We also believe that to make a lasting impact on health we need to encourage our consumers to make small changes to their everyday habits. We agree with the authors of this chapter that the Nordic population would benefit from a simple set of food-based dietary guidelines based on evidence based nutrition research as well as good knowledge of local food patterns. We also believe that the chapter makes an important bridge between the nutrition research addressing the impact of single nutrients on specific health outcomes, and the reality that people do not eat nutrient but eat foods, in certain patterns. To make the guidelines even clearer and easy-to-understand we would suggest some further clarifications and adding of scientific references as outlined below.</p> <p>Detailed comments</p> <p>DETAILED COMMENTS: • Rows 97: We propose to replace “spreads” by “vegetable oil based fat spreads”, as fat spread is the denominator used in the English version of the Council Regulation (EC) 1234/2007. This will also avoid any confusion with other types of spreads.</p> <ul style="list-style-type: none"> • Row 97-114: Consequently “spreads” should then be replaced by “vegetable oil based fat spreads” throughout this paragraph. • Row 99: We suggest adding other sources of oils such as “sunflower seeds” and “linseeds”. • Row 100: Margarine is a mixture of different vegetable oils and fat which also needs to be fully reflected. We therefore suggest making the sentence more explicit by stating “Margarine and fat spreads are mixtures of different vegetable oils and fats.” • Row 102-104: Adding of vitamin A and D is regulated by law. Thus this should also be reflected in the paragraph. Our suggestion is to rephrase: “Vitamin A and D are added to margarines and vegetable oil based fat spreads according to regulation”. This will also provide further clarity to consumers on the basis of adding of vitamin A and D. 	<p>Thank you for your very valuable comments; most of these have been considered together with other comments when systematically editing the text.</p> <p>We agree that the health effect of a whole range of different foods and food groups should be evaluated. In the project by Åkesson et al (2013) that systematically reviewed the health effects of food groups and chronic disease, it was absolutely necessary to limit the research questions due to the limited resources and persons involved (6 researchers and 2 librarians).</p> <p>The section now titled “Characteristics of dietary patterns” discusses some issues of concern related to Westernized diets, and cites published scientific papers that have suggested food group definitions according to the degree of processing.</p>

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	<ul style="list-style-type: none"> • Row 111-114: We suggest amending the sentences into: “Margarines and vegetable oil based fat spreads were previously sources of TFA, depending on the raw material used in manufacturing”. “However, the TFA content has during the last decade decreased considerably in the Nordic countries and today the TFA levels is much lower than in butter.” <p>Nutritional aspects should set the boundary conditions for health impact of foods and not the way of production or origin.</p> <p>Classifying foods as “processed” is thus scientifically not tenable. Further, since a lot of food are processed in one way or the other, the word processed is not very informative.</p> <p>References (Row 97-114)</p> <ul style="list-style-type: none"> – Intake of fatty acids in Western Europe with emphasis on trans fatty acids: The TRANSFAIR study. Eurpean Journal of Clinical Nutrition (1999) 53, 143±157. – The Elimination of Trans Fats from Spreads: How Science Helped to Turn an Industry Around. Onno Korver, PhD, and Martijn B. Katan, PhD. Nutrition Reviews_ , Vol. 64, No. 6: 275–279.June 2006 – Reformulation revolution: Trans fatty acids records big drop in UK foods since 2007. May 2013. https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/167938/Summary_Report.pdf – Stender S, Astrup A, Dyerberg J. A trans European Union difference in the decline in trans fatty acids in popular foods: a market basket investigation. BMJ Open 2012;2:e000859. doi:10.1136/bmjopen-2012-000859. <ul style="list-style-type: none"> • Row 170-185: Classifying foods as “processed” and/or “ultra-processed” is scientifically not tenable and is not helping consumers making healthy food choices. Manufactured foods make a significant contribution to the nutrient content of the diet and in terms of nutrition there are many benefits to modern processing techniques. They can minimise nutrient losses -where nutrients have been lost they can readily be restored - and they can also enable fortification to increase the nutritional value of food. Although we acknowledge the potential health adverse effects of eventual components (e.g. nitrites) added during manufacturing we believe nutritional aspects firstly should set the boundary conditions for health impact of foods and not the way of production or origin. In addition the paragraph lacks a definition for what is regarded as “processed” and/or “ultra-processed” leading to inconsistency when making references to different types of food. For example dried soups are seen as ultra-processed whereas dried meat is not. There is also no reference made to food that is high in salt and saturated fat such as for example sausages and cheese. The paragraph would therefore benefit from also having a recommendation to consume low salt and/or low saturated food to ensure consistency 	

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	<p>with overall guidelines for a healthy diet.</p> <ul style="list-style-type: none"> • Row 170: Thus, we propose to change the heading to “Energy dense food products”. This is aligned with previous NNR and is also better reflecting that processed food can have a balanced nutrition profile. • Row 175: Also, there have been reductions in the salt content of manufactured foods following the release of a technical World Health Organization report in 2003. In the expert consultations and technical meetings on sodium an explicit call was made to the food industry to help lower sodium intakes among populations. We believe this should be reflected in the paragraph as well as a recommendation to consume low salt and/or low saturated food to be amended to ensure consistency with overall guidelines for a healthy diet. <p>Reference: http://www.who.int/dietphysicalactivity/reducingsalt/en/)</p> <ul style="list-style-type: none"> • Row 178: Powder soup and sauces should be removed as a reference of “ultra-processed food products”. Drying is a form of mild processing, and especially dried vegetables have been shown to have good nutrient retention. Today’s technological advances mean that it is now possible to carefully treat food in such a way that it retains most of its nutritional content such as proteins, carbohydrates, fats, vitamins and minerals. This particularly applies to the dehydration process, which is one of the oldest methods for preserving foods being a truly natural preservation method. Removing of the water content delays spoilage reactions which in turn allows the food to be stored at room temperature for long period of time. No preservatives need to be added. Finally, dried soups and sauces have a short cooking time, ensuring the nutritional value and taste is preserved. Above is confirmed by a recent study by Basendowski, et al (submitted) comparing the nutrient retention of key nutrients and non nutrients in fresh and processed vegetables, supermarket versus industrial origin and also commercial dried soups versus homemade soups. Results concluded that industrial dehydrated soups are from a nutrient content point of view comparable to their corresponding homemade soups. References: <p>- Supplementary information from http://epic.iarc.fr Slimani S - Eur J Clin Nutr. (2009) Nov;63 Suppl 4:S206-225</p> <p>–Takeoka GR, Dao L, Flessa S, Gillespie DM, Jewell WT, Huebner B, Bertwo D, Ebeler SE. (2001) Processing Effects of Lycopene Content and Antioxidant Activity of Tomatoes. Journal of Agricultural and Food Chemistry, 49(8), 3713-3713 –</p> <p>Abushita AA, Daood HG, Biacs PA (2000). Change in Carotenoids and Antioxidant Vitamins in Tomato as a Function of Varietal and Technological Factors. Journal of Agricultural and Food Chemistry, 305(6), 2075-2081 - Basendowski S, Spraul M, Feunekes G (Submitted). Nutrient retention of selected marker compounds during</p>	

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	<p>dehydration and preparation of dried versus homemade soups. Journal of Food Composition and Analysis</p> <ul style="list-style-type: none"> Row 222: We believe the paragraph should be aligned with the WHO /FAO recommendations which include both short and long chain PUFA. Thus we suggest to amend the sentence into: “Many reports indicate that there is convincing evidence for the health benefit of replacing SFA in the diet with unsaturated fat, both PUFA from vegetable oils, vegetable oil based fat spreads and very long omega-3 PUFA from fish (Nasjonalt råd for ernæring 2011)”. <p>References: FAO report of an expert consultation on fats and fatty acids in human nutrition. Geneva, 2008. http://foris.fao.org/preview/25553-0e4cb94ac52f9a25af77ca5cfba7a8c.pdf WHO/FAO. Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO expert consultation, 28 January - 1 February 2002, Geneva, Switzerland (WHO Technical Report Series 916) http://www.who.int/nutrition/publications/obesity/WHO_TRS_916/en/index.html Explaining the 25-year decline of serum cholesterol by dietary changes and use of lipid-lowering medication in Finland.</p> <p>Valsta LM, Tapanainen H, Sundvall J, Laatikainen T, Männistö S, Pietinen P, Vartiainen E. Public Health Nutr. 2010 Jun;13(6A):932-8. Judd JT, Baer DJ, Clevidence BA, Muesing RA, Chen SC, Weststrate JA, Meijer GW, Wittes J, Lichtenstein AH, Vilella-Bach M, Schaefer EJ. Effects of margarine compared with those of butter on blood lipid profiles related to cardiovascular disease risk factors in normolipemic adults fed controlled diets. Am J Clin Nutr 1998 Oct;68(4):768-77.</p> <p>Tonstad S, Strom EC, Bergei CS, Ose L, Christophersen B. Serum cholesterol response to replacing butter with a new trans-free margarine in hypercholesterolemic subjects. Nutr Metab Cardiovasc Dis 2001 Oct;11(5):320-6.</p> <p>Gagliardi AC, Maranhao RC, de Sousa HP, Schaefer EJ, Santos RD. Effects of margarines and butter consumption on lipid profiles, inflammation markers and lipid transfer to HDL particles in free-living subjects with the metabolic syndrome. Eur J Clin Nutr 2010 Oct;64(10):1141-9.</p> <p>Lecerf JM, Luc G, Marecaux N, Bal S, Bonte JP, Lacroix B, Cayzeele A. Small, qualitative changes in fatty acid intake decrease plasma low-density lipoprotein-cholesterol levels in mildly hypercholesterolemic outpatients on their usual high-fat diets. Int J Food Sci Nutr 2009;60 Suppl 7:151-63. Seppanen-Laakso T, Vanhanen H, Laakso I, Kohtamaki H, Viikari J. Replacement of butter on bread by rapeseed oil and rapeseed oil-containing margarine: effects on plasma fatty acid composition and serum cholesterol. Br J Nutr 1992 Nov;68(3):639-54.</p> <ul style="list-style-type: none"> Row 233-236: For a non specialist reader, these two sentences might appear 	

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	<p>contradictory. It seems to be a health difference between consumption of milk and general consumption of other dairy products such as butter. We therefore suggest adding the mentioning of “Low-fat dairy consumption is recommended to sustain calcium intake while reducing saturated intake”. This is also in line with the overall conclusion of this chapter (row 561).</p> <ul style="list-style-type: none"> • Row 277-287: If the suggested references below are not already taken into account we would like to suggest the authors of the review (Akeson et al. 2013) to consider additional references regarding the food group “milk” and “milk products”, two of which are intervention trials performed in the Nordics countries. These references address the impact of butter vs. margarines on blood cholesterol and underpin the conclusion advising to limit butter and replace it by soft margarines and vegetable oil based fat spreads. References: Tonstad S, Strom EC, Bergei CS, Ose L, Christophersen B. Serum cholesterol response to replacing butter with a new trans-free margarine in hypercholesterolemic subjects. <i>Nutr Metab Cardiovasc Dis</i> 2001 Oct;11(5):320-6. Seppanen-Laakso T, Vanhanen H, Laakso I, Kohtamaki H, Viikari J. Replacement of butter on bread by rapeseed oil and rapeseed oil-containing margarine: effects on plasma fatty acid composition and serum cholesterol. <i>Br J Nutr</i> 1992 Nov;68(3):639-54. Judd JT, Baer DJ, Clevidence BA, Muesing RA, Chen SC, Weststrate JA, Meijer GW, Wittes J, Lichtenstein AH, Vilella-Bach M, Schaefer EJ. Effects of margarine compared with those of butter on blood lipid profiles related to cardiovascular disease risk factors in normolipemic adults fed controlled diets. <i>Am J Clin Nutr</i> 1998 Oct;68(4):768-77. Gagliardi AC, Maranhao RC, de Sousa HP, Schaefer EJ, Santos RD. Effects of margarines and butter consumption on lipid profiles, inflammation markers and lipid transfer to HDL particles in free-living subjects with the metabolic syndrome. <i>Eur J Clin Nutr</i> 2010 Oct;64(10):1141-9. Lecerf JM, Luc G, Marecaux N, Bal S, Bonte JP, Lacroix B, Cayzeele A. Small, qualitative changes in fatty acid intake decrease plasma low-density lipoprotein-cholesterol levels in mildly hypercholesterolemic outpatients on their usual high-fat diets. <i>Int J Food Sci Nutr</i> 2009;60 Suppl 7:151-63. • Row 306-317: In order to further clarify the perceived intention to address sugar based foods, we suggest that the headline is changed into “Foods high in sugar”. • Row 411: Besides rapeseed oils, the diet of the SYSDIET study also contained margarines based on vegetable oils (page 5 in article), with an overall diet containing >2/3 unsaturated fats. This should also be reflected in the text. • Row 561: We suggest the adding of “vegetable oil based fat spreads” as an example of a dietary pattern that is recommended to be adapted. To provide consistency as well as expand the food choices available for consumers the text in the Conclusion and the 	

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	<p>Table 1.1 (row 722) should also be aligned thus mentioning both “vegetables oils, soft margarines and vegetable oil based fat spreads with a high content of unsaturated fats”. First, the denominators for edible fats/butter/spreads/margarine are regulated in Council regulation 1234/2007. And by including vegetable oils based fat spreads to the choices this would give the complete variables of the soft fat group aiming to reduce saturated fats Second, the adding of the wording “fat spreads” shows an expanded choice from consumer perspective in line with the final conclusions. • Row 562-563: We suggest the removing of the “refined and highly processed food products”. Classifying foods as “processed” is scientifically not tenable and not helping consumers making healthy food choices. The health impact of foods should be evident from the nutrition panel, not from way of production or origin. This would also enable a better alignment between the Conclusion and Table 1.1 (row 722) where we suggest the wording to be: “Food products high in sugar, salt, saturated fatty acids and transfatty acids should be limited”. • Row 562-563: Consequently, we also suggest adding the mention of “ high fat dairy products” to ensure full alignment with the Table 1.1 (row 722).</p> <ul style="list-style-type: none"> • Row 564: We suggest an additional mentioning on the importance of reasonable portion sizes and balancing calorie intake. • Rows 558-564: In conclusion, we propose the Conclusion to read: “Overall the scientific evidence indicate that in order to promote future health and wellbeing in Nordic populations a dietary pattern should be adopted that is rich in vegetables (including dark green leaves, cabbage, onion, peas and beans, root and fruiting vegetables), fruits and berries, nuts and seeds, whole-grains, fish and seafood, vegetables oils, soft margarines and vegetable oil based fat spreads that are high in unsaturated fatty acids and low-fat dairy products. In contrast, a pattern characterized by high intakes of processed meats, red meat, high fat dairy products and of food products high in sugar, salt, saturated fatty acids and trans fatty acids should be limited . These dietary patterns should also ensure a balanced calorie intake (Table 1.1)”. • Row 722: Table 1.1 (summary table): First, we suggest to add the wording “vegetable oil based fat spreads” to ensure consistency with the Conclusions as well as expand the food choices available for consumers. Second, we suggest to change the heading “Drinks and food products with added sugar” to “Drinks and food products high in added sugar”. Third, to change “Food products with added fat and salt” to “Food products high in fat and salt content”. We believe this will provide better clarity and also better differentiate food where sugar and salt is added in a reasonable amount. 	

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University of Bergen, Norway	<p>Only general comments:</p> <p>The NNR 2012 guidelines on a healthy diet does well to present much useful information within the document balancing well with clarity and not overloading with information. Here are some few suggestions to a minor changes:</p> <p>In line 61, the sentence referring to plant foods stating “usually contain low amounts of food energy”, can be misinterpreted. Many types of plant foods are energy dense such as nuts, seeds, avocado, olives, legumes, bananas, and could all contribute substantially meet energy needs. It could thus be justified to delete the part of the sentence “usually contain low amounts of food energy”.</p> <p>In line 109, in the sentence acknowledging fish oils as a source of long omega-3 PUFA, algae products could be added, reading “fish oils and algae oils”, as algae oils are increasingly popular as supplements with comparable fatty acid distribution to fish oils. After line 143 it could be useful to add a paragraph on plant milks (such as soy, oat and rice milk), as they are used increasingly. In this paragraph it could e.g. be written that many of these products are enriched with calcium, vitamin B12 and vitamin D, and can be useful dietary sources of these vitamins. The macronutrient content could also be presented briefly (similarly as it is done for cow’s milk).</p> <p>The sentence in line 258-260 seems to be unclear “The Nordic study concluded that if this reduction was achieved the nutritional consequences should be negligible (Tetens, Hoppe et al. 2013)” seen in light of the preceding paragraphs and the meta-analysis on cancer incidence and intake of red and processed meat by Chan et al. published in PLoS One in 2011.</p> <p>Thus, it could be clearer to just delete the sentence in line 258-260.</p> <p>In line 299-301: “However, since meat consumption is an important contributor to iron intake, there is a concern that when replacing some meat with plant foods, that certain population groups may be at higher risk of mineral (i.e., iron) deficiency.”</p> <p>According to Hunt (Bioavailability of iron, zinc, and other trace minerals from vegetarian diets. Am J Clin Nutr 2003) and the position of the American Dietetic Association from 2009 (Position of the American Dietetic Association: vegetarian diets. J Am Diet Assoc 2009), this concern might not be markedly different from the general population. The American Dietetic Association argues that “Whereas many studies of iron absorption have been short term, there is evidence that adaptation to low intakes takes place over the long term, and involves both increased absorption and decreased losses. Incidence of iron-deficiency anemia among vegetarians is similar to that of nonvegetarians. Although vegetarian adults have lower iron stores than nonvegetarians, their serum ferritin levels are usually within the normal range.”</p>	<p>Thank you for your comments and advice which has been considered when editing the text.</p> <p>We would like to point out that a reduction in processed and red meat consumption does not mean that the whole population automatically will change to a well-balanced vegetarian diet. Thus the health consequences of any population wide change need to be considered carefully.</p>

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	Thus, it could be justified to delete the sentences from line 299 to 304.	
Danish Endocrinology Society	<p>Only general comments:</p> <p>Vitamin D Comments on effects of calcium and vitamin D (CaD) supplementation on risk of fracture p. 8. In the chapter on calcium, it is stated that “calcium supplementation in combination with vitamin D may be effective in reducing fractures in institutionalized populations although the effect in the general population is unclear”. Similarly, in the section on vitamin D it is stated that CaD “has been shown to reduce the risk of total fracture and hip fracture in institutionalized elderly, whereas supplementation with vitamin D alone has not shown a significant effect (Lamberg-Allardt et al. 2013)”. It should be noted that most of the studies included in the cited systematic reviews have been performed non-institutionalized elderly. Accordingly, the summary risk estimates, showing a reduced fracture risk in subjects randomized to CaD, are based on data, which to a large extent are derived for findings in non-institutionalized elderly. It may be considered to draw a conclusion saying that “SRs of randomized controlled trials on supplementation with calcium and vitamin D in combination have in general shown a reduced risk of fractures in response to supplementation in the elderly. The effect is apparently most pronounced in institutionalized elderly and/or in subjects with low vitamin D levels prior to the intervention. Supplementation with vitamin D alone has not shown significant effects”. Comments to “Lower intake level” of vitamin D p.14 A number of good arguments for defining the Upper intake level of vitamin D are stated. However, there are almost no reasons mentioned for defining the threshold for the lower intake level. One paper is cited concerning newborn Chinese infants in whom no clinical signs of rickets were evident at six months of age, following a minimum supplementation of 2.5 microgram/d. However, with an average 25OHD level (geometric mean) of approximately 30 nmol/l within this group, a notable proportion of the population had levels in the range known to be associated with an increased risk of rickets. The small study population and short study duration (6 mo.) are obvious limitations to this study. As the lower intake level (LI) is defined as a cut-off intake value below which an intake could lead to clinical deficiency symptoms in most individuals, this threshold is of specific importance to those with no or very limited sun-exposure. In adults (and, most likely also in children) without/with very limited sun-exposure, a daily intake of 2.5 microgram/d is unlikely to be sufficient to maintain a 25OHD concentration</p>	Thank you for your valuable comments, and references of published studies. We have updated the text in line with these and other comments.

Organisation Company	General and detailed comments	WG response & actions
	<p>> 27.5 nmol/l and is likely, in the long run, to result in rickets/osteomalacia in “several” individuals. Accordingly, it may be considered to increase LI to 7.5 (or at least 5) microgram/d – a footnote may be added saying that this is considered to be the minimum requirement in persons with very limited sun exposure. Comments to “Body weight” p. 12. It is stated that “an association between vitamin D intake and body weight is inconclusive”, but from several recent RCT’s it is clear that the results may be described as conclusive – there are no effects (if all negative results should be stated as inconclusive nothing will never be conclusive) Foods, food patterns and health outcomes – guidelines for a healthy diet. 1) page 7, line 222-231. Two studies published in 2012 and 2013 described no effect of daily n-3 fatty acid supplementation on cardiovascular events (morbidity or mortality) (http://www.ncbi.nlm.nih.gov/pubmed/23656645 and http://www.ncbi.nlm.nih.gov/pubmed/22686415) in ~12.000 subjects with a mean follow up of 6.2 yrs. 2) Page 9, line 306-317 (Sugar-containing foods) One study published in 2012 described that intake 1 L of sucrose sweetened soft drink per day for 6 months increased ectopic (liver and skeletal muscle) fat accumulation and visceral fat mass (http://www.ncbi.nlm.nih.gov/pubmed/22205311). Additionally, one study published in 2013 described that an intake of 336 g (12 oz) of sucrose sweetened soft drink per day increased the incidence of type 2 diabetes with 22 % (http://www.ncbi.nlm.nih.gov/pubmed/23620057). Thus, recent RCT’s and observational studies also in children and adolescents (Am J Clin 2013http://www.ncbi.nlm.nih.gov/pubmed/23719557 & http://www.ncbi.nlm.nih.gov/pubmed/23172617) strongly support that sugar-sweetened beverages increase cardiometabolic risks, independent of weight status – which should be a part of the conclusion of that paragraph.</p>	
Nofima AS - Norwegian Institute of Food, Fishery and Aquaculture	<p>Only general comments:</p> <p>It is good that this chapter is included in the NNR5, but the definition of processed food must be more unified. Our society is dependent of processed food, but there are misperceptions and miscommunication about the role of processed foods as it cited by Floros et al 2010.</p> <p>I have looked into four different papers which are dealing with classification of processed foods and compared the wording in this chapter. There is a mixture of words like food groups, processed (highly processed, processed stable/basic foods), modestly processed, non-processed, manufactured, ultra-processed and refined food which are defined in different ways. Here there is a need for a common agreement of definition and wordings.</p>	<p>Thank you for your valuable comments, and references of published studies. We have updated the text in line with these and other comments.</p> <p>The section now titled “Characteristics of dietary patterns” discusses some issues related Westernized diets and cites published scientific papers that have suggested food group definitions according to the degree of processing.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>Dietary assessments and dietary recommendations typically use classifications of food that largely ignore or minimize the significance of industrial food processing. The significance of food processing in itself is overlooked. In this way, foods with very different nutritional profiles and impacts on eating patterns and health, such as whole grains, breakfast cereals, flours, breads, cookies, crackers, and cereal based snacks such as “power bars”, are classified within the same food group of cereals and cereal products. The same applies to whole fresh fruits, sugared canned fruits, and reconstituted sugared fruit beverages (classified within fruits) as well as to fresh meat, chicken and fish, and processed products such as burgers, nuggets and fingers (classified within meat or meat and meat products). Such classifications, derived from the “big four” (or “five”) groups originally devised early last century, still dominate official and other authoritative information and education programmes, accounts of population dietary patterns, and reports designed to prevent diseases that are influenced by diet. Practically all food is processed in some way. (Monteiro et al 2010).</p> <p>In NNR5 food categories have been used:</p> <p>Line 61 Vegetables, fruits and berries</p> <p>Line 116 Fish and seafood</p> <p>Line 137 Milk and milk products</p> <p>Line 156 Meat and meat products</p> <p>Processed foods (processed staple/basic foods and highly processed foods)</p> <p>Processed foods that have been industrially prepared and involving a high degree of processing such as drying, flaking, hydrogenation, heat treatment, use of industrial ingredients and industrial deep frying, including foods from bakeries and catering outlets, and which required no or minimal domestic preparation apart from heating and cooking (e.g. breakfast cereals, commercial cakes, biscuits, cheese, commercial sauces, canned foods including jams, margarine etc). In this definition, we further distinguished between processed staple/basic foods (e.g. bread, pasta, rice, milk, butter, vegetable oils) and highly processed foods (e.g. cakes, biscuits, breakfast cereals, crisp bread, confectionary, processed meat and fish, yoghurt, cheese, cream).</p> <p>Modestly processed foods</p> <p>Moderately processed foods include 2 sets of foods. First, foods involving relatively modest processing and consumed with no further cooking (e.g. dried fruits, raw vacuum packed foods, frozen basic foods, extra virgin oil, fruits and vegetable canned in water/brine or in own juice). Second, foods processed at home and prepared/cooked from raw foods or moderately processed foods (e.g. vegetables, meat and fish cooked from raw fresh ingredients or vacuum-packed, deep frozen, canned in water/brine or in own juice).</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>Non-processed foods</p> <p>Non-processed foods are foods consumed raw without any further processing, preparation, preparation, except washing, cutting and squeezing (e.g. fruits, non-processed nuts, vegetables, crustaceans, fresh juices). (Chajes, V. et al 2011)</p> <p>In NNR5 Processed or manufactured food are used</p> <p>Line 73 processed grain or cereal</p> <p>Line 79 processed into different type of flour and bread, pasta and mixed and complex products like breakfast cereals, cakes and pizza</p> <p>Line 111 Processed fats</p> <p>Line 150 Processed meat has undergone some preservation process like smoking, salting or use of other chemicals for preservation. Examples of such processed meats are ham, bacon, salami, different kinds of sausages and smoked meat. Meat that is boiled, fried, dried, fermented or frozen is usually not categorized as processed</p> <p>Line 161 The salt content is low in raw, unprocessed meat, but much higher in processed meat Processed meat is very special since the definition differs between different processes and only few can be called processed meat. Processed meat has no general agreed definition in WCRF because the term is inconsistently used in epidemiological studies.</p> <p>Line 183 manufactured food products But food product which are only mentioned as such, are also processed</p> <p>Line 97 Vegetable oil, margarine, spreads and butter = processed foods</p> <p>Line 98 Mayonnaise, dressings, baked goods and soups = processed foods</p> <p>Line 136 Milk – food in itself and raw material for different dairy products (like cheese, butter, fermented milk, yoghurt or cream) = processed foods</p> <p>Ultra-processed products – non-core foods and everyday food</p> <p>Ultra-processed products are made from processed substances extracted or refined from whole foods—eg, oils, hydrogenated oils and fats, flours and starches, variants of sugar, and cheap parts or remnants of animal foods—with little or no whole foods. Products include burgers, frozen pizza and pasta dishes, nuggets and sticks, crisps, biscuits, confectionery, cereal bars, carbonated and other sugared drinks, and various snack products. Most are made, advertised, and sold by large or transnational corporations and are very durable, palatable, and ready to consume, which is an enormous commercial advantage over fresh and perishable whole or minimally processed foods. Consequently, their production and consumption is rising quickly worldwide.</p> <p>Ultra-processed products are typically energy dense; have a high glycaemic load; are low in dietary fibre, micronutrients, and phytochemicals; and are high in unhealthy types of</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>dietary fat, free sugars, and sodium. When consumed in small amounts and with other healthy sources of calories, ultra-processed products are harmless; however, intense palatability (achieved by high content fat, sugar, salt, and cosmetic and other additives), omnipresence, and sophisticated and aggressive marketing strategies (such as reduced price for super-size servings), all make modest consumption of ultra-processed products unlikely and displacement of fresh or minimally processed foods very likely. These factors also make ultra-processed products liable to harm endogenous satiety mechanisms and so promote energy overconsumption and thus obesity. (Moodie et al 2013)</p> <p>Processed foods now account for approximately 75% of the total sales. The extent to which foods are processed varies greatly from product to product. Some foods are extensively processed (or ultra-processed). Ultra-processed foods refers to those non-core foods such as confectionary, chocolate, crisps and soft drink, but also includes many everyday food such as bread, biscuits, breakfast cereals, soups, sauces, cheese, pre-prepared meals and processed meats. Ultra-processed foods often contribute unnecessary amounts of sodium, saturated fat, trans fat and energy to the diet. Reformulation of processed foods provides a realistic opportunity to improve the health of a population.</p> <p>The complexity of reformulating processed foods has been well documented in the literature. A collaborative approach between government, industry, researchers and public health bodies will help address the challenges associated with improving the nutritional characteristics of processed foods.</p> <p>Most foods are processed in some way.</p> <p>Processed foods = minimally processed or ultra-processed foods (Heart Foundation). In NNR5 Ultra-processed or refined food products</p> <p>Line 170 Energy dense and ultra-processed food products</p> <p>Line 177 ultra-processed food products (e.g. soft drinks, confectionary, candy bars, desserts, bakery goods, sugared cereals, sugared milk-products, powdered sauces and soups, snacks, deep-fried potatoes etcetera)</p> <p>Line 80 refined cereal products – low nutrient content and often added sugars, fat and salt</p> <p>Line 171 Many food products are manufactured from refined cereals/white bread and with additional sugar, fat and salt</p> <p>Other concerns</p> <p>Line 179 and 180 refined and processed foods can contain components be added (nitrite) during the manufacturing process or formed (acrylamide) during prolonged heat treatment which can have health effects</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>References</p> <p>Chajes, V. et al Ecological-Level Associations between Highly Processed Food Intakes and Plasma Phospholipid Elaidic Acid Concentrations: Results From a Cross-Sectional Study Within the European Prospective Investigation Into Cancer and Nutrition (EPIC) Nutrition and Cancer 2011, 63(8), 1235 - 1250</p> <p>Floros et al "Feeding the World Today and Tomorrow: The Importance of Food Science & Technology". Comprehensive Reviews in Food Science and Food Safety 2010, 9, 572 - 599</p> <p>Heart Foundation Australia. Rapid review of the evidence: Effectiveness of food reformulation as a strategy to improve population health. 2012</p> <p>Monteiro, C.A., Levy, R.B., Claro, R.M., de Castro, I.R.R., Cannon, G. A new classification of foods based on the extent and purpose of their processing Cad. Saúde Pública, Rio de Janeiro, 26(11):2039-2049, Nov, 2010 and Public Health Nutrition 2010, 14(1), 5 - 13</p> <p>Moodie,R., Stuckler, D., Monteiro, C., Sheron, N., Neal, B., Thamarangsi, T., Lincoln, P., Casswell,S. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries Lancet 2013; 381: 670–79</p>	
European lifelong learning program "Eating Together"	<p>General comments</p> <p>I write on behalf of the Lifelong learning program n° 2011-1-BE3-GRU06-03638 (http://www.dietethics.eu/en/nutrition/national-guidelines.php).</p> <p>The Danish Animal Alliance takes part in this European group, collecting official dietary guidelines for vegan diets around the world to help to protect people who choose to live on vegan diets. The Nordic Nutrition Recommendations are very interesting. We have requested an in depth study of your very good drafts and previous editions by Stephen Walsh (coordinator of the International Vegetarian Union's science committee). His suggestions on the "Foods, food patterns and health outcomes: Guidelines for a healthy diet" focuses from line 132 onwards to question the comparison of death rates of the general population of Finland to that of Finnish fishermen from an ecological study.</p> <p>Detailed comments</p> <p>"Food sources of nutrients and other bioactive substances" from line 132 to 134</p> <p>"However, the lower mortality of cardiovascular diseases and type 2 diabetes among fishermen indicates that the benefits of fish consumption seem to overcome the potential hazards (Turunen, Verkasalo et al. 2008)." This conclusion from an ecological study comparing death rates in the general population in Finland with those of Finnish fishermen seems unwarranted. Ecological studies provide very low level evidence as they cannot make allowance for multiple differences between populations: e.g., the</p>	<p>Thank you, your comments have been acknowledged. The text has now been edited and shortened.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>fishermen studied were all engaged in physically active employment as well as having higher fish consumption. A prospective cohort study from Finland provides higher grade evidence and a less encouraging picture http://atvb.ahajournals.org/content/25/1/228.long with mercury levels having comparable adverse associations to the beneficial associations with higher blood levels of omega-3 fatty acids.</p>	
Baljväxtakad emin, Sweden	<p>General comments About the chapter of foods, food patterns and health outcomes, the perspective of dietary patterns and specific foods are a well appreciated perspective in the recommendation of a healthy lifestyle. We suggest using “legumes” instead of “pulses”. According to FAO definition the term “pulses” excludes legumes used for oil extraction (soybean, peanut) and those harvested green for food (green pea, green bean); the latter being classified as vegetables. http://www.fao.org/es/faodef/fdef04e.htm#4.01</p> <p>Detailed comments As described (line 295-299), red and processed meat are risk factors for colorectal cancer, and therefore something that should be limited in a healthy diet. Meat is at the same time a important contributor for minerals like iron and if not replaced with suitable foods, deficiency might occur. Here (301), pulses should be noted as a good source of minerals, which adequately can replace meat to some or full extent. Instead of just stating that plant foods in general might give mineral deficiency, a differentiation would give a more accurate picture. Table 1.1 states that we should increase our consumption of vegetables, fruit and berries, nuts and seeds, wholegrain and fish and sea food. Here, pulses is mentioned (inside the parenthesis) as any vegetable. The fact is that pulses are unique in its nutritional character with its high content of protein, minerals and fiber as described earlier. If the general population (and health practitioners) ought to understand this fact, a separation of pulses from vegetables should be done, just as for fruit, berries, nuts and seeds which also is unique in certain nutritional aspects. We therefore suggest that pulses/legumes (beans, peas and lentils) will have its one line in table 1.1.</p>	<p>Thank you for your informative comments. The text has been edited accordingly.</p>

Organisation Company	General and detailed comments	WG response & actions										
National Institute for Health and Welfare (THL), Finland	<p>NNR5 Comments to the chapter “Foods, food patterns and health outcomes – Guidelines for a healthy diet” Title => change into “Food-based dietary guidelines”</p> <p>We suggest the new title because food patterns are not explicitly defined in the text. Food patterns seem to refer to combinations of foods. The conclusions focus on foods, not on food patterns. The main message (lines 559-564) can be expressed without mentioning food patterns at all. General comments, structure The chapter is too long and its organisation is not clear. There are overlaps between the different parts. The text can be structured as follows (recommended length in parentheses):</p> <p>1. Guidelines and reasoning behind them (1-2 pages) -What is new compared to previous Nordic recommendations (2004), why are changes needed/not needed? -Why are no quantities or frequencies recommended, e.g. 400g/sex portions of fruit and vegetables daily; bread, pasta, rice and potatoes several times/ day... XX g read meat /day?</p> <p>2. Foods as sources of nutrients in the Nordic countries (2 pages) -Delete details and construct a table summarising the contribution of foods as sources of nutrients in the Nordic diet. The nutrient content of a single food needs to be related to the average consumption of the food in the Nordic countries. E.g. rye bread and milk products are relevant sources of many nutrients in Northern Europe because they are consumed in large quantities. The comment on energy dense and ultra-processed foods (lines 171-185) can be shortened and presented below the table. Table xx. Foods as sources of nutrients in the Nordic countries</p> <table><tr><th>FOOD GROUP</th><th>RELEVANT SOURCE OF</th></tr><tr><td>Vegetables, fruit and berries (roots, legumes, nuts?)</td><td>Potatoes</td></tr><tr><td>Cereals</td><td>Vegetable oils, margarines, butter (edible fats?)</td></tr><tr><td>Fish and seafood</td><td>Milk Eggs Meat</td></tr><tr><td>Sugar-containing foods and drinks</td><td>Alcohol</td></tr></table> <p>3. Health impact of foods and their combinations (4 pages) -Organise similarly to chapter 3, according to the same food groups as presented in the suggested new table. Maybe a table could be constructed also here. –</p> <p>There is no generally accepted definition to “dietary pattern”. Therefore it is impossible to construct evidence based recommendation concerning certain dietary pattern. A recommended (=healthy diet) can be composed from various different combinations of individual foods. After presenting the role of specific foods discuss shortly the role of different types of food combinations= dietary patterns, included in the referred studies, such as the prudent (?) diet, healthy/recommended diet, Mediterranean diet, Nordic diet... Whether a dietary pattern follows the recommendations can be evaluated with the help of indicators, such as the share of fat and saturated fat, sugar and alcohol out of the total energy intake. –</p> <p>Delete the text from lines 263-547. Include the central outcomes of these lines (reviews on foods, dietary patterns) in the previous chapter. The systematic reviews need no</p>	FOOD GROUP	RELEVANT SOURCE OF	Vegetables, fruit and berries (roots, legumes, nuts?)	Potatoes	Cereals	Vegetable oils, margarines, butter (edible fats?)	Fish and seafood	Milk Eggs Meat	Sugar-containing foods and drinks	Alcohol	<p>Thank you for your valuable comments, which have together with those of many other professionals in the field systematically been incorporated into the text when appropriate.</p> <p>A major focus of this chapter is food patterns (or combinations of many foods), as described in a separate section of the chapter. This concept has emerged during the last 15-20 years mainly in parallel with the use of statistical data reduction methodologies in nutritional epidemiology.</p>
FOOD GROUP	RELEVANT SOURCE OF											
Vegetables, fruit and berries (roots, legumes, nuts?)	Potatoes											
Cereals	Vegetable oils, margarines, butter (edible fats?)											
Fish and seafood	Milk Eggs Meat											
Sugar-containing foods and drinks	Alcohol											

Organisation Company	General and detailed comments	WG response & actions
	<p>separate subtitles and the discussion on methodological problems (lines 477-506) is unnecessary. The reviews should, naturally, be properly cited and maybe included as appendixes. –</p> <p>The review on “population groups in transition” does not belong to this chapter. All population groups have dietary patterns. If there is a need to present the recommendations for specific population groups also other groups in addition to immigrants from non-Western origin should be included, e.g. men/women, elderly, children, adolescents, families, marginalized, socio-economic etc...</p> <p>5. Conclusion (1 page) and Table 1.1. -Table 1.1 is good in principle. Why not present pulses and potatoes separately? However, slogans or messages that are ready for applications in health education, publicity and media should be better than long list in parenthesis. Slogans give examples of healthy choices. Examples: Compare salt contents – choose food items with lower content. Fill half of your plate with vegetables, roots or mixed salad. Should we include portion sizes in the table: Avoid big portions.</p> <p>Detailed comments PLEASE, SEE ABOVE, THE MOST IMPORTANT COMMENTS ARE NOT STATED ACCORDING TO THE LINES.</p> <p>6. Minor remarks: Line 52, rather foods tend to contain more than one or two nutrients. Line 85, All plant foods (including vegetables etc) Line 111, Fat spreads and trans-fat content? Are there possibilities to have vegetable-fat-based spread with high content of TFA’s in the Nordic food market? Should this be mentioned? Line 116, The fish examples do not cover very well the Nordic fish market. Line 120, There are results (Mattila et al) from high Vitamin D values in lean fish also. The sentence needs some reformulation. There should be a sentence concerning sustainable fish species! Line 175, sugar should be mentioned together with energy and salt. Lines 295-304, the reference for WCRF/AIRC here, but not on page 7? Should be there too. Lines 345-9, a reference needed? Lines 417-9, Is the evidence strong enough?</p> <p>7. Other comments Will there be any recommendations concerning the frequency and regularity of eating = meal pattern= eating pattern= number of eating events/day. In regard to the prevention of caries and obesity the frequency of eating is relevant. In</p>	

Organisation Company	General and detailed comments	WG response & actions
	addition, a regular pattern of 4-5 eating events (meals and snacks) helps in the maintenance of ideal weight.	
Valio Ltd	<p>Only general comments:</p> <p>One health outcome associated with food patterns is bone health and consumption of dairy foods. This association should be mentioned in the chapter along with other health associations mentioned. There are several references available to demonstrate the beneficial effect of dairy on bone health, such as:</p> <p>De Beer et al. Dairy products and physical stature: A systematic review and meta-analysis of controlled trials. <i>Econ Hum Biol.</i>2012;10(3):299-309.</p> <p>Caroli et al. Invited review: Dairy intake and bone health: a viewpoint from the state of the art. <i>J Dairy Sci.</i>2011;94:5249-62.</p> <p>Fiorito et al. Girls' Calcium Intake Is Associated with Bone Mineral Content During Middle Childhood. <i>J Nutr</i> 2006;136:1281-1286.</p> <p>Goulding et al. Children who avoid drinking cow's milk are at increased risk for prepubertal bone fractures. <i>J Am Diet Assoc</i> 2004;104(2):250-3.</p> <p>Huncharek et al. Impact of dairy products and dietary calcium on bone-mineral content in children: results of a meta-analysis. <i>Bone</i> 2008;43(2):312-21.</p> <p>Moore et al. Effects of Average Childhood Dairy Intake on Adolescent Bone Health. <i>J Ped</i> 2008;153(5):667-673.</p> <p>Mouratidou et al. Associations of dietary calcium, vitamin D, milk intakes, and 25-hydroxyvitamin D with bone mass in Spanish adolescents: the HELENA study. <i>J Clin Densitom.</i> 2013;16:110-7.</p>	<p>Thank you for your comments. We agree with you that the link between diet and bone health was not covered satisfactorily in the draft chapter. Therefore the text has been expanded. However, we would like to point out that the literature indicates that not only dairy foods but many dietary components in combination with lifestyle factors like physical activity are important for optimal bone health.</p>
Norewegian Directorate of Health	<p>General comments</p> <p>Overall this chapter is important, informative and clear. Most chapters start with a conclusion or recommendation. This is very convenient for the reader.</p> <p>You may consider starting with the conclusion 556-64 and table 1. Thus the lay-out will resemble other main chapters. Information concerning health effects and advice concerning choice of drinks (tea, coffee and cocoa in general, and juice/squash in relation to energy intake) is lacking and may be added. We believe this is of interest for the reader.</p>	<p>Thank you for your many valuable comments, and references of published studies. We have systematically updated the text in line with your comments, and those made by other researchers in the field.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>Dietary supplements are a part of the dietary pattern for large groups of the populations. This may be commented and discussed. You may also consider adding more information about energy balance and energy density.</p> <p>Detailed comments</p> <p>170. It is relevant to discuss both energy dense and ultra-processed food products. However these two concepts are mixed in this paragraph. Please define the two concepts and differentiate them in the text</p> <p>171-85. Energy density was defined and reviewed by WCRF 2007. As a consequence of your introduction of energy density here, one may expect that this is discussed in later chapters concerning associations between dietary intake and health effects. However, this is hardly not done.</p> <p>188-90. Consider adding one or two sentences about the intention of this section. Which health effects and or diseases do you intend to review here? You may also consider adding subtitles regarding food groups as in the previous section line 61.</p> <p>200-05. Vegetables, fruit and berry is here referred to as a group. You may consider giving some short information about separate groups as vegetables; fruits; berries; nuts and legumes. As the main conclusion in this chapter is “a dietary patternrich in vegetables ...” you should review health effects of vegetarian diets, here or in another suitable paragraph or chapter.</p> <p>205. The sentence regarding potatoes seems a bit strange. You may add some more information.</p> <p>233-44. Some intervention studies have suggested that dairy products may influence body weight, but the results remain controversial. In addition the NNR SLR by Fogelholm concludes “suggestive evidence was found for a protective role against increasing weight fromhigh-fat dairy products ...”. Thus you may here add information that a meta-analysis of RCT by Chen et al does not support the beneficial effect of increasing dairy consumption on body weight and fat loss in long-term studies or studies without energy restriction. Chen M, Pan A, Malik VS, Hu FB. Effects of dairy intake on body weight and fat: a meta-analysis of randomized controlled trials. Am J Clin Nutr. 2012;964:735-47.</p> <p>254. The sentence about WCRF recommendations concerning meat may be split in two as the quantitative recommendations concerning red meat and processed meat is very different, red meat less than 500 g/week and very little if any of processed meat, respectively.</p> <p>256. You may add that this is average consumption of red meat.</p> <p>258. You may add “considerable” in front of reduction.</p> <p>316-7. You may consider to add information about associations between intake of sugar,</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>sugar-containing foods and body weight and BMI.</p> <p>339-49. You should here consider to add information about the influence of energy balance and energy density of foods and diet. See also our earlier comments regarding the NNR chapter on energy.</p> <p>386. A handful of Nordic trials... Consider to add experiences from Finish studies as “When and how to start prevention of atherosclerosis? Lessons from the Cardiovascular Risk in the Young Finns Study and the Special Turku Coronary Risk Factor Intervention Project.” Magnussen CG et al. <i>Pediatr Nephrol.</i> 2012 Sep;27(9):1441-52.</p> <p>560. “fruiting vegetables” is this a common and easy understandable expression?</p>	
The Finnish Heart Association	<p>General comments</p> <p>We propose that the Guidelines for a healthy diet should also include recommendations for daily amounts of each food group. The draft includes a recommendation to adopt a dietary pattern that is “rich in vegetables, fruits and berries, nuts and seeds, whole-grains, fish and seafood, vegetable oils and low-fat dairy products”. We believe that the definition for “rich” is variant for each individual. Some kind of specification is needed. We also propose that some kind of recommendation should be mentioned for eating rhythm and the amount of meals per day. And a moderate portion size is essential regarding to weight control.</p> <p>Detailed comments</p> <p>line 110: Butter should be included as a notable source of SFA.</p> <p>line 113: It’s misleading to say “TFA content has... decreased”. It should be clarified that currently most margarines and vegetable based spreads contain TFA only in a neglectable amount.</p> <p>line 120-121: Vitamin D is not dependent of the amount of fat in fish. Word “Fatty” should be removed.</p> <p>line 137-138: A sentence “Milk and milk product are important source of fat...” could be misunderstood. We propose “considerable” instead of “important”. And saturated fat should be included to the list.</p> <p>line 561: The list of “rich in” should also include “margarines with a high content of unsaturated fats” or “soft margarines”.</p> <p>table 1.1: Yellow column, row 2: “butter based spreads” should also be mentioned along with butter.</p> <p>red column, row 3: “Food products with added fat and salt” should be replaced by “Food products with high in fat and salt”. Almost all food products contain added fat and salt, e.g. bread. Salt should be included in red column as itself.</p>	<p>Thank you for your comments; these together with other comments from many professionals have been incorporated into the edited text.</p> <p>The food based guidelines of the NNR focus risk assessment and required dietary changes at the population level to reduce chronic disease risk. A specification of the amounts of food is a management issue. That is a task left for the National Food Agencies in each country.</p>
SNF Swedish	Only general comments:	Thank you for the comments and suggestions, which

Organisation Company	General and detailed comments	WG response & actions
Nutrition Foundation	<p>General aspects</p> <ul style="list-style-type: none"> • The inclusion of the chapter “Food and food pattern” in the NNR5 makes the boundary between risk assessment and risk management less distinct. To avoid confusion, it would be valuable if it could be clarified how the recommendations in the chapter on “Food and food pattern” is related to for example national dietary guidelines. • The terminology used to describe foods and food categories (e.g. “junk foods”, “ultra-processed food”) are sometimes not well defined. To increase usefulness of NNR5 for risk management, all terms used to describe foods and food categories should be defined as precisely as possible. Ideally, each single food product found in the market should be possible to classify according to the terminology used. Legal definitions of various food groups should also be considered and used, if possible. In case of conflict between the scientific definition and legal definition, this should be commented. • Nutritional properties of foods/food categories are more important than their way of processing, in relation to their health effects. This should be clarified. <p>Page 5 line 149-154</p> <ul style="list-style-type: none"> • Since there has been some discussions regarding the definition of “processed meat” we suggest NNR5 to further explore on the definition of this term. For example, in the Norwegian dietary advice from 2011 (Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer. Metodologi og vitenskapelig kunnskapsgrunnlag. Nasjonalt råd for ernæring 2011) minced meat is defined as processed meat. Is minced meat included as processed meat in NNR5? Another question is if white processed meat is included, for example smoked/boiled turkey? • It is also important to carefully choose the correct word when translating “processed meat” to the different Nordic languages, not to cause any further confusion. <p>Page 5 line 170 • The term “ultra processed” is unfamiliar for us. This term needs to be defined, in relation to other types of processing.</p> <p>Page 5 line 170-181 • In the present text the word “processed food” could be perceived as it always is energy dense and low in nutrients, which is not the case. Most foods are processed, and many of them are high in nutrients and low in fat, salt and sugars. This should be clarified.</p> <p>Page 7 line 246-255 General about meat and processed meat</p> <ul style="list-style-type: none"> • Different types of “processed meat products” could have very different nutritional properties (eg. fat content, salt content). Therefore, it could be questioned why “processed meat” is not divided into different subgroups. Is, for example, the associations between consumption of smoked, lean ham and risk of disease equal to the associations for salami? I.e is it possible to grade different processed meat products in relation to the disease risk? It would be useful for the reader if NNR5 adds a comment 	<p>together with other comments from many professionals has been incorporated into the text.</p> <p>We agree that the NNR and its base the SR:s should deal with risk assessment and not risk management. We need to point out that risk assessment does not only concern nutrient exposures. When scientific studies examine food groups or food patterns as exposures it is possible to use food groups and food patterns as the base for risk assessment.</p> <p>Current research tells us very clearly and convincingly that dietary health risks cannot solely be associated with single nutrients; rather dietary risks depend on the overall dietary pattern. The whole diet needs to be considered simultaneously. This means that the impact of a nutrient may depend on other dietary qualities, and also on non-nutrient food factors. So far we have only limited understanding of the details of these mechanisms. However, there are whole diet trials and food pattern studies that indicate health risks, thus it is possible to some extent to define overall dietary patterns associated with higher and lower health risks. A more detailed specification of the amounts of foods to include is a management issue. These tasks are left to the National Food Agencies in each country.</p> <p>Unfortunately, studies do not always give detailed definitions of “processed” meat. In this chapter we use the definition of (and cite) the WCRF/AICR.</p> <p>We agree there is a great need for more research on various aspects on how foods and food processing influence health. The responsibility for such research would seem to largely rest on those that produce and introduce these food products into the market.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>on why “processed meat” is not divided into different subgroups.</p> <ul style="list-style-type: none"> • It would be valuable if NNR5 could add a short comment on possible mechanisms behind the associations between red meat/processed meat consumption and increased risk of cardiovascular diseases, type II diabetes and cancer. • Several references that report an association between risk of disease and consumption of red meat and processed meat are from countries outside the Nordic countries. It would be valuable if NNR5 could comment on differences/similarities in meat processing between different countries, where the studies are performed. Could there be factors in the process that differ between countries that could effect the association between processed meat and risk of disease? • There are future needs of more research around mechanisms behind meat consumption and risks of disease. This would make it possible for the food industry to develop healthier food products, by for example by changing the process or use of ingredients. <p>Page 20 Table 1.1</p> <ul style="list-style-type: none"> • We suggest modifying the colours of the table 1.1. The red colour signals “stop” and the reader can get the impression that the red column represents foods that you should avoid completely, rather than limit the intake of. • The term “junk food” is, though commonly used, still diffuse and should be defined more closely. • As table 1.1 probably will be a key message from this chapter we find it relevant to add a comment in connection to this table, that it is the total food intake and not single food items, that determine a healthy diet. • All food categories mentioned in the table 1.1 on page 20 should be commented in the background text. For example, nuts are included in the table, but not much discussed in the text. 	
Public Health Epidemiology unit, University of Gothenburg, Sweden	<p>General comments on the section “Food and dietary patterns of population groups in transition” in “Foods, food patterns and health outcomes: Guidelines for a healthy diet”</p> <p>The problems (vitamin D deficiency, dental caries, overweight and obesity, iron deficiency) that are pointed out in the section “Food and dietary patterns of population groups in transition” are important public health issues. It is urgent to present a basis of knowledge that will allow health professionals to give individuals and minority groups adequate support in prevention and treatment. The basis of knowledge should be of a quality that makes it appropriate to communicate to the populations in question as an ingredient in self-care. By Table 1 in the cited review (Wändell 2013); immigrants from Africa and Asia add up to 1.037 000 people in the Nordic countries. Most of the data on</p>	<p>Thank you for your comments.</p> <p>This section has now been removed from this chapter.</p> <p>Instead some of these issues are discussed in the separate nutrient chapters.</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>which the statements in the section are based on are collected from a very small part of the populations to which they are generalized. Problems should be described, researched and addressed. However, to enable this, including the fine-tuned analysis that can guide searches for causality, “population groups in transition” have to be more specific. Otherwise there is a risk of masking differences between minority groups and to create unnecessary worry and misguided efforts, potentially stigmatizing groups that do not in reality have a higher risk of worse health outcomes. Also, with categories of population groups that do not reflect reality, there is a risk to neglect risk factors such as skin colour (see below on vitamin D). Renzaho et al demonstrated shortcomings of broad generalisations by showing substantial discrepancies within the sub-Saharan refugee group in Australia; concerning food habits, sedentary time and physical inactivity (1). The diverse lifestyles yielded significant differences in prevalence of childhood obesity within the group. The systematic review on population groups in dietary transition points out that there are no clear patterns for dietary changes. It is also worth underlining – which the review by Wändell (2013) does not – that many immigrants from non-Western countries had a “western diet” already in their home countries before migrating. Only few “ethnic” groups have been extensively studied (e.g. women from Sri Lanka and Pakistan living in Oslo) in this regard. For those “ethnic” groups where studies are limited or even non-existing, closer examination is needed before statements can be made. By focusing on migration there is a risk to miss other factors such as income, education, unemployment, discrimination and stress, that are linked to adverse health outcomes. Skin colour, which is relevant in relation to vitamin D status, is another example of factor that may be included when assessing the combined risk of developing a disease or vitamin D deficiency. Describing categories of populations groups thoroughly (preferably in cooperation with the groups in question) from an objective point of view, helps to avoid stigmatization of minority groups. One example of potentially stigmatizing categories is to compare “children to immigrant parents” with “children to Danish parents” as in the study cited in Wändell (2013) (p 7). It raises the question whether citizenship is not enough base for nationality, since many immigrants are also Danish citizens – or at least, they could be, by the current definition of “immigrant”. I suggest rewriting the section “Food and dietary patterns of population groups in transition”, with focus on the above mentioned risk factors for nutritional adverse outcomes. 1. Renzaho AM, Swinburn B, Burns C. Maintenance of traditional cultural orientation is associated with lower rates of obesity and sedentary behaviours among African migrant children to Australia. Int J Obes (Lond). 2008;32(4):594-600.</p> <p>Detailed comments on the section “Food and dietary patterns of population groups in</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>transition" in "Foods, food patterns and health outcomes: Guidelines for a healthy diet"</p> <p>Line 351 The title "population groups in transition" mirrors the confusion and unclear categorization of groups. "In transition" means "a change from one place or state or subject or stage to another" (www.tyda.se) and is, in several senses, attributable to most of the population all the time. However, the focus in this section lies on non-Western immigrants.</p> <p>Line 358 The statement: "...the risk of vitamin D deficiency is high in the Nordic countries among non-Western immigrants, especially women and children" rests on approximately 3000 female immigrants (Tamils, "Arabs", Pakistani and Somali) and 382 children (tables 3, 4 and 5 in Wändell 2013). If women constitute half of the immigrants from non-Western countries, the examined population correspond to 0,6 %, from a limited number of countries. Thus, it is questionable to generalize these findings to all non-Western immigrants.</p> <p>Lines 359-362 possible reasons for discrepancies in vitamin D status are discussed. Skin colour, which is a known factor to influence cutaneous production of vitamin D, is however not mentioned. The comment about low consumption of fatty fish and vitamin D enriched dairy products may be based on the statement in Wändell (2013)(p 19); "However, fish-eating dietary habits and vitamin D supplement use are not wide-spread among non-Western immigrants in the Nordic countries". However, it is unclear how Wändell comes to that conclusion, since that statement does not have a reference and since pages 16-18 show that the data on this is limited.</p> <p>Line 363 The statement "Dental caries is high among "immigrant children" in Nordic countries due to higher intake of sugary product" is based on 1425 subjects from Turkey, Pakistan, Somalia, Albania and "Arabs".</p> <p>Line 365 "Overweight and obesity, associated to higher risk of diabetes and CHD, are prevalent in many immigrant groups and need further attention". OW and obesity have an extremely heterogeneous aetiology. Income, education, occupation are important determinants, together with discrimination and stress. The category "immigrant status" is too crude to be of any help.</p> <p>Lines 366-367 The sentence "There are also reports that iron deficiency is more common among immigrant women" is based on data from 38 Pakistani women. Despite limited data, findings from one minority group are generalized to the immigrant population.</p>	

Organisation Company	General and detailed comments	WG response & actions
Swedish Food Federation	<p>General comments</p> <p>The Swedish Food Federation is positive to the inclusion of the chapter “Food, food pattern and health outcomes - Guidelines for a healthy diet” NNR 2012. It highlights the importance of linking the impact of single nutrient to food and food pattern. However, the inclusion of this chapter highlights the lack of clarity between risk assessment and risk management in NNR 2012. It is therefore desirable to clarify how the recommendations in the chapter are related to national food based guidelines. It is our opinion that NNR 2012 only should handle risk assessment. Risk assessment and risk management/food based dietary guidelines should be kept as separate tasks and food based dietary guidelines is in our opinion a task for the national authorities to take place on top of the recommendations. Differences between countries and regions (prevalence of nutrient imbalances, diet-related public health issues, dietary habits and traditions) require that the guidelines should be national (see EFSA:s Scientific Opinion on establishing Food-Based Dietary Guidelines, EFSA Journal 2010; 8(3):1460).</p> <p>The terminology used to describe foods and foods categories (processed, ultra-processed, junk-food, refined and highly processed) is not scientific terminology and should be replaced.</p> <p>The terminology should be focused on the nutritional properties of foods/food categories, e g “foods high in fat, sugar and salt”.</p> <p>We fully support the comments on the chapter sent by Lilia Ahrné, SIK/Chalmers. These comments show the need of more precise and scientific based terminology.</p> <p>Detailed comments</p> <p>Line 77 Replace “antioxidants” with “phytochemicals”; antioxidants are already mentioned (e g vitamin E and zinc) and whole-grains also contain phytochemicals with other biological functions than antioxidative.</p> <p>Line 82 Replace “low” with “lower”, and “high” with “higher”. No definition exists telling what is low or high.</p> <p>Line 149-154 and 246-255 The Swedish Food Federation and the Swedish Meat Industry Association fully support the comments sent by Swedish Nutrition Foundation (SNF).</p> <p>Line 170-185 and Line 558-564 The terminology used to describe foods and foods categories (processed, ultra-processed, highly processed, refined) is not scientific terminology and should be replaced. The terminology should be focused on the nutritional properties of foods/food categories, e g “foods high in fat, sugar and salt”.</p> <p>We fully support the comments on the chapter sent from Lilia Ahrné, SIK/Chalmers. These comments show the need of more precise and scientific based terminology</p> <p>Line 722 – Table 1.1 The colours used in table 1.1 should be modified. The use of colours</p>	<p>We have received several comments on the unscientific nature of the terms “processed” and “ultra-processed foods”, which to us are surprising since these issues are debated in the scientific literature and have been the subject of scientific scrutiny.</p> <p>The paragraphs now titled “Characteristics of dietary patterns” discusses such issues and cites scientific papers published in peer-reviewed scientific journals that have suggested food group definitions according to the degree of processing.</p> <p>We agree that the NNR and its base the SR:s should deal with risk assessment and not risk management. We need to point out that risk assessment does not only concern nutrient exposures. When scientific studies examine food groups or food patterns as exposures it is possible to use food groups and food patterns as the base for risk assessment.</p> <p>Current research tells us very clearly and convincingly that dietary health risks cannot solely be associated with single nutrients; rather dietary risks depend on the overall dietary pattern. The whole diet needs to be considered simultaneously. This means that the impact of a nutrient may depend on other dietary qualities, and also on non-nutrient food factors. So far we have only limited understanding of the details of these mechanisms. However, there are whole diet trials and food pattern studies that indicate health risks, thus it is possible to identify overall dietary patterns associated with higher and lower health risks.</p> <p>A more detailed specification of the amounts of foods to include in a diet is a management issue. This is a task for the National Food Agencies in each country.</p>

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	<p>in a scientific based risk assessment could be questioned. The red colour signals foods that should be completely avoided rather than limited. The term “junk food” is not scientific terminology and should be replaced and defined more closely.</p>	<p>The Table 1.1 has been edited based on discussions in the NNR Working group and comments from several other professionals in the field.</p>
AarhusKarlshamn AB	<p>General comments</p> <p>We agree with the conclusion that there is convincing evidence for health benefits of replacing saturated fatty acids with unsaturated fatty acids and, as stated in line 223, also that long chain omega-3 fatty acids seems to have specific health benefits. We do not see any strong evidence of health benefits from reducing total fat from the present level and as we read the report this is also the conclusion of the NNR expert group. As is pointed out by e.g. the FAO report from 2010 on fat and fatty acids, average E% from fat has decreased in industrialized countries and at the same time obesity is increasing. Regarding risk of CVD beneficial effects have been shown when replacing saturated fats with unsaturated, but not from simple reduction of total fat.</p> <p>Detailed comments</p> <p>Line 243: Refers to limited/suggestive evidence of a link between various forms of cancer and a high total fat intake. We would like to point out the fact that total fat may confound the effect of energy and that total fat per se does not contribute to cancer risk (FAO 2010 report).</p> <p>Line 442 - 448 & Table 1.1 We would like to challenge the suggestion to “limit products with added fat” in Table 1.1, as this may be misunderstood or even counterproductive. Recent research, among others Estruch et al, line 442 – 448, conclude that a high fat content, in this case 40%E, is not a risk factor compared to the fat composition. In the Nordic diet, a major part of saturated fat comes from natural sources like meat and dairy products, whereas food products containing added fat like margarine, spread, salad dressing and mayonnaise are major sources of (poly)unsaturated fatty acids. It is also a fact that the fat composition of these products with added fat has been and is continually being developed in the direction of the dietary guidelines, whereas it is often difficult or impossible to change the fat composition of food product having a natural high content of fat and saturated fatty acids.</p>	<p>Thank you for your comments.</p> <p>We agree that total energy intake and a positive energy balance are major risk factors for cancer and most chronic disease. In this context it is important to recognize that diets high in total fat will be more energy dense than those low in fat. Energy-adjustment is a major issue in the statistical analysis of nutritional epidemiology studies. Thus the conclusion that a high total fat intake is associated with an increased risk is the risk observed when high energy intake has been accounted (adjusted) for.</p> <p>Table 1.1 represents the interpretation of findings from <u>all</u> gathered evidence. Many aspects of dietary influences on health (i.e., energy density, micro-nutrient density and nutrient requirements, fat quality (SFA vs. MUFA+PUFA), carbohydrate quality, food selection and the overall dietary pattern), are here combined into a description of an optimal diet. Based on the suggestions from many professionals in the field and discussion in the NNR Working group we have edited the chapter and also Table 1.1.</p>

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Animalia - The Norwegian Meat and Poultry Research Centre	<p>Only detailed comments:</p> <p>Consumption of food is not a matter of eating nutrients or single food items, but a complex and varied diet. It is the entire diet that impacts health. Animalia - The Norwegian Meat and Poultry Research Centre appreciate the opportunity to comment on the chapter “Food, food patterns and health outcomes: Guidelines for a healthy diet” in NNR5.</p> <p>Line 246 The role that beef consumption specifically plays in promoting adverse changes in the diabetes or cardiovascular risk factor profiles is much more unclear than it is presented in the draft chapter. The chapter only presents a few of the studies conducted in this area, not rendering the diverging results in the different studies. We suggest the draft to include a wider selection of studies presenting the divergent results from studies evaluating the effect of red and processed meat on diabetes and cardiovascular disease (1-6). The results of the Fretts’ study find an association between processed meat and diabetes, but not with unprocessed red meat and diabetes (5), which we recommend to be included in the text. This is also in line with the results from the study of French women by Lajous et al (6). A meta-analysis of 8 randomized, controlled, clinical trials (RCTs) has evaluated the effects of beef, independent of other red and processed meats, compared with poultry and/or fish consumption, on lipoprotein lipids (2). The results demonstrated similar changes in the fasting lipid profile with beef consumption compared with those with poultry and/or fish consumption. The researchers concluded that this indicated that inclusion of lean beef in the diet increases the variety of available food choices, which may improve long-term adherence with dietary recommendations for lipid management. The nuances in the conclusions by Micha et al have neither been integrated into the draft chapter (7). The study is reporting a weak association between processed meats, but not red meats and coronary heart disease and diabetes mellitus. The discussion following their publication (8,9) should also be included in the chapter discussion, since Micha et al are clearly stating that their findings “suggest that both future research and policy efforts should separately consider unprocessed red and processed meats in relation to CHD and diabetes risk. Our findings, and those of others, also suggest that effects of dietary fats in these meats may be less relevant than other characteristics, such as type of processing and content of preservatives. These are not minor issues. Currently, parents and spouses around the world often choose processed “low-fat” deli meats for their loved ones, assuming that this is a healthier choice than unprocessed red meats. Our findings do not support this assumption, indicating a need for further rigorous evaluation of health effects of different types of meats and underlying reasons for these effects, to inform evidence-based preparations and processing of meats to minimize harms.”</p>	Thank you for your important comments. We agree with many of your suggestions, and have updated and edited the text accordingly.

Organisation Company	General and detailed comments	WG response & actions
	<p>Line 251 The association between intake of red meat and colorectal cancer was reduced from the Global report to the CUP publication. We propose this fact to also be covered in the chapter text (10).</p> <p>Line 470 Another publication from last year studied the effect on LDL cholesterol of cholesterol lowering diets with varying amounts of lean beef (3). The study design was based on the the DASH studies, and the participants in one of the arms of the study followed the DASH diet including 28 g beef/d. Another study arm was the DASH diet including a red meat more in line with the regular intake (BOLD - Beef in an Optimal Lean Diet), 113 g beef/d. The third arm was BOLD plus additional protein (BOLD+) including 153 g beef/d. These three arms were compared with a healthy American diet (HAD). The results showed a decrease in both total cholesterol (TC) and LDL-cholesterol concentrations after consumption of the DASH, BOLD and BOLD+ diets compared with after consumption of the HAD. Apolipoprotein A-I, C-III, and C-III bound to apolipoprotein A1 particles decreased after BOLD and BOLD+ diets compared with after the HAD, and there was a greater decrease in apolipoprotein B after consumption of the BOLD+ diet than after consumption of the HAD. The study concludes that low-SFA, heart-healthy dietary patterns that contain lean beef elicit favorable effects on cardiovascular disease (CVD) lipid and lipoprotein risk factors that are comparable to those elicited by a DASH dietary pattern. These results, in conjunction with the beneficial effects on apolipoprotein CVD risk factors after consumption of the BOLD and BOLD+ diets, which were greater with the BOLD+ diet, provide support for including lean beef in a heart-healthy dietary pattern. We propose this reference to be included in the draft chapter, to give a broader presentation of the picture. Further, these results indicates that it might be a high intake of processed meat or dietary of lifestyle factors associated with intake of processed meat rather than the lean beef itself,- indicating that it is room for lean beef in a healthy diet.</p> <p>Line 562 In our experience, the current evidence is too inconsistent and inconclusive to conclude about a causal relationship between the intake of red meat per se and diabetes, cardiovascular disease and other lifestyle diseases.</p> <p>Our suggestion is to remove the term “red meat” from the conclusion, line 562, and from table 1.1 until further evidence is obtained.</p> <p>References: 1. World Cancer Research Fund / American Institute for Cancer Research. (2007) Food, Nutrition, Physical Activity and the prevention of Cancer: a Global Perspective. Washington, DC, AICR and the update project http://www.dietandcancerreport.org/cup/index.php 2. McNeill S, et al. Red meat in global nutrition. Meat Sci. 2012 Nov;92(3):166-73. doi: 10.1016/j.meatsci.2012.03.014. www.ncbi.nlm.nih.gov/pubmed/22658075 3. Maki KC, et al. A meta-analysis of</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>randomized controlled trials that compare the lipid effects of beef versus poultry and/or fish consumption Journal of Clinical Lipidology 2012 volume 6 issue 4 Pages 352-361 DOI: 10.1016/j.jacl.2012.01.001 www.lipidjournal.com/article/S1933-2874(12)00015-3/fulltext 4. Roussell MA, et al. Beef in an Optimal Lean Diet study: effects on lipids, lipoproteins, and apolipoproteins. Am J Clin Nutr 2012;95:9–16. http://ajcn.nutrition.org/content/95/1/9.full.pdf 5. van Woudenberg GJ, et al. Meat consumption and its association with C-reactive protein and incident type 2 diabetes: the Rotterdam Study. Diabetes Care. 2012 Jul;35(7):1499-505. doi: 10.2337/dc11-1899. www.ncbi.nlm.nih.gov/pubmed/22596177 6. Fretts, AB, et al. Associations of processed meat and unprocessed red meat intake with incident diabetes: the Strong Heart Family Study. Am. J. Clin. Nutr 2012;95(3): 752-758 www.ncbi.nlm.nih.gov/pubmed/22277554 7. Lajous M, et al. Processed and unprocessed red meat consumption and incident type 2 diabetes among French women. Diabetes Care. 2012 Jan;35(1):128-30. doi: 10.2337/dc11-1518. www.ncbi.nlm.nih.gov/pubmed/22100967 8. Micha, R., G. Michas, et al. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes--an updated review of the evidence. " Curr Atheroscler Rep. 2012; 14(6): 515-524. www.ncbi.nlm.nih.gov/pubmed/23001745 9. Bryan NS. Letter by Bryan Regarding Article, "Red and Processed Meat Consumption and Risk of Incident Coronary Heart Disease, Stroke, and Diabetes Mellitus: A Systematic Review and Meta-Analysis". Circulation. 2011; 123: e16 doi: 10.1161/ http://circ.ahajournals.org/content/123/3/e16.long 10. Renata Micha R, et al. Response to Letter Regarding Article, "Red and Processed Meat Consumption and Risk of Incident Coronary Heart Disease, Stroke, and Diabetes Mellitus: A Systematic Review and Meta-Analysis". Circulation. 2011;123:e17, doi:10.1161 http://circ.ahajournals.org/content/123/3/e17.full</p>	
Dansk Industri	<p>General comments</p> <p>The Confederation of the Danish Food Industry appreciates this opportunity to provide specific comments to this chapter. We recognize there might be differences across population groups in terms of guidelines for a healthy diet but wants to highlight the importance of considering the ability to align with EU or WHO guidelines as the food supply is global. Although we appreciate the whole food approach we also believe it is not feasible to ignore what we know about the need for specific nutrients, and how the needs for these can be fulfilled. Thus, we argue for a multifaceted approach. How perfect foods may seem, they might not have the optimal composition as they are.</p> <p>Detailed comments</p> <ul style="list-style-type: none"> • Row 97: We propose to replace "spreads" by "vegetable oil based fat spreads", as fat 	<p>Thank you for your valuable comments; most of these have been considered together with other comments from many professionals when editing the text.</p> <p>We agree that the health effect of a whole range of different foods and food groups should be evaluated. In the project by Åkesson et al (2013) that systematically reviewed the health effects of food groups and chronic disease, it was absolutely necessary to limit the research questions due to the limited resources and persons involved (6 researchers and 2 librarians).</p>

Organisation Company	General and detailed comments	WG response & actions
	<p>spread is the denominator used in the English version of the Council Regulation (EC) 1234/2007. This will also avoid any confusion with other types of spreads.</p> <ul style="list-style-type: none"> • Row 97-114: Consequently “spreads” should then be replaced by “vegetable oil based fat spreads” throughout this paragraph. • Row 99: We suggest adding of other sources of oils such as “sunflower seeds” and “linseeds”. • Row 100: Margarine is a mixture of different vegetable oils and fat which also needs to be fully reflected. We therefore suggest making the sentence more explicit by stating “Margarine and fat spreads are mixtures of different vegetable oils and fats.” • Row 102-104: Adding of vitamin A and D is regulated by law. Thus this should also be reflected in the paragraph. Our suggestion is then to rephrase accordingly: “Vitamin A and D are added to margarines and vegetable oil based fat spreads according to regulation”. This will also provide further clarity to consumers on the basis of adding of vitamin A and D. • Row 111-114: We suggest amending the sentences into: “Margarines and vegetable oil based fat spreads were previously sources of TFA, depending on the raw material used in manufacturing”. “However, the TFA content has during the last decade decreased considerably in the Nordic countries and today the TFA levels is much lower.” <p>Nutritional aspects should set the boundary conditions for health impact of foods and not the way of production or origin.</p> <p>References (Row 97-114)</p> <p>- Intake of fatty acids in Western Europe with emphasis on trans fatty acids: The TRANSFAIR study. European Journal of Clinical Nutrition (1999) 53, 143±157. - The Elimination of Trans Fats from Spreads: How Science Helped to Turn an Industry Around. Onno Korver, PhD, and Martijn B. Katan, PhD. Nutrition Reviews, Vol. 64, No. 6: 275–279. June 2006 - Reformulation revolution: Trans fatty acids records big drop in UK foods since 2007. May 2013.</p> <p>https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/167938/Summary_Report.pdf - Stender S, Astrup A, Dyerberg J. A trans European Union difference in the decline in trans fatty acids in popular foods: a market basket investigation. BMJ Open 2012;2:e000859. doi:10.1136/bmjopen-2012-000859.</p> <ul style="list-style-type: none"> • Row 170-185: Classifying foods as “processed” and/or “ultra-processed” is scientifically not tenable and is not helping consumers making healthy food choices. Manufactured foods make a significant contribution to the nutrient content of the diet and in terms of nutrition there are many benefits to modern processing techniques. They can minimise nutrient losses -where nutrients have been lost they can readily be restored - and they can also enable fortification to increase the nutritional value of food. Although we 	<p>The section now titled “Characteristics of dietary patterns” discusses some issues of concern related to Westernized diets, and cites published scientific papers that have suggested food group definitions according to the degree of processing.</p>

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	<p>acknowledge the potential health adverse effects of eventual components added during manufacturing we believe nutritional aspects firstly should set the boundary conditions for health impact of foods and not the way of production or origin. In addition the paragraph lacks a definition for what is regarded as “processed” and/or “ultra-processed” leading to inconsistency when making references to different types of food. For example dried soups are seen as ultra-processed whereas dried meat is not. The paragraph would benefit from also having a recommendation to consume low salt and/or low saturated food to ensure consistency with overall guidelines for a healthy diet.</p> <ul style="list-style-type: none"> • Row 170: Thus, we propose to change the heading to “Energy dense food products”. This is aligned with previous NNR and is also better reflecting that processed food can have a balanced nutrition profile. • Row 175: Also, there have been reductions in the salt content of manufactured foods following the release of a technical World Health Organization report in 2003. In the expert consultations and technical meetings on sodium an explicit call was also made to the food industry to help lower sodium intakes among populations. We believe this should be reflected in the paragraph as well as a recommendation to consume low salt and/or low saturated food to be amended to ensure consistency with overall guidelines for a healthy diet. Reference: http://www.who.int/dietphysicalactivity/reducingsalt/en/ • Row 178: Powder soup and sauces should be removed as a reference of “ultra-processed food products”. Drying is a form of mild processing, and especially dried vegetables have been shown to have good nutrient retention. Today’s technological advances mean that it is now possible to carefully treat food in such a way that it retains most of its nutritional content such as proteins, carbohydrates, fats, vitamins and minerals. This particularly applies to the dehydration process, which is one of the oldest methods for preserving foods being a truly natural preservation method. Also removing of the water content delays spoilage reactions which in turn allows the food to be stored at room temperature for long period of time. No preservatives need to be added. Finally, dried soups and sauces have a short cooking time, ensuring the nutritional value and taste is preserved. Above is also confirmed by a recent study by Basendowski, et al (submitted) comparing the nutrient retention of key nutrients and non nutrients in fresh and processed vegetables, supermarket versus industrial origin and also commercial dried soups versus homemade soups. Results concluded that industrial dehydrated soups are from a nutrient content point of view comparable to their corresponding homemade soups. <p>References: - Supplementary information from http://epic.iarc.fr Slimani S - Eur J Clin Nutr. (2009) Nov;63 Suppl 4:S206-225 - Takeoka GR, Dao L, Flessa S, Gillespie DM, Jewell WT, Huebner B, Bertwo D, Ebeler SE. (2001)Processing Effects of Lycopene Content and</p>	

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	<p>Antioxidant Activity of Tomatoes. Journal of Agricultural and Food Chemistry, 49(8), 3713-3713 - Abushita AA, Daood HG, Biacs PA (2000). Change in Carotenoids and Antioxidant Vitamins in Tomato as a Function of Varietal and Technological Factors. Journal of Agricultural and Food Chemistry, 305(6), 2075-2081 - Basendowski S, Spraul M, Feunekes G (Submitted). Nutrient retention of selected marker compounds during dehydration and preparation of dried versus homemade soups. Journal of Food Composition and Analysis</p> <ul style="list-style-type: none"> • Row 222: We believe the paragraph should be aligned with the WHO /FAO recommendations which include both short and long chain PUFA. Thus we suggest to amend the sentence into: "Many reports indicate that there is convincing evidence for the health benefit of replacing SFA in the diet with unsaturated fat, both PUFA from vegetable oils, vegetable oil based fat spreads and very long omega-3 PUFA from fish (Nasjonalt råd for ernæring 223 2011)". References: FAO report of an expert consultation on fats and fatty acids in human nutrition. Geneva, 2008. http://foris.fao.org/preview/25553-0ece4cb94ac52f9a25af77ca5cfba7a8c.pdf WHO/FAO. Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO expert consultation, 28 January - 1 February 2002, Geneva, Switzerland (WHO Technical Report Series 916) http://www.who.int/nutrition/publications/obesity/WHO_TRS_916/en/index.html Explaining the 25-year decline of serum cholesterol by dietary changes and use of lipid-lowering medication in Finland. Valsta LM, Tapanainen H, Sundvall J, Laatikainen T, Männistö S, Pietinen P, Vartiainen E. Public Health Nutr. 2010 Jun;13(6A):932-8. Judd JT, Baer DJ, Clevidence BA, Muesing RA, Chen SC, Weststrate JA, Meijer GW, Wittes J, Lichtenstein AH, Vilella-Bach M, Schaefer EJ. Effects of margarine compared with those of butter on blood lipid profiles related to cardiovascular disease risk factors in normolipemic adults fed controlled diets. Am J Clin Nutr 1998 Oct;68(4):768-77. Tonstad S, Strom EC, Bergei CS, Ose L, Christophersen B. Serum cholesterol response to replacing butter with a new trans-free margarine in hypercholesterolemic subjects. Nutr Metab Cardiovasc Dis 2001 Oct;11(5):320-6. Gagliardi AC, Maranhao RC, de Sousa HP, Schaefer EJ, Santos RD. Effects of margarines and butter consumption on lipid profiles, inflammation markers and lipid transfer to HDL particles in free-living subjects with the metabolic syndrome. Eur J Clin Nutr 2010 Oct;64(10):1141-9. Lecerf JM, Luc G, Marecaux N, Bal S, Bonte JP, Lacroix B, Cayzele A. Small, qualitative changes in fatty acid intake decrease plasma low-density lipoprotein-cholesterol levels in mildly hypercholesterolemic outpatients on their usual high-fat diets. Int J Food Sci Nutr 2009;60 Suppl 7:151-63. Seppanen-Laakso T, Vanhanen H, Laakso I, Kohtamaki H, Viikari J. Replacement of butter on bread by rapeseed oil and rapeseed oil-containing 	

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	<p>margarine: effects on plasma fatty acid composition and serum cholesterol. Br J Nutr 1992 Nov;68(3):639-54.</p> <ul style="list-style-type: none"> • Row 233-236: For a non specialist reader, these two sentences might appear contradictory. It seems to be a health difference between consumption of milk and general consumption of other dairy products such as butter. We therefore suggest adding the mentioning of “Low-fat dairy consumption is recommended to sustain calcium intake while reducing saturated intake”. This is also in line with the overall conclusion of this chapter (row 561). • Row 277-287: If the suggested references below are not already taken into account we would like to suggest the authors of the review (Akesson et al. 2013) to evaluate the adding of additional references regarding the food group “milk” and “milk products”, two of which are intervention trials performed in the Nordics countries. These references address the impact of butter vs. margarines on blood cholesterol and underpin the conclusion advising to limit butter and replace it by soft margarines and vegetable oil based fat spreads. References: Tonstad S, Strom EC, Bergei CS, Ose L, Christophersen B. Serum cholesterol response to replacing butter with a new trans-free margarine in hypercholesterolemic subjects. Nutr Metab Cardiovasc Dis 2001 Oct;11(5):320-6. Seppanen-Laakso T, Vanhanen H, Laakso I, Kohtamaki H, Viikari J. Replacement of butter on bread by rapeseed oil and rapeseed oil-containing margarine: effects on plasma fatty acid composition and serum cholesterol. Br J Nutr 1992 Nov;68(3):639-54. Judd JT, Baer DJ, Clevidence BA, Muesing RA, Chen SC, Weststrate JA, Meijer GW, Wittes J, Lichtenstein AH, Vilella-Bach M, Schaefer EJ. Effects of margarine compared with those of butter on blood lipid profiles related to cardiovascular disease risk factors in normolipemic adults fed controlled diets. Am J Clin Nutr 1998 Oct;68(4):768-77. Gagliardi AC, Maranhao RC, de Sousa HP, Schaefer EJ, Santos RD. Effects of margarines and butter consumption on lipid profiles, inflammation markers and lipid transfer to HDL particles in free-living subjects with the metabolic syndrome. Eur J Clin Nutr 2010 Oct;64(10):1141-9. Lecerf JM, Luc G, Marecaux N, Bal S, Bonte JP, Lacroix B, Cayzeele A. Small, qualitative changes in fatty acid intake decrease plasma low-density lipoprotein-cholesterol levels in mildly hypercholesterolemic outpatients on their usual high-fat diets. Int J Food Sci Nutr 2009;60 Suppl 7:151-63. • Row 306-317: In order to further clarify the perceived intention to address sugar based foods, we suggest that the headline is changed into “Foods high in sugar”. • Row 411: Besides rapeseed oils, the diet of the SYSDIET study also contained margarines based on vegetable oils (page 5 in article), with an overall diet containing >2/3 unsaturated fats. This should also be reflected in the text. • Row 561: We suggest the adding of “vegetable oil based fat spreads” as an example of 	

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	<p>a dietary pattern that is recommended to be adapted. To provide consistency as well as expand the food choices available for consumers the text in the Conclusion and the Table 1.1 (row 722) should also be aligned thus mentioning both “vegetables oils, soft margarines and vegetable oil based fat spreads with a high content of unsaturated fats”. First, the denominators for edible fats/butter/spreads/margarine are regulated in Council regulation 1234/2007. And by including vegetable oils based fat spreads to the choices this would give the complete variables of the soft fat group aiming to reduce saturated fats Second, the adding of the wording “fat spreads” shows an expanded choice from consumer perspective in line with the final conclusions. • Row 562-563: We suggest the removing of the “refined and highly processed food products”. Classifying foods as “processed” is scientifically not tenable and not helping consumers making healthy food choices. The health impact of foods should be evident from the nutrition panel, not from way of production or origin. This would also enable a better alignment between the Conclusion and Table 1.1 (row 722) where we suggest the wording to be: “Food products high in sugar, salt, saturated fatty acids and transfatty acids should be limited”. • Row 722: Table 1.1 (summary table): First, we suggest to add the wording “vegetable oil based fat spreads” to ensure consistency with the Conclusions as well as expand the food choices available for consumers. Second, we suggest to change the heading “Drinks and food products with added sugar” to “Drinks and food products high in sugar”. Third, to change “Food products with added fat and salt” to “Food and products high in fat and salt content”. We believe this will provide better clarity and also better differentiate food where sugar and salt is added in a reasonable amount.</p>	
Minivent AB	<p>Only general comments: NNR 2012 –Foods, food patterns and health outcomes: Guidelines for a healthy diet Public consultation answer By Björn Hammarskjöld M.D., Ph.D. in Biochemistry Independent senior scientist in nutrition C.E.O. of Minivent AB 13. Foods, food patterns and health outcomes - Guidelines for a 14. healthy diet 15. 16. 17. 18. Introduction 19. Lines 37- 48 should be replaced with the following text. Much new original data from both observational and experimental studies on the impact of foods, whole diets and food patterns on health have been published during the last centuries. Several reports, reviewing the scientific basis for linking food intake and food consumption patterns with chronic disease are available. This chapter mainly summarizes information from some hundred years ago. The first scientific report of the food, food patterns and health outcomes from Nordic countries is from 1732 when Carl von Linné described the Sami diet and also the health outcome . In his report he described vividly that the Sami had reindeer meat, reindeer milk, fish and fowl. In season the Sami had egg and berries. Linné described that the Sami in their sixties still could roam around in the mountains in</p>	<p>Thank you for your interest in the revision of the Nordic Nutrition recommendations. Your comments have together with those of many professionals in the field of food and nutrition been considered when editing the text.</p>

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	<p>contrast to the fat, breadeating farmers from Scania To follow a diet is good for loosing weight and to avoid diabetes and obesity. There is no controverse which diet is the best. Already the old pig farmer knew how get a pig fat for Christmas. Just give the pig coarse milled grain, potatoes and some fat. NNR4 recommend coarse milled grain, rebranded fibre rich bread and fibre rich cereals, oil cooked potatoes, rebranded French fries/chips. Also the Japanese Sumo wrestlers had a more than 1 400 year of experience in weight gain, eating 3.3 kg rice, 50 fat and 250 g meat per day. NNR's have more than 35 years of experience of the effect of an extreme high carbohydrate diet. The number of persons having diabetes is at least 397 548 persons on 2012-12-31 That is an increase since 2006-12-31 of 79 649 cases. It's an increase of 13 275 cases per year. The obesity rate increase is similar. Just look at pony jumping shows. In 1990ies the girls were slender all of them. Nowadays, 25 % of those pony jumping show girls are, despite much harder physical training, overweight to obese. So the NNRs have the perfect method, well proven during millennia, how to increase obesity and diabetes frequencies. Recommend extreme amounts of carbohydrates. The conclusion is obvious. The NNRs (a.k.a. pig fodder) generate obesity and disease including diabetes. The modern pig farmer knows how to get a slender pig for Christmas. The pig has to eat protein and fat. Then the farmer adds enough carbohydrates for the pig to convert excess of carbohydrates to stored fat. This is since millennia a wellknown technology and physiology.</p> <p>Lines 57-162 should be replaced with the following text. for which human requirements have been less well defined. The description of major food groups and their nutrient contributions given below is largely based on information provided by the old fashioned physiology from previous centuries. First of all we have to identify the essential components of the food for humans. AcCoA is the central molecule in our metabolism. We can make AcCoA from proteins via amino acid deamination. We can make AcCoA from fats We can make AcCoA from carbohydrates We can use AcCoA as an energy source We can use AcCoA to make amino acids We can use AcCoA to make fats We can use AcCoA to make some carbohydrates. We can use AcCoA to make a lot of essential molecules like cholesterol So AcCoA is THE kingpin molecule in our metabolism. Among essential components of our food there are: 1. protein or rather the amino acids, the building stones of proteins, are partly essential as humans can produce about ten of the 20 amino acids. This means that we have to eat the remaining amino acids to survive. 2. Fats are essential as an energy source and as building material to convert different fatty acids to other fatty acids. Also, we have a very limited processing facility for the long chain polyunsaturated animal fats so they are regarded as essential. Most of the other fats we can synthesize from smaller fatty acids, mainly from AcCoA. But we have to have fat to be able to rebuild fat and for energy production. 3. Minerals are essential as we</p>	

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	<p>can not produce elementary atoms. 4. Vitamins are mostly essential. We are unable to produce most of them. Carbohydrates are nonessential as humans can produce all necessary sugars in the liver in enough amounts. The brain does have mitochondria in all cells and is thus using only AcCoA as fuel. But red blood cells (RBC) need 3-6 mmol/L glucose in the blood as they lack mitochondria. Then glucose is metabolized along a RBC specific pathway to degrade glucose via the Rapoport-Luebring pathway producing 2,3-diphosphoglycerate (2,3-DPG) and then to lactic acid and energy. The 2,3-DPG is required to release the oxygen from the haemoglobin molecule. So with a too low blood concentration of glucose the brain can not get enough oxygen to oxidize citric acid in the Krebs cycle. And there is a too low concentration of glucose for the brain cells to anaerobically produce energy from glucose. So the brain does die within minutes when the glucose level in the blood goes too low. Protein Our protein sources are from animal or vegetable origin. We need at least 0,5 g animal protein per kg per day. Our best sources of animal protein is from herbivores like cattle, elk, horse and other herbivores. Also, pig, fowl, fish, egg and game are animal sources of protein as well as fat. Vegetable proteins are always deficient in one or more amino acids so we have to eat several times more vegetable protein to satisfy the needs of all amino acids. Free ranging herbivores are excellent carbohydrate-to-fat-and-protein converters. They eat enough minerals and vitamins for their own needs. The only thing we have to do is to eat the meat and inner organs to satisfy all our needs regarding protein, fat, minerals and vitamins in perfect amounts. Fat Free ranging herbivores are excellent carbohydrate-to-fat-and-protein converters. The fat in most animal fat have the same composition, shy of 50 % saturated fat (SFA), shy of 50 % monounsaturated fat (MUFA) and about 5 % polyunsaturated fat (PUFA). So the best fat we can eat is animal fat as it is about the same composition as our own fat stores. They all have the same melting temperature and thus we have to process the fats minimally to save energy. The only fats that have been proven harmful are trans fats and PUFA of omega-6-type . SAT fats and MUFA fats are harmless and essential. Sources of food The best sources of food are of animal origin like egg, meat, fish and milk. Then we get all we need of protein, fat, minerals and vitamins. And our liver can make all necessary carbohydrates. Milk from ruminants is both a food in itself and raw material for different dairy products (like cheese, butter, fermented milk, yoghurt or cream). Milk and milk products are important sources of animal fat, protein, vitamin A, riboflavin, vitamin B12, calcium, and iodine. Fat soluble vitamins are often added to skim and low fat milk as the cows are not receiving enough sunshine to produce enough vitamin D3. Two thirds of the fat in whole milk is SFA but there are shorter SFAs to keep the melting temperature constant. The major unsaturated fatty acid is oleic acid (C18:1). Milk also contains short chain fatty acids and the odd-chain fatty acids C 15:0 and C17:0.</p>	

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	<p>Fat content varies from 0.1 to around 4 g/100g, protein about 3.0-3.5 gram per 100 gram; and an animal carbohydrate (lactose) about 4-5 gram per 100 gram. Whole milk and low fat milk contain about the same amounts of calcium. Cheese has a high calcium content. It is shown several times that a high intake of dairy fat (butter as spread and high fat milk and whipping cream) was associated with a lower risk of central obesity (OR 0.52, 95% CI 0.33–0.83) and those children having full fat milk are not as obese as the children having low fat milk , . So the drinkers and eaters of dairy products should have high fat milk and full fat butter to stay slim. Egg is relative to its energy content high in protein, riboflavin, vitamin A and D. Egg is low in fat, and the egg yolk contributes together with dairy products, meat and fish to the dietary intake of cholesterol. One normal egg contains about 0,2 g cholesterol. An adult of 70 kg has about 200-400 g of cholesterol in the body. So the cholesterol amount of an egg may increase the total cholesterol by less than one tenth of a percent. The cholesterol in food is totally negligible, cholesterol in food can never alter the total amount of cholesterol, the amount is too tiny. Meat from beef, pork, mutton and game (e.g. reindeer and moose) is generally defined as “red” meat, while meat from chicken and turkey are defined as “white”. Processed meat has undergone some preservation process like smoking; salting or use of other chemicals for preservation (likes nitrites). Examples of such processed meats are ham, bacon, salami, different kinds of sausages, and smoked meat. Meat that is boiled, fried, dried, fermented or frozen is usually not categorized as processed. Meat and meat products contain 20-35 % protein, and are usually good sources of vitamin B6, vitamin B12, iron, zinc and selenium. The content of energy, fat, fatty acids and salt may vary considerably between types. Fat content may vary from less than one per cent to more than 40%. Also, types of fatty acids vary between different animals; typical proportion of SFA is 30% in chicken, 35-40% in pork, and 40-55% in beef and mutton. The level of conjugated linoleic acid (CLA) is less than 1 % in chicken and pork, but 3-5% in ruminants (like beef and mutton). The salt content is low in raw, unprocessed meat but much higher in processed meat. Game meat usually has lower fat content. Pure meat, fresh or preserved the way our ancestors did, has never been shown to harm. On the contrary, pure carnivorous people like Sami, Eskimoos, American Indians like the Pima Indians were totally healthy as long as they stuck to their traditional diet. Fish and seafood contain 20-35 % protein. Lean fish like cod and plaice contain less than 2 grams of fat per 100 gram, medium fat fish like winter-mackerel and tuna contain 2-8 grams of fat per 100 gram, and fatty fish like herring, summer-mackerel, trout, salmon and eel contain more than 8 grams of fat per 100 gram. Medium fat and fatty fish are the major dietary sources of marine omega-3-fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Fatty fish is also a major source of dietary vitamin D, but</p>	

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	<p>some lean fresh-water fish (e.g. pike-perch) also contain high amounts. Fatty fish and especially cod liver contain high amounts of vitamin A (retinol). Fish and seafood are also good sources of vitamin B12, iodine and selenium. However, the nutrient content may vary between wild fish and farmed fish depending of the feed. Fish and seafood may contain different environmental toxins. In general fish captured in the open sea has lower concentrations of pollutants than fish from the Baltic Sea or Norwegian fjords. Some marine fish, e.g. large tuna and halibut, and freshwater fish from certain areas may contain elevated levels of methyl mercury. Lean fish generally contain low levels of persistent organic pollutants (POPs). Finnish studies indicate that fish consumption is related to higher blood concentration of dioxins, polychlorinated biphenyls and methyl mercury, as well as of omega-3-fatty acids (Turunen AW, Männistö S et al. 2010). However, the lower mortality of cardiovascular diseases and type 2 diabetes among fishermen indicates that the benefits of fish consumption seem to overcome the potential hazards (Turunen, Verkasalo et al. 2008). Pork fat in bacon has about the same amount of animal omega-3 fatty acids as fat fish like salmon when you compare the absolute amount, not percent. So we don't have to empty the seas from fish to get enough animal omega-3 fats. There is another way to normalize the ratio of omega-6 PUFA to omega-3 PUFA. Decrease the intake of omega-6 to below cancer causing levels and intakes. Vegetables, fruits and berries usually contain low amounts of food energy, plenty of water and dietary fibre, small amounts of vitamins like ascorbic acid (vitamin C), carotenoids (pre vitamin A), folate, vitamin E (tocopherol) and vitamin K, and minerals like potassium and magnesium. But fibres are irritating the intestines and are cancer causing . Fibres contain phytates that minimise mineral uptake . Carotenoids are not degradable by humans to vitamin A. Beans and peas are not very good sources of protein, minerals (iron, zinc, magnesium and potassium), B-vitamins (except B12). But beans and peas contain large amounts of fibre and starch, a.k.a. sugar, carbohydrates. So they may be eaten in small amounts. Nuts and seeds contain plenty of monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) (as well as some vegetable protein, magnesium, zinc, copper, potassium, vitamin E, B6, niacin and several antioxidants). Nuts and seeds also contain large amounts of carbohydrates and intake should be minimized Potatoes are comparatively rich in carbohydrates (starch), but is lower than meat in several minerals (like potassium and magnesium) and vitamins. Traditionally, potatoes were sources of vitamin C and protein among high consumers. We need at least 0,5 g animal protein per kg per day. Vegetable proteins are always deficient in one or more amino acids so we have to eat several times more vegetable protein to satisfy the needs of all amino acids. Carbohydrate intake in physiological amounts (less than 1 hg) are very low in vitamin C. Wholegrain is defined as intact and</p>	

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	<p>processed grain or cereal where the fractions endosperm, bran and germ are present in the same proportion as in the intact grain. Cereals are to be eaten in low amounts as they contain very large amounts of carbohydrates in the form of polymerized glucose easily digested to glucose and thus increases the glucose level very fast in the blood. Grain, a.k.a. cereals, flour, especially wheat, contain large amounts of gluten. The large increase of grain consumption during the last three decades may be the cause of IBS, gluten entropathy, morbus crohn, cholitis ulcerosa and “wheat belly”. All these diseases improve on a grain free diet and may even be states free from symptoms as long as the patient totally refrain from eating grain. Whole grain products do provide fibers and contain phytates that minimise mineral uptake . Resistant starch is a minor part of (less than 10 % of total starch) in cooked and hydrogenated starches and the amount is usually too low to make any difference at the blood glucose level. Cereals, especially those that are processed into a variety of products, should be avoided as much as possible. Because micro-nutrients and other bioactive compounds mostly are very scarcely found only in the germ and bran fractions, refined cereal products generally have very low nutrient content, and often high amounts of added sugar (see below). They are often correctly called empty calories and should be avoided. All plant foods (like vegetables, root vegetables, fruits, berries and nut and seeds, and whole-grains) naturally contain a wide variety of phyto-chemicals like polyphenols, salicylates, phytosterols, saponines, glucosinolates, monoterpenes, phytoestrogens, sulphides, terpenes and lectins. Most of these have important functions in the plant cells and may also negatively influence biological functions in the human body via a large number of defence mechanisms. Many are antioxidants with the potential to reduce the production of our own more efficient antioxidants, others may influence our signal systems, cell cycles, reparation systems and inflammation reactions. The number of bioactive phyto-chemicals has been estimated to around 100,000. A single plant based meal may provide around 25,000 different phyto-chemicals, but luckily comparatively small amounts of each. The observed ill health effects associated with vegetable, fruit, berry and whole grain consumption may likely be explained by the combined action of many phyto-chemicals and other nutrients. All plants try to defend themselves from being eaten as they can not run away from a plant eater. That is why plants always have toxins. So there are more than 100 000 reasons to minimize the food from the plant kingdom. Butter and some vegetable oils are used in cooking and with small amounts of bread. Margarine, spreads and vegetable oils are used by the food industry to produce foods like mayonnaise, dressings, baked goods and soups. Vegetable oils are manufactured by pressing oil from seeds or plants like rapeseeds, olives, palm fruit and coconuts. But there is also oil manufactured by omega-6 rich soya beans, maize kernels, sunflower and</p>	

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	<p>safflower seeds. Butter is made from the fat of cow's milk. Margarine and spreads are mixtures of different fats. Milk fats and vegetable oils, margarine and spreads contribute dietary energy, fat (essential and non-essential fatty acids) and fat soluble vitamins (i.e., A, D, E and K). Vitamins A and D are usually added to margarines and spreads. Vegetable oils contain fat (100%), while margarines and spreads contain varying amounts of fat due to dilution of fat with water and chemicals. The fatty acids composition may vary considerably depending on the fat source used in manufacturing. Soy bean, maize and sunflower seed oil are rich in omega-6 PUFA, while rapeseed oil and especially olive oil is rich in MUFA. Rapeseed and soybean oils have comparatively high content of omega-3 fatty acids but soybean oil has too much omega-6 to be classified as human food. Vegetable oils and fat from marine sources, e.g. fish oils, contain a lot more unsaturated fatty acids than fat from land living animals, e.g. lard and tallow. However, palm and coconut oils have high content of SFA. Fish oils as well as lard are generally rich in very long animal omega-3 PUFA. Fat from ruminants, e.g. tallow, have the same content of SFA, and animal fats contain tiny amounts of cholesterol. Butter and ruminant fat contain 3-5% CLA. Processed fats like margarines and spreads were previously sources of TFA, depending on the raw material used in manufacturing. However, the TFA content has during the last decade decreased considerably in the Nordic countries due to changes in raw material and processing. But still most of the vegetable oils are cheap but unhealthy. Then choose coconut, canola (rapeseed) or olive oils when you choose an oil. All other oils contain much too high amounts of inflammatory and cancer causing omega-6. Lines 170-260 should be replaced with the following text. Energy dense and ultra-processed food products Many food products in Westernized societies are manufactured from refined cereals/white bread and with additional sugar. Biomarker studies have demonstrated that Westernized dietary patterns are associated with extremely high carbohydrate concentration and low concentrations of micro-nutrients (Kant 2010) (Román-Viñas, Ribas Barba et al. 2009), and will likely provide less of other health enhancing substances found in plant-foods. The energy content is increased mainly as carbohydrates and disproportionate to the content of natural and essential animal protein, animal fat vitamins and minerals and to other bio-active substances of health importance in ultra-processed food products (e.g. soft drinks, confectionary, candy bars, desserts, bakery goods, sugared cereals, sugared milk-products, powdered sauces and soups, snacks, deep-fried potatoes et cetera). Many refined and processed foods may also contribute components with potential adverse health effects, added during the manufacturing process (e.g. carbohydrates), or formed during prolonged heat treatment (like heterocyclic amines or so called advanced glycation/lipidoxidation end-products, AGE/ALE). Studies within EPIC (European Prospective Investigation into Cancer</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>and Nutrition) indicate that the use of manufactured food products currently (i.e., data collected in the 1990s) is higher in northern and central European countries than in Mediterranean countries (Slimani, Deharveng et al. 2009) (Chajès, Biessy et al. 2011). The health impact of specific foods Observed associations between single nutrients and health outcomes may be difficult to interpret, because of the complexity of diet. Also, diet-related chronic diseases are caused by carbohydrates, especially fructose , and also excess of omega-6 rich oils . Therefore a search for the health effect of single nutrients may be misleading (Slattery 2010) (Jacobs Jr and Tapsell 2007) (Appel 2008). In recognition of these complexities we have to revert to old fashioned physiology, biochemistry and endocrinology instead of reading biased research from the last decades. As stated above, animal protein, animal and a few vegetable fats are essential. Carbohydrates, especially glucose, very quickly increases the blood glucose level which triggers a fast insulin response for the person to survive the massive glucose challenge. The old fashioned physiology tells us that a 70 kg person has 3-6 mmol/L or 1,5 to 3,0 grams of glucose in the whole blood volume. Having just 5 g glucose will rise the blood glucose level from 5 mmol/L to 15 mmol/L. If that person has more than 30 mmol/L - 50 mmol/L or 15-25 g glucose in the blood, that amount of glucose is lethal. Then NNR4 recommends that male person to eat 480 g of carbohydrates per day, a more than 20-fold lethal dose of carbohydrates per day. Fructose is 5-10 times more efficient to nonenzymatically glycate proteins than glucose. The most well known Advanced Glycated Endproduct (AGE) is HbA1c where glucose or fructose attaches to the lysine residue of haemoglobin. This sugar molecule sterically blocks the oxygen to attach to the haemoglobin molecule. Thus a high blood glucose level increases the HbA1c. Fructose is much more efficient in blocking oxygen transport. But we never measure the fructose level in blood so usually we have not a clue how much fructose there is in the blood. Another problem is that fructose normally is metabolized in the liver to saturated fatty acids and increases the rate of non alcohol fatty liver disease (NAFLD) and even non alcohol fatty liver cirrhoses (NAFLC). Excess carbohydrates are converted to SFA and stored in the fat depots and locked up by the high insulin levels. Several studies have shown a long time weight reduction by limiting only the carbohydrates , . There is no convincing evidence that consumption of milk or dairy products is related to increased risk of cardiovascular disease (Beck, Hoppe et al. 2010; Nasjonalt råd for ernæring 2011). However, studies have shown that high animal fat intake decreases the obesity rate of children , . Some reports indicate that milk consumption is related to a reduced risk of metabolic syndrome, type-2 diabetes, hypertension and stroke (Beck, Hoppe et al. 2010; Nasjonalt råd for ernæring 2011). The WCRF/AICR (2007) concludes that milk consumption probably reduce the risk of colorectal cancer. No conclusion can be made</p>	

Organisation Company	General and detailed comments	WG response & actions
	<p>on the link between milk and breast cancer. Lines 263-554 should be replaced with the following text. Macronutrients, foods and weight maintenance The old fashioned physiology, biochemistry and endocrinology implies that carbohydrates are toxic to us in larger amounts than 100 g per day in metabolically healthy individuals and may reduce the symptoms in metabolically deranged persons like metabolic syndrome, diabetes and overweight/obesity. Still, carbohydrates increase the blood glucose level that increases the insulin response that increases the fat deposition in fat tissue. Increased level of insulin also stops fat metabolism. Animal protein is not giving a weight gain according to all old studies from previous centuries. Animal fat and selected vegetable fats do not increase the blood glucose level and insulin which means fat are not making us fat, fat is making us satisfied for long. Just reread the old books in physiology, biochemistry and endocrinology. Those old books reveal the truth about metabolism. Lines 556-564 should be replaced with the following text.</p> <p>Conclusion The overall scientific evidence show that the old fashioned physiology, biochemistry and endocrinology still rules. As long as humans eat their species specific food as carnivores with normal amounts of animal protein, an enough amount of animal fat and a very limited ability to eat carbohydrates, the individuals as well as the populations will keep healthy. Just look at a newborn human baby. Human breast milk has evolved for millennia to be the ultimate food for babies. The milk is diluted to ensure that all children, regardless living in arctic region or tropical region, will have enough water. The amount of animal protein, 8 percent of total energy (E%), is enough for a newborn child to increase 50 % in length during the first year. The amount of carbohydrates, 36 E% of animal carbohydrates a.k.a. lactose, is enough for a newborn child to increase 300 % in weight during the first year. The amount of animal fat, 56 E% of animal fats, is enough for a newborn child's brain to develop normally during the first year. The same conditions apply for adults. So just decrease the amount of carbohydrates to a level that does give the endocrinological condition of normal low insulin response to stay healthy and keep the normal body weight.</p>	

Organisation Company	General and detailed comments	WG response & actions
The Norwegian Dairy Council	<p>General comments</p> <p>First of all, we would like to comment that it is positive that the 5th edition of the Nordic Nutrition Recommendations (NNR) set the whole diet in focus.</p> <p>General comment</p> <p>Regarding research on Mediterranean-style eating patterns, it is natural to describe the eating pattern in general terms. However, it should be emphasized in the text that a large number of cultures and agricultural patterns exist in countries that border the Mediterranean Sea, so the “Mediterranean diet” is not one eating pattern. No single set of criteria exists for what constitutes a traditional Mediterranean eating pattern. Even in studies, different criteria for this diet, is used. Own section about “Nutrient-dense food”</p> <p>This chapter has a section describing “Energy dense and ultra-processes food products”. When talking about a healthy eating pattern in terms of the Nordic diet, it should be stated that this is simply focusing on nutrient-dense foods. A healthy eating pattern focuses on nutrient-dense foods such as vegetables, fruits and berries, nuts and seeds, whole grains, fish and seafood, vegetables oils and low-fat dairy products prepared without added solid fats, sugars, starches, and sodium. These food groups combined can provide the full range of essential nutrients and fiber, without excessive calories. Hence, the nutrient-dense food should be pointed out in an own section.</p> <p>Dietary patterns in the different countries</p> <p>The NNR is referring to all of the Nordic countries. However, there are some differences regarding the intake of some groups of food. The recommendations should have a table showing the intake of the different food groups in the Nordic diet from each country. As a result it would be easier to interpret the major challenges in different areas of the diet from each country.</p> <p>Detailed comments</p> <p>Page 4 Line 141: When referring to the content of protein in milk, it should be stated that protein content is about 3.0 – 5.0 gram per 100 gram. The new milk “Styrk” from Tine has a higher amount of protein than the traditional milk on the market.</p> <p>Line 142: “Whole milk and low fat milk contain about the same amount of calcium. Cheese has ha high content of calcium”. In this section the amount of calcium per 100 gram should be referred to. Otherwise it is difficult to interpret what high content of calcium for cheese means.</p>	<p>Thank you for your comments and suggestions, which have been incorporated into the edited text.</p> <p>We agree with you the there are many varieties of the both the Mediterranean and Nordic diets. Although common traits, every region has their variety. We also agree that the common trait of the traditional Mediterranean-like-diet, healthy Nordic diets and recommended diets is the density of essential micro-nutrients.</p>

Vegetarian diets

Organisation/ Company	Comments	WG responses and actions
University of Bergen, Norway	<p>General comments: In general the NNR 2012 on vegetarian diets is generally well written. There are some few sections and sentences which could benefit from minor changes. Here are some comments and suggestions (see detailed comments)</p> <p>Detailed comments: In the introduction, it is written “A strict vegan diet consists only of plant foods, and avoids all food products of animal origin. Other diets may exclude meat and meat products, but include fish, dairy and eggs.” Even though it is partly clarified in a later table, a diet including fish is according to most dictionaries not vegetarian (but pescetarian). (http://www.merriam-webster.com/dictionary/vegetarian, http://oxforddictionaries.com/definition/english/vegetarian?q=vegetarian and http://dictionary.reference.com/browse/vegetarian?s=t) Thus, the word “fish” in line 13 would better be deleted. In the list of plant foods in line 17 and 18, it could be worthy to add “mushrooms” as some mushrooms including champignon are frequently eaten in Nordic countries also among vegetarians. In line 21, it could be nice to include a reference on bioactive substances. I suggest the following reference: Bohn SK, Myhrstad MC, Thoresen M, Holden M, Karlsen A, Tunheim SH, Erlund I, Svendsen M, Seljeflot I, Moskaug JO et al: Blood cell gene expression associated with cellular stress defense is modulated by antioxidant-rich food in a randomised controlled clinical trial of male smokers. BMC Medicine 2010, 8. The sentence in line 25-28 focus on potential problems with vegetarian diets: “However, there is a concern that if certain food groups are excluded from the diet, or when foods are not selected with care, dietary imbalances may occur that could result in health problems. This short review is based on a report of the Swedish National Food Agency (Johansson 2011) and some additional references.” With several studies and reviews suggesting substantial benefits of vegetarian diets (Tantamango-Bartley et al., Appleby et al., Barnard et al., Craig 2009, Key, Sabata and Wien, and Thorogood et al., to mention a few), it could be justified to re-phrase the sentence to e.g.: “As also the case for other diets, some knowledge on how to balance a diet well is beneficial to could optimise a vegetarian diet, such as ensuring sufficient intake of some micronutrients.” This sentence would in my opinion also cover the intention of the sentences in line 47-51, which could then be deleted. In line 38 it is written that “Since individuals following vegetarian diets tend to be more health conscious, more often non-smokers, and more physically active than non-vegetarians (Craig WJ & Mangels AR 2009), not only diet but also the overall lifestyle likely contributes to a better health status.” This is true; however, most studies investigating health effects from vegetarian diets have adjusted for likely confounders such as smoking and exercise. Thus, it could be justified to delete the sentence in line 38-41. Table 1 includes both vegetarian and non-vegetarian diets. This is fine if the title is changed to “e.g. Content of various diets”. Then a row for “omnivorous” diets could also be included and a column also for “meat”. It could also be worthy to add a row for “vegan” and a column for “plant based products” (the latter could be ticked-off for most diets). In line 57, it is written “but the proportions are not as optimal as in animal foods” referring to amino acids in plants. The protein digestibility-corrected amino acid scores are very similar for many animal food sources and plant sources. In fact, many soy products have a similar and in many cases higher scores than several animal food sources. A preferable distribution of amino acids can also be achieved through an intake of e.g. both grains and legumes combined. To avoid confusion, it could be justified to delete the part of the sentence mentioned above. Similarly, line 57 to 61</p>	<p>Thank you for your comments. The text has been edited taking into account your suggestions, and those made from several other professionals in the field.</p> <p>The definition of vegetarian diets has been clarified in the text as well as in the tables.</p> <p>It is well known that behaviours tend to cluster. Epidemiological studies therefore adjust in analysis for potential confounders like smoking or physical activity. Because of residual confounding, however, it may be very difficult to remove all influences of one behaviour when examining another in an epidemiological study.</p>

Organisation/ Company	Comments	WG responses and actions
	<p>could also be deleted. Several new staples (from a Nordic perspective) such as quinoa and amaranth are frequently eaten by vegetarians, contain several of the essential amino acids and are good sources of protein. These could also be mentioned, e.g. after line 66. In line 73, it is written that “Vegetarian diets excluding fish may provide limited amounts of long-chain omega-3 fatty acids.” This could be true, but there are also other sources of long-chain omega-3 fatty acids such as algae oils (Lenihan-Geels G, Bishop KS, Ferguson LR: Alternative sources of omega-3 fats: can we find a sustainable substitute for fish? <i>Nutrients</i> 2013, 5(4):1301-1315). Thus, the sentence could be re-phrased “Long-chain omega-3 fatty acids such as DHA and EPA are mainly found in fish and algae oils. Vegetarians excluding fish may benefit from including algae oils as an alternative supplement to fish oil. “ In line 90, it could be worthy to add the sentence “Similarly, plant milk (e.g. soy milk, oat milk and rice milk) is often enriched with vitamin D which could be an important source of vitamin D in vegans.” (see the content list of e.g. Alpro as an example http://www.alpro.com/uk/soya-plain). Similarly, after line 102, it could be reasonable to add that “Several plant milks are also enriched with riboflavin which could contribute to avoid deficiencies.” Similarly, as plant milks usually contains similar content of calcium as cow’s milk, the sentence in line 105, could be modified to e.g. “Milk and milk products are major sources of calcium, both those of made from cow’s milk and enriched plant milk alternatives.” In line 120, “lentils, peas, chick peas and mushrooms” could be added to the list of major sources of selenium (e.g. lentils often have several times the content of selenium compared to similar amounts of cows’s milk, http://www.matvaretabellen.no/linser-roede-hermetiske-06.131 and http://www.matvaretabellen.no/lettmelk-uspesifisert-01.229). Are there any studies suggesting that vegetarians in Nordic countries tend to be deficient in selenium? If so, references on this would be appropriate. In line 139-140, it could be worthy to reference the review by Messina & Mangels on vegan diets in children. Based on this review, growth among children on a vegan diet is not significantly in vegan children from the growth reference. In the review by Van Winckel et al. referenced, it is written that “Descriptive cohort studies have demonstrated an identical growth and weight evolution in lacto(-ovo)-vegetarian children and adolescents compared to their omnivorous peers, whereas vegan children tend to be leaner and smaller [7, 12, 21, 30, 32, 40, 41, 43, 49].” The growth comparison in vegan children is presented as a tendency and going into the background material, the references 7 and 49 refer to effects of macrobiotic diets (which are non-vegetarian and not representative of vegan diets). The references 12, 21, 30, 40 and 41 refer to effects of a lacto-ovo-vegetarian diet. We are then left with two studies on vegan diets and growth, reference 32 by Connell et al. and reference 43 by Sanders. Connell et al. writes the following “In this study, the growth of a group of children raised with a relatively strict form of vegetarian diet (vegan) was similar to that of the reference population”. The graphs show median height-for-age (HAZ) for girls and boys around the median in the reference population (50 percentile) with a healthy distribution and few if any outliers. Median weight-for-height (WHZ) is slightly below the median compared to the growth reference population (around the 30 percentile, which could be argued to be beneficial). The study included 288 vegan infants/children. Reference 43, a study by Sanders, is based on 37 vegan children, which had a height development quite close to the growth reference population, with no significance testing of differences. Thus, I suggest a minor change in the following sentence “the results indicating somewhat slower growth among some of the children” to rather read “with no clear differences found in growth of the children except a lower risk of</p>	

Organisation/ Company	Comments	WG responses and actions
	<p>childhood obesity (Sabate and Wien M 2010).” Regarding table 2: For protein: it is written “Careful combination of foods”. This might be interpreted to be more challenging than it actually is, see comment on proteins above. Thus, I suggest rather writing, “Generally adequate”. For “Essential fatty acids”, I suggest to rather write “Algae oil supplement is beneficial” as conversion from ALA to DHA is limited (Lenihan-Geels et al.) Regarding table 3: Macrobiotic diets are often non-vegetarian (including e.g. fish or fowl [Van Winckel et al.]). Fruitarians are few, and as mentioned in the table, raw food can be vegetarian or non-vegetarian. Thus, I suggest removing table 3 as it does not seem to be of much relevance to the chapter on vegetarian diets. References and sources: Bohn SK, Myhrstad MC, Thoresen M, Holden M, Karlsen A, Tunheim SH, Erlund I, Svendsen M, Seljeflot I, Moskaug JO et al: Blood cell gene expression associated with cellular stress defense is modulated by antioxidant-rich food in a randomised controlled clinical trial of male smokers. BMC Medicine 2010, 8. Tantamango-Bartley Y, Jaceldo-Siegl K, Fan J, Fraser G: Vegetarian diets and the incidence of cancer in a low-risk population. Cancer Epidemiol Biomarkers Prev 2013, 22(2):286-294. Appleby PN, Davey GK, Key TJ: Hypertension and blood pressure among meat eaters, fish eaters, vegetarians and vegans in EPIC-Oxford. Public Health Nutr 2002, 5(5):645-654. Craig WJ: Health effects of vegan diets. Am J Clin Nutr 2009, 89(5):1627S-1633S. Barnard ND, Cohen J, Jenkins DJ, Turner-McGrievy G, Gloede L, Green A, Ferdowsian H: A low-fat vegan diet and a conventional diabetes diet in the treatment of type 2 diabetes: a randomized, controlled, 74-wk clinical trial. Am J Clin Nutr 2009, 89(5):1588S-1596S. Key TJ, Fraser GE, Thorogood M, Appleby PN, Beral V, Reeves G, Burr ML, Chang-Claude J, Frentzel-Beyme R, Kuzma JW et al: Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. Am J Clin Nutr 1999, 70(3 Suppl):516S-524S. Craig WJ: Nutrition concerns and health effects of vegetarian diets. Nutr Clin Pract 2010, 25(6):613-620. Thorogood M, Mann J, Appleby P, McPherson K: Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. BMJ 1994, 308(6945):1667-1670. Craig WJ, Mangels AR: Position of the American Dietetic Association: vegetarian diets. J Am Diet Assoc 2009, 109(7):1266-1282. Messina V, Mangels AR: Considerations in planning vegan diets: children. J Am Diet Assoc 2001, 101(6):661-669. O'Connell JM, Dibley MJ, Sierra J, Wallace B, Marks JS, Yip R: Growth of vegetarian children: The Farm Study. Pediatrics 1989, 84(3):475-481. http://www.ncbi.nlm.nih.gov/pubmed/2771551 Van Winckel M, Vande Velde S, De Bruyne R, Van Biervliet S: Clinical practice: vegetarian infant and child nutrition. Eur J Pediatr 2011, 170(12):1489-1494. Content of soy milk, Alpro used as an example: http://www.alpro.com/uk/soya-plain Vegetarian in Webster's dictionary: http://www.merriam-webster.com/dictionary/vegetarian Oxford dictionary: http://oxforddictionaries.com/definition/english/vegetarian?q=vegetarian Cambridge dictionary: http://dictionary.cambridge.org/dictionary/british/vegetarian_1?q=vegetarian Vegetarian in dictionary.com: http://dictionary.reference.com/browse/vegetarian?s=t Sabate J, Wien M: Vegetarian diets and childhood obesity prevention. Am J Clin Nutr 2010, 91(5):1525S-1529S.</p>	

Organisation/ Company	Comments	WG responses and actions
Oslo University Hospital, Norway	<p>General comments: We appreciate this opportunity to comment on the Section on Vegetarian Diets in the Nordic Nutrition Recommendation (NNR) 2012. Our main comment is about the minor information on how to plan well-balanced and nutritionally adequate Vegetarian diets. In accordance with the Position of the American Dietetic Association (1), it is stated in the NNR 2012 (line 22-23) that a well balanced lacto-ovo-vegetarian or lacto-vegetarian diet is considered to provide sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, and children. However, after 10 lines about the health effects of the vegetarian diets approximately 100 lines of nutritional considerations follows. To enhance quality of the NNR 2013 on vegetarian diets, we suggest that a vegetarian food guide with recommended servings of the different food groups (2) based on the Nordic eating pattern is implemented in the NNR. Sincerely, Serena Tonstad and Mette Svendsen</p> <p>References 1. Position of the American Dietetic Association and Dietitians of Canada: vegetarian diets. J Am Diet Assoc. 2009;109:1266-1282. 2. Messina V, Melina V, Mangels AR. A new food guide for North American vegetarians. J Am Diet Assoc. 2003;103:748-765.</p>	<p>Thank you for your comments, which have been taken into account when editing the text.</p> <p>The formulation of food guides recommending serving sizes etc. is a management issue, which should be handled by the national food agencies in each Nordic country. This is not the task of the NNR 2012.</p>
Norwegian Directorate of Health	<p>Detailed comments: 106-7. "Since oxalic acid (OA) may inhibit calcium absorption, green leaf varieties with low OA content should be selected." This may be a good recommendation, but this information may be a bit difficult for the reader to relate to. If possible give examples of vegetables to be selected or avoided, and state what you mean with low and/or high levels of OA. References: you may add this recent review: Vegetarian diets, low-meat diets and health: a review. McEvoy CT, Temple N, Woodside JV. Public Health Nutr. 2012 Dec;15(12):2287-94.</p>	<p>Thank you for your comment. The text has been edited to clarify the issue.</p>
Unilever	<p>General comments: Helping consumers to make better choices and improving their health and nutrition requires consumer friendly, clear and evidence based information. We believe this is essential if consumers are to make informed healthier choices. We also believe that to make a lasting impact on health we need to encourage our consumers to make small changes to their everyday habits. To make the guidelines even clearer and easy-to-understand we would suggest some further clarifications</p> <p>Detailed comments</p> <p>Rows 75-76 • We suggest adding other sources of ALA, such as vegetable oil based fat spreads and soft and liquid margarines based on these oils. (...camelina oil have a have high content of ALA, as well as vegetable oil based fat spreads and soft and liquid margarines based on these oils.) Rows 77-79 • We suggest adding other sources of LA, such as to specify sunflower seed oil, as well as adding vegetable oil based fat spreads and soft and liquid margarines based on sunflower seed oil. The oils mentioned in the draft document, such as flax seed oil, soybean oil, hempseed oil, as well as walnuts are not used as such in very high amounts in the Nordic countries; hence they do not make a big impact on intake. In contrast, spreads and margarines with both ALA and LA are consumed in higher quantities on a more regular basis. Therefore, we believe it is important to add the information as suggested above.</p>	<p>Thank you for your comments. The text has been edited taking your suggestions into account as well as those of other professionals in the field.</p>

Organisation/ Company	Comments	WG responses and actions
Vegaaniliitto ry / Vegan Society of Finland	<p>Detailed comments: Line 21: AMENDMENT: “A well balanced vegan, lacto-ovo-vegetarian or lacto-vegetarian diet is considered to...” Lines 25-28. AMENDMENT: “However, there is a concern that if certain food groups like fruits and vegetables, are excluded from the diet, or when foods are not selected with care, dietary ...” RATIONALE: In line 25 the 'certain food groups' is too vague and allows for possible misinterpretations. It should be clarified which food groups are indicated. Leaving animal products like meat and dairy product groups is not likely to result in health problems in a well planned vegetarian or vegan diet, whereas leaving food groups like fruits or vegetables is much more likely to increase the risk of dietary problems. Lines 47-54: TO BE OMITTED. RATIONALE: Although the nutritional content of vegetarian and conventional, mixed diets are different, a well balanced vegan, lacto-ovo-vegetarian or lacto-vegetarian diet is considered to provide sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, as well as children. Line 54: AMENDMENT “All essential amino acids are found in plant foods. The protein intake is sufficient with a vegan diet, when energy needs are met and plenty of good sources of vegetable protein are included, like pulses, whole grain cereals, potatoes, nuts and seeds. Other plant foods also provide protein although in lower amounts.” RATIONALE: A well balanced vegan, lacto-ovo-vegetarian or lacto-vegetarian diet is considered to provide sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, as well as children. TO BE OMITTED (detail): “but the proportions are not as optimal as in animal foods.” RATIONALE: This fact is nutritionally unessential TO BE OMITTED (detail): “This should not be a problem in adults consuming varied vegetarian diets” RATIONALE: A well balanced vegan, lacto-ovo-vegetarian or lacto-vegetarian diet is considered to provide sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, as well as children. Line 59: TO BE OMITTED: “However, because the digestibility of vegetable protein is slightly lower compared to animal protein, subjects on vegan diets excluding all animal foods may require slightly higher protein intakes.” RATIONALE: Even if the digestibility of vegetable protein were slightly lower compared to animal protein, intake of protein in vegans is nutritionally sufficient. Therefore the general recommendations of protein intake are sufficient for vegans. Lines 63-66 TO BE OMITTED: RATIONALE: Pulses are only one of the major sources of vegetable protein. Instructions dealing with preparation of food items are not necessary in dietary recommendations. Lines 101-102 AMENDMENT: "If milk-products are excluded in the vegetarian diet, plenty of pulses, dark green leafy vegetables and wholegrain cereals should be used to ensure sufficient riboflavin intake. Some vegan products contain added vitamin B2." Lines 105-107 TO BE OMITTED: "Since oxalic acid (OA) may inhibit calcium absorption, green leaf varieties with low OA content should be selected." RATIONALE: It is more important to encourage consumption of a large variety of vegetables including dark green leafy vegetables than to restrict the use of some on the basis of OA. A good variety of dark green leafy vegetables should be recommended also to vegetarians. Line 131 TO BE OMITTED: "it cannot be considered a reliable source" TO BE REPLACED WITH: "the amount of iodine of the used algae should be known" Infants and children Lines 137-143: TO BE OMITTED TO BE REPLACED WITH: "Well-planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets are appropriate for all stages of the life cycle, including pregnancy and lactation. Appropriately planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets satisfy nutrient needs of infants, children, and adolescents and promote normal growth" SOURCE: ADA Vegetarian Diets</p>	<p>Thank you for your comments. The text has systematically been edited taking into account your suggestions and those made from several other professionals in the field.</p> <p>The definition of vegetarian diets has been clarified both in the text and in the tables.</p>

Organisation/ Company	Comments	WG responses and actions
LRF Mjölks/Dairy Sweden	<p>General comments: The following comments come from Dairy Sweden (LRF Mjölks) and are put together by Helena Lindmark Månsson and Annika Smedman. General comment: What is the position of the NNR on the use of supplements? Should a diet according to the NNR be sufficient without the use of supplements? Please include a discussion on this matter in the NNR publication.</p> <p>Detailed comments: The following comments come from Dairy Sweden (LRF Mjölks) and are put together by Helena Lindmark Månsson and Annika Smedman. Line 21: Please include definitions of the diets mentioned, and refer to Table 1. Page 3, sections “Vitamins” and “Minerals”: Please explain why only these nutrients chosen to be discussed in this section. Lines 95-102: Why is use of supplements mentioned and recommended for vitamin B12 but not for vitamin B6? Please motivate if this difference will remain. Line 105-107: Please include the bioavailability of calcium from different foods, e.g. milk, broccoli, spinach and almonds. Lines 112-113: “Milk products contain very little iron.” This sentence is odd in this context. Delete the sentence or include other foods with low iron content. For example the iron content in eggs, fish/fish dishes, snacks/nuts, fruits/berries and juices are of the same magnitude as in milk and milk products (ref: Livsmedelsdatabasen, Livsmedelsverket). Table 1: Informative table, but vegan diet is missing, please include it.</p>	<p>Thank you for your comments. The text has systematically been edited taking into account your suggestions and those made from several other professionals in the field.</p> <p>The definition of vegetarian diets has been clarified.</p> <p>A paragraph regarding vitamin B6 has been added.</p>
National Food Agency, Sweden	<p>Detailed comments: Vitamins (84-103) It would be valuable to mention in this section a reference to the chapter on B6 and bioavailability of the bound form of the vitamin in foods of non-animal origin. Vitamin B6 / Physiology and metabolism/ line 40-42 In most fruits, vegetables and grains, a part of the 40 pyridoxine occurs as a glucoside, which is considered to be less bioavailable than other non-glucoside forms (2, 4) This could be of importance for vegans, see reference Dietary intake of vitamin B6 and concentration of vitamin B6 in blood samples of German vegans. A Waldmann et al, Public Health Nutrition(2006) 9(6), 779-784 DOI:10.1079/PHN2005895 There are now methods for determination of free and bound vitamin B6 in vegetables, legumes and cereals, see reference Determination of total vitamin B6 in foods by isocratic HPLC: a comparison with microbiological analysis. M Kall Food Chemistry 82 (2003) 315-327 DOI 10:1016/S0308-8146(02)00568-X There are some data available, but not published in Sweden (Livsmedelssystemet) and I believe in Denmark. It would be valuable if these data were available, and more data determined, to enable evaluation on vegetarian diets and also make research on the matter possible.</p>	<p>Thank you for your comments. The text has systematically been edited taking into account your suggestions and those made from several other professionals in the field.</p> <p>A paragraph regarding vitamin B6 has been added.</p>

Organisation/ Company	Comments	WG responses and actions
National Food Agency, Sweden	<p>General comments: Traditionally vegetarians and vegans have had a tendency to be more health conscious than the general population. Nowadays it has, however, become more and more common to adopt vegetarian or vegan diets for ethical or environmental reasons. This means that individuals that choose these diets today may not be as health conscious as those who have been subjects for earlier studies. This might be good to mention in this chapter.</p> <p>Detailed comments: 20. Selenium is mentioned as an example of antioxidants that vegetarian diets in general provide enough of. As mentioned on line 126 exclusion of animal products from the diet may lead to a low selenium intake. It would therefore be better to erase selenium from the examples on line 20. 82. The sentence about dietary fibre is extremely categorical and could preferably be developed. Is it adequate for all groups, including children and underweight individuals? 107. Oxalic acid is highlighted as an inhibitor for calcium absorption. Therefore green leaf varieties with low oxalic acid should be selected. Is there any available information on oxalic acid content in different green leaves and has a risk benefit evaluation been done on this matter? 147. In the table on nutritional concerns protein and essential fatty acids are mentioned as critical nutrients followed by the recommendation "Careful combination of foods". What does this mean? Should all vegans calculate their amino acid score? A suggestion would be to recommend a varied diet with enough energy and plenty of pulses and whole grain products. 154. In the description of macrobiotic diets it says that they occasionally eat fish but that all animal products are excluded. It would be more correct to say "all animal products except some fish".</p>	<p>Thank you for your comments. The text has systematically been edited taking into account your suggestions and those made from several other professionals in the field.</p> <p>The text about macrobiotic diets is written as stated in the citation.</p>
Confederation of the Danish Food Industry	<p>General comments: We thank you for this opportunity to comment on the Nordic Nutrition Recommendations. We believe it is important to provide guidance in line with current habits and food supply, thus we would like you to consider adding more sources of ALA and LA in the vegetarian diet.</p> <p>Detailed comments: Lines 75-76: We suggest you add vegetable oil based spreads and soft and liquid based oils. Lines 77-79: We suggest you add sunflower oil as a source of LA as well as soft margarines and spreads based on sunflower oil. These products are more common than the products suggested in the text.</p>	<p>Thank you for your comments. The text has been edited taking your suggestions into account as well as those made by other professionals in the field.</p>

Organisation/ Company	Comments	WG responses and actions
Private	<p>General comments: Vegetarian diets in children, pregnant and lactating women should be in line with ADA (American Dietetic Association) position, in which says: "Well-planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets are appropriate for all stages of the life cycle, including pregnancy and lactation. Appropriately planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets satisfy nutrient needs of infants, children, and adolescents and promote normal growth " (ADA: Position of the American Dietetic Association: Vegetarian Diets. Journal of the American Dietetic Association 2009:109:1266-82.)</p> <p>Detailed comments: Line 11: remove word "strict", it is unnecessary adjective. Term "Vegan" is clear, it means person who excludes all animal products in his/her diet. Line 22: "A well balanced lacto-ovo-vegetarian or lacto-vegetarian diet is considered to provide"... - replace: "A well balanced lacto-ovo-vegetarian, lacto-ovo-vegetarian or vegan diet provide sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, as well as children." Line 102: Add: However, studies shows, that vegans riboflavin intake is sufficient. Many vegan products, like soya milks, contain fortified riboflavin. Line 105: Add: Nowadays fortified soy,- oat,- and rice milks and some juices are significant calcium contributors in vegan diet. Line 131: When amount of iodine in algae is known, it can be considered a reliable source. Iodine content in nori, typically used in sushi foods, is negligible, but for example in wakame and kombu there is lot of iodine (Kolb N et. al. Evaluation of marine algae wakame (Undaria pinnatifida) and kombu (Laminaria gigitata japonica) as food supplements. Food Technology and Biotechnology 2004;42:57-61.) Line 137: "A vegetarian diet including dairy has generally been associated with adequate growth, and a well- balanced lacto-ovo-vegetarian diet is nutritionally adequate for the growing child" Replace: "Appropriately planned vegan, lacto-vegetarian, and lacto-ovo-vegetarian diets promote adequate growth and are nutritionally adequate for growing child. Vegan diet needs always supplemental vitamin B12 and vitamin D."</p>	Thank you for your comments. The text has systematically been edited taking into account your suggestions and those made by several other professionals in the field.

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General and detailed comments	WG comments and actions
<p>MTT Agrifood Research Finland</p> <p>A rich biodiversity absorb fluctuations and stress in the ecosystem. Why not to refer to resilience, which is a professional term. Absorbing fluctuations does not help understanding. Biodiversity, in which conditions does grazing cattle increase biodiversity in which condition decrease? In an extensive production system with grazing cattle the biodiversity increases, but in overexploitation of grazing areas and in circumstances of uncontrollable land use biodiversity might drastically decrease.</p> <p>A positive development in greenhouse heating has been seen in for example the Netherlands and Sweden – and in Finland – we have been able to show that with modern greenhouse technology the climate change impact of e.g. tomato in Finnish greenhouses are at the same level as in field production in the Mediterranean countries. And by using</p>	<p>“Resilience” accepted</p> <p>Thank you. Interesting reference.</p>

General and detailed comments	WG comments and actions
<p>greenhouse products one can avoid all the toxic impacts that originate from the necessary use of plant protection products in warmer climate conditions. YRJÄNÄINEN, SILVENIUS, F., KAUKORANTA, T., NÄKKILÄ, J., SÄRKKÄ, L., TUHKANEN, E-M. 2013. Kasvihuonetuotteiden ilmastovaikutuslaskenta: Loppuraportti. MTT Raportti 83: 43 s. Abstract: Greenhouse, environmental impacts, carbon footprint, tomato, cucumber, salad, tulip, pot plant.</p> <p>“Butter is generally not recommended in a nutritionally adequate diet due to its’ high content of saturated fatty acids. But in a sustainable food system all food produced should be consumed. In the food industry butter has been substituted with plant oils, some being palm oil, appreciated for its’ technical properties. There is no nutritional advantage to replace butter with palm oil. Both contain approximately 50 per cent saturated fatty acids. In a more sustainable and plant-based diet, containing less saturated fatty acids from animal sources, it may be worth to re-evaluate the role of butter in the diet.” This is really complex way of expressing – who will understand this or how?</p> <p>When comparing the dietary results this article could also be refereed: SAARINEN, M., KURPPA, S., VIRTANEN, Y., USVA, K., MÄKELÄ, J., NISSINEN, A. 2012. Life cycle assessment approach to the impact of home-made, ready-to-eat and school lunches on climate and eutrophication. Journal of Cleaner Production 28: 177-186.</p> <p>Line 92 – 99 Line 159-162 Line 180-186 Line 218 further on PLEASE NOTICE - I SEND THIS SECOND TIME BECAUSE OF A SPELLING MISTAKE (GRACING - SHOULD BE GRAZING)</p>	<p>Been rewritten</p> <p>Too detailed information for chapter.</p> <p>It is changed</p>
<p>Elling Bere and Nina Øverby, University of Agder, Norway.</p> <p>We appreciate that the New Nordic Nutrition Recommendations now also include a chapter on sustainable food consumption. We have a few comments to this chapter: - Transportation of food is hardly mentioned. For environmental reasons, the food should be produced as close to where it is consumed as possible. See Bere & Brug (2009) for a discussion on this issue. - There are more conflict areas that need discussion: E.g. green house vegetables, vegetables by air, tropical fruits and milk – all foods with high climate impact (if the low energy density of these products is taken into consideration, as it should). Sugar also is a conflict area: low climate impact (table 1), but not very healthy. - Milk/butter. We find the conflict discussion on butter rather strange. This discussion should rather be on milk and milk products in general. - Another conflict not mentioned is that we produce most animal foods we eat ourselves in the Nordic countries, while a lot of the plant food is imported. As transportation probably is more important for the climate impact of plant foods than for animal foods, while type of production is more important for animal foods (e.g. see paper by Saxe et al. (2013) below) – this is clearly a conflict. - Wine is mentioned several times, however, beer is not mentioned. – There are no more/less suggestions in the four last rows in Table 2.</p> <p>References: Bere E, Brug J. Towards health promoting and environmentally friendly regional diets – a Nordic example, Public Health Nutrition, 2009, 12:91-96. – Saxe H, Larsen TM, Mogensen L. The global warming potential of two healthy Nordic diets compared with the average Danish diet, Climatic Change, 2013, 116:249-262.</p>	<p>Agreed. Will lift it. It does appear in Table 2.</p> <p>Butter discussion is changed. More and more of animal food imported, domestic production declines.</p> <p>Beer is now mentioned It is changed</p>

General and detailed comments	WG comments and actions
<p>Ministry of social affairs and health, Finland.</p> <p>lines 141-147 are unclear, especially the sources of saturated fatty (SaFa) acids in the Nordic diet. Current text gives impression that eating just 30% less meat can be a solution to high SaFa intake. However, according to National Findiet 2007 Survey in Finland most SFA comes from milk and dairy products (29% for women, 28% for men) and cereal and bakery products (19% for women, 21% for men). Fat spreads and dressings cover 16% for women, 18% for men. Meat dishes contribute to 18% of total SaFa intake among both men and women. As SaFa is 13 E% among men and 12% among women this problem cannot be solved by 30% cut in meat only. So please revise the text to give more comprehensive description of the situation in Nordic Countries</p> <p>lines 185-186 unclear what lines referring to butter in the diet mean. Health problems related to saturated dairy fat are well established. Excess butter can be utilized also outside food chain (candles etc.) lines Table 2, less dairy line, comments section: The sentence referring to butter consumption is very odd as butter has no health benefits and even reducing meat intake is not solution to SaFa problem (see my comment for lines 141-147)</p>	<p>% SFA from meat :</p> <p>DK 18% (2003-08)</p> <p>FI 18% (2007)</p> <p>NO 19% (1997)</p> <p>SE 17% (2010-11)</p> <p>UK 25% (a quarter of intake from meat + -products)</p> <p>Text added: "in UK where the intake of SFA from meat is 40% higher than in the Nordic countries, a decrease of 17 % in...was seen when meat was cut out".</p> <p>It has been altered.</p>
<p>European lifelong learning program "Eating together".</p> <p>General</p> <p>I write on behalf of the Lifelong learning program n° 2011-1-BE3-GRU06-03638 (http://www.dietethics.eu/en/nutrition/national-guidelines.php). The Danish Animal Alliance takes part in this European group, collecting official dietary guidelines for vegan diets around the world to help to protect people who choose to live on vegan diets. The Nordic Nutrition Recommendations are very interesting. We have requested an in depth study of your very good drafts and previous editions by Stephen Walsh (coordinator of the International Vegetarian Union's science committee). His suggestions on Sustainable food consumption are relevant to unessential red meat (line 139) and how to meet calcium requirements without milk (table 2 line 223).</p> <p>Details</p> <p>Line 139 "The degree to which red meat would be essential in our diet is unknown." This seems a surprising comment as epidemiological evidence indicates red meat to be associated with worse not better health (http://archinte.jamanetwork.com/article.aspx?articleid=414881) and studies on vegetarians indicate equal or better health with the complete exclusion of meat (http://ajcn.nutrition.org/content/70/3/516s.full.pdf). Red meat is clearly not essential to a healthy diet. Table 2 (Line 223) "Difficult to meet calcium recommendations without milk." Meeting recommended calcium intakes while cutting down on dairy products can be an issue if nothing else changes but can be readily resolved by fortification of milk substitutes or other foods. Fortification of plant-based substitutes for animal foods with a small number of key nutrients such as B12 and calcium would facilitate a safe shift to a more sustainable</p>	<p>Comments below support the aim of the chapter.</p>

General and detailed comments	WG comments and actions
plant-centred diet.	
<p>Unilever Nordic, Introduction: INTRODUCTION: To be truly sustainable we need to look at all the environmental, social and economic impact of the food we eat. We acknowledge this is a big task but crucial to be included when defining what consists a sustainable diet. As the proposal on “sustainable food consumption” currently stands there are several inconsistencies making the proposal less convincing. For example there is a discrepancy between the recommendation to reduce SAFA at the same as there is a suggestion to re-evaluate butter despite its high SAFA content without considering comparative life cycle assessments. We know that conflicting information often lead consumers confused about how to make the best choices for their health and the environment. Therefore we also believe any nutrition recommendations should be based on sound science as well as take a life cycle analysis approach to properly assess the impacts. We also believe nutritional aspects should set the boundary conditions for what constitutes a sustainable diet. Not the other way around. Thus we believe the chapter on Sustainable food consumption would benefit from a more explicit definition of what consists a sustainable diet as well as set targets to clarify what to achieve by promoting such a diet. To ensure consistency we also believe the paragraph on “conflicting areas of a sustainable diet and health” needs re-writing to reflect the addressed concerns and comments as outlined below. For further information about Unilever Sustainable Living Plan please visit www.unilever.com</p> <p>DETAILED COMMENTS:</p> <ul style="list-style-type: none"> • Row 43-45: Any recommendations in the area of sustainable diets aligned with NNR should be based on a more comprehensive assessment of sustainability factors. The current evidence in the literature is focused almost exclusively on the link between diet and greenhouse gases and this illustrates the current lack of knowledge/understanding. Unilever supports the need for further research in this area so that more aspects than green house gases are considered. • Row 92-93: To generally state that grazing animal helps biodiversity is overly simplistic and the effects are highly location and time-dependent. In the Nordics the link might be positive but this is not the case in many countries (e.g. beef production in Brazil) and we need to be aware of the global nature of many food chains. • Row 123-126: The text about the climate efficiency of protein production should be extended to include other dietary nutritional values of food. • Row 116-162: To fully understand the consequences of a shift in diet and the associated agricultural systems changes, a more explicit definition of sustainable diet is needed and what targets one want to achieve by promoting such a diet to the public. • Row 180-186: It is also important to focus on the “invisible fat” (in meat, cream, cheese, cakes etc.) since that is providing almost 2/3 of the fat in the diet and not get a bias towards the easy measurable and visible fat intake from butter, margarine and oils. • Row 181-186: It will be confusing when the scientific chapter on fat includes general recommendations to reduce SAFA and in the sustainability chapter of the NNR there is a comment to re-evaluate butter despite its high SAFA content. This shows discrepancy in scientific opinions in the NNR and opens the scientific papers for criticism of inconsistency, and will make the recommendations less convincing. 	<p>Definition of Sustainable foods is changed</p> <p>Butter comment is rewritten.</p> <p>Acknowledged, but early stated in chapter there is not enough information to enumerate various foodstuffs in relation to other environmental goals.</p> <p>Agree, it is place dependent</p> <p>A very good idea, but space limitations</p> <p>Redefined</p> <p>Palm oil is often an invisible fat in cakes and other processed food items.</p> <p>This is an exploratory paper bringing in environmental aspects and showing the conflicts that may occur.</p>

General and detailed comments	WG comments and actions
<ul style="list-style-type: none"> • Row 181: We agree with that the efficiency of food production systems should be optimized but we do not believe that adding butter to be part of a diet as a substitute for recommended low fat margarines is correct. If one does a simplified check, starting with the recommendation to drink 0,5L low fat milk per day, which produces two “by-products” 15g (2) meat and 17,5g milk fat. If one then follow the food pattern suggested in S.m.a.r.t. maten (3) approximately 14 g milk fat is consumed via cream, cheese and “indulgence food” (utrymmesmat). Remaining 3,5g could then of course be used for milk fat based light margarines for 1- 2 of the 5-6 sandwiches recommended. But there is still a need for 20-25g light margarine per day. This margarine, if well chosen, will also in a good way contribute to the intake of unsaturated fat, which should contribute with 2/3 of total fat according to previous and assumed new recommendations. Health should be prioritized and If there is a surplus of butterfat innovationcould help find a good use for it. • Row 181-184: The text claims that there is no nutritional advantage to replace butter with palm oil, since the content of saturated fat is the same. This is both confusing, and factually incorrect. Palm oil has not, and is not, used as a replacement for butter, since palm oil is used neither for spreading, cooking or baking in Nordic countries. Palm oil is used as an ingredient in foods. Palm oil is though often replacing partially hydrogenated fats/oils, which contain trans fatty acids. • Row 184: The fat quality should be compared per fat content and not per 100 g product. Butter is a product with 80% fat content. Butter fat contains more saturated fatty acids (SAFA) than palm oil. Butter fat contains 64 % SAFA, and with around 3% trans fatty acids included it is as much as 67 % of total fat. Palm oil contains 47 % SAFA and no trans fatty acids (Swedish National Food Administration’s data base). Based on these facts the suggestion to re-evaluate the role of butter in the diet has no support from nutritional point of view, and moreover it will also be very confusing for nutrition experts as well as consumers.(1) • Row 185-186: Considering the pressure that exists on the ecosystem for fish it might not be an option to have a further increase intake of fish, even if desired, to raise the intake of unsaturated fats. So a complement of unsaturated fat is needed from vegetable fat based products like margarines, liquid margarines and oils. So to replace current source of vegetable unsaturated fatty acids from margarines and oils with butter as suggested, would not improve the nutritional profile of the diet. • Row 180-186: Market data from 2012 Table 1 show that the oils from products like mélanges, spreads and margarines have the following volumes (4). Table 1: OIL TYPE OILS IN TONS butteroil 26670 rapeseed 17200 palm 8490 sunflower 3040 linseed 1290 coconut 860 olive 18 CO2 equivalents produced by these oils are shown in Table 2 (5) Table 2: OIL TYPE CO2 EKV butteroil 229000 rapeseed 33000 palm 35400 sunflower 5460 linseed 1730 coconut 370 olive 21 • Row 222-224: The basis of classification of food sources and ingredients in Table 2 is unclear. In the table palm oil is singled out and not grouped together with other vegetable oil and the diverse group of fruits and berries and vegetables are group together doesn’t not seem logical. <p>References: (1) Nilsson et al, 2010 - DOI 10.1007/s11367-010-0220-3. (2) http://www.kottguiden.se/ (3) http://www.folkhalsoguiden.se/Informationsmaterial.aspx?id=1068 (4) Nielsen retail Scan track DVH Yellow Fats FY 2012 and Nielsen Market trends 2011 (5) Butteroil deducted from CO2ekq for butter, LCA assessment of margarine and butter by SIK, Unilever Rapeseed:</p>	<p>The suggestion is to discuss if palm oil could be substituted with butter where palm oil already is used. The production of palm oil is detrimental in both ecological and social terms, and the production is criticized.</p> <p>Agree. It is used as an ingredient. The suggestion was: where palmoil is used, can butter be used instead? The food industry may use the individual fatty acids as they require. The essence is about two sources of fat containing high amounts of saturated fatty acids – could there be a discussion from an environmental point?</p> <p>The suggestion is to replace saturated fatty acids already used in the food industry with other already existing saturated fatty acids on a 1:1 basis.</p> <p>Valuable statistics. Acknowledged</p> <p>Food groups or food stuffs showing difficulties or being favourable to the environment are singled out. List will be improved.</p>

General and detailed comments	WG comments and actions
<p>Ecoinvent - publically available database (http://www.ecoinvent.org/database/)</p> <p>Palm: average of palm and palm kernel oil incl. cultivation on peat and land transformation Sunflower oil, cultivation in South Africa (Unilever data)</p> <p>Coconut: Ecoinvent – publically available database (http://www.ecoinvent.org/database/) & Shonfield (2007) Internal UL Agricultural LCA model</p> <p>Olive oil: Shonfield Shonfield (2007) Internal Unilever Agricultural LCA mode</p>	
<p>SIK – The Swedish Institute for Food and Biotechnology.</p> <p>General</p> <p>The document covers the area in a good way, considering the complexity of the issue and the limited number of pages. However, this leads to some overly simplified descriptions that are covered in the detailed comments below. It should be clearly stated that the point of departure is a nutritionally balanced diet; this is the ultimate goal that can't be compromised. Based on that the sustainability of a nutritionally balanced diet is discussed and possibly recommendations done. The aspect of global food security is poorly covered, and this should be a part of a sustainable diet in our view. The core issue is the limited area of possible farm land. This is one of the most important ultimate constraints to food production, so area intense production per se has negative consequences. At the same time there is an issue of land quality; for example ruminants might have a role in their ability to transform roughage feed (e.g. grass) into high quality meat, hence making it possible to use poorer land in less favorable climates for food production. Comments from SIK are given by Katarina Nilsson, Senior Consultant, and Ulf Sonesson, Research Manager at SIK and Senior Scientist. Both are from the Dep. of Sustainable Food Production.</p> <p>Details</p> <p>Row 19-35, "Conclusions": This is not really conclusions, but more of a summary. We think it is difficult to have a focused section with conclusions, a "Summary" would probably be better. If conclusions are presented it must be more precise. For example, as it is now vegan diets are mentioned, but is the conclusion that we should strive for veganism and is a vegan diet sufficient to present nutrient recommendations?</p> <p>Row 34: The discussion on butter seems very detailed and illogical; we suggest removing that part of the sentence.</p> <p>Row 38-60, "Introduction": We lack the global food security perspective. Mention clearly that climate is the main focus (which we have interpreted).</p> <p>Row 65-66: Many gases besides CO₂, CH₄ and N₂O also contribute, even if these three mentioned ones are the largest. For certain products (e.g. wild caught fish) emissions of refrigerants are the largest contributors to climate change.</p> <p>Row 98: The expansion of arable land and cropland is expected to take place in developing countries, at the same time arable areas in developed countries are expected to slightly decrease. This is mainly due to market mechanisms, where food production is cheaper in developing countries.</p> <p>Row 112-114, Table1: This table shows the climate impact from primary production, not for the whole supply chain. For some products the latter part of the chain might contribute significantly, especially if fossil fuels are used for electricity production. Dietary oil is a broad group from environmental point of view. Olive, rape seed and other oils should be in the middle group, palm- (if counted without contribution of land use change and methane produced from waste fraction</p>	<p>Changed to Summary.</p> <p>Rewritten Acknowledged. Will clarify climate focus.</p> <p>Acknowledged. Will include in final version.</p> <p>Acknowledged</p>

General and detailed comments	WG comments and actions
<p>at mill) and coco oil has lowest climate impact and fits into group one. Green house vegetables if produced in greenhouses using non fossil fuel might result in a climate contribution less than 1 kg CO₂/kg as well.</p> <p>Row 152: Reformulate “cereals and other animal sources” to “cereals or animal sources”</p> <p>Row 160: “spill” should be “residual”, change also in table two p 8.</p> <p>Row 170-178: Farmed fish or aquaculture could be more sustainable if herbivorous species were farmed instead of carnivorous. Laos farming of filtering organisms like mussels can be interesting.</p> <p>Row 180-186: Remove this entire section. It does not make sense as we see it, and is also very detailed compared to the rest of the document.</p> <p>Row 190-204: At several places in this part “environmental impact” is mentioned. Is it environmental impact or climate impact? If it is environmental impact then it should be mentioned how all different impacts are weighted together in order to have a single number for reduction.</p> <p>Row 208-212: The climate impact of vegan- and ovo-lacto diets are discussed, but you should also discuss their nutritional values. Otherwise it can be interpreted that they are similar to recommended diets from that point of view, hence be the best diets from all perspectives.</p> <p>Row 222-224, Table 2: This is a good summary but need to be more developed. 1. Split the column “environmental benefit/impact” into two columns. The first column should describe the env benefits, the second column negative env consequences. Uncertainties should be in the “comments” column. Avoid comparisons in the table (as for field vegetable, where a comparison with greenhouse grown vegetables are stated). 2. In the preceding text or in the caption clearly explain that the table is based on diet changes required to reach a more healthy diet as the starting point, and the environmental consequences are derived from that. It is not that we start with minimum environmental impact and list health consequences (which would be strange). 3. More fish and seafood. In the comments column mention herbivorous fish species and filtering organisms as interesting options. 4. Less savory snacks/sweets. Delete the comment on “less climate intense production”. We do not see any connections to that. Reducing the consumption of these products has health benefits but not many environmental benefits. 5. More fruits, berries and vegetables: Add that within this group you find several air-transported products.</p>	<p>Done</p> <p>Done</p> <p>It is rewritten.</p> <p>Changed to climate impact</p> <p>Acknowledged- Done</p> <p>Advice followed</p>
<p>RIVM – National Institute for Public Health and the Environment.</p> <p>General</p> <p>My compliments for the text and work. Some suggestions.</p> <p>188: check for typing error Experiences general: maybe you can add information of this recent publication Replacement of meat and dairy by plant-derived foods:</p> <p>Estimated effects on land use, iron and SFA intakes in young Dutch adult females. Temme EH, van der Voet H, Thissen JT, Verkaik-Kloosterman J, van Donkersgoed G, Nonhebel S. Public Health Nutr. 2013 Feb 21:1-8. [Epub ahead of print]</p> <p>Table 2. I miss statements for the drinks category. what about soft drinks? what about beer? Alcoholic drinks might be another conflict area. (raise HDL cholesterol, lower risk of CHD, medium-high climate impact)</p>	<p>Reference given, not published yet</p> <p>Drinks category improved in table.</p>
Mills, Norway.	

General and detailed comments	WG comments and actions
<p>General</p> <p>With reference to the document “ Sustainable Food Consumption NNR 2012” we would like to comment on the text presented on lines 180-186: Butter is generally not recommended in a nutritionally adequate diet due to its’ high content of saturated fatty acids. But in a sustainable food system all food produced should be consumed. In the 181 food industry butter has been substituted with plant oils, some being palm oil, appreciated for its’ technical properties. There is no nutritional advantage to replace butter with palm oil. Both contain approximately 50 per cent saturated fatty acids. In a more sustainable and plant-based diet, containing less saturated fatty acids from animal sources, it may be worth to re-evaluate the role of butter in the diet. We have the following comments:</p> <ul style="list-style-type: none"> • We agree that butter should not be recommended due to its high SAFA content. • We agree in principle that all food produced should be consumed to avoid unnecessary food waste, however this does not mean that a product should be promoted or recommended simply to avoid wastage. This would apply to any food product including margarine. Whether a product is recommended by NNR should solely be based on intrinsic nutritional or environmental features that set it apart from products it would be natural to make a comparison with. • It is also important to point out that in Norway palm oil has been replaced by alternative plant oils, such as soybean, rapeseed, sunflower, thereby vastly improving the overall fatty acid profile. This means that, from a nutrition viewpoint, plant oil based margarines on offer in the Norwegian market are greatly superior to butter. • Bearing in mind the unequivocal link between SAFA and a higher risk of CHR disease, it is vital that such recommendations provided by NNR should not contribute to an increase in butter consumption. This is particularly vital with the increasing popularity of trend diets such as “low carb” that promote the use of butter. Today we see that that SAFA contributes with 16 E% in the diet, a figure far exceeding the 10E% recommendation made NNR. Public health concerns should therefore considered seriously before making changes to recommendations that might increase this further. • The following factors must be taken into consideration before recommending butter from an environment standpoint: <ul style="list-style-type: none"> o Production of butter is dependent on the use of animal feed to supplement grass/fodder eaten by cows. This animal feed (kraftfôr) is based on such crops as soybean, maize, oats rapeseed and barley. These must be produced and transported over long distances and they make up a significant part of the resources needed to produce milk and subsequently butter. o The fraction of the crops mentioned above used in production of animal feed is much greater than that used to make plant oils. For example, oil extracted from soybean represents approximately 20% of the crop, while the remainder is used mainly for production of feed. There is in addition an conversion factor of 12:1 of feed to butter which must be taken into consideration. Conversely oils for margarine 	<p>Comment concerns the “butter-issue”. The section is rewritten.</p>

General and detailed comments	WG comments and actions
<p>production can be used more efficiently (1 kg oil = 1,2 kg margarine).</p> <p>o There are also rain forest issues associated with the production of soybean based feeds.</p> <p>o The livestock used in dairy farming also represent a significant environment factor. Methane emitted by cows is a major contributor to the total production of greenhouse gases. Methane is estimated to be 23 times more potent compared to CO₂ and a cow can produce as much as 100 – 200 L daily.</p> <p>o A LCA study published in 2010 in The International Journal of Life Cycle Assessment (*) concluded in the following way: The margarine products analysed here are more environmentally favourable than the butter products. In all three markets (UK, DE and FR) the margarine products are significantly better than the butter products for the categories global warming potential, eutrophication potential and acidification potential. These findings are also valid when comparing margarines and butters between the markets; for this reason they are likely to be of general relevance for other Western European countries where similar margarine and butter production systems are found. There can be no doubt regarding the serious nature of making general recommendations that can have long lasting public health consequences. The negative nutritional aspects of butter are clear and unmistakable. It is therefore of paramount importance that all the above mentioned factors are given serious consideration before making any recommendations for butter. This is particularly important since the sustainability conclusions presented in this hearing document are both unfounded and contradictory to the prevailing nutritional recommendations to limit the intake of butter.</p> <p>* Nilsson K et al. Comparative life cycle assessment of margarine and butter consumed in the UK, Germany and France. The International Journal of Life Cycle Assessment, November 2010, Volume 15, Issue 9, pp 916-926. (http://link.springer.com/article/10.1007%2Fs11367-010-0220-3)</p>	
<p>Norwegian Directorate of Health</p> <p>General</p> <p>This draft gives the impression of a very preliminary draft. The language and level of precision may be improved. Terms and concepts used should be more clearly defined e.g. sustainable; food systems; climate impact; effect on the environment (includes?) etc. You may also clarify if this chapter is a summary of current knowledge or if it reflects conclusions and recommendations from the NNR-project. You may also add more information from other Nordic countries. E.g. the Norwegian report on food based dietary guidelines from 2011, chapter 32 deal with “Dietary advice and environment”. It includes some references that still may be of interest.</p> <p>Details</p>	<p>I Kostråd till og fremme folkhälsan, 2 s (=plant food, transport, packaging, waste).</p>

General and detailed comments	WG comments and actions
<p>Line 18. Is this section a summary of the chapter or conclusions from the NNR-group? It is important that this section is improved in relation to readability and conclusions of documentation.</p> <p>L 20 “a nutrient balance” – what do you mean?</p> <p>L 23. “a lower impact.” – than what?</p> <p>L 31 you list several food groups – what about staple food as grain, bread, potatoes?</p> <p>L 32.”sensitive products” - define</p> <p>L 34 the conflict with butter seems artificial – lower production of milk will give less butter.</p> <p>L 39 define sustainable food consumption?</p> <p>L 39 without risking those of future – risking what?</p> <p>L 41. ,etc. - please complete the list.</p> <p>L 45. “climate impact can be measured and communicated” – we believe that also other environmental issues can be communicated even if they are difficult to measure.</p> <p>L 70 define food systems.</p> <p>L 92 vs 98. The sentences concerning the effects of grazing and grasslands in relation to biodiversity seem to contradict each other.</p> <p>L 112 Table 1. Climate impact of production of foods. This is useful information, but you should add information about the validity of these figures. You may add more data concerning differences in climate impact within the high CO2e food group. If possible also add CO2e values for processed foods as margarine, cakes and meat balls, and if there is a difference between wild and farmed fish? Later you mention organic production. What are the CO2e-values for organic and conventional production per kg?</p> <p>L 118. “half of the present impact” – of what (measure)?</p> <p>L 126.give reference to figures.</p> <p>L 142 “cutting the overall intake of meat ...” – do you mean to zero?</p> <p>L 177. Please expand what you mean with “fishing policies are often ignored ...etc”</p> <p>L 180-86. The conflict concerning butter seems artificial and constructed. Decreased production of milk will give less butter. Please clarify if production of butter is environmentally sound. You may add information about different environmental effects of milk production based on the use of grasslands and fodder in remote areas that cannot be used for other food production than meat and milk, in comparison with areas that can be used for production of cereals and other plants for direct human consumption. We agree that butter and palm oil both are high in saturated fats. Nutritionally we prefer that butter and palm oil is exchanged for other edible oils as rapeseed oil. However, you claim in table 2 that palm oil production is very efficient per ha.</p>	<p>Nutritional balance ..than present diet (changed) List has been changed, they are added.</p> <p>Perishable. Rewritten Done Sentence omitted. Word omitted. “Communicated” has been omitted.</p> <p>Food systems: Food production, food consumption, export, import, transport, storage, retail Rewritten</p> <p>Table has been expanded, but all food items cannot be presented. The variation in climate impact of a food item is discussed in text.</p> <p>Organic discussion to add. Climate impact LCA data from literature and nutrient data from NFAs food database. YES, that is what the authors meant. Rephrased (...policies..). I should not expand this section, I should rather decrease the section, in accordance with comments given.</p> <p>0.3 kg CO2e per kg oil- but no addition</p>

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<p>What is the CO2e for palm oil production?* You may add information about the Norwegian campaign on palm oil. The import of palm oil to Norway has decreased from 26 000 tons in 2005 to 15000 tons in 2011 and 5000 tons in 2012. http://www.regnskog.no/bevisst-forbruker/palmeolje/hva-er-probleme-med-palmeolje</p> <p>L 195 “by 15,5 per cent” – the use of one decimal seems strange here. L 195. “environmental impact” – define L 208 “the climate impact” – define. L 222, table 2. Suggested dietary changes – by whom? Less savory snacks – “Less climate intense production” – Which means? Please define. For some crops you mention pesticide use (potatoes) but not for others (roots, cereals). Is this correct? Page 8. “Rape seed production part of agricultural circle” – which means?</p>	<p>from land use change – can be substantial Useful information.</p> <p>Agree Climate impact</p> <p>Appropriate question. Been done earlier Table and list is improved. Points taken.</p> <p>Agricultural rotation.</p>
<p>Dairy Nutrition Council of Finland. General Row 77: All food production brings about varying environmental cost. However, it is important to consider this cost in the context of the nutritional value provided by the food. Available research, although still relatively new and complex, shows that milk scores significantly better than many other beverages in nutrient density to climate impact and that changing the structure of human diets by reducing the consumption of animal-based products is not a sufficient approach to significantly reducing emissions. It should be borne in mind that it is not merely an issue of greenhouse gas emissions but also of the nutritional density of food products. Smedman A et al. Nutrient density of beverages in relation to climate impact. Food & Nutrition Research, 54: 5170, 2010.</p> <p>Details Row 223 Table 2: Row less Dairy: This is confusing even to professionals, what does it mean. The consumption of milk and dairy doesn’t exceed the recommended level of current national recommendations. With less milk than that, it is difficult to meet the recommendation on calcium and vitamin D (milk is enriched in Finland) with more sustainable products. When compared to the nutrient density of beverages, the climate impact of milk is substantially better than of the other beverages studied, incl. soy and oat drink (see Smedman A et al. Nutrient density of beverages in relation to climate impact. Food & Nutrition Research, 54: 5170, 2010). Could it be here rather "maintain the present consumption level" than "less dairy"? Comments column: Add->Also other intake of essential nutrients decreases for instance high quality protein (all essential amino acids), potassium, magnesium, vitamin B12, zinc, selenium, folate, riboflavin, niacin equivalents, iodine and vitamin A (vitamin D). Also, dairy has a positive impact on health in many ways, for example diabetes prevention,</p>	<p>Nutritional density vs climate/environmental impact is a valuable way to illustrate the nutrient contribution from various food stuffs. There is unfortunately not space to expand more. There is a short discussion on protein and climate impact in text.</p> <p>Less consumption in relation to today.</p> <p>Not everyone agree on the level of milk consumption. Milk is a very nutrient dense and valuable food, but several of it’s nutrients can be found in foods with less climate impact. – has been added now.</p>

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<p>which is another reason why the consumption of milk and dairy should not be decreased. For example:</p> <ul style="list-style-type: none"> • Peter C. Elwood et al. The Consumption of Milk and Dairy Foods and the Incidence of Vascular Disease and Diabetes: An Overview of the Evidence. <i>Lipids</i> 2010; 45:925–939. DOI 10.1007/s11745-010-3412-5. • Frédéric Fumeron et al. The Data from the Epidemiological Study on the Insulin Resistance Syndrome (DESIR) Study Group. Dairy Consumption and the Incidence of Hyperglycemia and the Metabolic Syndrome. <i>Diabetes Care</i> 2011;34:813–817. • Marianne Hauge Wennersberg et al. Dairy products and metabolic effects in overweight men and women: results from a 6-mo intervention study. <i>Am J Clin Nutr</i> 2009;90:960–968. Vasanti S Malik et al. Adolescent dairy product consumption and risk of type 2 diabetes in middle-aged women. <i>Am J Clin Nutr</i> 2011;94:854–61. 	
<p>Finnish Meat Association. General Basic challenge in evaluating environmental impacts of food production is lack of scientific measurement methods. This recommendation is based on to GHG-calculations which covers unfortunately only one perspective of environmental impacts in food production chain. CO2-calculation is based on mathematic models which might not have any contact to visible environment. Calculation doesn't cover all environmental impacts the food production has, for example land use, biodiversitys, water intensity. etc. There's no common globally agreed CO2 –calculation method, so there's no guarantee that we have comparable results in different products. Conclusions made in recommendation cannot be based on calculation methods which is restricted in most remarkable issues in environment. Models which presents different diet comparisons doesn't take account the opportunity costs there is when moving more vegetable diet. The environmental costs decreased because of minimizing meat production will be fulfilled with other impacts on opportunity use of natural resources, because demand for protein doesn't decrease. From sustainable point of view the Nordic nutritional recommendation should also take account the local Nordic food production facilities and needs for local consumption. Optimal local food production in Nordic countries is based on integrated system (mixed livestock-crop) where vegetable/crop-production and meat production is in balance. Meeting the demand of growing population from sustainable point of view the local integrated production should be preferred. For example large areas in Nordic countries are most suitable for grassland (used by beef production) and not very suitable for crop production (because of the soil and short period of growth). Water is also one factor preferring local production. At the moment approximately 41% of Finnish water footprint consists of foreign origin. (www.vesijalanjalki.org). In Nordic countries most water used in agriculture is so</p>	<p>Calculating climate impact is an extensive calculation. There are differences in the methods used. Overall we are able to roughly categorize food stuffs after their climate impact. There is a continuous development. However there is much less consensus how to measure biodiversity and water intensity.</p> <p>Yes, it would be valuable to broaden the discussion.</p>

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<p>called "green water" (rainwater), no watering systems needed. In foreign countries there's need for watering in agriculture and system uses "blue water" (groundwater, rivers, lakes). The key element securing sustainable food consumption is to increase production efficiency both in crop production and animal protein with substantial reductions in the environmental impacts (Aland & Madec 2009)</p> <p>References: Sustainable animal production. The challenges and potential developments for professional farming. A.Aland & F. Madec. 2009. Wageningen Academic Publishers. The Netherlands.</p>	
<p>Swedish Food Federation. General Swedish Food Federations (Li) comments on "Sustainable food consumption – Nordic Nutritional Recommendations 2012" The Swedish Food Federation is positive to the inclusion of the area "Sustainable food consumption" in NNR 2012. It highlights the importance of knowledge about the relations between a healthy diet and environmental benefits/consequences. We fully support the comments on the chapter sent from SIK – the Swedish institute of Food and biotechnology (see below - under "detailed comments"). These comments show the lack of knowledge and the need of more research cooperation between the two scientific areas sustainability and nutrition. We strongly suggest that SIK's comments should be taken into account since sent out draft include inaccuracies. The scientific area is very young and premature - for example is the area almost exclusively focused on the link between diet and climate impact. The chapter should therefore more have the character of a summary of the current knowledge status than being communicated as recommendations</p> <p>Details SIK comments on "Sustainable food consumption – NNR 2012" General comments The document covers the area in a good way, considering the complexity of the issue and the limited number of pages. This leads to some overly simplified descriptions that are covered in the detailed comments below. It should be clearly stated that the point of departure is a nutritionally balanced diet; this is the ultimate goal that can't be compromised. Based on that the sustainability of a nutritionally balanced diet is discussed and possibly recommendations done. The aspect of global food security is poorly covered, and this should be a part of a sustainable diet in our view. The core issue is the limited area of possible farm land, this is one of the most important ultimate constraints to food production, so area intense production per se has negative consequences. At the same time there is an issue of land quality; for example ruminants might have a role in their ability to transform roughage feed (e.g. grass) into high quality meat, hence making it possible to use poorer land in less favorable climates for food production. Comments from SIK are given by Katarina Nilsson,</p>	<p>It has never been anything else than the current knowledge, but it is now more clearly pointed out.</p> <p>SAME COMMENTS AS THOSE FROM SIK.</p>

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<p>Senior Consultant, and Ulf Sonesson, Research Manager at SIK and Senior Scientist. Both are from the Dep. of Sustainable Food Production. Specific comments</p> <p>Row 19-35, "Conclusions": This is not really conclusions, but more of a summary. We think it is difficult to have a focused section with conclusions, a "Summary" would probably be better. If conclusions are presented it must be more precise. For example, as it is now vegan diets are mentioned, but is the conclusion that we should strive for veganism and is a vegan diet sufficient to present nutrient recommendations?</p> <p>Row 34: The discussion on butter seems very detailed and illogical; we suggest removing that part of the sentence.</p> <p>Row 38-60, "Introduction": We lack the global food security perspective. Mention clearly that climate is the main focus (which we have interpreted).</p> <p>Row 65-66: Many gases besides CO₂, CH₄ and N₂O also contribute, even if these three mentioned ones are the largest. For certain products (e.g. wild caught fish) emissions of refrigerants are the largest contributors to climate change.</p> <p>Row 98: The expansion of arable land and cropland is expected to take place in developing countries, at the same time arable areas in developed countries are expected to slightly decrease. This is mainly due to market mechanisms, where food production is cheaper in developing countries.</p> <p>Row 112-114, Table1: This table shows the climate impact from primary production, not for the whole supply chain. For some products the latter part of the chain might contribute significantly, especially if fossil fuels are used for electricity production. Dietary oil is a broad group from environmental point of view. Olive, rape seed and other oils should be in the middle group, palm- (if counted without contribution of land use change and methane produced from waste fraction at mill) and coco oil has lowest climate impact and fits into group one. Green house vegetables if produced in greenhouses using non fossil fuel might result in a climate contribution less than 1 kg CO₂/kg as well.</p> <p>Row 152: Reformulate "cereals and other animal sources" to "cereals or animal sources"</p> <p>Row 160: "spill" should be "residual", change also in table two p 8.</p> <p>Row 170-178: Farmed fish or aquaculture could be more sustainable if herbivorous species were farmed instead of carnivorous. Laos farming of filtering organisms like mussels can be interesting.</p> <p>Row 180-186: Remove this entire section. It does not make sense as we see it, and is also very detailed compared to the rest of the document.</p> <p>Row 190-204: At several places in this part "environmental impact" is mentioned. Is it environmental impact or climate impact? If it is environmental impact then it should be mentioned how all different impacts are weighted together in order to have a single number for reduction.</p> <p>Row 208-212: The climate impact of vegan- and ovo-lacto diets are discussed, but you should also discuss their nutritional values. Otherwise it can be interpreted that they are similar to recommended diets from that</p>	

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<p>point of view, hence be the best diets from all perspectives.</p> <p>Row 222-224, Table 2: This is a good summary but need to be more developed. 1. Split the column “environmental benefit/impact” into two columns. The first column should describe the env benefits, the second column negative env consequences. Uncertainties should be in the “comments” column. Avoid comparisons in the table (as for field vegetable, where a comparison with greenhouse grown vegetables are stated). 2. In the preceding text or in the caption clearly explain that the table is based on diet changes required to reach a more healthy diet as the starting point, and the environmental consequences are derived from that. It is not that we start with minimum environmental impact and list health consequences (which would be strange). 3. More fish and seafood. In the comments column mention herbivorous fish species and filtering organisms as interesting options. 4. Less savory snacks/sweets. Delete the comment on “less climate intense production”. We do not see any connections to that. Reducing the consumption of these products has health benefits but not many environmental benefits. 5. More fruits, berries and vegetables: Add that within this group you find several air-transported products.</p>	
<p>Finnish Heart Association Details</p> <p>Lines 33-34: It is misleading to say that “there is a conflict between nutritionally and environmentally sustainable diet with butter”. In table 1 butter is in high climate impact group. And from the nutritional point of view butter contains a lot of saturated fat.</p> <p>Lines 185-186: It is misleading to say that “it may be worth to evaluate the role of butter in the diet”. It is true that in food industry butter could be used instead of palm oil if suitable for particular food product. It is another thing to include butter in individual’s diet. If the term “diet” is used, recommendation could be interpreted in a way that consumers could use butter as itself in their own diet. It should be highlighted that it is not consumer’s responsibility to use the produced butter. It is only a choice for food industry to use excess butter instead of palm oil. Table 2: Line Less Dairy, column Comments “With smaller consumption of animal based food, smaller intake of saturated fatty acids, and therefore the produced butter may be consumed.”</p> <p>Reducing animal based food consumption is already a challenging goal. That is why it is unrealistic to assume that there would be space for saturated fat from butter in diet. And altogether it is challenging to reduce intake of saturated fat from dairy, at least in Finland. NNR should be considered primarily from the nutritional point of view. Why should the produced butter be consumed in diet if it is harmful for health?</p>	<p>OK, good point.</p> <p>Obviously the text is not clear enough, This has <i>never</i> been stated as a recommendation, only as a careful suggestion to discuss the matter in the food production.</p>
<p>National Institute for Health and Welfare, Finland General</p> <p>The round three in commenting procedure of the fifth Nordic Nutrition recommendations includes aspects for</p>	

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<p>sustainable food consumption. In summary, the main conclusion is, that there are excellent opportunities to have a nutritionally adequate diet in a sustainable way. However, some potential conflicts are stated in the choices of fish and butter from nutritional and from environmental view. The list of suggested dietary changes emphasizes the higher consumption of basic, vegetable-based food stuffs. The list would be more understandable if it would start from guidelines concerning vegetable-based food. THL, Nutrition unit (Finland) supports the following points: - A lower consumption of highly processed food is recommended. This concerns especially salty snacks and sweets. - Local root is appreciated. - Local cereal could be emphasized on recommended food items (Table 2) in more details by presenting cereals such as rye, barley and oat in the table. - Less dairy products seems to be too wide concept. Could the table separate milk drink in comparison with cheese, dairy fat and high-fat dairy products. It should be valuable to include oat milk as an vegetable-based alternative. But what is the environmental impact of the processing of oat milk? - Rape seed oil is presented in the table 2 as an ecological plant production. The health benefits of rape seed oil should also be listed. Poultry instead of beef and pork seems to have favorable impacts (see table 1) on the environment, but it is not separated in table 2. Is this logical?</p> <p>Details</p> <p>We suggest the following sentences to page 5, instead of rows 181-186: From environmental advantages, the use of palm oil as an ingredient of hard fat in industrial food products is not a sustainable solution. From nutritional point of view the fatty acid content of palm oil is equal to that of butter. It would be advisable to compare the fatty acid content and technological properties of different types of hard fats (e.g. hard fat made of rapeseed oil and other vegetable oils vs. butter or palm oil) in industrial food products. For sustaining environmental biodiversity, cattle breeding, grazing and living country side is important for Nordic countries. Thus there is a need to re-evaluate the role of butter in the industrialized food production and product development, but we hardly can recommend to re-evaluate the role of butter in the diet. Further, the term diet is not correct in the last sentence of this chapter. We suggest deletion of the last sentence and the reformulation of the previous section as presented above</p>	<p>Table 2 has been changed.</p> <p>Oat milk – to detailed and still a small product Health benefits are not highlighted in this text. Point taken. It is separated.</p> <p>Point taken and included.</p>
<p>Finnish Food and Drink Industries' Federation</p> <p>General</p> <p>Reference: NNR 5 working group: http://www.slv.se/en-gb/Startpage-NNR/Public-consultation11/ FINNISH FOOD AND DRINK INDUSTRIES' FEDERATION'S (ETL) COMMENTS ON THE CHAPTER ON SUSTAINABLE FOOD CONSUMPTION Sustainable food consumption general comments:</p> <ul style="list-style-type: none"> • The terms used in the draft recommendations are not clearly defined: What is meant by “sustainable” and “environmental”? Such official definition as “sustain-able food” does not exist and it may have various 	<p>Sustainable is defined now Environmental is described.</p>

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<p>meanings depending on the user. Hence, it remains unclear whether the focus is on purely environmental aspects or more widely on sustainability. In addition, the reasons for greater or lighter emphasis put on various environmental aspects are not transparent. Another question relates to the geographical focus: production circumstances and diets vary between the Nordic countries.</p> <ul style="list-style-type: none"> • While writing about environmental impacts one should define the scope in question. Climate change is only one of the impact categories. Life-cycle assessment of various environmental impacts related to foodstuffs should be the starting point for the analysis in order to be able to prioritize the most important impact categories and the corresponding actions. • The draft discusses only the trade-offs and synergies between food and sustainability/environmental/climate aspects without discussing the wider framework of food production and consumption. This analysis is done in isolation and therefore the resulting conclusions may be considered misleading. The overall framework of regional, national or global food production is missing and crucial matters such as national self-sufficiency (and the related geographical and other factors) are not included in the analysis. • Also the questions of consumer behavior and individual lifestyles and consumption patterns need to be taken into account. What is the price effect on consumers of the suggested measures and how would consumers react to these changes? • The draft is inconsistent and does not provide a logical overview and conclusions on the issue. It contains simplifications and does not succeed in setting the big picture and the related priorities or practical measures of how to tackle the twin challenge of improving consumer's nutritional choices and steering food production systems, including all types of raw material and end products, towards more sustainable operations. Thus, the suggested recommendations remain unconnected. • The difficulty of measuring environmental impacts of foodstuffs in order to create "sustainability criteria" for them is a widely recognized fact. Various reasons for this have been indicated for example by Oakdene Hollins (2011) in studying possibilities to create ecolabelling criteria for foodstuffs. Among these are the biological processes related to agriculture, huge variety of foodstuffs and the related production processes and combinations of ingredients as well as rapidly changing recipes and manufacturing formulas. The lack of consistent and widely recognized evaluation criteria makes it very challenging to produce comparable data related to the environmental impacts of various foodstuffs. This kind of information is a key factor in discussing nutritional and environmental benefits of various foodstuffs. • Diets and food manufacturing are not an environmental zero-sum game; demand for protein and other sources of nutrition will continue to increase. Thus, it is equally important to aim at improved environmental sustainability in all food production. Increasing energy and resource efficiency and minimizing waste in all parts of the food production and consumption value chain (i.e. producing more out of less and with less 	<p>Climate change is one of more environmental impacts - is stated</p> <p>Correct. Not enough space to bring up the issue with food production vs not using pesticides as well as askew distribution.</p> <p>Consumption patterns are discussed in chapter. Price effects are outside the scope, but nevertheless important.</p> <p>Important but outside the scope.</p> <p>Acknowledged. Ecolabelling so far is restricted to few foodstuffs in various systems/schemes. It is still an emerging area.</p>

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<p>impact) is more crucial than “blacklisting” various foodstuffs.</p> <ul style="list-style-type: none"> • To conclude, the contents of the draft exemplifies the fact how difficult it is to create a balanced, coherent and science-based view on environmental impacts related to foodstuffs. Adding the nutritional aspects to this makes the matter even more challenging, if not impossible (at least at this stage). FoodDrinkEurope: Draft position paper on food and sustainability • Diets cannot be seen in isolation and lifestyles have to be considered as a whole when talking about impacts on health and environment. The impacts people have on the environment and on their own health are influenced by a number of factors. In order for consumers to make informed decisions about how sustainable their choices are, they have to know what they are eating and drinking, how that contributes to their dietary needs and how their own activities affect their nutritional needs and the environmental impacts of food. This is often provided ‘on-pack’ but increasingly this is being provided by alternative, digital means (e.g. dedicated websites, social media, and smartphone applications). The report of the European Food Sustainable Consumption and Production Round Table on ‘Communicating environmental performance along the food chain’ http://www.food-scp.eu/files/ReportEnvComm_8Dec2011.pdf suggests that companies should use a mix of communication devices and should not rely on a single or limited set of communication tools. A broader consumer education on the environmental and health aspects of food consumption is needed to raise general awareness and ensure that the information provided is useful to the consumer. • Information provided to consumers should be simple, factual, objective, science-based and easy to access by consumers. Simplistic messages based on subjective criteria risk misinforming and misleading the consumer. Most importantly, however, it has to be ensured that any information that is communicated about the content and possible impacts of food and drink products, including the underlying criteria and mechanisms of such information, shall be scientifically reliable and consistent, understandable and not misleading, so as to support informed choice. For instance, the first step in moving forward in communicating the environmental impacts of food and drink products must be the finalisation of the Envifood Protocol by the European Food SCP Round Table. Strong public control to make sure that the information put on the market is in line with these principles is needed to ensure a level playing field and to avoid green washing. • Education is a key element in supporting the factual information about food and drink products, whether ‘on pack’ and/or through alternative means of communication, consumers are now given to make choices in line with their needs. Not only is it necessary to motivate the consumer to absorb the information; the consumer should also be able to process the information, placing the information in its appropriate context, in order to make a truly informed choice. Consumers who are given a choice are making healthier food choices than they used to. The food and drink industry is delivering increased choice to help consumers make appropriate food choices to suit their individual health needs and sustainability demands. 	<p>OK</p>

General and detailed comments	WG comments and actions
<p>Details</p> <ul style="list-style-type: none"> • Lines 19 and 197: What is preferable or theoretical is not the same as realistic. • Lines 22-23 and 213-214: It is stated here that persons following the (Nordic) nutrition recommendations tend to have a diet with lower climate impact. If so, why the focus can't be simply in promoting the existing recommendations? This would be useful and cost-efficient as consumers have difficulties in following the current nutritional recommendations. • Line 32: The key function of minimizing the environmental impacts of foodstuffs is waste minimization. • Lines 43-45: No general method for calculating and communicating climate im-pact exists. • Table 2: Suggested changes are described as "less" or "more". It remains un-clear what the reference point to "less" and "more" is in this case. • Table 2: Water is pointed out only as a matter of bottled water although the role of water supplies is crucial for food production. • Table 2: Fish and seafood is considered only as the capture and intake of wild fish stocks. The role of aquaculture is rapidly increasing due to which focusing only on wild fish stocks is misleading. • Table 2: The columns "environmental benefit/impact" and "comments" have partly overlapping information. In addition, the logic according to which certain environmental aspects have been highlighted remains unclear. Moreover, the table doesn't define the scale or internal importance of the various listed aspects. 	<p>Absolutely, that is a step on the way, but it is not enough, with the knowledge we have today, it shows we must go even further.</p> <p>Yes, waste reduction is very important, but it is not enough.</p> <p>No, but still there are methods and therefore it can be discussed.</p> <p>It has been rewritten-</p> <p>Acknowledged. Water supply was pointed out as a crucial factor at start. Table 2 has been revised.</p> <p>Table 2 has been revised according to comments.</p>
<p>LRF Dairy, Sweden</p> <p>General</p> <p>It is positive that the Nordic Nutrient Recommendations include a chapter on environmental impacts of diets. However, the aim of this chapter should be defined, as well as the methods used when preparing it. A complexity that is unfortunately not addressed is how you replace high-impact food items such as animal foods from a full nutritional perspective. This is particularly true if the full nutritional dimension of nutrient dense foods such as milk is taken into consideration. Replacing the protein of milk may be done quite easily with foods with lower environmental impact per kilo but replacing the full nutrient content is more challenging, without increasing the energy intake or using enriched products or supplements. The environmental impacts of fortification and supplements are commonly not quantified in studies on the environmental impacts of food. Therefore, only pointing to the advantages of a vegan diet from an environmental point of view needs to be carefully discussed and clearly referenced to capture the uncertainty in the analysis. Comments from LRF Dairy Sweden (LRF Mjöl, former Svensk Mjöl) are given by Anna-Karin Modin Edman, PhD and environmental expert, Helena Lindmark-Månsson, adjunct professor and nutrition</p>	

General and detailed comments	WG comments and actions
<p>expert, and Annika Smedman, PhD and nutrition expert.</p> <p>Details</p> <p>A discussion considering the willingness of the population to change dietary habits according to the suggestion should be included. The environmental impact of sweets, desserts, chips/crisps et cetera should be discussed (Nilsson et al. 2011). Relevant references should be included in the text, also on the combination of nutrition and climate impact (eg Buttriss et al. 2013, Vieux et al. 2012 and Smedman et al. 2010).</p> <p>Page 1, line 24-25: The sentence that those who choose a diet with less dairy products have a further lower climate impact lack references needed to support such a statement. In order for this to be a desirable strategy from a societal point of view, the study must ensure that these consumers fulfill daily requirements for all essential nutrients.</p> <p>Page 1, line 24-25: The sentence regarding the benefits of a vegan diet lacks a reference. It is therefore impossible to judge the quality of such a study. The environmental impacts of enrichment and supplements commonly required to make a vegan diet nutritionally adequate are usually not quantified in studies of the environmental impact of diets. This is an important weakness of many studies comparing diets and should be addressed in this document.</p> <p>Page 2, line 74-75: When discussing the climate impact of land use change it is also important to address carbon sequestration, which is an important mitigation possibility for agriculture, in particular grass based dairy and beef production (e.g. Schulze et al. 2009, Soussana et al. 2007, Cederberg, et al. 2012.)</p> <p>Page 3, line 89-90: A reference would add to the possibility of reviewing the argumentation. See comment and references above relating to the mitigation potential of carbon sequestration in agricultural soils, in particular grasslands that form the basis for dairy production.</p> <p>Page 3, table 1: Dietary oil is a vague concept. A reference to the carbon footprint of palm oil would be beneficial to confirm that this is true for all kinds of vegetable oil.</p> <p>Page 5, line 174-175: The vegetable feed in the case of farmed fish is often soy. This aspect of farmed fish needs further discussion.</p> <p>Page 7, table 2: What scientific evidence is the content of table 2 based on?</p> <p>Page 7, table 2: Please refer to relevant literature.</p> <p>Page 7, table 2: “Dairy” is a nutritionally heterogenous group. Please define more closely which dairy products are meant.</p> <p>Page 7, table 2, last three rows, first column (“Change in consumption”): in what direction should these food groups be changed?</p> <p>Page 7, table 2: The suggestions regarding change in consumption of potatoes and dairy does not agree with the conclusions in the NNR 2012 chapter on energy.</p>	<p>This is a summary. Reference to studies mentioned in text.</p> <p>This is a summary. Reference to studies mentioned in text.</p> <p>Add sequestration – if possible</p> <p>Reference FAO Lifestock’s long shadow</p> <p>More details are added.</p> <p>Good point.</p> <p>Summary from several LCA analysis, national studies and food surveys. Done. Table 2</p> <p>Done.</p> <p>NNR doesn’t consider climate impact and other environmental impacts.</p>

General and detailed comments	WG comments and actions
<p>References</p> <p>Smedman, Lindmark-Månsson, Drewnowski and Modin Edman. Nutrient density of beverages in relation to climate impact. Food and nutrition research 2010; 54: 5170.</p> <p>Schulze et al. Importance of methane and nitrous oxide for Europe's terrestrial greenhouse-gas balance. Nature Geoscience 2009; 2:842-850.</p> <p>Soussana et al. Full accounting of the greenhouse gas (CO₂, N₂O, CH₄) budget of nine European grassland sites. Agriculture, Ecosystems and Environment 2007; 121: 121-134.</p> <p>Cederberg, C., Landquist, B. and Berglund, M. Potentialer för jordbruket som kolsänka. SIK-Rapport Nr 850, 2012.</p> <p>Buttriss, J. & Riley, H. Sustainable diets: Harnessing the nutrition agenda. Food Chemistry 2013. http://dx.doi.org/10.1016/j.foodchem.2013.01.083</p> <p>Vieux, F., Darmon, N., Touazi, D. & Soler, L.G. Greenhouse gas emissions of self-selected individual diets in France: Changing the diet structure or consuming less? Ecological Economics 2012; 75: 91–101</p> <p>Katarina Nilsson, K., Sund, V & Florén, B. The environmental impact of the consumption of sweets, crisps and soft drinks. TemaNord 2011:509.</p> <hr/> <p>Raisio Nutrition Ltd, Finland</p> <p>General</p> <p>Row 181-186: This paragraph gives a misleading impression that butter is/could be replaced by palm oil. This is not the case in practice. Butter should be compared with margarine to avoid misunderstanding, since palm oil is used only as an ingredient in foods in Nordic countries.</p> <p>Row 181: Butter is generally not recommended in a nutritionally adequate diet due to its' high content of saturated fatty acids. If there is a surplus of milk fat produced along the food chain, new innovative solutions could also be found for its utilization.</p> <p>Row 222-224 (Table 2 Less Dairy comments): If the nutritional aim is to decrease the intake of saturated fatty acids in the diet , it is confusing to suggest that one should decrease the intake of saturated fatty acids from some foods but increase from others.</p>	<hr/> <p>Section revised</p> <p>Good, any examples of new innovative solutions</p> <p>Point taken that is not clear enough. Has been rewritten.</p>
<p>MIFU – The Association of Danish Margarine Manufacturers and Importers</p> <p>General</p> <p>MIFU fully supports the comments made by Unilever. It is very important to have a clear distinction between sustainability and nutrition, especially regarding fats and the importance of alignment with the "fat-chapter". Also we would like to draw your attention to the Danish "CO₂ Pyramid"</p>	<p>Danish pyramid interesting.</p>

General and detailed comments	WG comments and actions
www.vb.dtu.dk/download.php?fileID=191	
<p>Valio Ltd. General</p> <p>In general, a comment to the suggested decrease of dairy: Consuming more than three servings of dairy per day leads to better nutrient status and improved bone health and is associated with lower blood pressure, reduced risk of cardiovascular disease and type 2 diabetes. For these reasons, decrease in the consumption of milk and dairy should not be encouraged.</p> <p>Examples of references: Rice et al. Meeting and exceeding dairy recommendations: effects of dairy consumption on nutrient intakes and risk of chronic disease. <i>Nutr Rev</i> 2013;71:209–223. Peter C. Elwood et al. The Consumption of Milk and Dairy Foods and the Incidence of Vascular Disease and Diabetes: An Overview of the Evidence. <i>Lipids</i> 2010;45:925–939. Frédéric Fumeron et al. The Data from the Epidemiological Study on the Insulin Resistance Syndrome (DESIR) Study Group. Dairy Consumption and the Incidence of Hyperglycemia and the Metabolic Syndrome. <i>Diabetes Care</i> 2011;34:813–817. Marianne Hauge Wennersberg et al. Dairy products and metabolic effects in overweight men and women: results from a 6-mo intervention study. <i>Am J Clin Nutr</i> 2009;90:960–968. Vasanti S Malik et al. Adolescent dairy product consumption and risk of type 2 diabetes in middle-aged women. <i>Am J Clin Nutr</i> 2011;94:854–61</p> <p>Details</p> <p>Row 77: All food production brings about varying environmental cost. However, it is important to consider this cost in the context of the nutritional value provided by the food. Available research, although still relatively new and complex, shows that milk scores significantly better than many other beverages in nutrient density to climate impact and that changing the structure of human diets by reducing the consumption of animal-based products is not a sufficient approach to significantly reducing emissions. It should be borne in mind that it is not merely an issue of greenhouse gas emissions but also of the nutritional density of food products. (Smedman A et al. Nutrient density of beverages in relation to climate impact. <i>Food & Nutrition Research</i> 2010;54:5170.) Animal foods, including dairy products, not only are excellent sources of many micronutrients but contain few components that inhibit the bioavailability of the nutrients they provide. In particular, phytates and polyphenols, found in plant foods, are two compounds that negatively impact the bioavailability of certain micronutrients. In populations with primarily plant-based diets, common in low-</p>	

General and detailed comments	WG comments and actions
<p>income countries, these compounds can have a major negative nutritional impact on nutrient adequacy. (Michaelsen KF et al. Choice of foods and ingredients for moderately malnourished children 6 months to 5 years of age. Food Nutr Bull 2009;30(3 Suppl):S343-404.)</p> <p>Row 112, Table 1 It is not sensible to present climate impact per kilo edible product: cheese, for instance, is nutritionally extremely dense food stuff for a very broad range of nutrients. The climate impact for cheese is the same as for milk when the nutrient content is taken into account.</p> <p>Row 223 Table 2: Row less Dairy: The consumption of milk and dairy does not exceed the recommended level of current national recommendations. With less milk than that, it is difficult to meet the recommendation on calcium (and vitamin D in countries where milk is enriched with vitamin D) with more sustainable products. When compared to the nutrient density of beverages, the climate impact of milk is substantially better than of the other beverages studied, incl. soy and oat drink (Smedman A et al. Nutrient density of beverages in relation to climate impact. Food & Nutrition Research, 54: 5170, 2010). Could it be here rather "maintain the present consumption level" than "less dairy"? The "Comments" column: The intake of many essential nutrients decreases, for instance high quality protein (all essential amino acids), potassium, magnesium, vitamin B12, zinc, selenium, folate, riboflavin, niacin equivalents, iodine and vitamin A (vitamin D).</p>	<p>The issue was brought up in text: protein vs climate impact. Important point.</p> <p>Table is rewritten.</p>
<p>Private person. General</p> <p>The chapter is very generalized and needs extensive editing. - There is almost no information or recommendation regarding food waste. Sustainable consumption is much more than the CO2 that was used to produce it. Making the consumer buy food products with low emissions is one thing, but what happens with the food in the household (processing, throwing away because of best before date, throwing away unconsumed cooked food) is a major contributor to sustainable consumption. This can not be ignored!!</p> <p>- A lot of the data that is presented in this chapter is very limited to one country, or comes from sources that are not peer-reviewed papers. In general, the amount of data is low and not specified to Nordic countries. There is a large difference between countries in how food is produced, bought and consumed and this needs to be taken into account.</p> <p>- Overall, the chapter focuses on reducing animal protein. While many people in the Nordic countries may eat too much of this according to nutritional guidelines, this can not be so generalized. If all people would suddenly start eating a vegan diet, many people would not know how to eat properly, and run into nutritional</p>	<p>Waste – more emphasis is added.</p> <p>Needs to clarify this is an exploratory report. It is not about excluding animal protein, it is about decreasing the amount.</p>

General and detailed comments	WG comments and actions
<p>problems with a lack of advice. Just eating more plants isn't enough: any vegan can tell you that it takes planning, preparation and going to specialty stores to eat healthy as a vegan. - The text itself needs a lot of work. There are references missing or incomplete and the English needs work (Experiences in the title of the section starting on page 5 needs to be Experiences). In Table 2 the wording is inconsistent: one can read that one has to eat more organic, more peas, etc, but once one comes to the water, the recommendations aren't clear. It reads as if we should drink more alcohol, yet fails to mention effects of overconsumption.</p>	<p>Table 2 rewritten</p>
<p>SLU – Swedish Agricultural University, Cecilia Sundberg, Department for Energy and Technology General</p> <p>The title “sustainable food consumption” requires a wider discussion of environmental impacts. The text is too focused on climate impact. The description of environmental impact should give a fuller picture of the relation between the environment and nutrition. The focus on climate in recent years is important, but should not be used as an excuse for omitting other important environmental aspects of food. The text should be strengthened regarding eutrophication. Agriculture and wastewater, the beginning and end of food, together account for most of the emissions of N and P to water, which is an enormous challenge to drinking water and aquatic ecosystems all over the world, including Sweden. See any report from the Swedish EPA on eutrophication. The description on environmental impact should include some environmental impacts that have a direct health effect. In addition to dioxins, mercury (Hg) and cadmium (Cd) should be covered. These are toxic metals that are available in concentrations in food that have a direct negative health effect on some groups of the population (http://www.kemi.se/sv/Innehall/Nyheter/Benbrott-for-miljardrar-fran-kadmium-i-mat/) http://www.slv.se/grupp1/Mat-och-naring/Kostrad/Rad-om-fisk/#fororen .</p>	<p>Acknowledged, but hasn't been possible to add all.</p>
<p>MTK ry – Centralförbundet för lant- och skogsbruksproducenter, Finland. General</p> <p>“Those who choose a diet with less meat and dairy products have a further lower climate impact.” A dairy product does not mean necessary more GHG's as ruminants are animals that can transfer herbaceous biomass (grasses) to protein. Grasses cannot be used for food without ruminants. Grasses are good carbon sinks and protect waters by controlling erosion. These facts need to be included when ruminants (beef and milk products) are considered. “With a general vegan diet it is possible to half the climate impact from what we eat. “should thus be thus reviewed. Emissions of CH₄ are tied to production of meat, milk and rice. CH₄ is produced when organic material decomposes in anaerobic conditions such as in digestion by ruminant livestock, from stored manures and from rice grown under flooded conditions. In milk and beef production methane emissions come from grasses, and carbon sink by grasses need to be considered when total impact is evaluated. Regarding flooded soils it is crucial to enhance good drainage management in agricultural land</p>	<p>This is a general description of measures to be done in agriculture</p>

General and detailed comments	WG comments and actions
<p>under humid conditions. Under good drainage conditions, methane and also nitrogen gases are minor. Good yields from well aerated soils mean fewer emissions. Regarding manure, there is technology to reduce methane emission. New technology in manure management and soil application should be highlighted more. Emissions of N₂O are tied to any crop and to animal products. Nitrous oxide is generated by the microbial transformation of nitrogen in soils and manures. Emissions occur also during the production of synthetic fertilizers. Regarding N₂O emissions it is crucial that soil has good aeration and water supply for agricultural crops. When soil is growing well, the emissions are less. Modern subsurface placement technology of inorganic fertilizers and manures decrease the emissions from soils. Also fertilizer production technology has improved considerable last 5 years. The environmental impact is a complex web, where each factor is supporting each other in an upward or a downward spiral. This is very true and the weather conditions affect a lot the impacts of agriculture, as well as the modern technology.</p> <p>Details</p> <p>“Those who choose a diet with less meat and dairy products have a further lower climate impact.” A dairy product does not mean necessary more GHG’s as ruminants are animals that can transferee herbaceous biomass (grasses) to protein. Grasses cannot be used for food without ruminants. Grasses are good carbon sinks and protect waters by controlling erosion. These facts need to be included when ruminants (beef and milk products) are considered. “With a general vegan diet it is possible to half the climate impact from what we eat. “should thus be thus reviewed. Emissions of CH₄ are tied to production of meat, milk and rice. CH₄ is produced when organic material decomposes in anaerobic conditions such as in digestion by ruminant livestock, from stored manures and from rice grown under flooded conditions. In milk and beef production methane emissions come from grasses, and carbon sink by grasses need to considered when total impact is evaluated. Regarding flooded soils it is crucial to enhance good drainage management in agricultural land under humid conditions. Under good drainage conditions, methane and also nitrogen gases are minor. Good yields from well aerated soils mean fewer emissions. Regarding manure, there is technology to reduce methane emission. New technology in manure management and soil application should be highlighted more. Emissions of N₂O are tied to any crop and to animal products. Nitrous oxide is generated by the microbial transformation of nitrogen in soils and manures. Emissions occur also during the production of synthetic fertilizers. Regarding N₂O emissions it is crucial that soil has good aeration and water supply for agricultural crops. When soil is growing well, the emissions are less. Modern subsurface placement technology of inorganic fertilizers and manures decrease the emissions from soils. Also fertilizer production technology has improved considerable last 5 years. The environmental impact is a complex web, where each factor is supporting each other in an upward or a downward spiral. This is very true and the weather conditions affect a lot the impacts of agriculture, as well as the modern technology.</p>	<p>To highlight this new technology will be too detailed in this short chapter.</p> <p>This section is the same as “General”</p>

General and detailed comments	WG comments and actions
<p>NFE - Norsk Forening for Ernæringsfysiologer.</p> <p>General</p> <p>Norsk forening for Ernæringsfysiologer (NFE) appreciate the opportunity to comment on the chapter “Sustainable food consumption” in NNR5. With a growing population the demand for food is rising, and food production challenges the environment. NFE strongly supports that sustainability in food choices is addressed in the official nutrition recommendations.</p> <p>Details</p> <p>We would like the following comments to be considered: Reducing food waste is probably one of the most effective ways the consumer can help reducing environmental impact from food production. A reduced food waste will result in less demand for as well as a lower food production. How to achieve this should be stated more clearly in the chapter. Also, excess intake of calories is a challenge to public health, in addition to causing an unnecessary burden on the climate. A recommendation to strive for adequate calorie intake should be stressed more highly. The term "less" used in e.g. Table 2 is ambiguous and need to be clarified when used in recommendations to alter intake of food groups that bring important nutrients to the diet. Meat and dairy are important sources of iron, calcium, iodine and vitamin B12 in the Norwegian diet. It should be assessed how the recommendations may challenge the intake of these nutrients.</p> <p>The paragraph “Conflict areas of a sustainable diet and health” (line 165-186) addresses two major areas of conflict areas and a paragraph describing the “Nutritional benefits of a sustainable diet” is included (line 116-162). However, we miss a paragraph describing the “Nutritional challenges of a sustainable diet”. The paragraph should e.g. address challenges in meeting the nutritional demands for calcium and iron with a sustainable diet and how to meet these challenges. In the draft this is only briefly commented, but should be elaborated both in the text as well in a separate column in Table 2.</p> <p>Further, we have some suggestions to revise two paragraphs in the chapter “Conflict areas of a sustainable diet and health”. Firstly, in the second paragraph, nothing is mentioned regarding fish and the method of capture. This should be outlined, as the fishing method greatly influences the sustainability of a fish product. We also recommend including some guidelines on what fish/seafood that is considered a sound choice with regards to sustainability. Secondly, in the last paragraph, butter is mentioned in an ambiguous matter. It is first said that “Butter is generally not recommended in a nutritionally adequate diet due to its’ high content of saturated fatty acids”, but towards the end of the paragraph it is said that “ it may be worth to re-evaluate the role of butter in the diet “ based on the notion that palm oil and butter contains approximately the same amount of saturated fatty acids. We suggest excluding or at least revising this paragraph, as butter and palm oil both generally are considered being unhealthy food choices. Instead, it would be more appropriate to</p>	<p>Food waste has been expanded.</p> <p>Suggested paragraph. The contents of this is partly discussed in the referred studies where diets that meet nutritional demands and simultaneously have lower climate impact. Topic FISH. To mention fishing methods.</p> <p>Topic BUTTER. This para is revised.</p> <p>Topic Margarine is lacking.</p>

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<p>suggest different plant oils that are good substitutes for butter and palm oil both with regards to health and sustainability. An also; what edible fat is recommended in a sustainable diet: nothing is said about e.g. margarine. In Table 1 fish is mentioned both in the column for “medium” and “high” climate impact, which is confusing. If this is due to differences regarding how the fish is caught or whether it is wild type or farmed; it should be more clearly stated. We find Table 2 quite unclear. We recommend that the column headings in Table 2 should be revised, as they come across as quite confusing. The headings are not consistent; some places it is stated “less”, other “more”, but it is confusing when neither is stated; e.g. with “dietary fats and oils”. As previously mentioned, we also recommend a separate column in this Table, describing “Nutritional challenges of a sustainable diet”. Additionally, we do not understand the basis for describing “palm oil” separately; as it is it seems more logical to include this under “dietary fats and oils”.</p> <p>When searching after literature in the databases Medline and Embase with keyword “Sustainable diet” and combined keywords “Sustainable diet AND nutrition”, 125 and 41 hits, respectively, were found. Several of these seem to be relevant for this NNR chapter. A new literature search should be conducted, to ensure all relevant new literature to be included. Also, references are missing after numerous statements. Finally, we find the language used in this chapter being not “up to par”, both grammatically and academically. We strongly suggest that the whole text should be proofread in a thorough manner before printing.</p>	<p>Table 1, there are many fishes, now they are exemplified. Table 2 has been revised.</p> <p>Palm oil was treated separately due to it’s extraordinary efficiency but also it’s detrimental effect on forest and social societies.</p> <p>Search: Same search was done, only a few references were relevant.</p> <p>Language: all chapters will be language checked.</p>
<p>TINE SA, Norway General</p> <p>Production of food impacts the environment, and TINE recognizes the responsibility that the food sector has in this important matter. We appreciate the opportunity to comment on the chapter “Sustainable food consumption” in NNR5. The demand for food is rising, and the impact that the production of it has on the environment calls for swift action. At the same time, one must not loose of sight the fact that the primary aim of food is to meet the individuals need for nutrients. Official nutrition recommendations for the Nordic countries is an important tool on which national recommendations are built. They have a potentially strong impact on the food choices of individuals, and is thus of major importance for the food industry. Sustainability in food and dietary patterns is a relatively new field of research, and there is restricted scientific research available upon which to build recommendations. Nonetheless, given the impact that such recommendations have for both individuals (nutrient intake) and for food industry, it is of utmost importance that the recommendations are well scientifically substantiated. In order to make the process more transparent and the outcome predictable for the industry, we ask for the selection criteria for studies and how these are weighted in the conclusion to be included in the document. Moreover we propose that the fact that there is a restricted amount of research on the field is discussed in the paper, and that this fact is reflected in the conclusions.</p>	<p>The focus is on how nutritionally adequate diets can be sustainable</p> <p>Is does not aim to give recommendations, it is an exploratory chapter.</p> <p>Waste reduction in all food chain may reduce climate impact substantially, but</p>

General and detailed comments	WG comments and actions
<p>Reducing food waste is by far the most effective way to reduce environmental impact from food production. As written, the text gives the impression that eating less or more of certain food items is the most important way for the consumer to contribute to a more sustainable diet. Thus, the importance of food waste should be pointed out more strongly. Also, studies indicate that restricting calorie consumption to adequate intake would be more effective than changing dietary structure(1). Given the uncontroversial nature and important environmental implication of these matters they should be given a more prominent focus in the discussion and conclusion. Overall impression from reading the chapter is that consumption of dairy products should be reduced. Apart from high quality protein, milk and other dairy foods contribute with calcium, iodine, vitamin B12, riboflavin and other essential nutrients to the diet. In Norway, dairy contributes with approximately two thirds of dietary calcium (Norkost 3), and 60 % of iodine. A recently launched study on iodine intake in pregnant woman concludes that public health strategies are needed to improve and secure the iodine status of pregnant women in Norway (2). While nutritionally adequate dairy-reduced diets are achievable, they can be difficult, particularly for children. Modeling analyses from the US (3) show that when dairy products are excluded, calcium, magnesium, phosphorus and other nutrients drop below 100 % of goals in most food patterns. Moreover, The United States 2010 Dietary Guidelines Advisory Committee points to the fact that “the amount of many potential alternatives to provide sufficient calcium would provide too many calories and/or be a large amount to consume daily.” Although conclusions from studies on dietary patterns in the US are not necessarily transferable to the Nordic countries they indicate that recommendations to reduce dairy intake, without further clarification (as fx. to what levels) should be considered with care.</p> <p>Details</p> <p>Conclusions:</p> <p>Line 23 “Those who choose a diet with less meat and dairy products have a further lower climate impact”. TINE cannot find that the text substantiates that those who eat less dairy have a lower climate impact. Moreover, the word “less” is ambiguous and could refer either to a lower intake than recommended, a lower intake than the individual’s current intake, or lower than mean intake. As this conclusion very likely would be interpreted as a recommendation for individuals to lower their intake of dairy we strongly ask for an assessment how this recommendation will impact the intake of calcium, iodine, riboflavin, vitamin B12 and</p>	<p>additionally food choices are equally is important. Waste is pointed out stronger.</p> <p>Restricted or adequate food intake is still another point to add to the report. Other studies have shown that a population being 40 % obese requires 19% more food energy for its total expenditure (Edwards P & Roberts I 2009). Reference 1 did not discuss food choices within food groups.</p> <p>The chapter suggests less dairy, not no dairy. Calcium from dairy can be compensated by a higher consumption of field vegetables containing 50-150 mg Ca per 100 g) and legumes (containing 40-80 mg Ca per 100 g cooked) and herring and other fish and shellfish – the intake increases. The consumption of these food groups are recommended to be increased to reach a healthier diet. Iodine is high in many sustainable and safe fishes for pregnant women, especially cod. Iodine from milk and dairy is via fodder.</p> <p>Suggested level of intake: It all depends on the pattern of eating and all other components.</p> <p>The text refers to lower than the mean intake. The nutritional impact from consuming less dairy will be of a concern for Ca and Iodine, but riboflavin and B12 is no problem. However it depends on what we eat instead. Sustainable food stuffs like green cabbage, cooked legumes, spinach, broccoli, Brussel sprouts contain 50 mg Ca and more per 100 g (milk has double) and can compensate. Fish is rich in Iodine. A recent scenario concerning a</p>

General and detailed comments	WG comments and actions
<p>other nutrients to which dairy is a unique source. We would like to draw attention to the LiveWell for Life report (4) recently launched by WWF. The report demonstrates that, in each of these countries, it is possible to develop a model diet which decreases GHGe by 25 % from the current average, costs no more, meet nutritional guidelines and still resembles current diets sufficiently to be widely and easily accepted. In all the model diets consumption of dairy products was kept similar to now. Although not peer-reviewed, this report substantiates the need for the advice to consume less dairy that comes out of this report should be given more concern. In table 2 it is stated that “combined meat and milk production is more efficient than only meat production”. This is the prevalent way of milk- and beef production in Norway, contributing to a lower environmental impact both from dairy and beef than what could be concluded from the numbers displayed in the document. As NNR5 should seek to encompass the food production systems that are most relevant in the countries they apply for we propose that this fact should be given more emphasis in the document, and that the implications should be mirrored in the conclusion.</p> <p>Line 117 “It is shown that no food group affects the environment through the production as much as meat and dairy (FAO 2006a).” The theoretical basis for this statement should reflect that there has been made a follow-up research report by the FAO in collaboration with IDF (International Dairy Federation) in 2010 (5). This report, which has a higher level of precision than the initial report of 2006, shows that dairy products alone account for a limited amount of GHG emissions: 2.7% for dairy in contrast to 18% for the whole livestock sector. This new knowledge should be reflected in the recommendations. This also indicates that dairy products not necessarily should be referred to in the same context as meat when it comes to levels of environmental impact. Environmental impact can be divided in two main dimensions: emission of pollutants and resource usage. The latter should also be given thorough attention: to feed a growing population, it is crucial that resources are utilized in the most efficient way, and that all nations seek to make use of available national resources. 3 % of Norway’s total area is agricultural land, whereof 2/3 are only suited to cultivate grass (6). To be able to utilize this scarce resource one is dependent on ruminants. Not utilizing this resource would be irresponsible in a world where the demand for food is increasing.</p> <p>Table 1 For the purpose of dietary planning and recommendations we propose to use another functional unit for comparison of CO2e from foods than kg. As present, table 1 could give the impression that it is meaningful to choose foods on the basis of how much CO2 is emitted per kg produce. Comparison by weight is not suitable for foods that serve different purposes in the diet and are eaten in very different amounts. We ask for a more relevant functional unit to be used, that in a better way takes in to account the foods ability to meet dietary needs (fex per calories, protein or other essential nutrients). Work by Briseid et al show how CO2e associated with production of foods change when calculated on the basis of MJ instead of kg (6). In 2010</p>	<p>7 day sustainable menu resulted in half the present climate impact when almost 200 ml milk was included per day and 4 g cheese.</p> <p>less climate impact published FR LiveWell menu increased dairy products with 12 %, Spain’s LiveWell menu contained 67 % of current milk, dairy and milk substitutes, Sweden’s LiveWell meny contined 84% of current milk and cheese consumption. In Sweden only 50-60% of beef comes from milk-meat production.</p> <p>Correction: the global milk production, processing and transportation to total anthropogenic emissions is estimated at 2,7 %. The global dairy sector contributes 4,0 % to the total global anthropogenic GHG emissions</p> <p>It is not easy to separate milk and beef production. There is a strong element of positive and negative environmental impact to weigh together in beef/milk production.</p> <p>Another functional unit is very valuable to use. One example in line 123 to 126. This could be expanded to key nutrients. Smedman’s report is a valuable contribution to tackle this task.</p>

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<p>Smedman et al introduced the concept of GHGe associated with production in relation to nutrient density (7). Such an approach would also address the problem that sweets, snacks and other products that bring practically nothing but calories to the diet stand out to be better choices than staple foods that are associated with higher CO₂e per kg. In a society where overconsumption and excess intake of calories is a major health challenge one of the most uncontroversial ways to reduce CO₂e would be to cut the intake of “empty calories”, regardless of whether the emission associated to production are high or low. Furthermore, in table 1 milk and yoghurt are classified as a medium impact products together with e.g. rice, fish and fruits. In the same table meat and butter are classified as high impact products. We believe that this implies that the use of the term “meat and dairy” throughout the document is somehow misleading as it could give the impression that dairy products in general have the equal environmental impact as meat, which is clearly not the case. This is also reflected in the updated report from FAO and IDF (5).</p> <p>Line 197 – 203 The paragraph describes what theoretically can be achieved by manipulating the diet in different ways. Single model studies are used as basis. In this context it would be relevant to address the question what happens in a real life situation if one reduces one component in the diet. This is also raised in the conclusion (line 21). We are not aware of studies from the Nordic countries on this field but would again like to draw the attention to the Vieux’ study “Greenhouse gas emissions of self-selected individual diets in France: Changing the diet structure or consuming less?” (1). The study indicates that cutting meat intake could lead to a higher GHG output from the total diet, due to intake of a larger volume of other foods to compensate for the calories. Table 2 Table 2 gives an overview of suggested dietary changes to reach a sustainable diet. In line with the above, TINE is of the opinion that optimization of calorie intake is the most powerful and uncontroversial way to reduce environmental impact of food. In line with the conclusion in the before mentioned Vieux’ study the impact of different food patterns is negligible compared to the effect of reducing intake to meet nutritional needs. We are of the opinion that total calorie intake and the importance of optimization should be put in context with the other suggested changes in consumption. Given the extreme potential of reducing food waste on GHG emissions, we propose that this should be emphasized more highly, either in connection with table 2 or elsewhere in the document.</p> <p>References:</p> <p>(1) Vieux et al. Greenhouse gas emissions of self-selected individual diets in France: Changing the diet structure or consuming less? Ecological Economics Volume 75, March 2012, s 91–101 http://www.sciencedirect.com/science/article/pii/S0921800912000043</p> <p>(2) Brantsæter et al. Risk of Suboptimal Iodine Intake in Pregnant Norwegian Women Nutrients 2013, 5, 424-440</p> <p>(3) Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010.: US</p>	<p>Still we cannot produce milk without raising cattle. The climate impact of meat and beef depends on how the allocation between the two is done. It could be done on a volume basis, protein basis or economical basis with different results.</p> <p>There are several European and Swedish studies “manipulating” existing consumption data. These will be presented in the report. Concerning Vieux, reference 1, her conclusion about fruits and vegetables compensating the withdrawn meat will not help to decrease the climate impact of the diet. However, it depends on what you compensate with. Preferably sustainable fruits and vegetables or roots, or even to leave out compensation, it’s energy intake exceeds requirement.</p> <p>Food Waste globally: A third of food production is thrown away in the food chain (FAO 2011). Distinguish between food losses and waste. High income countries have more waste in the later stages. 95-115 kg per capita food waste per year in Europe and North America. In SE calculated 1 million ton food waste of which 67 % in the household (SMED 2011). 65 % is unavoidable and 35 % is unnecessary . It is calculated that food chain emits 20 million ton CO₂e and food waste is estimated to account for 2</p>

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<p>Department of Agriculture, Center for Nutrition Policy and Promotion.;2010.</p> <p>(4) A balance of healthy and sustainable food choices for France, Spain and Sweden, 2013 WWF Live Well for LIFE report http://livewellforlife.eu/livewell-plate/reports</p> <p>(5) FAO and IDF, 2010. Greenhouse gas Emissions from the Dairy Sector: A Life Cycle assessment http://www.fao.org/agriculture/lead/themes0/climate/emissions/en/</p> <p>(6) Briseid et al., 2008: Klimagasser fra landbruket https://www.slf.dep.no/no/dokumenter/publikasjoner/_attachment/264?_ts=12824856ea0&download=true</p> <p>(7) Smedman et al. Nutrient density of beverages in relation to climate impact. Food & Nutrition Research 2010, 54</p>	<p>million ton CO2e, i.e 10 % of the food chain. If the unnecessary food waste is 35 % all through the food chain, then 3,5% of the emissions from the food chain comes from unnecessary food waste. Vegetables and bread are wasted the most, meat the least.</p>
<p>Danish Agriculture and Food Council General</p> <p>Danish Agriculture and Food Council appreciates the opportunity to comment on the draft chapter "Sustainable food consumption" in NNR5. In our opinion it should be considered carefully if the chapter on sustainable food production should be included in the NNR. The official nutrition recommendations are an important tool, and have a large impact on individuals, governments and business in the Nordic countries. For the recommendations to remain a strong tool they should be based on solid and conclusive science.</p> <p>Sustainability in food and food consumption is a developing science. Research is limited and non conclusive, and there is a lack of a commonly agreed methodology. Further, in our view, it is difficult from the draft chapter on sustainable food consumption to see the basis on which the editors have chosen one study from another and how the different conclusions in this still somehow scattered field of research have been weighted against each other. Further the chapter and its implicit recommendations to reduce intake of meat and dairy products suppresses in our view the positive role of meat and dairy in Nordic nutrition and for different groups of the population. If, however, the chapter on sustainable food consumption is kept in the publication we would propose that it includes a specific mentioning that background data of different food groups are limited and heterogenic, and are based on methods including different parts of the value chain (production, use and waste) of a food group - thereby making them in some cases non-comparable. Finally we would like to question the focus of the chapter to increase the sustainability of food consumption by changing dietary patterns. It will be more efficient for governments and individuals to focus the efforts where the largest potential gains are to be found; i.e. by promoting ways for households and governments to - reduce household food waste - reduce overconsumption - encourage to prioritize intake of nutrient-rich foods and avoid overconsumption of sweets and savory, to ensure nutrient requirements are met - choose food in season</p>	<p>Dietary changes are needed in addition to suggested changes.</p> <p>Food in season! Good idea!</p>

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<p>Details</p> <p>Page 1, line 19: Many public health scientists would agree that a largely, not solely, plant based diet is preferable as long as consumers are able to fulfill daily requirements for all essential nutrients with this diet.</p> <p>Page 1, line 23-24: Relevant references should be added to the statement that argue that those who choose a diet with less meat and dairy products have a further lower climate impact.</p> <p>Page 2, Table 1: Table 1 simply considers climate impact of different specific foodstuffs, and ranges them into groups as either low, medium or high CO2e values per kg edible weight. Unfortunately, this table does not consider the nutrient density and contribution of essential nutrients. A Swedish study (Smedman et al., 2010) shows the need to take both greenhouse-gas emission and nutrient density of foods and beverages into account when calculating climate impact. This study estimated nutrient density of selected beverages in relation to climate impact, using the Nutrient Density to Climate Impact (NDCI) index. The higher NDCI index, the better. They concluded that due to a very high-nutrient density, the NDCI index for milk was considerably higher than for the other beverages.</p> <p>Page 2, Table 1: Furthermore, table 1 gives an impression of sugar being a better option than for instance milk and yoghurt. While sugar contributes with no essential nutrients in the diet, milk and yoghurt contributes with a range of nutrients, such as protein, calcium, riboflavin, vitamin B12, iodine and phosphorus.</p> <p>Page 7, Table 2: Table 2 suggests “Less Dairy” as a dietary change to reach a sustainable diet. It is not defined what “Less Dairy” refers to. In the Norwegian dietary guidelines “Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer” it is recommended to eat low-fat dairy products on a daily basis. Therefore, a definition of what “Less Dairy” means is needed.</p> <p>Page 7, Table 2: A definition of which dairy products that is included in “Dairy” is also essential, since dairy is a nutritionally heterogonous group.</p> <p>Page 7, Table 2: As it is argued in table 2 it can be difficult to meet the calcium recommendations without milk. This statement should also include others dairy products. In a Norwegian national nutrition survey (Norkost 3) from 2012 it is shown that 67 percent of the population’s intake of calcium was due to dairy. If one should claim that a sustainable diet includes “Less Dairy”, it is necessary to demonstrate how one could achieve the daily intake of calcium with less dairy and with more sustainable alternatives. Besides, milk and dairy also contain other nutrients which need to be provided from other, more sustainable food groups.</p> <p>References: Nasjonalt råd for ernæring. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer: metodologi</p>	<p>Largely – is added.</p> <p>These are presented in the chapter.</p> <p>True. Climate impact is the first step to present the differences between food stuffs. In the report, line 123-126 , an additional calculation based on protein is presented. Smedman’s report is an example how to embrace several nutritional aspects and climate impact in creating an index.</p> <p>Food items are eaten in different amounts. 100 ml of milk is easily ingested but 100 g of sugar, oil or cheese is not. This is a crude list dividing food items into three different groups.</p> <p>Agree. Less than the average consumption. This will be changed in the report.</p> <p>The group has been divided into milk and cheese</p> <p>The most crucial nutrients from dairy are Ca and iodine. Many of the other nutrients are plentiful in other food stuffs. B12 is not a problem to reach. Iron recommendations will probably be easier to meet without too much dairy. The suggestion is not no dairy, only less in comparison to average intake. A scenario shows a diet with less climate impact but fulfilling the nutritional recommendations and food based dietary advices choosing</p>

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<p>og vitenskapelig kunnskapsgrunnlag. Oslo: Helsedirektoratet, 2011.</p> <p>Norwegian Directorate of Health (Helsedirektoratet). Norkost 3. En landsomfattende kostholdsundersøkelse blant menn og kvinner i alderen 18–70 år, 2010-11. Oslo: Helsedirektoratet, 2012.</p> <p>Smedman, Lindmark-Månsson, Drewnowski and Modin Edman. Nutrient density of beverages in relation to climate impact. Food and nutrition research, 2010; 54: 5170.</p>	<p>sustainable food stuffs (Jordbruksverket, Naturvårdsverket och Livsmedelsverket 2013)</p>
<p>Norwegian Dairy Council General</p> <p>Comments in this paper are given by Heidi Kathrine Ruud, clinical dietitian (RD), and Ingvild Steen Nøding, public health nutritionist on behalf of the Norwegian Dairy Council (Melk.no). Climate changes are regarded as a vast challenge of our time. Hence, including a chapter about sustainable food consumption in Nordic Nutrient Recommendations is relevant. Still, sustainable food consumption is a complex issue. Therefore, it is important that the aim of this chapter is clearly defined. Methods and criteria for studies and analyses selected as references should be discussed and clearly referenced. Food waste and overconsumption are important aspects when discussing sustainable food consumption. There is a need of reducing food waste worldwide, including the Nordic countries. Overweight and obesity have reached epidemic proportions globally, and this results in enormous health problems also in the Nordic countries. Since the fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended, it is important to address overconsumption in relation to both health and sustainable food consumption.</p> <p>Detalis</p> <p>Page 1, line 19: Many public health scientists would agree that a largely, not solely, plant based diet is preferable as long as consumers are able to fulfill daily requirements for all essential nutrients with this diet.</p> <p>Page 1, line 23-24: Relevant references should be added to the statement that argue that those who choose a diet with less meat and dairy products have a further lower climate impact.</p> <p>Page 2, Table 1: Table 1 simply considers climate impact of different specific foodstuffs, and ranges them into groups as either low, medium or high CO2e values per kg edible weight. Unfortunately, this table does not consider the nutrient density and contribution of essential nutrients. A Swedish study (Smedman et al., 2010) shows the need to take both greenhouse-gas emission and nutrient density of foods and beverages into account when calculating climate impact. This study estimated nutrient density of selected beverages in relation to climate impact, using the Nutrient Density to Climate Impact (NDCI) index. The higher NDCI index, the better. They concluded that due to a very high-nutrient density, the NDCI index for milk was considerably</p>	<p>Acknowledged.</p> <p>Food Waste and over consumption and obesity will be included.</p> <p>These are the same comments as provided by Danish Agriculture and Food Council.</p>

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<p>higher than for the other beverages.</p> <p>Page 2, Table 1: Furthermore, table 1 gives an impression of sugar being a better option than for instance milk and yoghurt. While sugar contributes with no essential nutrients in the diet, milk and yoghurt contributes with a range of nutrients, such as protein, calcium, riboflavin, vitamin B12, iodine and phosphorus.</p> <p>Page 7, Table 2: Table 2 suggests “Less Dairy” as a dietary change to reach a sustainable diet. It is not defined what “Less Dairy” refers to. In the Norwegian dietary guidelines “Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer” it is recommended to eat low-fat dairy products on a daily basis. Therefore, a definition of what “Less Dairy” means is needed.</p> <p>Page 7, Table 2: A definition of which dairy products that is included in “Dairy” is also essential, since dairy is a nutritionally heterogonous group.</p> <p>Page 7, Table 2: As it is argued in table 2 it can be difficult to meet the calcium recommendations without milk. This statement should also include others dairy products. In a Norwegian national nutrition survey (Norkost 3) from 2012 it is shown that 67 percent of the population’s intake of calcium was due to dairy. If one should claim that a sustainable diet includes “Less Dairy”, it is necessary to demonstrate how one could achieve the daily intake of calcium with less dairy and with more sustainable alternatives. Besides, milk and dairy also contain other nutrients which need to be provided from other, more sustainable food groups.</p> <p>References: Nasjonalt råd for ernæring. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer: metodologi og vitenskapelig kunnskapsgrunnlag. Oslo: Helsedirektoratet, 2011. Norwegian Directorate of Health (Helsedirektoratet). Norkost 3. En landsomfattende kostholdsundersøkelse blant menn og kvinner i alderen 18–70 år, 2010-11. Oslo: Helsedirektoratet, 2012. Smedman, Lindmark-Månsson, Drewnowski and Modin Edman. Nutrient density of beverages in relation to climate impact. Food and nutrition research, 2010; 54: 5170.</p>	
<p>Private person</p> <p>General</p> <p>We are pleased to find out there will be a chapter about sustainable food consumption in the new Nordic Nutrition Recommendations, however we urge that considerably more space should be given to this important topic. We suggest integrating sustainability in all chapters, where applicable. It is important that several environmental impacts are described in the chapter. Please add information about water use and acidification. The following Swedish paper reflects over different environmental impacts of meat production: Can carbon footprint serve as an indicator of the environmental impact of meat production? Rööf E, Sundberg C, Tidåker P, Strid I, Hansson P. Ecological Indicators 24 (2013) 573–581. http://dx.doi.org/10.1016/j.ecolind.2012.08.004</p>	<p>Several more environmental impacts could be discussed, but are not covered in chapter.</p>

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<p>We suggest that every food group should be discussed separately, both in texts and tables. Table 1 is great! Legumes should be separated from the vegetables and given more space in the chapter due to their nutritional and environmental benefits and the low consumption in the Nordic countries.</p> <p>Please take the following references about legumes/pulses into consideration: The nutritional value and health benefits of pulses in relation to obesity, diabetes, heart disease and cancer. Curran J. Br J Nutr. 2012 Aug; 108 Suppl 1:S1-2. doi: 10.1017/S0007114512003534. Pulse consumption in Canadian adults influences nutrient intakes. Mudryj AN, Yu N, Hartman TJ, Mitchell DC, Lawrence FR, Aukema HM. Br J Nutr. 2012 Aug;108 Suppl 1:S27-36. doi: . We would also like to see information about Fish Guides in the chapter (in Sweden for example WWF, Miljöstyvningsrådet, Naturskyddsföreningen), as well as information about certifications for fish (ASC and MSC). Keep up the good work!</p> <p>Detail Line number 125: We suggest using the term “legumes” instead of “pulses”. According to FAO definition the term “pulses” excludes legumes used for oil extraction (soybean, peanut) and those harvested green for food (green pea, green bean); the latter being classified as vegetables. http://www.fao.org/es/faodef/fdef04e.htm#4.01.</p> <p>Line number 157: Specify that this refers to heated greenhouses. Table 1: Fish is now both in medium and high, this need to be explained!</p> <p>All the text below refers to Table 2. Dairy: We agree with the suggested change in consumption (less dairy) and suggest that some alternatives to cow’s milk should be mentioned (oat drink, soy drink, almond drink, etc) and that it is important to choose products enriched with calcium and vitamin D.</p> <p>Fish and seafood: We urge not to recommend a higher consumption of fish and seafood (with the exception of mussels/clams, of which the consumption can increase). Of all fish populations worldwide 80 % are fully or overexploited. We suggest mentioning microalgae oil (algal-docosaheanoic acid) as a sustainable source of DHA, please see the two references below.</p> <p>Omega-3 fatty acids for nutrition and medicine: considering microalgae oil as a vegetarian source of EPA and DHA. Doughman SD, Krupanidhi S, Sanjeevi CB. Curr Diabetes Rev. 2007 Aug;3(3):198-203. Review. PMID:</p>	<p>Limited space to discuss in greater detail.</p> <p>High-light legumes! Added in final version.</p> <p>This is very national.</p> <p>Legumes will be the term.</p> <p>The fishes in the two categories are clarified.</p> <p>We will need climate data on the suggested drinks.</p> <p>Fish should be chosen according to recommended sustainability aspects.</p>

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<p>18220672 Clinical overview of algal-docosahexaenoic acid: effects on triglyceride levels and other cardiovascular risk factors. Ryan AS, Keske MA, Hoffman JP, Nelson EB. Am J Ther. 2009 Mar-Apr;16(2):183-92. doi: 10.1097/MJT.0b013e31817fe2be.</p> <p>We also suggest mentioning supplementation with vitamin D. We would like the Swedish National Food Agency (as well as the NFA:s in the other Nordic Countries) to evaluate the possibility to enrich additional foods with vitamin D, for example vegetable oils. Eggs are missing in the table. We suggest the recommendation to be to eat less eggs.</p>	<p>Supplementation is not within the scope of this task.</p> <p>Eggs are added to table 2</p>
<p>Confederation of the Danish Food Industry. General The Nordic Nutrition Recommendations put much emphasis on the scientific basic of the recommendations. Therefore, we recommend the chapter on sustainable food consumption should be taken out of the publication. First of all, we consider the knowledge base in the area too weak to guide a recommendation. We do not agree climate impact can be measured. In addition, we consider the approach taken to the concept of sustainability too narrow, and potentially misleading. Furthermore, we believe it is necessary to apply generally accepted international standards. Finally, we do not agree, climate impact can be communicated to the public in a meaningful way.</p> <p>Details</p> <ul style="list-style-type: none"> • Row 43-45: Any recommendations in the area of sustainable diets aligned with NNR should be based on a more comprehensive assessment of sustainability factors. The current evidence in the literature is focused almost exclusively on the link between diet and greenhouse gases and this illustrates the current lack of knowledge/understanding. XX supports the need for further research in this area so that more aspects than greenhouse gases are considered. • Row 92-93: To generally state that grazing animal helps biodiversity is overly simplistic and the effects are highly location and time-dependent. In the Nordics the link might be positive but this is not the case in many countries (e.g. beef production in Brazil) and we need to be aware of the global nature of many food chains. • Row 123-126: The text about the climate efficiency of protein production should be extended to include other dietary nutritional values of food. • Row 116-162: To fully understand the consequences of a shift in diet and the associated agricultural systems changes, a more explicit definition of sustainable diet is needed and what targets one want to achieve by 	<p>Expanded in final version.</p> <p>A wider and more comprehensive approach is wanted.</p> <p>We can't be comprehensive. Protein is an example.</p> <p>Sustainable diet definition is changed.</p>

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<p>promoting such a diet to the public.</p> <ul style="list-style-type: none"> • Row 181-186: It will be confusing when the scientific chapter on fat includes general recommendations to reduce SAFA and in the sustainability chapter of the NNR there is a comment to re-evaluate butter despite its high SAFA content. This shows discrepancy in scientific opinions in the NNR and opens the scientific papers for criticism of inconsistency, and will make the recommendations less convincing. • Row 184: The fat quality should be compared per fat content and not per 100 g product. Butter is a product with 80% fat content. Butter fat contains more saturated fatty acids (SAFA) than palm oil. Butter fat contains 64 % SAFA, and with around 3% trans fatty acids included it is as much as 67 % of total fat. Palm oil contains 47 % SAFA and no trans fatty acids (Swedish National Food Administration's data base). Based on these facts the suggestion to re-evaluate the role of butter in the diet has no support from nutritional point of view, and moreover it will also be very confusing for nutrition experts as well as consumers.(1) • Row 185-186: Considering the pressure that exists on the ecosystem for fish it might not be an option to have a further increase intake of fish, even if desired, to raise the intake of unsaturated fats. So a complement of unsaturated fat is needed from vegetable fat based products. • References (1) Nilsson et al, 2010 - DOI 10.1007/s11367-010-0220-3. 	<p>We do NOT recommend to eat over the limit, we talk about utilizing already existing saturated fatty acids instead of palm oil.</p> <p>Discussion has been altered.</p> <p>We may need to eat further down in the fish chain.</p>
<p>Stabburet, KLF, Nortura, Norway.</p> <p>General</p> <p>On behalf of the meat industry in Norway; Stabburet, Kjøtt- og fjørfebransjens landsforbund (KLF), Nortura, Opplysningskontoret for egg og kjøtt (Matprat), NHO Mat og Landbruk and Animalia (The Norwegian Meat and Poultry Research Centre), we appreciate the opportunity to comment on the chapter "Sustainable food consumption" in NNR5.</p> <p>Conclusions and recommendations: Our main recommendation for the draft is to omit the chapter from the NNR publication. If kept, then we highly recommend;</p> <ol style="list-style-type: none"> 1. to recognize that the background data on the different food groups is too limited and heterogenic, and that they are based on methods that include different parts of the value chain (production, use and waste) of the food groups, making them non-comparable, making it too difficult to form an acceptable body of scientific evidence on which to base advices 2. to mainly focus on health and sustainable efficient consumption by <ol style="list-style-type: none"> a. promoting reduced household food waste and overconsumption b. encouraging to prioritize intake of nutrient-rich foods and avoid overconsumption of sweets and 	<p>Overall good comments.</p> <p>These are mentioned</p>

General and detailed comments	WG comments and actions
<p>savory snacks, to ensure that nutrient requirements are met</p> <p>c. choosing food in season</p> <p>d. basing the diet on food produced in line with the natural resources in the region / country.</p> <p>General and detailed comments:</p> <p>In our opinion, a chapter on sustainable food consumption is not a natural part of a book regarding recommendations on intake of vitamins, minerals and other nutrients. The recommendations should concentrate on the body's need for nutrients, no matter how the delivering food is produced. Our main proposal to the editors is to consider omitting this chapter from the final publication. In the next sections we will elaborate why sustainability not should be a part of this book. The human need for nutrients is more or less consistent among the Nordic countries. In contrast, sustainability is a very complex issue, compiled of factors varying a lot both globally and within the Nordic countries. One aspect of sustainable food production is to use a country's nature-given resources for food production. The climate, soil and landscape vary between the Nordic countries, and this variation facilitates and limits the production of specific food items in the different countries. In a world with a high population growth, every country should exploit its own resources to contribute to adequate food supply in the world. This is also in accordance to recommendations in the newly published Nordic climate report (1) and the Norwegian white paper Velkommen til bords (2).</p> <p>Data from the UK shows that food production contributes 11 % of the emissions of GHG (3). Norwegian data on CO₂-emissions from households shows that the largest contributors to GHG emissions are transportation, shoes/clothes and chemicals (paints, detergents, fertilizers, pesticides, personal hygiene products). Out of the five categories food, clothes/shoes, furniture, residential and transportation, food amounted for roughly 5 % of the total CO₂ emission for a person through a lifespan (0-80 years of age) (4). To summarize, food is a minor contributor to the total CO₂ emission, and the contributions from the different food groups are even smaller pieces in the whole puzzle. It is not clear from the proposed text or the NNR website on what basis the included articles were selected and how they are weighted in the conclusion. The background data are not strong enough to support general advice on sustainability. To limit sustainable food production and particularly consumption to data regarding only greenhouse gas emissions, is the same as choosing one piece in a puzzle and ignore the totality. In addition to the factors mentioned in the opening of the chapter, factors like water, use of area, culture landscape, settling, animal welfare, use of fertilizers, etc., are of utmost importance. There are several Norwegian life cycle analyses (LCA) and other relevant studies that are not included in the chapter. An extensive LCA analysis on grain, milk and meat was recently completed. This is the most comprehensive study conducted in this research area in the Nordic countries and should be included (5). We therefore strongly suggest that the text elaborates on the data selection criteria, the fact that there are a</p>	<p>Refer to "Konsumtionens klimatpåverkan", Swedish Environmental Protection Agency. Food is a much larger part than the British reference indicates for UK. Swedish numbers included imported food.</p>

General and detailed comments	WG comments and actions
<p>lot of other factors affecting the sustainability of the diet beyond greenhouse gas emission, and includes Norwegian as well as separate data from each of the Nordic countries where available. It has not been possible to agree on a standard method of performing LCA analyses, even within Norway. Different research groups include different stages of the value chain when calculating the climate impacts of a food / food group. This increases the risk of comparing apples and pears when comparing results from different studies. Before drawing conclusions there is a need for more systematic LCA research. In addition, food production and the input factors to the models vary from region to region. For any one product, the environmental impact will depend on where, how and even when it is grown. In agriculture, practices are adapted to best suit climatic, geographical and market conditions and consequently, impacts measured in one region may not be applicable to another region. Impacts are also likely to vary over time as producers need to change their production system to suit seasonal conditions or market demand. Because of these reasons, the results from studies outside Norway need to be interpreted with great caution. Analysis of environmental effects of different foods and production methods (organic or conventional), shows that the conclusions are dependent on which factor the analysis is based on. Positive effects based on one factor, can be changed to negative effects when another background factor is chosen. Variations of emission between foods within the same food group are larger than between the different food groups. Given the complexity of food production systems, a multi-impact approach is recommended since mitigation of one impact can generate another impact (6). - Example: An ecological farm with mixed production of milk and beef has 5 % lower emissions of greenhouse gasses, lower terrestrial ecotoxicity, but needs 49 % more area to produce the same amount of food (5).</p> <p>(Line 195-196) Reports are estimating 30 – 50 % of all food produced to be wasted before consumption (7, 8, 9). Data from the Norwegian ForMat project indicated that we yearly waste more than 300 000 tons of food from households and grocery stores that could have been eaten (10). Household food waste represents the total loss of the product's nutritional value and the natural resources required throughout the entire production process, and consequentially, have a greater GHG impact than waste earlier in the production chain (11). The chapter per se is only briefly mentioning food wastage. According to The Global Waste Report, the potential to provide 60–100 % more food by simply eliminating losses, while simultaneously freeing up land, energy and water resources for other uses, is an opportunity that should not be ignored (9). FAO underlines that there is a need for a change in consumer behavior to decrease food wastage and reduce pressure on the environment (12). Given the impact that reducing food wastage will have on sustainable food consumption the topic should be emphasized in the chapter.</p> <p>Restricting calorie intake to meet individual energy needs would be more effective in reducing climate impact than focusing on which foods are and are not included into the diet. Reducing caloric intake to adequate</p>	

General and detailed comments	WG comments and actions
<p>levels could reduce diet-associated GHGE by approximately 2.4% (13). Reducing overbuying and overconsumption, which ultimately reduced obesity as well, reduces GHG impact at many levels, including resources required to produce wasted food; household waste; and emissions from food transport (14). Based on the fact that reducing overweight will have both environmental and health effects, this issue should be given a more prominent focus in the text. This will also be in line with the priorities from the expert group UK Sustainable Development Commission, which gives reduced consumption of food and drinks first priority in their advices in how to achieve a sustainable diet (15). Some of their other recommendations are reducing food waste, eating foods when they are in season and not drive a car when grocery shopping.</p> <p>Norway has only 3 % arable land, of which 73 % is not suitable for producing grains of human food quality (2). Most of the arable land in Norway is grassland, of which most cannot be utilized to produce grains, legumes or vegetables, because of the landscape, the short growth season and the climate. To make use of this land we are dependent on ruminants. The benefit of ruminant production systems in producing nutritious food using non-arable land and converting human inedible inputs into human edible outputs is unique and critical for food security, particularly in countries where arable land is scarce, such as Norway (16). This is relevant considering most other protein foods need arable land for their production.</p> <p>(Line 98/99) Grazing is a way for Norway to contribute to food production that is needed to feed the increasing population in the world. To not exploit this land and way of food production is unethical. Another aspect of grazing is the maintenance of the farmland and the biodiversity. We support the emphasis the text is putting on preserving the biodiversity (17). The numbers presented in the text regarding an increase in grassland are not relevant to Norwegian conditions however, nor is the statement that cropland is expected to increase. Unlike many other countries, Norway has experienced increasing overgrowth and forestation of the farmland and a reduced biodiversity in the last decades, mainly due to reduced grazing (19-22). During the last 100 years the land areas covered with forest has increased more than 50 % (20).</p> <p>(Table 1) The choice of functional unit in the table determines how the food groups are emerging. CO2 per kilo (weight) food is not an appropriate unit, due to the large differences in water and nutrient content in the food items. Figure 2 in the Bioforsk 2008 report (23) illustrates how the results are modified and the ranking of the foods changes when the functional unit is switched from CO2 per kg to CO2 per MJ. When CO2 is measured per kilo, beef has the highest emission. When CO2 is measured per MJ, tomatoes and cucumbers grown in greenhouses have the highest emissions. The same picture is recovered in the recent results from Bioforsk assessing the total global warming potential (GWP) (instead of only CO2) from a cow producing both</p>	<p>Impact of overweight is added to text.</p> <p>The illustration is good. This discussion is highlighted in the chapter.</p>

General and detailed comments	WG comments and actions
<p>milk and beef (5). When GWP per MJ is chosen, beef is responsible for the highest emission. When the GWP is rated against the amount of protein (kg) produced, milk is responsible for the largest amount of emission. These examples demonstrate the complexity of the topic. A food is rarely valuable in the diet by the content of only one nutrient, but as a result of the nutrient package it supplies to the diet. The Swedish study on climate impact of different drinks illustrates a different approach from the traditional methods (24). The total content of nutrients in the food is rated against the emission. Especially when sustainability is treated in a nutritional context, the functional unit should reflect the nutrient value of the food to integrate nutrition and sustainability.</p> <p>(Table 2) In table 2 there is a comment on combined milk and beef production being more efficient than specialized meat production. This is the dominant production of milk and beef in Norway, and separates us from most of the countries in the rest of the world. When GHG emissions due to milk and beef production are calculated, the individual distribution between the food groups vary depending on which method of allocation and which functional unit is chosen. Nevertheless it is important to keep in mind that the Norwegian combined production gives reduced emissions per amount of food produced compared to separate production. 75 - 80 percent of the beef we consume in Norway has its origin in dairy cows and the calves they encompass to maintain milk production (25). The increase in beef cattle production seen the last years is mainly due to reduced milk consumption in Norway. Beef cow herds can, to a greater extent than dairy farms, utilize land that can only be harvested by grazing and not mechanically. On the other side; securing an adequate milk intake to meet nutritional needs like calcium and iodine will simultaneously maintain the combined production, and hence preserve the most sustainable production of milk and beef. The fact that the Norwegian production of milk and meat has a higher environmental efficiency is not featured in the text, we encourage this fact to be specifically outlined in the text, and to ensure this also is taken into account in table 1.</p> <p>The overall impression of the chapter is “eat less meat and dairy”. Both the milk and the meat intake differ between genders and age groups, and there are population groups with an intake well below the recommendations. This could be a challenge with regard to securing the nutrient requirements. Norwegian women’s average red meat intake is slightly below the recommended level in the dietary guidelines (26, 27). This is not reflected in the current text. The Norwegian dietary guidelines underlines that the meat intake should be kept around recommended intake of 500 g red meat per week to prevent iron deficiency (28). With regards to meat, the current text should be nuanced, and focus on reducing the overconsumption in groups of the population with an intake above the level recommended in the dietary guidelines, rather than giving a general recommendation for the whole population.</p>	<p>A very good point, in comparison to SE, NO has a high milk/cow share among cattle. SE has lower, 50-60% of all cattle coming from cow/milk production. Not discussed to keep chapter short.</p> <p>A lower intake of red meat among women is discussed.</p>

General and detailed comments	WG comments and actions
<p>(Line 138) The draft text questions the degree to which red meat is essential in our diet. Beef and lamb are one of the most nutrient dense foods, providing a nutrient package consisting of high-quality protein, iron and zinc of high bioavailability, and several other minerals and vitamins such as vitamin B2, B3, B6 and B12, phosphor, selenium and potassium. Relative to the energy it contributes, the impact of red meat on the nutritional quality of the human diet via its contribution of protein and key micronutrients has ended up in the shadow of nutrient guidance (29). Iron deficiency is the most common kind of anemia seen by GPs and amongst women in fertile age 1/5 has iron deficiency (30). Evidence is demonstrating a positive association between red meat consumption and iron status, particularly in vulnerable population groups including infants (31-33), toddlers (34-38), adolescent girls (39) and women of child bearing age (40-45). Whilst single meal studies suggest enhancers, such as vitamin C, improve absorption of non-haem iron, only haem containing foods have been shown to be predictors of iron status in the context of the whole diet (40, 46). There is also evidence of a positive association between intake of meat and zinc status (47-49) and of meat intake with both iron and zinc status. The bioavailability of zinc depends on the quantity of total dietary zinc and phytate where dietary [phytate]:[zinc] molar ratios >15 have been associated with suboptimal zinc status (50). A study in premenopausal women showed that beef was a positive predictor of iron stores and zinc pool sizes, whereas bran breakfast cereals was a negative predictor (51). Zinc deficiency is a risk in predominantly plant-based diets where intakes of meat are low (52). This is not an exhaustive list of studies addressing the role of red meat in diet, but it gives an idea of the scientific literature on this topic. On this basis, we would like to suggest the sentence in the lines 138-139 to be removed.</p> <p>The Nordic calculations of the effect on the diet of excluding all processed meat and reducing red meat intake to 500 or 300 g a week, substituting it with either white meat and fish or an increased intake of all food groups, showed no reduction in iron content in the diet (27). But one aspect not considered in the report is the bioavailability of haem versus non-haem iron. This theoretical change in diet will lead to a reduced absorption of iron from the food.</p> <p>(Line 188) The assumption that a reduced meat intake will lower greenhouse gas emissions has to be better explored. The Bioforsk report estimated the impact on the GHG emissions with modeling different changes in diet. Even the most extreme model, based on excluding all meat from the diet, resulted only in a 5-6 % reduction of the emissions (23).</p> <p>We would like to draw attention to the French study by Vieux and researchers. This analysis is based on real diets representative of the French population. The results show a strong positive association between diet-</p>	<p>It is omitted.</p>

General and detailed comments	WG comments and actions
<p>associated greenhouse gas emissions (GHGE) and the amount of food and calories eaten (Vieux 2013). Differences in the total quantity of foods consumed by each individual explained a larger part of the diet-related GHGE variance than the carbon intensity or the energy density of the individual's diet. This means that the impact of substituting high carbon impact foods with foods with a lower impact is dependent on the amount of food required to match its energy and nutrient content. The authors showed how an isocaloric substitution of red meat with lower energy dense foods, such as fruit and vegetables, kept the diet-associated GHGE stable, or even increased it, despite its lower carbon intensity. These results indicate that it is necessary to perform more research on the effects of changing intake of the different food groups, before it is advisable to give detailed recommendations on the food groups to the public. By now, the advices should focus on more overarching topics, like food waste, avoid overconsumption and eat foods in season.</p> <p>See references below</p>	<p>In the study, meat was compensated by vegetables – and depending on how sustainable they are the climate impact varies considerably.</p>

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General and detailed comments	WG observations and actions
<p>Grenaa Akupunktur Klinik, Denmark</p> <p>Ved at indtage mere vand vil de fleste borgere kunne få en bedre livskvalitet og være plaget af færre sygdomme. Vi indtager generelt alt for lidt vand. Ved at drikke mere vand kan man reducere hyppigheden og styrken af følgende sygdomme: Højt blodtryk Natte vandladninger Tør hud og rynker Svedeture Konfusion Svimmelhed Hovedpine Blodpropper Ledsmerter Muskelsmerter O.m.a.... Jeg plejer at anbefale ca. en liter vand pr. 30 kg kropsvægt. Er man meget fysisk eller psykisk aktiv, så skal der drikkes mere end hvis man ikke er aktiv. Er ens urin mere end helt svag gul, så drikker man for lidt. Er ens tunge meget revnet eller rød, så skal der drikkes mere end gennemsnittet.</p>	<p>Acknowledged.</p>
<p>Høgskulen i Sogn og Fjordane, Norway</p> <p>Line 94-96: Sentence <i>"dehydration in excess of 3% to 5% of body weight decreases endurance and strength and is the primary cause of heat exhaustion"</i> is not correct. 3-5% dehydration may not reduce performance. In addition dehydration is not the primary cause of heat exhaustion.</p> <p>Detailed comment</p> <p>Line 94-96 could be replaced by: <i>"People should be in fluid balance at least every 24 hour and prior to physical activity. During physical activity drinking to thirst is recommended and dehydration up to 4% (Goulet 2011) or even up to 10% (Beis et al 2012) may not reduce performance."</i></p>	<p>Not relevant for general population.</p> <p>Text amended in final version: <i>"dehydration in excess of 3% to 5% of body weight <u>may</u> decrease endurance and strength and <u>contributes to</u> (is the primary cause of) heat exhaustion "</i></p>
<p>Swedish Dairy Association</p> <p>General comments</p> <p>In the chapter of fluid and water balance there is mentioned hydration status in relation to coffee and alcohol. Why is not physical activity mentioned? And what is the best way to recover after physical activity? Water, milk and carbohydrates may be a good choice.</p>	<p>The guiding value for fluid intake covers moderate physical activity. No specific recommendations are given for e.g. fluids, but general advice on food choice is given in chapter on "Food, food patterns and health outcomes – Guidelines for a healthy diet"</p>
<p>Danish Cancer Society</p> <p>Line 52. It is very unlikely that a healthy individual will need parenteral nutrition!</p>	<p>The statement refers to an estimation of total water requirement, not to a requirement for parenteral nutrition.</p>

NNR 2012: Public consultation – Chapter 8 (Energy)

Organisation Company	Energy Comments	WG responses and actions
Norwegian University of Life Sciences	<p>The report is an interesting read, and despite some critical comments as specified below, it is a useful and scientifically sound document. In the discussion of determinants of obesity and weight control, however, it would have been useful if the report was taking as a starting point for the discussion the fact that positive energy balance is the key to understanding obesity and weight control. Thus, it would have been useful if this chapter started with a discussion of general topics such as energy density of various foods, and the effect of various foods on satiation and satiety. It is the energy density and the effect on appetite which determines to what extent a food contributes to e.g. obesity. This would have made this chapter more scientifically sound. As it stands now, the authors are to a too large extent basing their reasoning on statistical associations as determined to a large extent on epidemiological data, and thus the conclusions are weak at best, and maybe even erroneous at worst. For example, the conclusion seems to be that whole-grain cereals are protective against obesity, since they are included in the category "Fibre-rich foods". However, as demonstrated recently by Kristensen et al. (2012, Journal of Nutrition 142, 710), replacing refined flour with whole-grain flour did not result in any considerable difference in weight. This is not surprising, since cereal products, independently on whether they are from refined flour or whole-grain flour, are very often used in products with a high energy density and a relatively low satiating effect. In the discussion of energy content of foods, it would have been useful if the limitations to the methods used would be discussed in more detail. The two most important limitations are that protein results in a significantly higher diet-induced thermogenesis than e.g. starch and sugars, and thus is overestimated as an energy source in the current energy calculation system. Secondly, as already discussed to some extent, the carbohydrate should be separated into starch, sugars and non-starch polysaccharides before being used for energy calculations. It would have been useful if the severe inaccuracy of not doing so - and the inaccuracy of using an arbitrary factor of either zero or 8 for fibre - had been problematised to a larger extent. Maybe it should be suggested that foods declarations should include starch and sugars?</p> <p>Detailed comments</p> <p>In line 45-51 the methods used for measuring energy expenditure are being presented. It may be true that the method of double-labelled water is the gold standard for measuring energy expenditure due to physical activity, but as</p>	<p>The main focus in the chapter in to lead to reference values for energy intake. Obesity is very closely related to the topic, but it would be beyond the aims of the chapter to go to, e.g., policies to tackle the increasing epidemic of obesity. The conclusions regarding foods and obesity are based on the systematic review by Fogelholm et al., published in Food & Nutrition Research as a background paper for the NNR. The evidence linking whole-grains in population studies is clear and there are no specific reasons to dispute this. The study by Kristensen et al. is a weight-reduction study and hence not within the scope of data used to build NNR (we have specifically stated that weight reduction is not included in the NNR and hence these studies are excluded from, e.g., systematic reviews).</p> <p>The point on different DIT's affecting the actual energy available from different foods is well taken. Although DIT for difference macronutrients are explained earlier in the chapter, a sentence was added to the end of 9.10.1.</p> <p>The chapter deals with total energy expenditure and hence the presentation of EE assessment is related to daily EE in free-living individuals. Hence, methods such as respiration chambers are less important in this context.</p> <p>The bullet points on lines 53—56 are precisely as they are in the great majority of publications we have on components of energy expenditure in humans, that is, BEE, DIT and PAEE (e.g. Hall et al. Am J Clin Nutr 2012;95:989–94). Maintaining body temperature in humans is included in BEE. Adaptive thermogenesis could be included as a fourth point, but</p>

Organisation Company	Energy Comments	WG responses and actions
	<p>discussed by e.g. Ainslie et al. (2003, Sports Medicine 33, 683), other methods can be equally or better suited for other purposes such as measurement of diet-induced thermogenesis. Anyway, calorimeter methods for direct measurement of energy expenditure and respiration chambers for in-direct measurements of energy expenditure must be included in line 49-50. In line 53-56, the statements are unprecise. If basal energy expenditure is used, as suggested in line 54, the potential energy used for maintaining body temperature must be included as a bullet point. The statement in line 102-103 may be questioned by more recent research than the reference used. It may be true that de novo lipogenesis is insignificant for a physically active individual on a average Nordic diet, but as shown by e.g. Parks et al. (2008, Journal of Nutrition 138, 1039) this pathway can be very significant in some situations, e.g. in sedate individuals on a high-carbohydrate (e.g. sugar) diet. It is suggested to delete this statement, or alternatively make it more correct by including "healthy, physically active" as a further description of individuals in line 102.</p>	<p>this is typically not done when discussing human EE.</p> <p>It is correct that in extreme sugar consumption de novo lipogenesis can be significant. The study by Parks et al. does not, however, bring any data against the statement (line 102—103) that this is in Nordic countries. The word "healthy" was included in the revised text.</p>
Uppsala University,	<p>My comment is about the uncritical adoption of the WHO cut-offs for BMI. Normal weight is suggested to be BMI 18.5-25, which is an adjustment from WHO for the cut-offs to be relevant all over the world. Asians have in general lower BMI than Caucasians. There are extremely few grown-ups in Sweden that have BMI 18.5-20 and still being healthy. Most people within this BMI-range are lean due to illness. There are no data that indicates survival benefits in the Western societies by BMI in this range. The study by Flegal et al JAMA 2013 recently showed that BMI 18.5-25 were not related to survival, rather they had lower survival than those in the range of 25-30. This result was most likely related to the fact that sick people with underweight, i.e. BMI 18.5-20 was included among those with "normal weight". My suggestion is at least to problematize the cut-offs.</p>	
Dansk Endocrinologisk Selskab	<p>Tak for en fin rapport vi har ikke mange kommentarer til det i øvrigt fine arbejde DES kan tilslutte sig forslaget om at ændre BMI grænsen for udregning af energitrit fra 21,7 til 23. Man vil dog foreslå, at denne ændring og begrundelsen herfor fremgår klart i starten af afsnittet om Energy</p>	<p>We agree that 21.7 would have been a BMI-value between 18.5 and 24.9. However, it was decided in the project group to use the arbitrary and more realistic value of BMI = 23 (at least 70% on the population in Nordic countries have a BMI higher than this value). This is clearly stated in the text, as well as in the legend of the corresponding table.</p>

Organisation Company	Energy Comments	WG responses and actions
Norwegian Directorate of Health	<p>1) The chapter concerning energy is evaluated together with the related SLR done within the NNR-project, the draft chapter 1 of June 2012 concerning overweight and obesity, and the January draft chapter concerning physical activity. Furthermore, the NNR reference values for energy should be related to the EFSA dietary reference values for energy published in January 2013. We believe that these DRV will be widely used in Europe. Difference between the NNR and the EFSA DRV should be explained or commented in the final NNR publication. 2) NNR Chapter 1, page 21. Overweight and obesity (draft June 2012) The reason for including a section concerning overweight in this chapter with main dietary recommendations is not so clear. We agree that obesity is a major and rising problem. However, CVD, cancer and hypertension are also major diet related challenges. If you decide to include information about diet related health problems in chapter 1 please cover more than just overweight and obesity. We suggest that this section is either: - rewritten to a section concerning energy balance and what may be done to prevent undesirable increase in BMI in populations, or - exchange the section concerning overweight with text concerning diet related health effects as NCD and the global strategy for preventing NCD. In the current text we miss information concerning energy intake, energy density and reference to the SLR concerning determinants of weight gain by World Cancer Research Fund (WCRF) 2007. Regarding energy density in addition to SLR by WCRF you may add the conclusions by the US 2010 Dietary Guidelines Advisory Committee, see: Pérez-Escamilla R, Obbagy JE, Altman JM et al. Dietary energy density and body weight in adults and children: a systematic review. J Acad Nutr Diet 2012;112:671 – 84. If you choose to focus on energy balance you should explain the components of energy balance and also quantify the effects of energy imbalance (both positive and negative). You should also explain what we may expect in magnitude of weight change following positive or negative energy balance, and give reference to papers as: Energy balance and its components: implications for body weight regulation. Hall KD, Heymsfield SB, Kemnitz JW, Klein S, Schoeller DA, Speakman JR. Am J Clin Nutr. 2012 Apr;95(4):989-94. Quantification of the effect of energy imbalance on bodyweight. Hall KD, Sacks G, Chandramohan D, Chow CC, Wang YC, Gortmaker SL, Swinburn BA. Lancet. 2011 Aug 27;378(9793):826-37. The current text is mainly based on one SLR by Fogelholm et al. We suggest that you use also other SLR, as the NNR SLR by Sonestedt and WCRF 2007. Furthermore recent reviews as Hauner et al. Evidence-Based Guideline of the German Nutrition Society: Carbohydrate Intake and Prevention of Nutrition –Related Diseases. Ann Nutr Metab 2012; 60 (suppl) 1-58. Hooper et al. Effect of reducing total fat intake on body weight: systematic review and meta-analysis of randomised controlled trials and cohort studies. BMJ 2012;345:e7666 Te Morenga et al.</p>	<p>The work has been very much based on decisions taken by EFSA, e.g., regarding the equation to estimate REE. Since the reference values use population-based weights, it is evident that NNR has different final numbers. Referring to the SR of Fogelholm et al. may seem biased, but this paper was specifically done as a background for NNR. We have added a couple of newer references, e.g. the review by TeMorenga et al. and Chen et al. Moreover, aspects from the consensus statement for energy balance (Hall et al. 2012) were also added to the text.</p> <p>Mixing self-reported and weighed BMI can, unfortunately, not be avoided here due to the lack of comparable data in Nordic countries.</p>

Organisation Company	Energy Comments	WG responses and actions
	<p>Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2012 Jan 15;346:e7492 Chen et al. Effects of dairy intake on body weight and fat: a meta-analysis of randomized controlled trials. Am J Clin Nutr 2012;96:735-47. 3) Chapter Energy – general comments We suggest that you expand the sections concerning energy balance and the dynamics of weight increase or decrease (ref Hall 2011 and 2012 above). You may also give more information concerning the association between dietary patterns and the risk of increased BMI, see comments regarding references in section 2) above. Please do not mix figures for prevalence of obesity that are measured and self-reported as you do in table 9.2.</p> <p>Detailed comments Line 126-131 regarding optimal BMI. This topic may be expanded as several reviews concerning the complexity of the association between BMI and mortality and chronic diseases has been published the last years. This complexity includes differences between cultures regarding the optimal BMI. E.g. differences between people living in Europe, South east Asia and the Indian subcontinent. You should also consider including information concerning the calculation of BMI and its relation to health in the Sami and Inuit populations. L 137. Consider to include also a reference to the WHO Child Growth Standards. L 139-140. You may also include information concerning differences in resting energy expenditure between populations, see references given by EFSA 2013 page 17. L 141-150. This section is difficult to read. Please consider to rephrase. L 157-158. We suggest that you consider to exchange the following sentence: “Unfortunately the data are inadequate to make any precise recommendations for optimal BMI among the elderly” with “However, we suggest a cut-off level in the range of 25-30 based on Norwegian data (Kvamme et al: Body mass index and disease burden in elderly men and women: The Tromsø study. Eur J Epidemiol 2010; 25:183-193. L 170 table 9.2. Here you mix self-reported and measured data. We suspect that the figures concerning prevalence of obesity for Norway are measured data. According to self-reported data from Statistics Norway the prevalence of obesity (BMI 30+) was 12-13 % in men aged 25-66 years and 9-10 % in women 25-66 years in 2008, see http://www.ssb.no/helseforhold In connection with table 9.2 you may consider to add a section with information about BMI based on measured data from Nordic health surveys. L 190-95. You should consider to refer further recent reviews, see also comments to BMI and health I 126-31. L 211-245. This text is mainly based on one SLR by Fogelholm et al. We suggest that you use also other SLR, as the NNR SLR by Sonestedt and WCRF 2007, see comments under 2) above. L 245 figure x. The figure is difficult to understand. Please improve title and include unit on the x-axis. L 255-59. Social differences in prevalence of obesity is an important topic. Please consider to use a separate section, and give Nordic facts concerning these differences e.g. a separate table in line with table 9.2. In Statistics Norway health interview surveys 2008 (self-reported data) the prevalence of obesity (BMI 30+) was 10 % among women with</p>	<p>Line 126-131 This is true. We have now added a few considerations on ethnicity and BMI. (Noahsen P, Andersen S. Ethnicity influences BMI as evaluated from reported serum lipid values in Inuit and non-Inuit: raised upper limit of BMI in Inuit? Ethn Dis. 2013 Winter;23(1):77-82. Chiu M, Austin PC, Manuel DG, Shah BR, Tu JV. Deriving ethnic-specific BMI cutoff points for assessing diabetes risk. Diabetes Care. 2011 Aug;34(8):1741-8.)</p> <p>Line 137: The WHO Growth Standards is a global tool. A more general note on growth charts was added.</p> <p>Line 139-140 We consider this to be beyond the focus of the chapter.</p> <p>Line 141-150: The paragraph has been rewritten.</p> <p>Line 157-158 The sentence has been rewritten and the reference by Kvamme et al. was added. However, we kept the final statement inconclusive.</p> <p>Line 170 Good, thank you for this information. We will check the table once again</p> <p>Line 190-195 We decided to keep this as it is. There are a few more recent meta-analyses on BMI and health (e.g. Strazzullo et al. Stroke 2010), but the message is the same as in Guh et al.</p> <p>Line 211-245: Referring to the SR of Fogelholm et al. may seem biased, but this paper was specifically done as a background for NNR. We have added a couple of newer references, e.g. the review by TeMorenga et al. and Chen et al.</p> <p>Line 245: This has been done.</p> <p>Line 255-59 and 274-77: The chapter is already lengthy and a more detailed</p>

Organisation Company	Energy Comments	WG responses and actions
	<p>short education (high school) and 7 % among women with long education (university and college). Among men corresponding figures were 12 % and 9 %.</p> <p>http://www.ssb.no/helseforhold L 274-77. Please give some more information concerning what the use of DLW have learnt us e.g. in relation to energy expenditure in relation to obesity etc. L 289. Remember to include EFSA 2013. Table 9.4. Specify “BEE” (only used the abbreviation in the table). L 315. You may include a comment regarding the growth spurt during adolescence. L 337-345. Could maybe include some new references. L 359-73. This section is difficult to understand. You should consider rephrasing it. Table 9.6. Should there be included a text in the heading over “Physical activity level”? L 404 MET is mentioned for the first time. Definition of MET may be included under definitions in the beginning of the chapter. L 452. You mention that tables 9.9 shows reference weights based on data in Denmark Are these data measured, and from what time period? Why not include Norwegian data? We suggest that you include a table with basic Nordic data for measured weight and height and BMI. See EFSA 2013 table 4 page 32. Then it may be easier to understand how you have calculated the reference weights in table 9.9. You may also comment the difference in reference weight between NNR Table 9.9. and EFSA Table 4, and furthermore corresponding differences between NNR table 9.10 and table 12 in EFSA 2013 concerning reference energy requirements. L 491-8. Expected weight change in relation to changes in energy balance is of great interest for many users of NNR. You may assess the information you have given in this section in relation to the suggestions of Hall et al that are cited above in 2). In contrast to NNR the American Society for Nutrition (Hall et al 2012) recommended that the 3500 kcal per pound rule should no longer be used. They suggest a new model (rule of thumb) for calculation of expected weight loss following decreased energy intake as well as increased energy expenditure. Furthermore, Hall et al claim that it is inappropriate to use the 3500 kcal per pound rule to model the effects of interventions. To illustrate this problem they give this example, a 40-kcal/d (170-kJ/d) permanent reduction in energy intake resulting from taxing sweetened beverages has been predicted to result in ;20 lb (9 kg) of weight loss in 5 y according to the 3500 kcal per pound rule, whereas only 4 lb (2 kg) of weight loss is predicted using the new rule of thumb. Models for calculating effects of nutrition policy measures as taxes or subsidies are of great political interest. It is not obvious that we shall use the same models and factors to predict effects of changes in energy balance for weight reduction among a group of obese, and when we shall predict effects of price policy on consumption and subsequent increase or decrease in body weight in the whole population. Thus it is important that we have as much consensus as possible regarding how to calculate and predict effects of dietary changes. Please include in NNR facts both concerning energy costs of building body tissue and fat tissue, as well as energy gained when losing body weight. Please also add your recommendations regarding models and factors that can be used for calculating effects of changes in energy balance, both in relation to weight reduction and in relation to predict effects of policy measures in populations. L 569-78.</p>	<p>description of DLW goes beyond our scope.</p> <p>Line 289: EFSA is referred to. Abbreviation is explained in the legend.</p> <p>Line 315, L337-345, L 359-73: The legend of table 9.6. was changed so that it refers now to energy requirements, not expenditure.</p> <p>L 409: MET is now introduced earlier in the chapter.</p> <p>L 452: The reference weights are based on population studies with data on height. Based on height, the weight corresponding BMI = 23 was then calculated.</p> <p>L491-8: Yes, indeed this is true. I made a calculation for a hypothetical 90-kg man by using the Henry equation for REE and PAL 1.5. The predicted daily EE is 2795 kcal. Then I assumed that energy intake would be reduced by 500 kcal to a fixed value of 2295 kcal/day. I also assumed the 500 kcal energy deficit would lead to a 0.5 kg weight loss. Then the reduced weight would affect the REE and daily EE calculations and reduce the weight loss. This is, as far as I understood, the main critique in the statement paper by Hall et al. After 1 mo, the weight loss would be 450 g/wk (that is, 50 g slower than predicted). At 1 year the estimated weekly weight loss is no more than 160 g/week and after 2 years 53 g per week. This problem is now clarified in the chapter and it is clarified that the “500 g weight reduction for each 3500 kcal energy intake decrease” should only be used during the first month of weight reduction.</p> <p>The “rule of the thumb” in the paper by Hall et al. was considered less helpful in this context. It shows the relation between and initial energy deficit and what can be achieved, but we don’t think this is very helpful in planning weight-reduction diets. Therefore, while pointing out the problems in the simple method, the new “rule” was left out of the NNR 2012.</p> <p>L569-78: A reference from the MoBa was added.</p> <p>L597: We have earlier indicated some figures on obesity in Nordic countries and they are considered adequate in this chapter. After all, the main point of the chapter is to justify the reference values for energy intake.</p> <p>Table 9.13: Yes, this was corrected.</p> <p>L643: The study by Bertz et al. is now added to the text (short note) and consequently also as a reference.</p>

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	<p>You may add data from the Norwegian Mother and Child Cohort Study (MoBa). This is a unique study where more than 90,000 pregnant women were recruited between 1999 and 2008. Over 70,000 fathers have also participated.</p> <p>http://www.fhi.no/eway/default.aspx?pid=240&trg=Main_6664&Main_6664=6894:0:25,7372:1:0:0:::0:0 We also suggest that you add up-dated information from The Medical Birth Registry, a national health registry containing information about all births in Norway.</p> <p>http://www.fhi.no/eway/default.aspx?pid=240&trg=Main_6664&Main_6664=6898:0:25,7840:1:0:0:::0:0 L 597. You claim that obesity is common among Scandinavian women and that this should be avoided in reproductive ages. Here you may give some facts about prevalence in Scandinavia. Table 9.13. Should “exclusive breastfeeding” be used instead of “complete breastfeeding” in title, table and footnote? L 643 In relation to handling of obesity among lactating women you may refer to this paper: Diet and exercise weight-loss trial in lactating overweight and obese women. Bertz F, Brekke HK, Ellegård L, Rasmussen KM, Wennergren M, Winkvist A. Am J Clin Nutr. 2012 Oct;96(4):698-705. Epub 2012 Sep 5. L 667. If possible you may add information related to this question: Does energy expenditure in relation to defined physical activities change with increasing age? E.g. in the chapter about Physical activity page 9 line 306 you mention that climbing stairs may be perceived as light activity for a 30-year-old and hard for a 70-year old. Phrased in another way: How do we interpret the information and figures given in table x.1 in the physical activity chapter concerning energy requirements as percentages of cardiorespiratory fitness ...? Numbers given for climbing stairs 20-39 years is 42 and for 80+ years is 77. L 693-7. Challenges concerning false reporting of energy intake and expenditure, both under and over reporting, is an important methodological question that you might expand in a separate section and not only hide it in the section of low energy intake. L 734-55. This section is difficult to read and understand. You should consider rephrasing this section. Please also give a clear conclusion concerning your recommendation regarding the energy value for dietary fiber that we shall use for calculating energy intake. Is it zero or 8 kJ/g?</p>	<p>The energy factor for fibre is an average value based on a previous FAO report on food energy and is also adopted for labelling in EU.</p>
LRF Dairy Sweden	<p>Line 213. The statement "Weight gain is caused by positive energy balance." should be discussed in more detail, also including factors causing a positive energy balance.</p>	<p>This is partly covered in chapters on macronutrients, but also refer to behavioral factors mainly outside the scope of the current NNR revision.</p>
Danish Cancer Society	<p>Very well written chapter! To some extent I miss descriptions of the associations between high energy diets, consumption of energy dense foods, positive energy balance food s and different chronic diseases, i.e. diabetes, CHD and cancers. There is a good description of determinants of obesity and weight control, (line 120-259) but I think it should be extended to the large chronic diseases. Of course there is an indirect link to obesity.</p>	

Organisation Company	Energy Comments	WG responses and actions
Selskab for Ernæringsforskning, Denmark	Detailed comments p.3 line 124: Add BMI "have" p.4 line 167: replace NNR 204 with NNR 2004 p.16 line 545: Add energy requirement "/24 hours"	Corrections done.
Minivent AB, Sweden	Please replace the text from line 1 to the last with this text. Unfortunately, the formatting capabilities of this form seems to be nonexistent. Please publish the formatted version I send by ordinary e-mail NNR5 Energy The human organism is always striving for survival and homeostasis. In order to survive the person needs to eat food. Food consists of macronutrients and micronutrients. The micronutrients are covered elsewhere. The macronutrients are the energy suppliers as well as building material for the body. There are three macronutrients, protein, fat and carbohydrates. They all contain carbon as an energy source. Protein Proteins consist of the building blocks amino acids. There are about 20 amino acids in the animal body. Humans can make about ten amino acids from scratch and the remaining about ten amino acids the person has to eat to be able to use them. So protein is an essential part of the food. Without protein we will not survive for long. The amount of protein in the food should be at least 0.5 grams of animal protein per kg body weight. This is enough for a newborn child to increase 50 % in length during the first year and should be enough for the rest of the life . We have a limited storage facility of free amino acids. Any excess of protein is deaminated and the rest is converted to short fatty acids or short alcohols that can be used in the metabolism. Those short fatty acids and short alcohols can enter the central metabolism in the body, more about that later. We have tens of kg of muscles with about 20 % protein. On the cellular level cells contain a lot of different structural, signalling, building and other kinds of protein molecules, all necessary for survival. Muscle cells contain great amounts of contracting proteins to be able to move the body and parts. No normal animal protein is toxic to humans. Fat The major functions of fat is energy production, energy source, energy storage and padding delicate organs in the body. We have tens of kg of fat in the body. We can store several hundred kg of fat. Fats consist mainly of triglycerides (three fatty acids connected to a glycerol molecule) but there are free fatty acids, monoglycerides, diglycerides, phospholipids as well as other lipids and lipid like molecules. The major composition of fat is shy of 50 % saturated fats (SAT), shy of 50 % monounsaturated fats (MUFA) and the rest, about 5 %, is polyunsaturated animal fats (PUFA) with carbon atom numbers of at least 20 C. The fatty acid composition is about the same for all warm blooded animals as	Comments acknowledged. Some basic physiology and metabolism is covered in chapters on macronutrients. These chapters also deal with various health effects.

Organisation Company	Energy Comments	WG responses and actions
	<p>they have the same body temperature around 37°C. This is because fats must have a correct melting temperature for proper function. Arctic fish have much higher PUFA concentration as PUFAs have much lower melting temperature compared to animal fat. This is shown beautifully by placing butter, olive oil and canola oil taken from the fridge on a plate and let it sit on the table. The canola oil is still liquid at fridge temperature, the olive oil is partly solid at fridge temperature and the butter is definitely solid at fridge temperature. At room temperature both oil fats are liquid but butter is solid. When temperature rises above 30° C then also butter melts into liquid form. So arctic fish must have higher concentration of PUFA to survive due to it's lower organism temperature. Salmon has 3.3 g PUFA and bacon has 2.8 g PUFA. This means that 100 g salmon can be replaced by 118 g bacon to achieve the same amount of PUFA. Also 100 g salmon can be replaced by 35 g lard. So as long as we eat animal fat we will always get enough PUFA. We also have to realize that vegetable PUFAs usually are nonelagable to animal long chain PUFAs in our body. Vegetable omega-6 are inflammation producing and if more than 20 g per day also causing cancer, well known by the old transplantation surgeons as well as Livsmedelsverkets civil servants. Animal fats in the form of phospholipids are essential to our cell membranes as they separate cells and organelles from each other and intracellular as well as extracellular environments. Also in all membranes there are about the same number of cholesterol molecules as phospholipids molecules to make the cellular and other membranes stable and supportive. So the lipids of all kinds are essential to life. Lipids and fats are also necessary making up the cushioning around delicate structures in the body. Fat is also an excellent energy source for the body. Also fat tissue and fat cells in muscles is normally the major source of acetylcoenzyme A (AcCoA or activated acetic acid) which is the only energy producing molecule in the cellular mitochondria. See the Krebs cycle for more biochemical and energy details. This means that all animal fat are essential to us and they are nontoxic as they consist of the same fats in the same proportions as our own fat. The animal fats also requires the least remodeling and exchange due to the same reason. Fat is also the most energy dense macronutrient so persons can eat smaller portions to have enough energy. Also, fat is the most satisfying macronutrient. Carbohydrates</p> <p>Carbohydrates are the third kind of macronutrients. Carbohydrates consists of sugars. So carbohydrates are equivalent to sugars. Fibers consists of polymers of glucose that no warm blooded animal can digest. Herbivores are using microbes to digest fibers like cellulose to short chain fatty acids, less than 7 carbon atom</p>	

Organisation Company	Energy Comments	WG responses and actions
	<p>long chains. Those short or volatile fatty acids are completely water soluble and can thus be taken up directly in the blood and then transported directly to the liver for further use and can enter the central metabolism in the body, more about that later. Humans are unable to digest fibers as we lack the fermenting microbes and fermentation tank like the 1 hL four parted ventricle of a normal cow. So fibers lack energy in humans, easily controlled by eating pure fibers and check the lack of hydrogen gas production the digestion of fibres always cause. Starches all consists of polymers of glucose easily degraded in the intestinal tract by a lot of enzymes down to single units of glucose. Other common disaccarides are sucrose (consists of glucose and fructose), lactose (glucose and galactose), maltose (glucose and glucose). Disaccarides can not be absorbed by the intestines, so they have to be digested by common enzymes to their monosaccarides. Those monosaccharides like glucose (most abundant), galactose (animal sugar) and fructose (vegetable sugar). A human of 70 kg has a low glucose concentration of 3-6 mmol/L in the blood to supply the Red Blood Cells (RBC) with energy as RBC lack mitochondria. The normal blood concentration of 3-6 mmol/L is equal to the amount of 1.5 to 3 grams of glucose in the total blood volume of 5.6 L. The RBC utilize glucose anaerobically via the RBC-specific Rapoport-Luebring pathway including 2,3-diphosphoglycerate (2,3-DPG) that is required to release oxygen from the hemoglobin molecule. Then the 2,3-DPG is transformed to lactic acid and 2 molecules energy rich ATP. When having 5 g glucose, without insulin and a theoretical immediate uptake, the glucose level will increase to around 15 mmol/L which is a toxic concentration of glucose. By having more than 15 to 25 g glucose in the blood (>30-50 mmol/L) the normal person will very shortly die from acute glucose intoxication. Glucose and fructose are toxic at higher concentrations than 6 mmol/L the monosackarides are nonenzymatically glyating the lysine amino acid residue in proteins and thus changing the morphology and may change the function of the protein. The most famous glycated protein is HbA1c where the attached glucose/fructose molecule destroys the oxygen transport function of hemoglobin. This glycation of proteins is the major problem with carbohydrates. When the person has carbohydrates the blood glucose rises immediately which releases insulin. The insulin has a lot of actions in order to normalize the blood glucose level. The insulin slows down the intestinal uptake of nutrients, inhibits the AcCoA production from fat and protein because the monosaccaride concentration just has to be normalized as the body otherwise will die from acute monosaccaride intoxication. Read more about the wonderful insulin</p>	

Organisation Company	Energy Comments	WG responses and actions
	<p>molecule and functions at reference below . There are no essentiality of eating carbohydrates. All sugars the human body needs is happily produced by the liver. And the liver can always produce enough sugar. The logic behind NNR4 recommendation to have 50-60 % of the energy (%) is totally missing. As excess of carbohydrates increase the insulin level for long times thus decreasing the fat energy expenditure and that carbohydrates are partly converted to fat and thus increasing body weight the amount of carbohydrates has to be minimized. It is also after the introduction of NNR with a in the 1980-ies that the epidemy of diabetes and obesity rose inexplicably. The only change in the environment is the exchange of fat to carbohydrate as major energy source. It's OK to have vegetables ad libitum as they do contain small absolute amounts of carbohydrates. The amount of fruit should be limited due to the fact that most fruit contains the same amount of sugars as soft drinks, about 10-12 grams per 100 g The amount of grain and grain products like bread, pizza, pasta contains 30-90 g carbohydrates per 100 g of grain product. Thus grain products should be restricted to small amounts realizing that many persons are gluten intolerant and should then be totally excluded. Energy The intake of energy is recommended by NNR4 to 2.5 Mcal (2 500 kcal) for women and 3.2 Mcal for men. As long as the carbohydrate intake is less than 100 g per day the population will normalize the body weight. So this recommendation can be propagated to NNR5. All weight reduction diets have a common denominator: Carbohydrate reduction. Just compare a NNR4 diet with 50 E% carbohydrates and 0.8 Mcal to a low carbohydrate diet with 2.5 Mcal and 16 E% carbohydrates. This means that the NNR4 starvation diet contains 100 g carbohydrates, the person loses weight, is always hungry and the person will starve to death, usually within a year. On the other hand the low carbohydrate diet contains 100 g carbohydrates, the person loses weight, is always satisfied and the person can eat that kind of food for decades. Both diets are shown to give the same weight decrease in the short term but the famine dieter is unable to continue for more than a few months or the diet will be lethal. The lowcarber can have delicious food for decades and keep the weight loss. The first law thermodynamics tells us that in an isolated system energy in equals energy out. But the human body is not an isolated system. And we have to realize that we also have to obey the second law of thermodynamics as well as the hormonal control of the body. This means that the body can regulate the efficiency between 0 and up to less than 100 %. The most part will be heat. We also have to realize that fat does not increase the insulin level. Excess protein may and carbohydrates always increase</p>	

Organisation Company	Energy Comments	WG responses and actions
	<p>the insulin level to normalize the glucose level. So insulin changes the normal fat burning metabolism to carbohydrate burning and fat generating metabolism to survive. The obesity care have tried to have obese patient to eat less and run more. This hypothesis has been disproven many times during the last 35 years. But it seems that the obesity care units were unable to realize these results. So by having ad libitum food consisting of normal amounts of protein, maximum 100 g of carbohydrates and 70-90 E% mostly animal fat the population will in a few years regain normal weight, decrease disease burden and a better general health. NNR5 macronutrient recommendation Proposal of NNR5 nutrition recommendations to healthy individuals based on the old trustworthy sciences physiology, biochemistry and endocrinology: Summary of NNR 2012 recommendations to healthy individuals NNR 2012 recommend an adequate intake of essential nutrients of protein, fat, minerals and vitamins. NNR 2012 recommends a normal daily intake of essential animal protein of at least 0,5 g/kg bodyweight. NNR 2012 recommends an upper limit of 100 g of carbohydrates/sugars per day . NNR 2012 recommends a sufficient intake of essential fats to satisfy the energy and metabolic requirements. NNR 2012 recommends an upper limit of 20 g per day of vegetable omega-6 polyunsaturated fats. , . NNR 2012 recommends an upper limit of fiber intake of 25 g per day to an adult and an upper limit of 0,35 g/kg to a child. NNR 2012 recommends an upper limit of one fruit per day. The conclusion of food consumption changes between NNR4 and NNR5 to promote health in Nordic populations Increase . Unchanged . Limit to per day . Fat, preferably animal Protein from meat . Limit Carbohydrates to 100 g. Vegetables . Limit Omega-6 PUFA to 20 g. Limit fiber to 25 g . Limit fruit to one . Background information for NNR 2012 recommendations From NNR5 Recommendation for carbohydrates, fibre and added sugar http://www.slv.se/upload/NNR5/NNR5%20Carbohydrates.pdf Mikael Fogelholm The data on health effects of dietary fibre and fibre-rich foods are very strong. Recommendations in NNR 2012: Adults: Intake of dietary fibre should be at least 25-35 g/d, i.e. approximately 3 g/MJ. Children: An intake corresponding to 2-3 g/MJ is appropriate for children from 2 years of age. From school age the intake should gradually increase to reach the recommended adult level during adolescence. Comment According to present recommendations in NNR4 children should have about double the amount of fiber per kg bodyweight as an adult. Adults are recommended about 30 g/day or 0,4 to 0,6g/kg while children are supposed to have 3 g/MJ which will be 12 g</p>	

Organisation Company	Energy Comments	WG responses and actions
	<p>fiber per day or 1,2 g/kg. This is due to recommendations are associated to the amount of energy eaten instead of kg bodyweight. Children have a higher energy intake per kg bodyweight due to the fact that they need more energy to grow and need more energy to compensate for a larger skin surface heat loss per kg bodyweight than adults. As fibers are associated to an increased risk of colon cancer , , there is an increased risk that children and adults get colon cancer with the present recommendations. So the new recommendations should be: NNR 2012 recommends an upper limit of fiber intake of 25 g per day to an adult and an upper limit of 0,35 g/kg to a child. Mikael Fogelholm Intake of added sugars should be kept below 10 E%. Limitation of the intake of added sugars from particularly sugar-sweetened beverages and sugar-rich foods is recommended in order to reduce the risk for type 2 diabetes, weight gain and dental caries. •A limitation of the intake of added sugars is also necessary to ensure an adequate intake of essential nutrients and dietary fibre, especially in children and adults with a low energy intake. Comment In NNR4 there is a recommendation to keep intake of added sugars to below 10 E%. It seems that the NNR4 have disregarded that carbohydrates and sugar are synonyms as they recommend a limit of carbohydrates in the form of “added sugar” but recommend 50-60 E% of carbohydrates in a day. It is good that NNR 2012 will limit the intake of sugar to ensure an adequate intake of essential nutrients. All kinds of carbohydrates consists of sugar which are nonessential and contains 1 to 1/1 000th (or less) of minerals and vitamins compared to food of animal origin . Then we have to re-establish what the essential nutrients are. So the new recommendations should be: NNR 2012 recommend an adequate intake of essential nutrients of protein, fat, minerals and vitamins, preferable in the form of animal proteins and fats which contain enough minerals and vitamins. Vegetables are insufficient in amino acids, all fatty acids including long chain polyunsaturated fatty acids, minerals and vitamins but vegetables are excellent to improve the visual impression of food. Fruit with 10 % sugar content may decrease to maximum one fruit per day Also, the NNR 2012 finds no justifications to include a diet-based recommendation for GI in NNR. Comment This is correct as GI is a partial and indirect measurement of carbohydrates, we are just measuring the glucose level in the blood, we are not measuring all other sugars like fructose, galactose nor are we measuring the continuous insulin level that delays the uptake of sugars in an attempt to normalize a too high blood glucose level. Also, the SBU report Mat vid diabetes of 2010 found no justification to include a diet-based recommendation for GI So an added</p>	

Organisation Company	Energy Comments	WG responses and actions
	<p>recommendations should be: NNR 2012 finds no justifications to include a diet-based recommendation for GI in NNR. Also, NNR4 recommends specific “quality” of fat. As all mammals have the same temperature of 37°C, they all have the same animal fat composition with the same melting temperature for correct function. This means animal fat has shy of 50 % saturated fat, shy of 50 % monounsaturated fat and about 5 % polyunsaturated fat. This is the ideal mixture of fats and the fat has a correct melting temperature to support correct function of fat in tissues and cell membranes.</p>	

NNR 2012: Public consultation – Chapter 9 (Physical activity)

Organisation/ Company	General and detailed comments	WG response and actions
The Directorate of Health expert group on physical activity, Iceland	<p>Our main concerns regarding this proposal are threefold. First of all the fact that the Nordic Physical Activity Guidelines are still presented as just one of many chapters in the Nordic Nutrition Recommendations, which we believe does not grant these guidelines the true weight they deserve. This key independent determinant of health deserves to stand alone as a section, and it should also be acknowledged by giving the proposed publication a more descriptive name, e.g. Nordic Nutrition and Physical Activity Recommendations 2012(3?). This, we believe, will more strongly emphasize the importance of physical activity as one of the major health determinants and build a more solid ground for having this publication serve as a primary foundation for physical activity recommendations presented by each of the Nordic countries.</p>	We agree - indeed. However this is a more political issue and we cannot change the mandate of NNR 2012.
	<p>The second concern we would like to raise is that although we noticed that more current references have been added to the precursor to these current guidelines (NNR 2004) we still feel that the authors may build upon somewhat outdated references. We agree that it is important to acknowledge the work of those who were in the forefront of physical activity research in recent decades, but likewise it is important to update information when it's due. Thus, perhaps it would strengthen the drafted proposal even further to more thoroughly describe the state-of-the-art to the reader for each part. This is particularly evident for the chapter on the mental health and quality of life, elderly and pregnancy.</p>	<p>We have now included several key references in the text. As we state in the chapter: We do “not intend to cover the entire body of literature regarding the effects of physical activity.”</p>
	<p>The third general point we would like to make concerns the argumentation for the changes proposed in these new guidelines. In particular the presentation of the recommendations for</p>	Acknowledged – we have made an effort to do so. Also, we have mentioned leading

Organisation/ Company	General and detailed comments	WG response and actions
	adults, elderly and pregnancy might seem to some to be drastic. Thus, it would be very helpful if the authors would more clearly convey the evidence for them, and to mention the leading international recommendations in relation to the updated Nordic recommendations.	international recommendations.
	<p>Detailed comments</p> <ul style="list-style-type: none"> Line 52: Box 1. Since the terms aerobic activities and muscle-strengthening activities (and bone-strengthening activities) are important part of the key recommendations, should they maybe be included in the definition list (aerobic activities instead of endurance training?)? Line 57: Physical inactivity. See for example: * Nordic monitoring: Physical inactivity = Not meeting recommendation, less than 15 min/week (page 84, http://www.norden.org/is/utgafa/utgefid-efni/2012-552/at_download/publicationfile) * USA: Physical inactivity = no activity beyond baseline activities of daily living, (page 5, http://www.health.gov/paguidelines/pdf/paguide.pdf) Line 196: "...the amount of physical activity to avoid weight gain is about 60 minutes of moderate intensity (daily?) or somewhat..." Also, perhaps update references concerning this point? Line 222: ...lower limb (?) muscle strength ... Line 224: See references: 68 (69)-71 Line 225: Physical activity can counter(act) Line 247: The current reference is since 2001. Does more recent evidence allow us to take this a step further? "However there is (insufficient evidence about?) the dose-response relationship between PA and depression and anxiety..." See for example USA background report (Part G. Section 8: Mental health. Page 10 and page 16, http://www.health.gov/paguidelines/Report/pdf/G8_mentalhealth.pdf) Line 264: Which recommendations? Line 303: Table x. The Borg scale is good addition. Could it be even clearer to remove the intensity categories in the left column (but keep the activities) and represent it rather in steps with for example different shades or lines depending on what applies for different age groups? See for ex. Appendix1, page 55 (relative intensity) http://www.health.gov/paguidelines/pdf/paguide.pdf 	We have considered the detailed comments and wherever appropriate corrected, clarified or changed the wording according to the many useful comments.

Organisation/ Company	General and detailed comments	WG response and actions
	<ul style="list-style-type: none"> Line 315: "Those who are physically inactive..." Readers can misunderstand this sentence! Maybe add to it "...may achieve the greatest health gains by (increasing their physical activity)." Line 341: "...insulin(and) glucose ratio ..." Line 364: Should screen-time be mentioned specifically? Line 373: "...and others." Add reference. Line 373: "WHO also (specifically?) recommend..." Line 394: Paragraph, move point 4 down one line. Line 396: Italic Line 447: "...on uneven terrain (())which is..." 	
Swedish Professional Associations for Physical Activity (YFA)	<p>1. There is a need to summarise the sources of the recommendations (children, adults and elderly). It is suggested to include a short paragraph, where the sources/references and possible amendments/additions are described for the recommendations given in NNR 5. We are missing the committee report, see below US Department of Health and Human Services. Physical Activity Guidelines Advisory Committee Report, 2008. Washington,, DC: ODPHP Publication No. U0049. 2008. 683 p. Available from: http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.</p> <p>2. We are aware that the recommendations on sedentary behavior are similar to those stated in UK. However, we cannot neither in the text nor in the literature find any scientific motivation for stating that sedentary time should be minimised. It is well known that a balance between rest and activity is needed and minimising sedentary might yield other problems. Reduced could be ok, but of course that depends on how much each individual is sitting and if a majority of the population is sitting too much. Based upon objective measures of sitting (sedentary time) adults in Sweden and Norway are sedentary in 8 hours per day (mean) and active at low to higher intensities for 7-8 hours. The sedentary time is on average interrupted 13-14 times per day, i.e. indicating that on average people are sitting no more than 30 minutes in a row. For example, the study by Van Der Ploeg et al Arch Intern Med 2012 did not find any support for a link between self-reported sitting less than 8 hours a day and all-cause mortality. Thus, stating reduced and especially minimising are quite strong and not motivated based upon neither population prevalence nor the literature.</p> <p>3. Definitions and concepts need to be revised and made clearer and used consistent.</p> <p>a) Define the concepts that are important and part of the recommendations and use already</p>	<p>We have now included several key references as suggested.</p> <p>Acknowledged. There is an ongoing debate concerning this issue. We have made a compromise and deleted "minimize" and reworded the text with respect to this topic.</p> <p>We have done an effort to adhere to this important general comment. For instance</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>existing definitions. For example we miss definitions on aerobic physical activity as well as on muscle--strengthening activity which are the terms used in the NNR recommendations. We suggest taking out the definitions from Box 1 that could be read or explained in the text. For example those on fitness, exercise, cardio-respiratory fitness that are not part of the recommendations.</p> <p>b) Use the stated definitions consistent throughout the chapter. For example, low, moderate and vigorous is used in different ways. We suggest to use the concept light intensity physical activity, moderate intensity physical activity, and vigorous intensity physical activity or aerobic physical activity of light, moderate or vigorous intensity. For “physical inactivity” it might be less confusing if the concept “insufficient physical activity” is used to define those not meeting the guidelines and “sedentary behavior” when relating to sitting.</p>	<p>“physical inactivity” is replaced by “insufficient physical activity”. The use of the different definitions should now be consistent.</p>
	<p>4. There is a need to clarify the concepts of both absolute and relative intensity. The absolute intensity definitions in box 1 (METs) are commonly used in epidemiological studies to describe PA volume and intensity. However, when it comes to individual exercise prescriptions, one has to apply relative intensity measures such as RPE (e.g. the Borg scale), %HR-max or talk test (talk/sing). In fact, in most epidemiological studies, PA questionnaires are used and information on PA habits is originally collected on the basis of relative intensities and we have to come back to this in order to individualize PA prescriptions. This is highlighted in ACSM – 2011 pp1341-42 (American College of Sports Medicine. Position stand: Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal and neuromotor fitness in apparently in healthy adults. Med Sci Sports Exerc. July2011 - Volume 43 - Issue 7 - pp 1334-1359. See also Fletcher et al Circulation. 2001;104:1694-1740, Table 7). We suggest leaving out table x1. See also comment below Line 30. Possible substituting table x1 with a table describing moderate and vigorous intensity in relative terms such as RPE (e.g. the Borg scale), %HR-max or talk test (talk/sing). (12-13 moderate, 14-16 vigorous etc). Some typical activities can be mentioned for the various intensity levels without giving the METs values. Those are of more interest when doing calculations of energy expenditure as already mentioned in NNR 5.</p>	
	<p>Specific comments Line 19: Table (no number): a) Recommendations --- italics missing for minutes per day above children.</p>	<p>Thank you for all these very useful specific comments. We have considered the detailed comments carefully. Wherever appropriate</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>b) Footnote unclear.</p> <p>c) see general comments above (1)</p> <p>d) We suggest using/defining aerobic physical activity instead of physical activity</p> <p>Line 36: Segments? Language?</p> <p>Line 37, 39:</p> <p>a) Inconsistent use of concepts: In the first sentence physically inactive are used as per definition in box 1, in the second sentence physical inactive are used instead of time spent sedentary. This is different. Maybe use the concept “insufficient physical activity” for those not reaching the guidelines.</p> <p>b) We also miss a sentence that states that the former and current recommendations are based upon self---reported data, i.e. comparison of prevalence’s using objective data should be made more carefully.</p> <p>Line 40: Language?</p> <p>Line 47: Indeed the authors have included key references, but we miss systematic review papers as they can give a better picture of the state of the art. One example is the physical activity guidelines advisory committee report (PAGAC-report) that was conducted prior to forming the guidelines on physical activity in the US in 2008 and used for the WHO recommendations. See point 1 above.</p> <p>Line 73: MET and line 83 Resistance training should be on a new line. And is resistance training equal to the concept used in the recommendations, i.e. muscle---strengthening activity?</p> <p>Line 77: Light intensity cannot be < 3 MET, if so also sedentary is included as light. I.e. it has to be from something MET> 1.3 or 1.5 as per definition of sedentary.</p> <p>Line 104: References missing</p> <p>Line 116---123: For clarity we suggest to add that the extra EE should be on activities that are</p>	<p>we have corrected, clarified or changed the wording according to the many useful comments.</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>at least on moderate intensities.</p> <p>Line 141---148: Ref 20 shows that there was no benefit of fitness if one is inactive. Compare with lines 104---105 "Physical fitness is also related to a genetic profile that could prevent CVD without exercise (no reference). Inconsistent?</p> <p>Line 178---179: "PA increases muscle mass", too unspecific for an increase in muscle mass a certain and specific PA need to be performed. Please define and elaborate more on this issue.</p> <p>Line 222: Lower limb? Should it be leg muscle strength or?</p> <p>Line 244: Mental health. This section could be extended as there is an extensive literature on the topic and also on exercise and the brain (much focus upon this area, eg WHO's Global Mental Action Plan 2013---2020)</p> <p>Line 297--- : How to handle this if intensity is only defined as absolute intensity- see comments on the table and general comments.</p> <p>Line 277---301: We miss a paragraph on muscle---strengthening activities and its link to the recommendations.</p> <p>Line 303 and on. Table x.1 See general comments +</p> <p>a) In general, this kind of table is not very helpful for individual exercise prescription. If it stays in the chapter it has to be checked regarding the data as well as consistency of headings etc.</p> <p>b) Not clear what source that have been used for this table. It does not seem totally accurate. For example: 6.4 km/h too low METs factor should be 5 (according to Ainsworth MET compendium 2000). RPE 12-13 seems also to be too low for the age group 80+ at the walking speed of 6.4km/h. Is the VO2max for this age group overestimated?</p> <p>c) Confusing to mix lifting with vigorous aerobic intensity activities, relevant activity?</p> <p>Line 312-327: Check the language. We suggest a less negative writing, for example instead of not linear (line 314), write curve-linear (especially as the Figure on page 10 is referred to and</p>	

Organisation/ Company	General and detailed comments	WG response and actions
	<p>used) and instead of no straight forward (line 323) is complicated.</p> <p>Line 355: Unclear. What is refined in WHO and PAG2008?</p> <p>Line 364 and 396: See general comments on recommendations on sedentary behavior. Line 382-384: Unclearly formulated. Quickly read, one thinks that 2-3 h of vigorous exercise is optimal</p> <p>Line 387: Specify</p> <p>Line 394: New line for muscle strengthening + there is a need to justify this in the text</p> <p>Line 399-406: Fig x2 “Physical activity” and “exercise”. Somewhat confusing to differ between “moderate exercise” and “moderate physical activity”. We suggest to only use the term “aerobic physical activity” , which is used in the NNR-recommendations and to define that term instead of make all definitions in Box 1 (line 53---87). A=aerobic physical activity – moderate intensity, B=aerobic physical activity – vigorous intensity See also general comments above</p> <p>Line 426: “Follow” instead of “use”</p> <p>Line 466-468: The wording could be misinterpreted as if 150 min is an upper limit.</p>	

NNR 2012: Public consultation – Chapter 10 (Fat and fatty acids)

Org/ Comp	General and detailed comments	WG response and actions
BoostOn Ltd, Finland	<p>General comments only</p> <p>Congratulations for getting such a huge task done. The chapter and the systematic review on fats will be a great resource for dieticians and other health care professional in coming years. I highly appreciate the work done by the group. As dietician who is interested in evidence based nutrition I want to point some possible inaccuracies in the text. These inconsistencies may not change the overall conclusions but they might become a subject of criticism and turbulent discussions. I hope the points I make herein will be addressed in the name of scientific pursuit and integrity.</p>	The working group acknowledges the comments.

Org/ Comp	General and detailed comments	WG response and actions
	<p>Cardiovascular disease (lines 409-480 in the chapter on fats) The group has chosen not to present any data on CHD mortality. This line of action is matter of debate. CHD mortality is an easy to detect and a reliable endpoint in the field of cardiology whereas composite CVD endpoint used in the text is rather weak end point (Ferreira-González et al. 2007). CHD mortality is cited as the most important endpoint when health authorities argue the effects of life style changes and North Carelia project in Finland and elsewhere. Therefore data on CHD mortality should also be presented. Hooper et al. (2012) showed in their systematic review that replacing saturated fat with unsaturated fat has no effect on CHD mortality. This information is lacking. The discordance in CHD incidence between different systematic reviews is not addressed in the text. It remains unclear why authors have chosen the systematic review of Mozaffarian et al. 2010 as primary source of data on CHD. Even if the authors cite Hooper et al. 2012 in other contexts they dismiss the findings of Hooper et al. 2012 on CHD incidence. Interestingly, Hooper et al. 2012a showed no significant effect on CHD when SFA was replaced with unsaturated fat. Discrepancy between the results of Mozaffarian and Hooper is explained by the selection of the randomized studies into the respective meta-analyses. Mozaffarian included non-randomized Finnish Mental Hospital Study (FMHS) and excluded Rose Corn Oil Study where as Hooper excluded FMHS and included Rose Corn Oil and further some other studies. Finnish Mental Hospital Study was not randomized study, and does not even fulfil the modern criteria of a cluster randomized trial. This is the reason why Hooper et al. excluded FMHS. Referring exclusively to any systematic review (ie. Mozaffarian et al. 2010) including a non-randomized study is not a right path to take. Furthermore, the group does not cite either in the draft text, or in their systematic review (Schawab et al. 2013), existing two other recent systematic reviews by Ramsden et al. (2011 & 2013 both based on RCTs). The group has excluded Ramsden et al. (2011) because its alleged poor quality (C). It's questionable if this rating is based on scientific facts because the latter systematic review by Ramsden et al. (2013) basically re-produced the results of the 2011 review and was published in high quality journal with high impact factor, British Medical Journal. Furthermore, one can argue that Mozaffarian's meta-analysis is poorest in quality because it contains one non-randomized trial (FMHS) and one Mediterranean pattern trial, ie. Oslo Diet Heart. Neither of them do fulfil the pre-determined criteria set by Mozaffarian et al. themselves. These two systematic reviews by Ramsden et al. showed that in terms CHD mortality only replacement of SFA with mixed n-6/n-3 oils seems to decrease CHD mortality. Replacement of SFA by sheer n-6 vegetable oils had no significant effect on CHD mortality. Ramsden et al. (2013) were also able to recover the old CHD mortality data from Sydney Diet Heart Study for the first time and therefore use it in the meta-analysis. In fact, Sydney Diet Heart Study is the only published randomized study favouring the use of SFA instead of PUFA in prevention of cardiac deaths. I cannot see any reason why this</p>	<p>CHD mortality has been presented in the chapter on CVD when appropriate. Regarding CVD outcomes in general, results from both RCTs and epidemiological studies vary with respect to effects on total CVD events or sub-categories. Although the SR by Hooper et al. (2012) did not find a significant effect on CHD, this was found for replacement of SFA with PUFA in the SR by Mozaffarian et al. (2010), also supported by the pooled analysis of cohort studies by Jakobsen et al. (2009). Studies using biomarkers of fat intake have also shown a protective effect of increased linoleic acid tissue concentration (Laaksonen et al. 2005, Clarke et al. 2009).</p> <p>Regarding the SRs by Ramsden et al. the review from 2010 was evaluated as low-quality study (C), and thus not included in the NNR5 SR. It actually also includes the Finnish Mental Hospital trial. The subsequent paper from 2013 was not included in the searches. It includes additional data from an Australian study, previously published and included in the SR by Hooper et al. (2012). It has not improved the quality of the study. In the updated analysis, some studies, which were included in the 2010 paper, have been excluded, without any explanations. The study was also commented by the British NHS, see http://www.nhs.uk/news/2013/02february/pages/omega-6-fat-diet-heart-disease-death-risk.aspx</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>new and crucial information should be left out from the analysis of the group even if the closing date for publication was February 2012. It is a common practise to leave a post-script or amendment at the final stage if something essential is emerging (see Skeaff & Miller's last paragraph 2009). In conclusion, both the lacking data on CHD mortality and data on CHD incidence by Hooper et al. challenge the groups's conclusion "convincing evidence that partial replacement of SFA with PUFA decreases the risk of CVD...". It seems that "probable" might be better estimation of overall evidence in terms of CVD (see also the discussion below regarding strokes that are part of CVD). Actually the group itself uses wording probable in the abstract of systematic review (Schwab et al. 2013 page 2) but in the draft text (lines 428) evidence is deemed convincing. These wordings are in conflict. Authors present the data on coronary heart disease (CHD) and cardiovascular disease under the same subtitle (Cardiovascular Disease). For some reason, which is not clear, stroke data is presented under separate subtitle "stroke". It would be better if composite cardiovascular end point such as Hooper et al. 2012 data would be presented under separate subtitle and coronary heart disease data also under separate subtitle, exactly how the group has done in case of strokes. Mixing CVD and CHD data without having stroke data in the same context blurs clarity.</p> <p>Strokes (subtitle, lines 482-485) In the text, the effect of all other fatty acids but omega-3 fatty acids on strokes is lacking. It is of interest that only stroke mortality is mentioned in the text and no data on stroke incidence is presented. This is in contrast to on how data on coronary heart disease (CHD) is handled. The stroke chapter also conveys a false signal, as if there was no data on strokes and saturated fat. Systematic review by Hooper et al. 2012, which is used as a key reference in the text elsewhere, concluded that replacing saturated with unsaturated fat has no effect on stroke incidence. Furthermore, in a systematic review of prospective cohorts by Siri-Tarino et al. 2010 saturated fat intake had no effect on strokes. In conclusion, saturated fat seems to be neutral in terms strokes and this should be stated in the text. It is difficult to see why the Siri-Tarino's meta-analysis was excluded due to alleged poor quality (C). Siri-Tarino's results regarding CHD and cohorts are almost perfectly in line with Skeaff & Miller 2009 (Figures 5-7) which is used as a key reference. It seems that uncomfortable systematic reviews have been excluded and comfortable ones included in order to being able to reach more coherent conclusion on saturated fats.</p> <p>Type 2 diabetes (lines 487) The text and systematic review by Schwab et al. do refer to two recent and actually largest RCTs showing that neither replacement of SFA with MUFA (Jebb et al. 2011) nor reduction of SFA content of diet (Tierney et al. 2011) has any effect on insulin sensitivity. Jebb et al. concluded "This study did not support the hypothesis that isoenergetic replacement of SFAs with MUFAs or carbohydrates improves Si." The effect of SFA on T2D incidence in prospective cohort studies has shown null effects in a meta-</p>	<p>In the abstract there was an unfortunate discrepancy with the text and summary table on this topic. The abstract has been corrected to be in line with the text and the summary table.</p> <p>In conducting the SRs, the evaluation of all studies was based on the overall quality of the individual studies as described in Chapter 3. The meta-analysis by Siri-Tarino was excluded based on the criteria described. Results for stroke from Hooper et al. 2012 have been included.</p> <p>In conducting the SRs, the evaluation of all studies was based on the overall quality of the individual studies as described in Chapter 3. These studies were excluded based on the criteria described.</p>

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	<p>analysis (an integral part of a narrative review , Micha & Mozaffarian 2010). Dietary pattern and multi-faceted studies were to be excluded in the systematic review, but still Finnish Diabetes Prevention Study is referred to in the draft text. Using the same analogy one could argue that Mediterranean diet studies such as Predimed-Reus study should also be presented in diabetes and CHD chapters.</p> <p>Cancer (lines 523-556) A recent meta-analysis of prospective cohorts and case-control studies showed that extra virgin olive oil (high MUFA) is associated with 34 % reduction in cancer rates. The effect was observed almost for all cancer subtypes (Psaltopoulou et al. 2011). This systematic review is lacking in the text.</p> <p>Cold pressed oils Common people are very interested in if cold pressed oils are better than refined ones. It would be valuable if the group commented on possible variable effects of refined and extra virgin oils. There is evidence that extra virgin oils rich in polyphenols may produce better health outcomes than refined vegetable oils. For example, extra virgin olive oil has delivered reductions in CRP and IL-6 in RCT (Fito et al. 2008). This study seems not to be part of the analysis. In order to increase the scientific transparency and trustworthiness of the recommendations, addressing these potential inaccuracies might be of value. References Fitó M, et al. SOLOS Investigators. Anti-inflammatory effect of virgin olive oil in stable coronary disease patients: a randomized, crossover, controlled trial. Eur J Clin Nutr. 2008 Apr;62(4):570-4 Hooper L, et al. Reduced or modified dietary fat for preventing cardiovascular disease. Cochrane Database Syst Rev 2012a;5:CD002137. Jebb et al. Effect of changing the amount and type of fat and carbohydrate on insulin sensitivity and cardiovascular risk: the RISCK trial. AJCN 2011; 010 Oct;92(4):748-58 Ferreira-González I et al. Problems with use of composite end points in cardiovascular trials: systematic review of randomised controlled trials. BMJ 2007;334:786 Micha R & Mozaffarian D. Saturated fat and cardiometabolic risk factors, coronary heart disease, stroke, and diabetes: a fresh look at the evidence. Lipids. 2010 Oct;45(10):893-905 Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing 1043 polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of 1044 randomized controlled trials. PLoS Med 2010;7:e1000252. Psaltopoulou T et al. Olive oil intake is inversely related to cancer prevalence: a systematic review and a meta-analysis of 13,800 patients and 23,340 controls in 19 observational studies. Lipids Health Dis. 2011 Jul 30;10:127. Ramsden CE et al. n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials. Br J Nutr. 2010 Dec;104(11):1586-600. Ramsden C et al. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. BMJ 2013;346:e8707. Skeaff CM, Miller J. Dietary fat and coronary heart disease:</p>	<p>Studies on single foods were excluded</p> <p>The effects of food items are not included in the fat chapter. This is a general principle with all nutrients. There is a separate chapter food based dietary guidelines about recommended food choices. This is why the effects of olive oil – or other oils – are not included in this chapter.</p>

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	summary of evidence from prospective cohort and randomised controlled trials. Ann Nutr Metab. 2009;55(1-3):173-201. Schwab et al. 2013. Systematic review on fats for NNR5. Manuscript Siri-Tarino P et al. Saturated fat, carbohydrate, and cardiovascular disease. American journal of clinical nutrition 2010;91;502-509 Tierney et al. Effects of dietary fat modification on insulin sensitivity and on other risk factors of the metabolic syndrome—LIPGENE. Int J Obes (Lond). 2011 Jun;35(6):800-9	
University of Bergen, Norway	<p>detailed and general comments are given below</p> <p>In general the chapter on fat and fatty acids in the NNR 2012 gives a good background for the given recommendations. Here are some few comments and suggested changes: It could be reasonable to add a sentence about algae as source of DHA and EPA (e.g. after line 321). “Several algae oils are also rich sources of DHA and EPA, often with a similar distribution of very long- chain n-3 fatty acids as found in fish.” (Lenihan-Geels G, Bishop KS, Ferguson LR: Alternative sources of omega-3 fats: can we find a sustainable substitute for fish? Nutrients 2013, 5(4):1301-1315).</p> <p>In line 415-417 it is written “A pooled analysis of data from 11 American and European prospective cohort studies showed a 20 % decreased risk of CHD in both men and women when 5 E% PUFA was substituted for equal amounts of SFA (Jakobsen et al. 2009).” This sentence could be misunderstood/interpreted reverse of what I assume is meant (and the findings from Jakobsen et al. 2009). I thus suggest re-writing this to e.g. “A pooled analysis of data from 11 American and European prospective cohort studies showed a 20 % decreased risk of CHD in both men and women when 5% of the energy was replaced from SFA to PUFA (Jakobsen et al. 2009).”</p> <p>In line 493, there is a missing space between the words “evidencefor”.</p>	<p>Algae oils may contain long-chain n-3 PUFAs. However, some contains negligible amounts of EPA and also contains DPA (n-6).</p> <p>This sentence has been modified as suggested.</p> <p>This has been corrected.</p> <p>Corrected</p>
National Food Agency, Sweden	<p>Detailed comments only</p> <p>90 Please revise this sentence as it is not clear out of what TFA is 2-4 % or out of what 18:1 n7 is 30-50% 120 must be provided by foods/ in the diet</p> <p>149 To relate “very high” to current consumption would improve understanding. The limited availability of n3, relative to n6 in our diet result in a difference in intakes, but not necessary an imbalance. Could intakes of n6 in the diet in the Nordic countries reach levels reported as “very high” and cause disturbances of metabolism? If the levels at which metabolism is disturbed is not known, but unlikely obtained in diets, suggest writing “extremely high” or “at levels obtained in experimental diets only” For example in Bjermo 2012, despite a very high intakes of LA (n6:n3 ratio was 14:1), arachidonic acid did not increase in comparison with a diet rich in SFA.</p> <p>174 The conversion of EPA and DPA is probably limited, and further desaturation to DHA is even more limited. It is not clear whether the sentence at line 176 refers to the conversion to EPA and DPA or the step to DHA, and 21 % should not be called limited. We</p>	<p>This has been clarified.</p> <p>This has been corrected.</p> <p>This paragraph has been modified as suggested.</p> <p>The references refer to conversion of ALA to EPA and DPA, and further to DHA, which is pointed out in the previous sentence. In this specific sentence it is also mentioned that the conversion <i>may be</i></p>

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	<p>suggest removing the numbers of conversion, and to write that conversion is limited and higher in women of reproductive age (ref Burdge, 2002). For example Mozaffarian, 2011 JACC, summarize the conversion as limited referring to literature suggesting much lower conversion than 21%.</p> <p>421 suggested</p> <p>554 Could the relation between “prudent” dietary patterns and dietary fat quality be mentioned briefly here?</p> <p>602 The overall cis-PUFA recommendation of 5-10 E% (out of which at least 1% n3) is not mentioned here. This might confuse the reader. Please clarify in the text, that out of all cis-PUFA (sum of n6 and n3) n3 fatty acids should be at least 1% and out of the recommended 3 % essential cis-PUFAS n6 (LA) and n3 (ALA), n3 should be at least 0.5%</p>	<p>limited. So, 21 % is not considered limited itself. The estimated conversion rate varies among studies and it is affected by several factors, as presented in the paragraph. Furthermore, it is of note that DHA is quite readily incorporated in cell membranes and this is why it may not be measurable in plasma lipids. In erythrocyte membranes the proportion of DHA is about 4-5 times higher than the one of EPA and about twice as high as the one of EPA (Takkunen et al., in press). The estimated conversion rates may be of interest to some of the readers and this is why they are kept in the final version.</p> <p>This has been corrected. A new sentence has been added in this paragraph according to the suggestion. The text has been modified according to the suggestion.</p>
Unilever Finland Oy	<p>General comments: We would like to express our support to the proposed NNR recommendations for total fat and fatty acids intake and welcome their good alignment with international guidelines. This consistency between health authorities is very important in our view both to make sure that populations receive consistent messages and to provide industry with clear guidelines for their products (re)formulations. We acknowledge that this fat chapter of the NNR recommendations has been based on a very thorough review of literature (Schwab et al.) mostly based on new evidence published between 2000 and 2010. We would however recommend that in some specific cases (indicated in our detailed comments) the present chapter on fat and fatty acids should also take into account scientific evidence published before or after this timeframe, to ensure that the NNR recommendations fully reflect the current advancement of science.</p> <p>Detailed comments: Rows 41-42: We suggest including an additional sentence indicating that saturated fat intakes in the Nordic countries are still above recommendations. Rows 83-86: We agree that plant sterols are poorly absorbed from the intestine. In this chapter it is mentioned that plant sterols are absorbed (5-15 %) from the intestine. But based on the literature less than 5% of plant sterols are absorbed (1.9% for campesterol, 0.5% for sitosterol) from the intestine. References: Bosner MS, Lange LG, Stenson WF, Ostlund RE Jr. Percent cholesterol absorption in normal women and men quantified with</p>	<p>The text has been modified as suggested.</p> <p>The sentence has been modified</p>

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	<p>dual stable isotopic tracers and negative ion mass spectrometry. J Lipid Res 1999; 40:302-8 Ostlund et al. Gastrointestinal absorption and plasma kinetics of soy Delta(5)-phytosterols and phytosterols in humans. Am J Phys Endo Metab 2002; 202:E911-6</p> <p>Rows 88-89: We propose to reformulate the sentence into: "TFA are chemically formed by partial hydrogenation of vegetable and fish oils (i.e. industrial TFA). They are also formed by natural biohydrogenation of fats in the rumen of cattle and sheep (i.e. ruminant TFA)".</p> <p>Rows 91-92: We recommend to include "partially" in this sentence: "Industrially partially hydrogenated oils", as TFA are not produced during full hydrogenation.</p> <p>Rows 148-150: We suggest that very high dietary intake of LA & ALA should be defined, ideally by giving a % of energy. Otherwise, such statement remains open to various interpretations.</p> <p>Rows 154-155: According to the FAO/WHO expert consultation of 2008, "Based on the evidence and conceptual limitation, there is no rational for a specific recommendation for n-6 to n-3 ratio, or LA to ALA ratio, if intakes of n-6 and n-3 fatty acids lie within the recommendation established in this report." We would therefore suggest to replace the sentence "there is no consensus about the optimal n-6/n-3 ratio in the diet" by "there is no evidence nor rational to define an optimal n-6/n-3 ratio in diet". Reference: Fats and fatty acids in human nutrition. Proceedings of the Joint FAO/WHO Expert Consultation. November 10-14, 2008. Geneva, Switzerland.</p> <p>Rows 157-158: We suggest specifying the high intakes of PUFA: what kind of PUFAs and what amount is considered as high intake? If the suggested reference below is not already taken into account we would like to suggest the authors of review the recent systematic review of randomized controlled trials (Johnson et al. 2012) which found no association between dietary linoleic acid intake and markers of inflammation in healthy persons. References: Johnson GH, Fritsche K. Effect of dietary linoleic acid on markers of inflammation in healthy persons: a systematic review of randomized controlled trials. J Acad Nutr Diet. 2012 Jul;112(7):1029-41.</p> <p>Rows 284-289: We are surprised to by the conclusions of this paragraph. The well-known meta-analysis of 60 randomized controlled trials from Mensink et al. in 2003 clearly showed that replacing carbohydrates or saturated fatty acids with monounsaturated fatty acids or polyunsaturated fatty acids lowered LDL-cholesterol as well as the ratio of serum total to HDL-cholesterol. We also propose examples of individual studies in the references below (e.g. Mensink Lancet 1987; Grundy AJCN 1988). We suggest reconsidering this paragraph taking into account the totality of scientific evidence, including older studies as this field has been heavily studied before 2000. References: Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr 2003 May;77(5):1146-55. Mensink RP, Katan MB. Effect of</p>	<p>The sentence has been modified as suggested.</p> <p>The word "partially" has been added.</p> <p>Paragraph has been revised with new references.</p> <p>The text has been modified according to this suggestion</p> <p>The recommended intake of PUFA is 5-10 E%.</p> <p>The effect of replacing SFA by cis-MUFA or PUFA on total/HDL cholesterol ratio has been added</p>

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	<p>monounsaturated fatty acids versus complex carbohydrates on high-density lipoproteins in healthy men and women. Lancet. 1987 Jan 17;1(8525):122-5. Grundy SM, Florentin L, Nix D, Whelan MF. Comparison of monounsaturated fatty acids and carbohydrates for reducing raised levels of plasma cholesterol in man Am J Clin Nutr. 1988 Jun;47(6):965-9.</p> <p>Row 344: We suggest adding a few more recent references here. After KANWU, two large RCT's (LIPGENE and RISCK) were published that assessed the effect of MUFA in comparison to SFA and/or carbohydrates. Both studies did not find an overall effect on insulin sensitivity. As a result, we suggest to dampen the current conclusion of a probable positive effect of cis-MUFA in comparison with SFA or carbohydrates (line 345-348). References: Tierney AC, McMonagle J, Shaw DI, Gulseth HL, Helal O, Saris WH, et al. Effects of dietary fat modification on insulin sensitivity and on other risk factors of the metabolic syndrome--LIPGENE: a European randomized dietary intervention study. Int J Obes (Lond) 2011 Jun;35(6):800-9. Jebb SA, Lovegrove JA, Griffin BA, Frost GS, Moore CS, Chatfield MD, et al. Effect of changing the amount and type of fat and carbohydrate on insulin sensitivity and cardiovascular risk: the RISCK (Reading, Imperial, Surrey, Cambridge, and Kings) trial. Am J Clin Nutr 2010 Oct;92(4):748-58.</p> <p>Row 361: We suggest including also the most recent evidence here. One recent study (Bjermo et al, 2012) showed a benefit of PUFA on fasting insulin when compared to SFA. Reference: Bjermo H, Iggman D, Kullberg J, Dahlman I, Johansson L, Persson L, et al. Effects of n-6 PUFAs compared with SFAs on liver fat, lipoproteins, and inflammation in abdominal obesity: a randomized controlled trial. Am J Clin Nutr 2012 May;95(5):1003-12.</p> <p>Row 520: We propose to replace 'Diet rich in fat' by 'Diet rich in saturated fat' as lowering saturated fat and not total fat was part of the lifestyle modification.</p> <p>Rows 525-526: We suggest to take into account the FAO/WHO 2008 expert consultation which says (p.102) : " Most epidemiological studies do not show any association of LA and n-6 FA with colorectal cancer, prostate cancer and breast cancer. There are no data providing evidence of a link between essential fatty acids and cancer risk, and thus no recommendation can be made." Reference: Fats and fatty acids in human nutrition. Proceedings of the Joint FAO/WHO Expert Consultation. November 10-14, 2008. Geneva, Switzerland.</p> <p>Row 584: We suggest amending the sentence into: "Detailed recommendations on intakes of individual saturated fatty acids are so far poorly justified".</p> <p>Row 632: We suggest very high PUFA intakes should be specified (similarly to row 148).</p>	<p>In conducting the SRs, the evaluation of all studies was based on the overall quality of the individual studies as described in Chapter 3. Both the LIPGENE study and the RISCK study were excluded in the SR on fat based on the criteria described, but are mentioned in the SR discussion.</p> <p>In the study by Bjermo et al. the subjects were abdominally obese, 15 % of the subjects had type 2 diabetes. Majority of the subjects had increased liver fat percentage. So, the inclusion criteria were not met for including this study in the SR.</p> <p>The sentence has been modified by replacing the word "rich" by the word "high". The sentence is now in line with the preceding sentence. This comment is acknowledged.</p> <p>The text has been modified accordingly.</p> <p>The high PUFA intake has been defined as >10 E%, line 634 in the version available at public consultation.</p>

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Private, Sweden	<p>General comments only</p> <p>In the described studies of the effects of different kinds of fatty acids on CVD, there is no referrens to the amount of carbohydrates consumed by the persons who participated in the research. Recent studies on the effect of carbohydrate intake on CVD show that it is carbohydrates, not the SFA, who are the culprits. Carbohydrate consumption is closely followed by the release of insuline into the blood vessels. The insuline transformes all the carbohydrates that are not immedeately consumed, into fat (for later use- but often never used...). Whereas any excessive SFA you eat is transported out of the body via the rectum, which shows in the different nature of the faeces. So, it is not the SFA intake that leads to CVD, it is the fat derived from the carbohydrates, via insuline. There is not much use in comparing the effects of SFA, MUFA, PUFA etc on CVD, if you don't take the carbohydrate intake into consideration. It is time to let the NNR take a different path. The NNR 2012 is only "more of the same". But we can no longer disregard the 50 000+ people out there who have benefited from high intake of SFA and low intake of carbohydrates. The results arrive very soon, figuring effects like significantly lower bloodpressure and cholesterol levels, no more Diabetes2, in many cases dramatic weightloss. It is a mistake to believe that the good effects of the low-carb, high-fat diet is due to weightloss alone. There are people who have not lost any weight at all (mainly because they were not overweight in the first place), but they follow the diet to have normalised bloodpressure and cholesterol levels without medication, which they usually have after 3-4 months on the diet. I recommend reading some of the new blogs on the subject, where innumerable people are telling us about their success stories. http://www.kostdoktor.se/ http://traningslara....m-epidemiologi/</p>	<p>There is convincing evidence that the quality of dietary fat affects the risk of CVD. There are several references in the text regarding the health effects of the quality of dietary fat. The quality of carbohydrates is also of significance. This issue has been dealt with in the carbohydrate section.</p>
Lantmännen Ekonomisk Förening, Sweden, Norway and Denmark	<p>Detailed comments only</p> <p>441 carbohydrates, no beneficial effect on CHD risk has been found. Include: However, the consumption of high quality carbohydrates from cereals such as oat and barley are proven to lower the risk of CVD due to the soluble fiber betaglucan (EFSAs approved health claims)</p>	<p>This comment is not considered relevant for the fat chapter. This is commented in the carbohydrate chapter.</p>

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LRF Dairy Sweden	<p>General comments:</p> <p>Dairy Sweden is concerned about the time frame for commenting the fifth batch of nutrition recommendations. In total 807 pages are published in batch 5 to be read and commented on by 18 July the latest, in the Swedish holiday season. This is not respectful to the scientific community and stakeholders giving feedback on the recommendations. This procedure lowers significantly the reliability of the final recommendations and also on the process and transparency. If not the background material is open for comments, it is of no use to comment on the chapters on recommendations based on the background material. Several of the systematic reviews are not even submitted to Food & Nutrition Research, and have therefore not gone through the review process. The order of the total process of establishing nutrition recommendations is questionable. LRF Dairy Sweden comments therefore on both the background material and on the chapter on recommendations on fat and fatty acids. General comments on Effects of the amount and type of dietary fat on cardiometabolic factors and risk of cardiovascular disease, type 2 diabetes and cancer: s systematic review. Schwab et al. Manuscript to be published in Food & Nutrition Research: LRF Dairy Sweden is impressed over the transparent systematic review of the literature, especially the transparent procedure to identify and include/exclude published papers. The evaluation of papers is, however, not transparent. It is not evident how the evaluations of the results of the publications were made, and how the final decisions regarding strength of evidence was made. Further, this text is lacking a reflection regarding the number of publications available within the area in question and their quality. The criteria convincing, suggestive, probable unlikely should be defined in the paper to help the reader understand the result. If the expressions are not defined and differ across the paper, the final conclusions could be misleading. The conclusions on different effect of fat and fatty acids on biomarkers and health outcomes are not satisfactory. Just counting numbers of papers with positive/negative/neutral effect not taking the number of participants in each study into account is misleading. To be reliable the evaluation on health outcomes of fatty acids should take into account the effects of individual fatty acids and not only saturated fatty acid, monounsaturated acids and polyunsaturated fatty acids. This is done for omega-fatty acids. Saturated fatty acids are a heterogeneous group with different metabolic pathways and effects on health. It is unclear how the risk factors are judged in relation to actual health outcomes. This should be explained in the paper.</p> <p>General comments</p>	<p>This comment is acknowledged.</p> <p>The background and principles for setting dietary reference values are described in Chapter 3, which has been published. It also includes criteria for the quality and evidence grading. The specified inclusion criteria and number of studies included in the quality grading has been clearly presented in the SR which was available as a supporting material during the consultation period.</p> <p>The whole range of SFAs is present in the diet in Nordic countries. The effects of some individual SFAs (and PUFAs) on serum lipids are mentioned in the chapter. Effects of individual SFAs, were, however, not considered relevant to include in the SR, as it focuses on the whole diet. Effects of individual SFAs are largely food-related, which is covered in the chapter on dietary patterns and foods. The intake of omega-3 and omega-6 fatty acids can vary quite much in the Nordic countries, e.g. due to fish consumption. Omega-3 fatty acids are also available as supplements which affect the ratio of omega-6/omega-3 in the diet.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>Fat and fatty acids: The most important background document evaluating the effects of amount and type of dietary fat on cardiometabolic risk factors and risk of cardiovascular disease, type 2 diabetes and cancer has not yet been submitted to the Food & Nutrition Research for review and publication. To base nutrition recommendations on a paper not reviewed is unfair to the scientific community and stakeholders giving feedback on the recommendations. The reliability of the recommendations is therefore lowed and makes the recommendations less significant. The effect of fat and fatty acids on biomarkers and health outcomes are classified as limited, probable, convincing etc. These terms are not defined. They should be defined to make the final recommendations understandable. The references especially in the first part of the chapter on fat and fatty acids should be updated. References to support statements are often missing. Please improve this. In the systematic review of Schwab et al. the procedure of identifying high quality papers to take into account is described extensively. The evaluation procedure is however not well described and accomplished. It is not sufficient and reliable only to count papers reporting positive/negative/neutral results without taking the number of participants into account. This makes the systematic report less reliable than if a proper meta-analysis had been performed. To base the recommendations on this systematic review in its present state in inappropriate. The paper should be improved before the recommendations are finalized. Not only individual PUFAs should be evaluation but also the effect of individual saturated fatty acids should be evaluated. Individual fatty acids have different metabolic pathways and effects on biomarkers and health outcomes.</p> <p>Recommendations on individual fatty acids has been made by the French Food Safety Agency, AFSSA– Request no. 2006-SA-0359, OPINION of the French Food Safety Agency on the update of French population reference intakes (ANCs) for fatty acids http://www.anses.fr/sites/default/files/documents/NUT2006sa0359EN.pdf Regarding saturated fatty acids AFFSA states: “Saturated FAs cannot be regarded as a unit because they differ in their structure, their metabolism, their cell functions and even their deleterious effects in case of excess. From now on it is advisable to distinguish the subgroup of ‘lauric, myristic, and palmitic acids’ which is atherogenic in the event of excess. Based on observation studies and not formal intervention studies, AFSSA established a maximum intake of 8% of EI for this subgroup. Other saturated FAs, particularly the short and medium chains, have no known harmful effect and some of them even have rather beneficial effects. However, at present, it is not possible to establish recommendations for them and AFSSA considers it prudent to maintain an intake of total saturated FAs lower than 12% of EI.” LRF Dairy Sweden recommends the same approach as the French Food Safety Agency. The recommendation on <10E% of saturated fatty acids is not substantiated. The evidence for this recommendation should be included in the text.</p>	<p>It is unfortunate that the SR was not published before the public consultation of the fat chapter. However, the SR was available as a supporting material during the consultation in a form which had been modified according to the reviewers’ comments.</p> <p>These terms used for evidence grading are defined in Chapter 3, please see response above.</p> <p>The number of participants has been taken into account. The specified inclusion criteria and number of studies included in the quality grading has been clearly presented in the SR, please see response above.</p> <p>Regarding the non-inclusion of individual SFAs in the SR, please see response above.</p> <p>The whole range of SFAs is present in the diet in Nordic countries. The effects of some individual SFAs (and PUFAs) on serum lipids are mentioned in the chapter. Effects of individual SFAs, were, however, not considered relevant to include in the SR, as it focuses on the whole diet. Effects of individual SFAs are largely food-related, which is covered in the chapter on dietary patterns and foods.</p> <p>Furthermore, there is data in abundance to show the beneficial effect of replacing part of SFA with unsaturated fat. There is also general agreement in diet recommendations throughout the Westernized world on this issue. The proportion of fatty acids groups, i.e. SFA, MUFA and PUFA has to be taken into account. If the intake of SFA is high, even in the case most of them would be short and medium chain fatty acids, the intake of unsaturated fatty acids is usually lower than recommended or the diet is higher in fat than recommended.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>To give nutrition recommendations on individual nutrients without taking individual foods and their matrix into account is misleading. Regarding milk/dairy foods an adequate intake is associated with a reduce risk of ischemic heart disease, myocardial infarction, stroke, hypertension, type 2 diabetes, metabolic syndrome and colorectal cancer. It is also associated with improved bone mineral density and there is no association between consumption of dairy foods and weight change/obesity. It is therefore misleading to restrict one component in milk as the fat and individual fatty acids. This should seriously be taken into account when making the recommendation on fatty acids. LRF Dairy Sweden strongly suggest that it should be made clear that concerns about saturated fats do not relate to core/foundation diet foods such as regular-fat milk, yoghurt and cheese as these foods are associated with a reduced risk of myocardial infarction, ischemic heart disease and stroke, not an increased risk. In Riksmaten the contribution of fat from milk and fermented milks was 8% as from cheese. From milk and fermented milk the contribution of saturated fatty acids was 13% and from cheese 12 %. Discretionary food like cakes, confectionary, sauces and snacks contributes with 20 %of the total fat intake and 18 % of the saturated fatty acids. Fat and saturated fatty acids from milk and dairy product are accompanied by a high amount of most vitamins and minerals, which is not the case for discretionary foods. It is thus very important to discriminate between core foods and discretionary foods in the overall dietary guidelines as well as in recommendations regarding individual nutrients.</p> <p>Detailed comments:</p> <p>Page 2, line 48-55. Results from Sweden should be included.</p> <p>Page 2, lines 65-78. The metabolism of individual saturated fatty acids should be included.</p> <p>Page 3, lines 88-98. The information in this paragraph should be updated with the latest information on trans fatty acids of industrial and animal origin (ruminant), respectively. Some studies indicate that there are different physiological effects of industrial and ruminant TFAs. This is an area where active research is going on. Is there actually evidence enough to draw the conclusion that industrial and ruminant TFAs have the same physiological effects? Until scientific evidence can conclude the physiological effects of industrial and ruminant TFAs this should be written in the actual nutritional recommendations.</p> <p>Further, in the FNR manuscript, page 3, last line it is written that studies on TFA solely was not included. If not, why is then TFA mentioned in the recommendations? Please explain this. Suggestions on recent references on TFA: • Trans fatty acids and cardiovascular disease. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. N Engl J Med. 2006 Apr 13;354(15):1601-13. • Ruminant or industrial sources of trans fatty acids: public health issue or food label skirmish? Willett W, Mozaffarian D. Am J Clin Nutr. 2008 Mar;87(3):515-6 • World-wide consumption of trans fatty acids. Craig-Schmidt MC.</p>	<p>The chapters on nutrients primarily focus on effects of single nutrients. However, most foods contain a mixture of nutrients, and this aspect is covered in the chapter on dietary patterns, which includes associations between some food groups and dietary patterns with health effects. Based on data for individual nutrients, foods and dietary patterns, some general characteristics of a healthy Nordic diet are given.</p> <p>Regarding the population based evidence of the association of e.g. yoghurt and CVD, no firm conclusions can be made at nutrient level. It is of note that high intake of some food item is usually associated with lower intake of some other food item(s).</p> <p>These results have been included.</p> <p>This comment is acknowledged.</p> <p>The recommendation of TFA is set at as low as possible. This is why the source of TFA was not considered relevant. It is also of note that the food items high in TFA are generally also high in SFA.</p> <p>Studies on TFA were not included in the SR due to current low intakes in the Nordic countries. However, it was considered important to point out in the recommendations that the intake of TFA should be as low as possible, since there is evidence on the detrimental health effects of TFA. This is in line with diet recommendations throughout the Westernized world.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>Atheroscler Suppl. 2006 May;7(2):1-4 • Intake of ruminant trans fatty acids in the Danish population aged 1-80 years. Jakobsen MU, Bysted A, Andersen NL, Heitmann BL, Hartkopp HB, Leth T, Overvad K, Dyerberg J. Eur J Clin Nutr. 2006 Mar;60(3):312-8. • Intake of ruminant trans fatty acids and risk of coronary heart disease. Jakobsen MU, Overvad K, Dyerberg J, Heitmann BL. Int J Epidemiol. 2008 Feb;37(1):173-82. • Motard-Bélanger A, Charest A, Grenier G, Paquin P, Chouinard Y, Lemieux S, Couture P, Lamarche B. Study of the effect of trans fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease. Am J Clin Nutr. 2008 Mar;87(3):593-9. • JM, Destailats F, Malpuech-Brugère C, Moulin J, Bauman DE, Lock AL, Barbano DM, Mensink RP, Bezelgues JB, Chaumont P, Combe N, Cristiani I, Joffre F, German JB, Dionisi F, Boirie Y, Sébédio JL. Do trans fatty acids from industrially produced sources and from natural sources have the same effect on cardiovascular disease risk factors in healthy subjects? Results of the trans Fatty Acids Collaboration (TRANSFACT) study. Am J Clin Nutr. 2008 Mar;87(3):558-66. • Ruminant and industrially produced trans fatty acids: health aspects, Steen Stender, Arne Astrup and Jørn Dyerberg, Citation:Food & Nutrition Research 2008. DOI: 10.3402/fnr.v52i0.1651 • Ganguly R, Pierce GN. Trans fat involvement in cardiovascular disease. Mol Nutr Food Res. 2012 Jul;56(7):1090-6. • Lacroix E, Charest A, Cyr A, Baril-Gravel L, Lebeuf Y, Paquin P, Chouinard PY, Couture P, Lamarche B. Randomized controlled study of the effect of a butter naturally enriched in trans fatty acids on blood lipids in healthy women. Am J Clin Nutr. 2012 Feb;95(2):318-25. http://ajcn.nutrition.org/content/early/2011/12/28/ajcn.111.023408.full.pdf+html • Wang Y, Jacome-Sosa MM, Proctor SD. The role of ruminant trans fat as a potential nutraceutical in the prevention of cardiovascular disease. Food Res Int. 2012 Jul;46(2):460–468. • Bendsen NT, Christensen R, Bartels EM, Astrup A. Consumption of industrial and ruminant trans fatty acids and risk of coronary heart disease: a systematic review and meta-analysis of cohort studies. Eur J Clin Nutr. 2011;65:773-783. • Gebauer SK, Chardigny JM, Jakobsen MU, Lamarche B, Lock AL, Proctor SD, Baer DJ. Effects of Ruminant Trans Fatty Acids on Cardiovascular Disease and Cancer. A Comprehensive Review of Epidemiological, Clinical, and Mechanistic. Adv Nutr. 2011 Jul;2(4):332-54. http://advances.nutrition.org/content/2/4/332.full.pdf+html • Wang Y, Proctor SD. Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels. Br J Nutr. 2013 Apr 18:1-15. [Epub ahead of print] Page 3, line 89: “forestomach” should be replaced with “rumen”. Page 3, line 105. It is important to mention that the trans 10, cis 12 CLA is mainly found in industrial -fat, and the content naturally occurring in milk fat is in very low proportions. Please correct this sentence. Include text and references on other inflammation markers and oxidative stress. Pages 3-4, lines 217-243. Insert references on the health effects of cholesterol in the diet.</p>	<p>The source of t10,c12 CLA has been mentioned.</p> <p>Since cholesterol was not included in the SR, it was considered justified to refer to the recommendations of other instances.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>It is not relevant just to cite other organizations recommendations.</p> <p>Page 6, lines 224-232. According to Riksmaten 2010-11 the daily intake of cholesterol is on average 288 mg. The content of cholesterol in milk fat is 3.2 mg/g fett (Lindmark-Månsson, H., Fondén, R. & Pettersson, H.-E. 2003. Composition of Swedish dairy milk. International Dairy Journal, 13, 409-425). Also according to Riksmaten the contribution of fat from milk and fermented milk is 8% of the total intake of 77.3 g/day. This means that 6.2 g of fat originates from milk and fermented milk. The contribution of cholesterol from milk and fermented milk is thus 6.2 g fat x 3.2 mg cholesterol/g fat=19.8 g cholesterol per day. The percentage contribution from milk and fermented milk is 6.9 per day. The contribution from cheese is similar so in total 14% of the dietary cholesterol comes from dairy products. The sentence on line 224 should be specified with the daily contribution of cholesterol from different food sources or omitted.</p> <p>Page 6, line 245. The new systematic review has been done to constitute the scientific base for the recommendations on fat and fatty acids. This new review should be the foundation to the new recommendations. Older recommendations should therefore not be used as references or scientific material. Older findings/views should thus be omitted.</p> <p>Page 6, line 249. Delete the reference Valsta et al 2010, or make a more trustworthy evaluation of the decline in cholesterol levels. Many factors influence the prevalence of CVD, such as lifestyle factors including physical activity, overweight/obesity, smoking etc. These factors are not included in the included reference.</p> <p>Page 7, line 273. The effect of substituting saturated fat with UFA on the ration total cholesterol should be included in the evaluation since this is a marker taking both LDL and HDL into account at the same time. In the included studies the intake of SFA was between 13-19 E%, which is almost the double recommended intake. This intake is not relevant.</p> <p>Page 7, line 310: Why is it supposed that long N-3 PUFAs should be hypocholesterolemic? Please omit "are not hypocholesterolemic, but".</p> <p>Page 10, line 445. Referring to old studies for China like Campell et al 1998 and Campbell and Chen 1999 is irrelevant for the present Nordic situation, see comment on Page 8, line 342.</p> <p>Page 11, lines 488-496. The systematic review of Schwab et al, page 14, concludes that "there is limited –suggestive evidence of a direct association between 18:0 and total SFA with the incidence of T2DM, but this should be interpreted with caution since major SFA including 18:0 are weak biomarkers of dietary SFA intake." This conclusion should be</p>	<p>This comment is acknowledged.</p> <p>The SR is the major basis, but other scientific reports/papers are also used, where appropriate.</p> <p>It is agreed that other factors affect CVD risk as well. The reference of Valsta et al. is considered as relevant and the suggestion of omitting this reference is not accepted.</p> <p>Replacing part of SFA with unsaturated fat had a favourable, i.e. decreasing effect, on LDL-C concentration. The effect on HDL-C concentration was neutral according to the SR. The meta-analysis by Mensink and Katan (1992) suggested that replacing saturated fat by cis-MUFA may even be favourable, i.e. increasing effect.</p> <p>This sentence has been modified as suggested.</p> <p>It is agreed that it is striking that old studies conducted in China are referred to. However, these references were considered relevant within the context of the sentence in question, i.e. very low intake of both fat and saturated fat.</p> <p>There is clarifying text included in the section starting on line 506. Furthermore, it is of note, that the text which has been referred to in this comment deals with the proportion of fatty acids in plasma or plasma fatty acids, not the intake of single fatty acids as such. Regarding C18:0, please see the response above.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>included in the chapter instead of results from other random studies, line 488 "Total and SFA intake were associated with a higher risk of type 2 diabetes..." .</p> <p>Page 12, line 521. The systematic review of Schwab et al concludes on page 26 that "In summary, evidence for a direct association between total fat and the risk of T2DM is limited and inconclusive." Also the systematic review of Schwab et al, page 14, concludes that "there is limited –suggestive evidence of a direct association between 18:0 and total SFA with the incidence of T2DM, but this should be interpreted with caution since major SFA including 18:0 are weak biomarkers of dietary SFA intake." To suddenly not taken these conclusions seriously and replacing it with an individual study of Lindström et al 2012 is misleading. Doing so means that the systematic review is invalid and should not be referred to. Delete the Lindström reference and quote the systematic review correctly.</p> <p>Page 12, line 542. The conclusion on fat and fatty acids in relation to ovarian cancer is confusing; please check of the conclusion is correct. According to the systematic review of Schwab et al concludes on page 19 the conclusions on the relation between fat and fatty acids and ovarian cancer is suggestive. This conclusion is based on 4 publications. In Prentice et al 2007 found in the manuscript of the systematic review, ovarian cancer is not mentioned. In Genkinger et al. 2006 it is concluded in the abstract and in the text "A weakly positive, but non-linear association, was observed for saturated fat intake (pooled multivariate RR=1.29, ...). This statement is not substantiated in the results.</p> <p>Page 13, line 590. Please inset the scientific evidence for the recommended intake of SFA and TFA to less than 10E%, and distinguish between industrial and natural trans fat.</p> <p>Page 13, line 593. It is important to distinguish between industrially produced TFA and natural TFA as the scientific evidence for the harmfulness of industrial trans fatty acids are substantiated. This is not the case for natural trans fatty acids.</p> <p>Page 14-15: In the recommendations to children 6-23 months text is refereeing to other recommendations. How come? Has this area not been subject to a literature review? Is this explained somewhere in the NNR text? If not, please state that.</p> <p>Page 26, figure 1: Why is not the n-9 series included?</p>	<p>The reference of Lindström et al. is considered relevant, since it is conducted in a Nordic country, it is well controlled, and long term. It is not published after the search and this is why it is not included in the SR.</p> <p>Ovarian cancer is included in the paper by Prentice 2007. Risk was reduced over time.</p> <p>The recommendation of saturated fat has been set to <10 E% and the one of TFA as low as possible. This is why the source of TFA is not considered relevant. It is also of note that the food items high in TFA are generally also high in saturated fat, which are not recommended, please see the chapter on food based dietary guidelines.</p> <p>The evidence for distinguishing between ruminant and industrial TFA is far from evident, especially at the current low intakes. It is of note that sources of ruminant TFA are also high in SFA.</p> <p>The recommended fat intake for 6-23 months was reviewed and updated according to new literature updates such as other organizations' and countries' recommendations. There is evidence to a change similar to that for adults and the recommendation of total fat intake for this age group is now 30E% - 40E% instead of 30E% - 35E% in the NNR 2004. A systematic review for fat intake of small children and infants especially was not made.</p> <p>In figure 1, the desaturation and elongation of n-3 and n-6, i.e. essential fatty acids, has been presented, since these are the focus of that section. Metabolism of SFA and n-7 and n-9 unsaturated fatty acids can be found in text books.</p>

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National Institute for Health and welfare, Finland	<p>- systematic review (in press) seems to be thorough and extensive</p> <p>Detailed comments:</p> <p>- P 4, line 245. The text in the following chapter is rapidly moving from dietary fat to associations between serum cholesterol and chronic diseases. Could be add the term serum where appropriate? Dietary fat and fatty acid composition has been linked to risk of cardiovascular diseases, certain types of cancer, obesity and gallstones. The relatively high amount of fat, especially SFA, of the diet in the Nordic countries during the 1960s and 1970s has contributed to the high prevalence of cardiovascular diseases (Valsta et al. 2010). LDL-cholesterol has been identified as an important and causal risk factor for atherosclerosis, while a high HDL- cholesterol concentration and a low LDL-to-HDL-cholesterol ratio are associated with the reduced risk of atherosclerosis.</p>	The text has been modified as suggested.
Norwegian Directorate of Health	<p>General comments:</p> <p>We endorse the main conclusions and recommendations in this chapter. However, we have some comments that we ask the NNR-project group to take into account when revising the draft. Please clearly state NNR-conclusions regarding intake of EPA+DHA 250 mg/d (EFSA 2010), and the ratio of n-6/n-3, respectively. The description of trends in dietary content of fatty and fatty acids should be updated.</p> <p>Detailed comments:</p> <p>36-40. The description of trends in dietary content of fatty and fatty acids should be updated, and trends for trans fatty acids mentioned. The following suggestion is mainly based on Norwegian dietary data, and the described trends may not be valid for all Nordic countries. The dietary content of fat and fatty acids has changed much in the Nordic countries during the last decades. The total fat content decreased from the 1970s until the 1990s. After being rather stable for several years the dietary fat content has again increased in recent years in some Nordic countries. The content of saturated fatty acids have shown a similar trend as total fat, e.g. first it decreased, then leveled off, and in</p>	<p>The intake of EPA+DHA has been brought up more clearly, as well as the trends of the intake of fat and fatty acids.</p> <p>The text has been modified according to this suggestion.</p>

Org/ Comp	General and detailed comments	WG response and actions
	<p>recent years increased in some countries. The dietary content of trans fatty acids has decreased in all Nordic countries since the 1990s, caused by reduced use of partially hydrogenated oils in food production. Now the dietary content of trans fatty acids is on average below 1 E%. The dietary content of polyunsaturated fatty acids increased from the 1960s til the 1980s and has since been rather stable.</p> <p>46-7. When NNR does not give a recommendation for n-6/n-3 ratio, you should delete this line from table 11.1. Foot note ** for TFA Norway, 2000 may be changed to 2005-9. PUFA Norway: you may just use the average for men and women together e.g. 6.2 g/d.</p> <p>48-55. In this section the formulations and the systematics may be improved. - Information about Swedish trends is lacking - Iceland only information about dairy fat, if you have data about fatty acid trends please include. - Norway please do not mix together saturated and trans fat. Suggestion: "In Norway the dietary content of saturated fatty acids decreased from 16 E% in 1980 to 14 E% in the 1990s, and in recent years it has again increased to 16 E% in 2011. The dietary content of trans fatty acids decreased from 4 E% in the 1970s to 1 E% round year 2000, and it has been below 1 E% the last decade." 55. "But recently fat intake has stagnated." What do you mean? The decline in fat continues or do you mean that the decline has stalled?</p> <p>88-9. You may add that trans f.a. also can be formed in the process of deodorization of edible oils and that fat from animals with only one stomach as pigs also contain trans f.a. However, the proportion of trans f.a. is lower in fat from pigs than in fat from ruminants.</p> <p>125. Please add a reference.</p> <p>147-50. It is a bit difficult to follow the logic here. "...n-3 ..higher affinity for the enzymes. Therefore, a high intake of LA may disturbof n-3.."</p> <p>155. Please clearly state the conclusion by NNR regarding n-6/n-3 ratio. See also line 634-5.</p> <p>176. "Conversion – up to 21 % vs. 8 %. Please explain this more clearly.</p> <p>522. Do you have some data concerning intake of trans f.a. and risk of type 2-diabetes?</p> <p>578. "not exceed 40 E%." – Please comment or explain why.</p> <p>593-4. Please do not mix saturated and trans f.a. in this sentence. Furthermore you may consider using the same expression regarding the one-third vs. to-third proportions as in the main recommendation in chapter 1.</p> <p>634-5. Please state NNR-conclusions regarding intake of EPA+DHA 250 mg/d (EFSA 2010), and the ratio of n-6/n-3, respectively.</p>	<p>The intake data from Norway has been modified according to the suggestion.</p> <p>The text has been modified as suggested.</p> <p>The text has been modified as suggested.</p> <p>The text has been modified.</p> <p>The text has been modified.</p> <p>The texts have been modified.</p> <p>The text has been modified.</p> <p>This issue was not included in the SR. This is why it is not included in the chapter either.</p> <p>Explanation has been added.</p> <p>The text has been modified as suggested.</p> <p>Conclusion is included. A statement on EPA+DHA has been included.</p>
AarhusKarlshamn AB, Sweden	<p>General comments:</p> <p>Line numbers 59 and 96 - 98. In the document, hydrogenated vegetable oils are referred to as a major contributor to the intake of trans fatty acids in the Nordic countries. To the best of our information there is no supply of partly hydrogenated vegetable oils to the</p>	<p>The text on line 59 in the version under consultation refers to SFA in addition to TFA and this is why it has not been modified. The latter comment has been taken into account and the sentence has been</p>

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	<p>Nordic food industry. This is also the case for countries from which import of finished packed food products to the Nordic market is relevant. Therefore we must conclude that dairy products and meat are the only relevant source of trans fatty acids in the Nordic diet.</p> <p>Detailed comments:</p> <p>Line 147 - 155 Though no consensus on the optimum ratio of n-6 / n-3 has been established there is an indication that the ratio n-6 / n-3 might be too high in a modern, Western diet. Consequently a continued focus on poly unsaturated fatty acids without a simultaneously focus on alpha-linolenic acid may be counterproductive.</p>	<p>modified accordingly.</p> <p>The comment is acknowledged.</p>
Vitenskaps-komiteen for mattrygghet, Norway	<p>General comments only</p> <p>It would be helpful if it was commented on why the systematic review of NNR on fats and fatty acids come to other conclusions than previous reports from other competent bodies e.g EFSA's "Scientific Opinion on the substantiation of health claims related to eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), docosapentaenoic acid (DPA) and maintenance of normal cardiac function (ID 504, 506, 516, 527, 538, 703, 1128, 1317, 1324, 1325), maintenance of normal blood glucose concentrations (ID 566), maintenance of normal blood pressure (ID 506, 516, 703, 1317, 1324), maintenance of normal blood HDL-cholesterol concentrations (ID 506), maintenance of normal (fasting) blood concentrations of triglycerides (ID 506, 527, 538, 1317, 1324, 1325), maintenance of normal blood LDL-cholesterol concentrations (ID 527, 538, 1317, 1325, 4689), protection of the skin from photo-oxidative (UV-induced) damage (ID 530), improved absorption of EPA and DHA (ID 522, 523), contribution to the normal function of the immune system by decreasing the levels of eicosanoids, arachidonic acid-derived mediators and pro-inflammatory cytokines (ID 520, 2914), and "immunomodulating agent" (4690) pursuant to Article 13(1) of Regulation (EC) No 1924/2006" (http://www.efsa.europa.eu/en/efsajournal/doc/1796.pdf) and VKMs "Evaluation of negative and positive health effects of n-3 fatty acids as constituents of food supplements and fortified foods", 2011(http://www.vkm.no/dav/c7a41adb79.pdf).</p>	<p>The cited references refer to reports on evaluation of scientific basis for health claims, which is somewhat outside the scope of NNR.</p>
NIFES, Norway	<p>General comments only</p> <p>The main conclusions in this systematic review are due to the inclusion/exclusion criteria somewhat in contrary to the current/established knowledge from other competent bodies like EFSA and VKM. As stated in this SR there is a challenge to find enough eligible studies according to the inclusion criteria. This challenge will become even greater when assessing the potential health impact of a complex matrix like food.</p>	<p>This comment is acknowledged.</p>

Org/ Comp	General and detailed comments	WG response and actions
Margarine Foreningen MIFU, Denmark	<p>General comments:</p> <p>MIFU fully supports the comments made by Unilever. We would like to add that according to Danskernes Kostvaner, trans from animal fats is the main contributor to the intake of trans fatty acids in the diet.</p>	The text has been modified accordingly.
Dunder-backens Vårdshus, Sweden	<p>General comments:</p> <p>Hello! Since 5 years I follow the LCHF way of eating. I thereby have lowered my blood sugar, reduced my weight, have no problems with gases in my stomach any more and feel just fine. This way of eating is far from your recommendations. I can not understand why you are not interested in the LCHF way of eating. You may say there are no long term studies. But there are. Then you may say there are too few participants in these studies. But to show statistically significant results you do not need many participants if the difference between the control group and the other is big. So once again it seems that other forces than scientific ones dictate new nutrition guidelines. Jan Darenius</p>	Thank you for comment. LCHF was not a focus of the SR nor the chapter on fat, as most studies deal with intentional weight reduction among overweight and obese.
Minivent AB, Sweden	<p>(More info may follow)</p> <p>Detailed comments only</p> <p>This text is intended to replace lines 1 to 1 100 in NNR5 –Fat and Fatty acids The macronutrient composition of NNR4 is proven unscientific by SBU – Swedish Council on Health Technology Assessment in their report ”Mat vid diabetes” from May 2, 3, 5 and August 2010. This means that the non-scientific and rejected NNR4 recommendations has to be completely discarded and rejected, they can not be used as a basis for NNR5. NNR5 has to use science from previous centuries to establish a scientific base from physiology, biochemistry and endocrinology to be able to recommend any intake of macronutrients. Some basic facts seem to be necessary here. There are three macronutrients, protein and fat are both of them essential and the third macronutrient is the nonessential and toxic carbohydrate. Fat and fatty acids 24 25 Recommendation Age 6-11 mo 12-23 mo Adults and children from 2 years of age Animal fat Major part Major part Major part Cis-PUFA <5 E% <5 E% <5 E%# - n-3 >1 E% >1 E% >1 E% TFA animal unlimited unlimited unlimited TFA vegetable As low as possible As low as possible As low as possible Total fat 50-70 E% 60-80 E% >70 E% Introduction 29 Fat provides the organism with energy in a concentrated form. In addition to energy, dietary fats provide essential fatty acids and fat-soluble vitamins. Lipids, mainly phospholipids and cholesterol, are included in cell membranes and triglycerides are stored in adipose tissue as energy reserves. Certain fatty acids serve as a source of eicosanoids. In food items, fats are usually in the form of triglycerides. 35 Dietary sources and intake Habitual fat intake and composition in the Nordic countries has changed during the last decades. Total fat intake was at its highest during the 1960s and 1970s but has declined during the past 20-30 years. The fatty acid composition has also</p>	The WG thanks for comments. However, most comments lack scientific substantiation. The SBU report focuses on treatment of type-2 diabetes, which is outside the scope of NNR.

Org/ Comp	General and detailed comments	WG response and actions
	<p>changed with a reduction of the intake of saturated fatty acids (SFA) and increase in the intake of unsaturated fatty acids. The most important sources of fat are 1) spreads, butter, oils and other fats, 2) milk and milk products, and 3) meat and meat products. Fat-containing dairy products, butter, butter based spreads, meat products and partially hydrogenated fats in processed products are the main sources of TFA. Soft margarines and vegetable oils are the main sources of cis-polyunsaturated fatty acids (PUFA), while cis-monounsaturated fatty acids (MUFA) are derived from several product groups. 63</p> <p>Physiology and metabolism Most of the naturally existing fats are mixtures of triglycerides composed of one molecule of glycerol esterified with three fatty acid molecules, mainly fatty acids with 16-18 carbon atoms. Non-esterified fatty acids are uncommon in the diet. Fatty acids account for about 95 % by weight of triglycerides. The effects of fatty acids depend on the length of the carbon chain, the degree of saturation, the number, position and structure of the double bonds, and to some extent also on their position in the triglyceride molecule. The unsaturated fatty acids are characterised by the number of double bonds: MUFA have only one double bond whereas PUFA have 2 to 6 double bonds. The positions of the double bonds are calculated either from the carboxy-terminal end of the carbon chain or the methyl end. The human organism is capable of synthesising SFA and MUFA – including n-7 and n-9 series MUFA - from acetate, whereas n-3 and n-6 series PUFA are required from the diet. Linoleic acid (n-6, LA) and alpha-linolenic acid (n-3, ALA) are metabolised (desaturated and elongated) further in the organism by the same enzyme systems (Figure 11.1). Naturally occurring unsaturated fatty acids in plants and free-living fish are mainly cis-fatty acids. All fats are made from fatty acids and glycerol that are called triglycerides. There are also mono- and diglycerides as well as phospholipids. All cell membranes are of fatty origin with about equal amounts of phospholipids and cholesterol. Our fat deposits are made from triglycerides. Fatty acids are of three major types, saturated fatty acids, usually straight carbon chain molecules, monounsaturated fatty acids (MUFA), usually bent at the unsaturated or double bond between two carbon atoms and polyunsaturated fatty acids (PUFA), usually bent at every double bond between two carbon atoms. So the PUFAs are like balls, MUFAs are like bananas and saturated fats are like rods. Fatty acids can have from one to 24 carbon atoms length. The longer a fatty acid is the higher melting temperature the fatty acid has. Also, MUFAs have a lower melting temperature than a saturated fat of the same length and PUFAs have even lower melting point. All animal fat have the same melting temperature of about 37°C and about the same chemical composition with shy of 50 % saturated fat, shy of 50 % MUFA and the rest about 5 % PUFA. Fih have a lower melting point as the fish lives in a lower temperature environment and so fish have a higher percent of PUFAs than animal fat. Coconut fat is high in saturated fats live in tropical temperatures. 79 In addition to triglycerides, dietary fats comprise of phospholipids and</p>	

Org/ Comp	General and detailed comments	WG response and actions
	<p>cholesterol. The most common dietary phospholipid is phosphatidylcholine (lecithin). Cholesterol is found in foods of animal origin and is synthesised in the human organism, which is true for phospholipids as well. Plants contain small amounts of plant sterols, mainly sitosterol and campesterol, which are poorly absorbed (5-15 %) from the intestine, and interfere with the absorption of cholesterol. The absorption of the corresponding saturated sterols sitostanol and campestanol 85 is around 1-3 %. 86 87 We have to eat protein, preferably of animal origin, as we are unable to produce about ten out of twenty amino acids, the building blocks of protein . We have to eat fat, preferably of animal origin, as we have a limited production of long chained polyunsaturated fatty acids of animal origin. We also need fat as the major energy source for the body as well as fat makes the structural entity of cell membranes and constitutes padding fat tissue protecting delicate organs in the body . Biochemistry All fats are made from fatty acids and glycerol that are called triglycerides. There are also mono- and diglycerides as well as phospholipids. All cell membranes are of fatty origin with about equal amounts of phospholipids and cholesterol. Our fat deposits are made from triglycerides. Fatty acids are of three major types, saturated fatty acids, usually straight carbon chain molecules, monounsaturated fatty acids (MUFA), usually bent at the unsaturated or double bond between two carbon atoms and polyunsaturated fatty acids (PUFA), usually bent at every double bond between two carbon atoms. So the PUFAs are like balls, MUFAs are like bananas and saturated fats are like rods. Fatty acids can have from one to 24 carbon atoms length. The longer a fatty acid is the higher melting temperature the fatty acid has. Also, MUFAs have a lower melting temperature than a saturated fat of the same length and PUFAs have even lower melting point. All animal fat have the same melting temperature of about 37°C and about the same chemical composition with shy of 50 % saturated fat, shy of 50 % MUFA and the rest about 5 % PUFA. Fih have a lower melting point as the fish lives in a lower temperature environment and so fish have a higher percent of PUFAs than animal fat. Coconut fat is high in saturated fats live in tropical temperatures. Proteins and fats of animal origin are non-toxic and self regulate the energy intake. Animal fat has the same composition, properties and melting temperature as our own fats and are optimal . Excess of vegetable polyunsaturated omega-6 fatty acids may be cancerous The background is that the current major lifestyle diseases mainly result from over consumption of nonessential carbohydrates. Already Hippocrates knew 400 B.C. that over consumption of carbohydrates had an obesity and disease creating effect. Also the old pig farmer knew how to get a pig fat until Christmas, he fed the pig coarse milled grain ("gröpe" in Swedish) together with boiled potatoes and cow's milk. If you eat a high carb diet, the liver converts the sugar to fat. The fat is then stuffed into VLDL and excreted from the liver. When VLDL is excreted from the liver apolipoproteins are taken from HDL and stuck onto VLDL, and the HDL, as a consequence, drops. In short,</p>	

Org/ Comp	General and detailed comments	WG response and actions
	<p>carbs raise VLDL and lower HDL (a high fat diet reverses this). On the other hand, exercise uses up carbs, insulin levels fall, carbs are not turned into fat in the liver, VLDL levels to down and (as a direct result) HDL goes up. This is also why VLDL goes up and HDL goes down in a state of insulin resistance - it must. It is why VLDL goes up, and HDL goes down in Cushing's disease, and in those given steroids because it must. Steroids and Cushings create insulin resistance, insulin resistance causes insulin levels to rise, high insulin levels drive the production of fat from glucose/glycogen in the liver, more fat is produced, the liver gets fatty, VLDL levels rise, HDL levels - as a consequence - fall. The logical measure would be limiting the consumption of carbohydrates from the rejected NNR4</p> <p>recommended level of 480 grams of carbohydrates per day to less than 100 g for healthy population, the way diabetes was treated efficiently 100 years ago with at least 100 years if not at least 2 500 years of practical experience. The recommended intake of macronutrients is based on an overall assessment of the present knowledge about the impact of macronutrient intake on health and/or risk of disease. This requires various types of scientific data, primarily from the physiology, biochemistry and endocrinology of previous centuries , . The research behind NNR4 is rejected by SBU already in 2010 and can thus not be used by NNR5. Here the protein intake is minimum 0.5 g animal protein/kg body weight to secure essential amino acid supplies. Excess protein intake leading to amino acid excess is converted to short chain fatty acids. The carbohydrate intake ought to be limited to a physiological level. Healthy populations ought not have more than 100 g carbohydrates per day regardless of weight and populations with a metabolic disease like metabolic syndrome, diabetes or obesity are to be recommended a much lower carbohydrate intake. This implies that the rest of the energy requirements should be satisfied by fat intake . Polyunsaturated fats of omega-6 type should be limited to less than 20 g/day Randomized controlled clinical trials (RCTs), prospective cohort studies and other epidemiological studies are not applicable in nutritional research. RCT can not be used as there are always at least two parameters changed at the same time, which means we can usually not tell which parameter that is causative. Prospective cohort studies are difficult to control and thus unreliable. Epidemiological studies are always unreliable and may be hypothesis generating but never give causal relationship. So RCTs, prospective cohort studies and epidemiological studies can not be the basis of nutritional recommendations. Good nutritional observations and research are available from the old fashioned physiology, biochemistry and endocrinology from previous centuries , as well as modern studies from this century , . In these cases, the recommended intake of macronutrients is based on an overall assessment of the scientific, this means physiological, biochemistrical and endocrinological evidence and includes specific considerations about known patterns of intake of nutrients and foods and actual composition of available foods in the Nordic countries. The recommended</p>	

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	<p>intake of macronutrients refers to average intakes for groups and should be considered as goals for dietary changes and dietary advice (planning). Using acceptable ranges for the percentage of energy provided by carbohydrate, protein, and fat and their subclasses in the diet, refers to appropriate ranges of usual intake in the majority of the population.</p> <p>Food based dietary guidelines Food based dietary guidelines (FBDG) are based on an overall assessment of the present knowledge about the impact of food/food groups on health and/or risk of disease. Setting food based dietary guidelines requires various types of scientific data, especially evidence from physiology, biochemistry and endocrinology instead of non-scientific prospective cohort studies and other epidemiological studies and controlled clinical trials (RCTs) including the by SBU rejected NNR4. These guidelines are considered as a translation of nutrient recommendations into foods. They also take into consideration the habitual dietary patterns and scientific evidence of the effects of foods on different health outcomes. Since 150 years we know what is a cofactor in causing obesity, heart disease, diabetes, cancer and dementia. The knowledge shows that the extremely high carbohydrate Western diet is the common denominator if not the cause. Also, all weight reducing diets have a common denominator, they all reduce the carbohydrate content of the food. A causal relationship between food intake and risk of diseases is not always available from the scientific data. The food based dietary guidelines are therefore based on an overall assessment of the scientific evidence and include physiology, biochemistry and endocrinological knowledge and also specific considerations about known patterns of intake of foods and food groups and actual composition of available foods in the Nordic countries. On this basis, the FBDG should be considered as 'optimal' in Nordic countries. Suggested protein intake is as previously between 10 and 20 E% Suggested carbohydrate intake is less than 100 g per day Suggested fructose intake is less than 10 g per day Suggested animal fat intake is more than 70 E%. Suggested omega-6 PUFA should be limited to less than 0.3 grams per kg and day.</p>	

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Organisation/ Company	General and detailed comments	WG response and actions
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Organisation/ Company	General and detailed comments	WG response and actions
University of Bergen, Norway	<p>detailed and general comments are given below:</p> <p>In general the chapter on carbohydrates in the NNR 2012 gives a good background for the given recommendations. Here are some few comments and suggested changes: In line 633-634, it is written that "Some studies report lower growth rate among children consuming a vegan diet, but it is not clear if this can be attributed to the fibre content." This refers to a review by Edwards and Parrett (Edwards CA, Parrett AM. Dietary fibre in infancy and childhood. Proc Nutr Soc. 2003;62:17-1038 23.) In the review Edwards and Parrett, the statement is based on the study by Sanders from 1988 (Sanders TA. Growth and Development of British vegan children. Am J of Clin Nutr. 1988. Suppl. 3) First, the study of Sanders presents the finding as a tendency, and there are not used statistical significant tests. In terms of anthropometric measurements, nearly all the vegan children were inside the normal range both for height and weight, most quite close to the 50-percentile. In a review by Messina and Mangels, it is shown that growth among children on vegan diets with few exceptions are within the normal range (Messina V, Mangels AR: Considerations in planning vegan diets: children. J Am Diet Assoc 2001, 101(6):661-669.). Thus, it seems reasonable to delete the statement in line 633-634.</p> <p>In line 706, there is a spelling error "Fkemrequent". I assume it is meant to be "Frequent".</p>	<p>Sentence modified in final draft. The few studies, mainly on British vegan children indicate slower growth in some of the children (ref. in Sanders & Reddy 1994). As stated by Parret this may not be related to the high fibre content (5 g/MJ in the study by Sanders & Manning 1992).</p> <p>Corrected</p>
Danish Cancer Society and Danish Nutrition Society	<p>General comments</p> <p>Regarding the evidence for dietary fiber and cardiovascular disease (CVD), it is mentioned that there is clear evidence that dietary fiber is associated with lower risk of CVD. Furthermore, several mechanisms underlying this association are proposed. However, you judge that the evidence is "suggestive". The association with colorectal cancer is judged to be "convincing". Comparing the evidence regarding dietary fiber and CVD and colorectal cancer, there seems to be at least as clear evidence for CVD as for colorectal cancer. You could consider to classify CVD as "convincing" as well, or at least mention why the evidence for CVD is not as clear as for colorectal cancer. I understand and fully agree that the evidence for dietary fiber and colorectal cancer should be classified as "convincing" as I is in the newest update by World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR).</p> <p>Detailed comments</p> <p>Line 514-528: Please mention the possible underlying mechanisms that can explain the association between dietary fiber intake and blood pressure and blood glucose attenuation.</p> <p>Line 572: You mention that WCRF/AICR in their 2007 report concluded that dietary fiber is "convincingly" associated with lower risk of colorectal cancer. THIS IS NOT CORRECT. In the 2007 report, dietary fiber was classified as "probable". In 2011 the meta-analysis by Aune et al (that you also mentioned) reviewed the newest evidence, and this lead to that the evidence regarding dietary fiber was re-classified as "convincing" in the Continous Updrate project (CUP) of WCRF/AICR. WCRF/AICR (2011) Continuous Update Project Interim Report Summary. Food, Nutrition, Physical Activity, and the Prevention of Colorectal Cancer, available at: http://www.wcrf.org/cancer_research/cup/key_findings/colorectal_cancer.php</p>	<p>This is updated and amended in the final draft.</p> <p>Text added</p> <p>Deleted in final draft. Reference to Continuous update project added in connection with Aune (2011)</p>

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LRF Dairy Sweden	Page 2, lines 59-60. Dairy products are not mentioned as sources of added sugars in Riksmaten 2010-2011. How come the conclusion in the NNR chapter on carbohydrates include dairy and exclude buns, cakes and pastries that represent a larger source in Riksmaten 2010-2011?	The section on dietary sources is based on intake data from dietary surveys in the Nordic countries, including children and adults. Sweet bakery products are mentioned in the final draft..
Lantmännen Ekonomisk Förening, Sweden	<p>General comments only</p> <p>61 Cereals (wholegrain), fruits, berries, vegetables and fiber enriched food products.</p> <p>115 Tabell xx.1. Include arabionxylan as examples of other Non-Stach Polysaccharides.</p> <p>115 Tabell xx.1 It would be great if you can add one column with sources for the Components.</p> <p>127 Include betaglucan and arabinoxylan</p> <p>262 Include after their composition (amylose/amylopectin ratio).... and the amount of resistant starch (carbohydrate source and formation during the heating and chilling process)</p> <p>507 Viscous types of soluble fibre lower plasma total and LDL-cholesterol levels. Include Betaglucan from oat and Barley are examples of viscous types of soluble fibers.</p> <p>600 Include and rewrite effect of satiety. Our proposal is; results from Clinical studies shows an increase in satiety from an isocaloric diet with whole grain rye porridge for breakfast. Participant kept satiated to lunch and in some cases even until dinner (Satiating Effects of Rye Foods - Doctoral thesis No 2011:99 SLU Hanna Isaksson).</p> <p>723 Is it possible to include in the recommendation EFSA's approved Health claim for dietary fiber; we have tried to copy from EFSA's homepage unfortunately the format is not compatible. our recommendation is that you collect information directly from EFSA. Below you find the claims as compact text. Betaglucan: Claim type Nutrient, substance, food or food category Claim Conditions of use of the claim / Restrictions of use / Reasons for non-authorisation Health relationship EFSA opinion reference / Journal reference Commission Regulation Status Entry ID Art.13(1) Beta-glucans Beta-glucans contribute to the maintenance of normal blood cholesterol levels The claim may be used only for food which contains at least 1 g of beta-glucans from oats, oat bran, barley, barley bran, or from mixtures of these sources per quantified portion. In order to bear the claim information shall be given to the consumer that the beneficial effect is obtained with a daily intake of 3 g of beta-glucans from oats, oat bran, barley, barley bran, or from mixtures of these beta-glucans. maintenance of normal blood cholesterol concentrations 2009;7(9):1254, 2011;9(6):2207 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 754, 755, 757, 801, 1236, 1299, 1465, 2934 Art.13(1) Beta-glucans from oats and barley Consumption of beta-glucans from oats or barley as part of a meal contributes to the reduction of the blood glucose rise after that meal The claim may be used only for food which contains at least 4 g of beta-glucans from oats or barley for each 30 g of available carbohydrates in a quantified portion as part</p>	<p>Fiber-enriched products are not a major source</p> <p>Not considered</p> <p>Not considered</p> <p>Beta-glucan added, arabinoxylan is a hemicellulose</p> <p>Added</p> <p>Oats and barley are mentioned on p. 5</p> <p>Dietary fibre together with pectins</p> <p>These are mainly short-term studies, and will not be discussed further.</p> <p>The opinions on health claims are generally product and dose specific, and thereby largely outside the scope of NNR, which refer to the whole diet.</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>of the meal. In order to bear the claim information shall be given to the consumer that the beneficial effect is obtained by consuming the beta-glucans from oats or barley as part of the meal. reduction of post-prandial glycaemic responses 2011;9(6):2207 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 821, 824 Art.14(1)(a) Barley beta-glucans Barley beta-glucans and lowering of blood cholesterol and reduced risk of (coronary) heart disease. Information shall be given to the consumer that the beneficial effect is obtained with a daily intake of 3 g of barley beta-glucan. The claim can be used for foods which provide at least 1 g of barley beta-glucan per quantified portion. Q-2011-00798 Commission Regulation (EU) 1048/2012 of 08/11/2012 Authorised N/A Art.14(1)(a) Oat beta-glucan Oat beta-glucan has been shown to lower/reduce blood cholesterol. High cholesterol is a risk factor in the development of coronary heart disease Information shall be given to the consumer that the beneficial effect is obtained with a daily intake of 3 g of oat beta-glucan. The claim can be used for foods which provide at least 1g of oat beta glucan per quantified portion. Q-2008-681 Commission Regulation (EU) 1160/2011 of 14/11/2011 Authorised N/A Fibre: Claim type Nutrient, substance, food or food category Claim Conditions of use of the claim / Restrictions of use / Reasons for non-authorisation Health relationship EFSA opinion reference / Journal reference Commission Regulation Status Entry ID Art.13(1) Arabinoxylan produced from wheat endosperm Consumption of arabinoxylan as part of a meal contributes to a reduction of the blood glucose rise after that meal The claim may be used only for food which contains at least 8 g of arabinoxylan (AX)-rich fibre produced from wheat endosperm (at least 60 % AX by weight) per 100 g of available carbohydrates in a quantified portion as part of the meal. In order to bear the claim information shall be given to the consumer that the beneficial effect is obtained by consuming the arabinoxylan (AX)-rich fibre produced from wheat endosperm as part of the meal. reduction of post-prandial glycaemic responses 2011;9(6):2205 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 830 Art.13(1) Barley grain fibre Barley grain fibre contributes to an increase in faecal bulk The claim may be used only for food which is high in that fibre as referred to in the claim HIGH FIBRE as listed in the Annex to Regulation (EC) No 1924/2006. increase in faecal bulk 2011;9(6):2249 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 819 Art.13(1) Oat grain fibre Oat grain fibre contributes to an increase in faecal bulk The claim may be used only for food which is high in that fibre as referred to in the claim HIGH FIBRE as listed in the Annex to Regulation (EC) No 1924/2006. increase in faecal bulk 2011;9(6):2249 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 822 Art.13(1) Rye fibre Rye fibre contributes to normal bowel function The claim may be used only for food which is high in that fibre as referred to in the claim HIGH FIBRE as listed in the Annex to Regulation (EC) No 1924/2006. changes in bowel function 2011;9(6):2258 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 825 Art.13(1) Wheat bran fibre Wheat bran fibre contributes to an acceleration of intestinal transit The claim may be used only for food which is high in that fibre as referred to in the claim HIGH FIBRE as listed in the Annex to Regulation (EC) No 1924/2006. In order to bear the claim information shall be given to the consumer that the claimed effect is obtained with a daily intake of at least 10 g of wheat bran fibre. reduction in intestinal transit time 2010;8(10):1817 Commission</p>	

Organisation/ Company	General and detailed comments	WG response and actions
	<p>Regulation (EU) 432/2012 of 16/05/2012 Authorised 828, 839, 3067, 4699 Art.13(1) Wheat bran fibre Wheat bran fibre contributes to an increase in faecal bulk The claim may be used only for food which is high in that fibre as referred to in the claim HIGH FIBRE as listed in the Annex to Regulation (EC) No 1924/2006. Increase in faecal bulk 2010;8(10):1817 Commission Regulation (EU) 432/2012 of 16/05/2012 Authorised 3066</p>	
National Institute for Health and welfare, Finland	<p>General comments: A good summary for carbohydrates with classifications, - energy conversion factors Should we express these factors in kcal in a footnote? - The energy proportions for available carbohydrates reasonable (45-60 en%) -How should the publisher of the recommendations encourage public and non-profit organizations to produce material for planning and implementation of healthy diet and vegetable-based diet</p> <p>Detailed comments - P 2, line 58. Cereals as food ingredients contain starch and fibre while sweet bakery products contain also added sugar - P 11, starting l 445 May be added? The aim of a healthy diet is to have low energy density but diet with nutrient-dense foods. Nutrient-dense foods are foods that provide substantial amounts of vitamins and minerals and relatively few calories</p>	<p>Kcal could be given in a footnote. Derivation of food-based guidelines for various population groups is mainly a management issue, largely outside the scope of NNR</p> <p>Sentence amended</p> <p>A general statement is included in the summary chapter</p>
Norwegian Directorate of Health	<p>General comments: We endorse the main conclusions and recommendations in this chapter. However, we have some comments that we ask the NNR-project group to take into account when revising the draft. We support the use of the term “added sugar” and your definition of this term. However, we suggest that you add the following expressions to the definition: “Honey, concentrates of fruits and berries, maltose and maltodextrins are also regarded as added sugars.”</p> <p>We suggest that the fiber recommendation is reformulated. We believe it is important that NNR clearly state that it is the intake of foods rich in fibre that is the prime focus in the recommendation.</p> <p>62-3. the lower value for intake of added sugar may be changed to 7 E%. Norwegian adults have on average had an intake of added sugars below 10 E% for several decades. In Norkost 2010-11 the mean was 7.3 E% from added sugar for adults. 95. We support the use of the term “added sugar” and your definition of this term. However, we suggest that you add the following expressions to the definition: “Honey, concentrates of fruits and berries, maltose and maltodextrins are also regarded as added sugars.” 198-99. This clarification is very important and should be repeated in chapter 1.</p> <p>304 + 324-8. The title covers lipids, glucose and insulin, but the summary just covers lipids. What about</p>	<p>The definition already includes maltose and maltodextrins as “other isolated sugar preparations”. Honey and fruit concentrates will not be mentioned.</p> <p>This is clarified</p> <p>This could be a matter for national application and management issues</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>glucose and insulin?</p> <p>362-89. Body weight – it seems as the quality of carbohydrates and the quality of the foods that provide dietary carbohydrates are more important than total carbohydrates. This should be further discussed here. We suggest that you include short summaries/conclusions concerning the evidence in relation to increased and/or decreased body weight for added sugar, sugar containing beverages, fibre and foods and dietary patterns rich or poor in fibre, added sugar and refined foods, respectively. We recommend that you add information from the relevant NNR-SLRs and also use reviews as: Hauner et al. Evidence-Based Guideline of the German Nutrition Society: Carbohydrate Intake and Prevention of Nutrition – Related Diseases. Ann Nutr Metab 2012; 60 (suppl) 1-58., and Te Morenga et al. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2012 Jan 15;346:e7492 376-9. References stop at 2009, and may be updated.</p> <p>710. We suggest that you use the same expression here as in the recommendation in chapter 1, and thus delete (sucrose, fructose, starch hydrolysates) on line 710. You may in the text above the box repeat the definition of added sugar used in chapter 1.</p> <p>717-9. This summary of the evidence “There is convincing evidence for a protective effect of dietary fibre against colorectal cancer and suggestive evidence for a protective effect against breast cancer, cardiovascular disease and type-2 diabetes.” is not in line with your conclusions on line 594 and 680. Line 594: In conclusion, there is probable evidence for a protective effect of dietary fibre from various, 594 foods against CVD. Line 680: In summary there is probable evidence that dietary fibre intake is inversely associated with 680 type-2 diabetes. It is only for breast cancer that you use the grade suggestive (line 578).</p> <p>724. We suggest that the fiber recommendation is reformulated. We believe it is important that NNR clearly state that it is the intake of foods rich in fibre that is the prime focus in the recommendation. E.g.: “Intake of foods rich in dietary fibre, as wholegrain cereals, vegetables, pulses, nuts, fruits and berries, that provide a total intake of dietary fibre of at least 25 g/d for women and 35 g/d for men, i.e. approximately 3 g/MJ.” For children we suggest the following expression: “An intake of foods rich in dietary fibre that provide a total intake of dietary fibre corresponding to”</p> <p>Please add the section “Reasoning behind the recommendations”. This has been very useful in other chapters.</p>	<p>Heading reformulated in final draft</p> <p>A summary text has been added in the section on recommendations - total carbohydrates</p> <p>The German report has been reviewed, and generally is in line with the conclusions of the NNR SR. The SR by Te Morenga is cited.</p> <p>See above</p> <p>Amended in final draft</p> <p>Amended</p> <p>Added</p>
SNF Swedish Nutrition Foundation	<p>General comments only</p> <ul style="list-style-type: none"> • In all places when associations between intake and health outcome is mentioned, it should be made clear if the association is positive or negative (inverse). • It would be helpful if “high/low intake” and “frequent intake” could be defined.” <p>Page 9 • It would be helpful if NNR could discuss the mechanism behind the probable association between sugar sweetened beverages and the increased risk of type-2 diabetes Page 10, body weight: • The text states: “... less refined grains, meat and sugar-rich foods and beverages were associated with</p>	<p>Amended in final version</p> <p>Addressed in final version</p> <p>Further discussed in final draft</p>

Organisation/ Company	General and detailed comments	WG response and actions
	<p>less weight gain (Fogelholm et al. 2012).” However, according to the NNR SR by Fogelholm et al, the associations between weight change and ”CHO from foods with simple sugars”, ”SSSD” (sugar sweetened soft drinks) and ”sugar and confectionary” were graded as ”no conclusion”. We suggest that the findings by Fogelholm et al are commented and discussed, and that the background to the statement regarding sugar-rich foods and drinks is explained. See also comments to Chapter 1, page 10 (added sugars) and page 19 (overweight and obesity) and Chapter Carbohydrates page 17 (added sugars).</p> <p>Page 12, pregnancy outcome: • It would be helpful if NNR could specify the sentence on line 492-493 about GI and fetal growth. The sentence can be misinterpreted that it is positive that the mother has high glucose levels so there is glucose available for the fetus. We suggest that NNR explain for the reader that the insulin sensitivity during pregnancy is decreased and according to McGowen et al 2010 a low GI diet could therefore reduce the postprandial glucose peaks and as a consequence reduce the available maternal glucose that can be used by the fetus. An increased transport of macronutrients (as glucose) to the fetus increases the risk of having a large for gestational age baby.</p> <p>Page 12, GI/GL: • Page 12-15: It would be helpful if the discussion on health effects of GI/GL was completed with a paragraph summarizing the conclusion, according to the SR NNR by Øverby et al, eg ”There is not enough evidence that choosing foods with low GI will decrease the risk of chronic diseases in the population overall. However, there is suggestive evidence that ranking food based on their GI might be of use for overweight and obese individuals. Issues regarding methodology, validity and practicality of the GI remain to be clarified. It is still unclear how much of the possible health effects are due to the GI per se, and how much additional benefit a low GI diet may offer after compliance with recommendations to increase intake of DF, whole grains, legumes and fruits and vegetables.”</p> <p>Page 17, added sugars: • The text states: ”Consumption of sugar-sweetened drinks has been associated with an increased risk of type-2 diabetes and excess weight gain and should therefore be limited”. However, according to the NNR SR by Fogelholm et al, the associations between weight change and ”CHO from foods with simple sugars”, ”SSSD” (sugar sweetened soft drinks) and ”sugar and confectionary” were graded as ”no conclusion”. We suggest that the findings by Fogelholm et al are commented and discussed, and that the background to the statement regarding sugar-sweetened drinks and weight is explained. See also comments to Chapter 1, page 10 (added sugars) and page 19 (overweight and obesity) and Chapter Carbohydrates page 10 (body weight).</p> <p>Page 18, total carbohydrate: • We acknowledge the added upper range limit for total carbohydrate, compared to the version of chapter 1 published 5 June 2012.</p>	<p>Text added in final draft</p> <p>Included in final draft</p> <p>Further discussed in final draft. Evidence from SR and meta-analysis by Te Morenga et al. 2013 added.</p>
DI Fødevarer, Denmark	<p>General comments:</p> <p>We welcome this possibility to comment on the summary of the recommendations. The Nordic Nutrition Recommendations have wide implications for the food, nutrition and health policies in the Nordics. Therefore, transparency of the process is important. We believe there is a need to reconsider the recommendation on added sugar. We agree with Sondestedt there is not sufficient scientific evidence to</p>	<p>The recommendation on added sugars is based on several aspects including nutrient density, caries, type-2 diabetes and weight</p>

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	<p>establish a recommendation to limit added sugar intake to a specific percentages, and do not find it justified to do so anyhow. Sondestedt argue for a pragmatic interpretation in line with the USDA. However, the intake of added sugar in the US is approximately 50% higher than in the Nordics, and thus not comparable. The food patterns in the two regions are not comparable either. Taken into consideration the rationale behind the NNR to develop recommendations in line with local food patterns, the recommendation to look at USDA seems odd. Finally, EFSA in 2010 concluded, there is no scientific basis for establishing a recommendation for added sugar. Therefore, we urge you to reconsider the recommendation. This is also needed to ensure consistency in how the scientific reviews are implemented into the recommendations. There are no major differences in the outcome of the scientific review of glycaemic index and the scientific review of added sugar. But it is concluded no recommendation on glycaemic index can be included, but the recommendation to limit added sugar is maintained. That is inconsistent. The fact that previous versions of NNR had similar a recommendation is not an argument to keep it. On the contrary it would be fair to discuss and question it, especially at times when so much effort is made on promoting the science base behind the recommendation. Labeling of foods is regulated by EU law and based on the scientific advice of EFSA. As of December 2014 it will no longer be allowed to label the amount of added sugar on the food label. Instead the label will inform about the content of total sugar. Therefore, we find it would be more relevant to have a recommendation on total sugars, if any. We notice the way the findings of the carbohydrate chapter is changed in the summary chapter. We urge you to ensure consistency across and within the chapters.</p> <p>Detailed comments:</p> <p>Lines 702-704 We question if the reduction in intake of added sugar is needed for all population groups to ensure sufficient supply of essential nutrients.</p> <p>Line 456 suggest there may be a link for specific groups. If the recommendation is not abandoned for all age groups it should be considered if it should not only cover groups of people with very low energy intakes.</p> <p>Line 703 Similary line 457 suggests there may be a link between added sugar and dietary pattern related to fruit and vegetables. We do not believe this is sufficient evidence to claim restrictions in added sugar will support healthy dietary pattern.</p>	<p>maintenance. The SR by Fogelholm et al. focused on macronutrients, but also included studies on foods among adults. The associations between each of the different sugar-rich food groups were assessed as “no conclusion”, except for sweets and desserts (suggestive). However, 5 of 7 studies reported positive associations. A recent SR and meta-analysis, found that reduced intake of sugars was associated with less weight gain among adults. Among children high intake of SSB was associated with risk of overweight and obesity in prospective studies (Te Morenga et al. 2013). Effects seem to be mediated by a higher total energy intake. The 2007 WCRF report concluded that there was probable evidence that intake of energy dense foods and sugary drinks of increased risk of weight gain. The association with sugar is clear for dietary fibre is clear, for some other nutrients in studies from Nordic countries and Germany. It is stressed that the recommendation on added sugars is “especially important for children and persons with a low energy intake”. This to achieve an adequate nutrient density. Limitation of added sugars is one basis for deriving FBDG.</p> <p>See comment above.</p>

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	<p>Line 705 There are inconsistent results when it comes to linking added sugar with diabetes and weight gain. Furthermore, systematic biases have been identified for several of the studies investigating the link between soft drinks and weight gain. We do not believe there is sufficient evidence to support the statement. We suggest you write foods and drinks with a high energy (or sugar (and fat) content) has been associated with diabetes and weight gain. That would at least allow for the scientific doubt. We trust the review of the chapter on carbohydrates will be taken into account in the Summary chapter.</p>	
Finnish Food and Drink Industries' Federation	<p>General comments only</p> <p>Comments on the Nordic Nutrition Recommendation on “added sugars” 1. Comments on premises for the recommendation regarding “added sugars”. The premises are found in an article from Food & Nutrition Research 2012, vol. 56: 19104 by Sonestedt et al.</p> <p>a. National dietary surveys for the Nordic countries show that sugar intake is not rising in the Nordic Region. (Unfortunately I couldn't find a way to attach an annex here).</p> <p>b. Multiple international literature reviews have found no convincing evidence for a link between added sugars and diabetes or cardiovascular disease. This is also the conclusion in the article by Sonestedt et al.</p> <p>c. The link between added sugars and the development of overweight is not included in Sonestedt's article – it refers to a literature review done by the WHO's Nutrition Guidance Expert Advisory Group (NUGAG). The WHO's Expert Advisory Group published its review in British Medical Journal, January 2013. According to a press release from the British Medical Journal, Jim Mann (one of the authors and NUGAG experts) concludes that no strong scientific link was found between the intake of added sugars and the development of overweight. Nevertheless, he concludes that consuming less added sugar will result in less overweight. However, the focus should not be exclusively on added sugars, since the fact that a lower consumption of energy will lead to a smaller weight gain is not unique to added sugars. This applies to all energy-containing food and drink products. The focus should therefore be on overall energy intake (see link to press release) http://www.bmj.com/press-releases/2013/01/14/cutting-down-sugar-has-small-significant-effect-body-weight). d. The article by Sonestedt et al. concludes “overall, specific cut-offs for sugar intake based on strong scientific evidence cannot be made, but pragmatic interpretation of the evidence as was done by the USDA would support limiting added sugars and sugar-sweetened beverages intake, e.g. 10 % as recommended in NNR4. But US intake of added sugars cannot be compared to intake in the Nordic Region. - Why should the NNR be mirrored by US dietary guidelines (conclusion of the article by Sonestedt et al.)? Food patterns in the USA and the Nordic Region are not comparable. Average intake of “added sugars” in adults grams per day USA *77 g/day (2007-2008) - see annex 1 - Finland 54 g/day (2007) -“- Sweden 53 g/day (2010-2011) -“- Denmark 48 g/day (2003-2006) -“- Norway 42 g/day (2010-2011) -“- -</p> <p>Why was international literature used as a basis for the recommendation on “added sugars” in NNR, when the premise of the NNR is that they should be based on Nordic literature?</p> <p>*http://www.ncbi.nlm.nih.gov/pubmed/21753067 In addition, the aforementioned US dietary guidelines are expressed as an interval, not an exact value. Further, the interval is based on the political</p>	<p>The recommendation on added sugars is based on several aspects including nutrient density, caries, type-2 diabetes and weight maintenance. The SR by Fogelholm et al. focused on macronutrients, but also included studies on foods among adults. The associations between each of the different sugar-rich food groups were assessed as “no conclusion”, except for sweets and desserts (suggestive). However, 5 of 7 studies reported positive associations. The recent BMJ SR and meta-analysis found that reduced intake of sugars was associated with less weight gain among adults. Among children high intake of SSB was associated with risk of overweight and obesity in prospective studies (Te Morenga et al. 2012). Effects seem to be mediated by a higher total energy intake. The WCRF report 2007 concluded that energy dense foods and sugary drinks probably promote weight gain. NNR cannot solely be based on Nordic studies.</p>

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	<p>recommendation, not the scientific recommendation. See the differences in point 3, b. 2. If the NNR does not adhere to the scientific evidence, why then perform “systematic reviews”? See the conclusion in the attached article from Food & Nutrition Research 2012, vol. 56: 19104 by Sonestedt et al.</p> <p>3. Scientific comments a. The premise for the revised NNR is that the recommendations should be based on scientific evidence. Systematic reviews are therefore performed every 4 years to assess whether any new literature provides grounds for a revision. If the recommendations for added sugars, regardless of the conclusion of the scientific review, are still based solely on a “pragmatic interpretation”, why then perform a scientific review? As there is no scientific evidence for an exact reference value for added sugars, at a minimum an interval should be stated that reflects both the uncertainty of the scientific evidence and the fact that the amount of energy a person can consume from added sugars varies according to the individual, as it depends on the individual’s overall energy intake and energy consumption. b. The Sonestedt et al. article (page 2, column 1) points to the fact that the NNR is more or less in line with equivalent estimates of reference values from the EFSA and USDA in regard to carbohydrates, fat, protein and fibre, but that the NNR reference value for added sugars is not in agreement with the EFSA and USDA. - EFSA, EU, 2010, concludes that based on the existing literature there is insufficient scientific evidence to establish an upper reference value for intake of added sugars. - The USDA Scientific Committee, 2010, USA, concludes that the upper limit for added sugars should be 25 E% or less. - The USDA, policy document, 2010, recommends that maximal intake of saturated fat and added sugars be limited to 5-15 E%. Since the same scientific literature has been reviewed in NNR, it is odd that the recommendations for added sugars are not the same. c. The Sonestedt et al. article (page 2, column 1) points out that a recommendation for added sugars of 10 E% in 2004 was primarily based on the link between sugar and dental caries. Several scientific literature reviews, including the EFSA and IOM (Institute of Medicine, USA) have established that it is the frequency of consumption of fermentable carbohydrates that is significant in the development of dental caries, rather than the amount – a fact that is not affected by an upper limit for intake of added sugars. d. From a nutritional point of view, there is no difference between added sugars and other glycaemic carbohydrates. It is therefore misleading and incorrect to only have an upper limit for “added sugars”, when there is no equivalent recommendation for other glycaemic carbohydrates. e. The article by Sonestedt et al. refers to the WHO’s literature review of sugar and overweight (Jim Mann et al.). The review concludes that a reduction in sugar intake would lead to a small amount of weight loss. The fact that a reduction in energy intake leads to a reduction in body weight is not a property specific to added sugars. It applies to all food and drink products containing calories. The review (Jim Mann et al.) also includes several studies – which also include “trial products” containing nutrients other than added sugars. It is therefore impossible to know how much of the change in body weight can truly be attributed to a modified intake of added sugars. The conclusion of the WHO literature review does not correspond to comparable reviews from the Institute of Medicine, USA, 2010, the European Food Safety Authority, EU, 2010, the National Health and Medical Research Council, Australia, 2003, and a report from the FAO/WHO Consultation on Carbohydrates in Human Nutrition,</p>	<p>Regarding intake of added sugars it is mentioned that the average mean contribution is 10-16 E%, with higher intakes among children</p>

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	<p>Rome, 1997. Further, none of the literature reviews has been able to establish an exact reference value as to when the intake of added sugars increased the risk of overweight. g. Sugar intake of sugar supply is stable despite the increase in soft drinks The article by Sonestedt et al. suggests that sugar consumption is rising. This is not correct. The overall sugar supply and sugar intake in the Nordic Region has largely remained stable for several decades (see annex 1) , but the pattern of consumption has changed, so that there has been an increase in sugar-containing processed products such as soft drinks, while there has been a decrease in home-made products containing sugar. This is shown in several years of dietary surveys from the Nordic countries and in supply figures (see annex 1) In addition, the article (Sonestedt et al.) does not mention that there are problems with bias in several of the studies regarding the intake of soft drinks and overweight.</p>	
Private person	<p>The conclusions are NOT based on science (at least not all available science)!!! Tired!</p>	
Swedish Food Federation	<p>General comments Added sugars (Page 17, lines 701-710) 1. Comments on premises for the recommendation regarding “added sugars”. The premises are found in an article from Food & Nutrition Research 2012, vol. 56: 19104 by Sonestedt et al. a) National dietary surveys for the Nordic countries show that sugar intake is not rising in the Nordic Region. Data/basis could be sent on request - not possible to enclose here. b) Multiple international literature reviews have found no convincing evidence for a link between added sugars and diabetes or cardiovascular disease. This is also the conclusion in the article by Sonestedt et al. c) The link between added sugars and the development of overweight is not included in Sonestedt’s article – it refers to a literature review done by the WHO’s Nutrition Guidance Expert Advisory Group (NUGAG). The WHO’s Expert Advisory Group published its review in British Medical Journal, January 2013. According to a press release from the British Medical Journal, Jim Mann (one of the authors and NUGAG experts) concludes that no strong scientific link was found between the intake of added sugars and the development of overweight. Nevertheless, he concludes that consuming less added sugar will result in less overweight. However, the focus should not be exclusively on added sugars, since the fact that a lower consumption of energy will lead to a smaller weight gain is not unique to added sugars. This applies to all energy-containing food and drink products. The focus should therefore be on overall energy intake (see link to press release) http://www.bmj.com/press-releases/2013/01/14/cutting-down-sugar-has-small-significant-effect-body-weight). d) The article by Sonestedt et al. concludes “overall, specific cut-offs for sugar intake based on strong scientific evidence cannot be made, but pragmatic interpretation of the evidence as was done by the USDA would support limiting added sugars and sugar-sweetened beverages intake, e.g. 10 % as recommended in NNR4. But US intake of added sugars cannot be compared to intake in the Nordic Region. - Why should the NNR be mirrored by US dietary guidelines (conclusion of the article by Sonestedt et al.)? Food patterns in the USA and the Nordic Region are not</p>	<p>See response to comments by Finnish Food and Drink Industries' Federation</p>

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	<p>comparable. Average intake of “added sugars” in adults grams per day USA *77 g/day (2007-2008) Finland 54 g/day (2007) Sweden 53 g/day (2010-2011) Denmark 48 g/day (2003-2006) Norway 42 g/day (2010-2011) Data/basis could be sent on request - not possible to enclose here.</p> <p>*http://www.ncbi.nlm.nih.gov/pubmed/21753067 - Why was international literature used as a basis for the recommendation on “added sugars” in NNR, when the premise of the NNR is that they should be based on Nordic literature? In addition, the aforementioned US dietary guidelines are expressed as an interval, not an exact value. Further, the interval is based on the political recommendation, not the scientific recommendation. See the differences in point 3, b. 2. If the NNR does not adhere to the scientific evidence, why then perform “systematic reviews”? See the conclusion in the attached article from Food & Nutrition Research 2012, vol. 56: 19104 by Sonestedt et al. 3. Scientific comments a) The premise for the revised NNR is that the recommendations should be based on scientific evidence. Systematic reviews are therefore performed to assess whether any new literature provides grounds for a revision. If the recommendations for added sugars, regardless of the conclusion of the scientific review, are still based solely on a “pragmatic interpretation”, why then perform a scientific review? As there is no scientific evidence for an exact reference value for added sugars, at a minimum an interval should be stated that reflects both the uncertainty of the scientific evidence and the fact that the amount of energy a person can consume from added sugars varies according to the individual, as it depends on the individual’s overall energy intake and energy consumption. b) The Sonestedt et al. article (page 2, column 1) points to the fact that the NNR is more or less in line with equivalent estimates of reference values from the EFSA and USDA in regard to carbohydrates, fat, protein and fibre, but that the NNR reference value for added sugars is not in agreement with the EFSA and USDA. EFSA, EU, 2010, concludes that based on the existing literature there is insufficient scientific evidence to establish an upper reference value for intake of added sugars. The USDA Scientific Committee, 2010, USA, concludes that the upper limit for added sugars should be 25 E% or less. The USDA, policy document, 2010, recommends that maximal intake of saturated fat and added sugars be limited to 5-15 E%. Since the same scientific literature has been reviewed in NNR, it is odd that the recommendations for added sugars are not the same. c) The Sonestedt et al. article (page 2, column 1) points out that a recommendation for added sugars of 10 E% in 2004 was primarily based on the link between sugar and dental caries and lower nutrient density of the food with increasing sugar intake in the Nordic countries. Several scientific literature reviews, including the EFSA and IOM (Institute of Medicine, USA) have established that it is the frequency of consumption of fermentable carbohydrates that is significant in the development of dental caries, rather than the amount – a fact that is not affected by an upper limit for intake of added sugars. d) From a nutritional point of view, there is no difference between added sugars and other glycaemic carbohydrates. It is therefore misleading and incorrect to only have an upper limit for “added sugars”, when there is no equivalent recommendation for other glycaemic carbohydrates. e) The article by Sonestedt et al. refers to the WHO’s literature review of sugar and overweight (Jim Mann et al.). The review concludes that a reduction in sugar intake would lead to a small amount of weight loss. The fact</p>	

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	<p>that a reduction in energy intake leads to a reduction in body weight is not a property specific to added sugars. It applies to all food and drink products containing calories. The review (Jim Mann et al.) also includes several studies – which also include “trial products” containing nutrients other than added sugars. It is therefore impossible to know how much of the change in body weight can truly be attributed to a modified intake of added sugars. The conclusion of the WHO literature review does not correspond to comparable reviews from the Institute of Medicine, USA, 2010, the European Food Safety Authority, EU, 2010, the National Health and Medical Research Council, Australia, 2003, and a report from the FAO/WHO Consultation on Carbohydrates in Human Nutrition, Rome, 1997. Further, none of the literature reviews has been able to establish an exact reference value as to when the intake of added sugars increased the risk of overweight. f) Sugar intake of sugar supply is stable despite the increase in soft drinks The article by Sonestedt et al. suggests that sugar consumption is rising. This is not correct. The overall sugar supply and sugar intake in the Nordic Region has largely remained stable for several decades (Data/basis could be sent on request - not possible to enclose here), but the pattern of consumption has changed, so that there has been an increase in sugar-containing processed products such as soft drinks, while there has been a decrease in home-made products containing sugar. This is shown in several years of dietary surveys from the Nordic countries and in supply figures (Data/basis could be sent on request - not possible to enclose here.) In addition, the article (Sonestedt et al.) does not mention that there are problems with bias in several of the studies regarding the intake of soft drinks and overweight.</p> <p>Detailed comments Page 2, lines 59-60. Sources of added sugar are not in accordance with the results in Riksmaten 2010-11 (e g includes dairy but not buns, cakes and pastries).</p>	
Minivent AB	<p>General comments only</p> <p>This is to replace lines 1-742 in the public consultation of Carbohydrates - NNR 2012 Carbohydrates Introduction Chemical classification Biochemistry Physiology Endocrinology Nutritional classification Toxicology Energy content Requirement Recommended intake References Recommended intake of carbohydrates: Digestable carbohydrates for healthy persons less than 100 grams per day. Fructose, restrict to less than 10 grams per day. Fruit restrict to less than a fruit per day Persons with carbohydrate metabolic disease << 100 g carbohydrates per day, no fructose Cellulose in vegetables can be eaten freely, Roots and tubers may be eaten in moderation include in <100 grams of carbohydrates per day. Bran and other grain fibres Avoid to reduce the risk of intestinal cancer Introduction Carbohydrates are nonessential as a food source for humans as we can make all necessary carbohydrates in enough amounts for long time survival. Humans (the standard body weight is assumed to be 70 kg) need between 1.5 and 3.0 g glucose in the total blood volume for red blood cell (RBC) energy supply and function, see Physiology and Requirement. Carbohydrates or sugars are all composed of sugar molecules from single sugar molecules like fructose, galactose, glucose to polymers like starch, fructans and</p>	Generally not in line with current scientific evidence.

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	<p>cellulose. Carbohydrates also differ significantly regarding solubility, taste and digestability. Chemical classification Carbohydrates and sugars are classified in several ways. Carbohydrate types</p> <p>Monosaccharides consists of simple sugar molecules like glucose, galactose and fructose. Monosaccharides are water soluble and are the only carbohydrates that can be transported through the mucosa from the buccal mucosa to anus. Disaccharides consists of two sugar molecules like sucrose (built up from glucose-fructose) and lactose (glucose-galactose). Also disaccharides are water soluble but they are nonabsorbable but often digestable in humans. Oligosaccharides consists of 3-9 sugar molecules, usually from glucose polymers. Oligosaccharides are partly water soluble but they are nonabsorbable but often digestable in humans. Starch consists of more than nine glucose molecules, often branched chains of glucose like amylopectin or helical straight like amylose. Starch is not very water soluble at room temperature and they are nonabsorbable but often digestable in humans. Cellulose consists of long chains of glucose and are water insoluble and they are nonabsorbable and nondigestable in humans. Other carbohydrates like sugar alcohols and other carbohydrate like molecules may be converted to monosaccharides and should be regarded as carbohydrates . Fibers like bran and other stiff fibrous materials may increase the risk of cancer in the intestinal tract. This is due to the rough material in roughage that mechanically destroys the surface of the intestines which is responding appropriately by excreting more fluid and increase the passage speed to get rid of inappropriate material Biochemistry All carbohydrates consists of simple sugars and can remain as simple sugars like glucose, galactose and fructose These small monosaccharides are water soluble and easily absorbed into the blood stream . Glucose intake increases blood glucose level and that enhanced blood glucose level increase the release of insulin, a pancreatic hormone necessary to normalize the blood glucose level as fast as possible. There are two major pathways to break down glucose to normalize an increased blood glucose level Path number one is glycolysis to 1,3-diphosphoglycerate and then to acetylcoenzyme A. Acetylcoenzyme A (can be called activated acetic acid or simplified to acetic acid or abbreviated to AcCoA) can be used to produce energy in the Krebs' cycle in the mitochondria. But AcCoA can be a source to make fatty acids, many amino acids, cholesterol and other hormones, coenzyme Q10 and very many other molecules. The major source of energy in the body is fat derived AcCoA. Proteins can also be metabolized to amino acids and finally AcCoA. So AcCoA can be produced by fat, proteins and carbohydrates and AcCoA can produce a lot of molecules needed in the body. This means that AcCoA is the central metabolism molecule in the body. Galactose and fructose are normally not converted to glucose even as there are minor pathways making it possible. Both monosaccharides are metabolized in the liver and the final metabolism product is AcCoA which is converted to fatty acids in the liver. So galactose and fructose are converted to saturated fatty acids and fructose may give rise to non alcohol fatty liver disease and eventually non alcohol liver cirrhosis. Disaccharides (and higher polymers) are non absorbable in the intestines and the di- and polymers require enzymatic breakdown into monosaccharides to be absorbed. Starch and glycogen are easily transformed to glucose. Consequently those polymers cause the blood glucose to increase and thus their intake should be limited. Cellulose and other fibers consisting of animal</p>	

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	<p>nondegradable glucose polymers. Herbivores have a fermenting tank, cloven-footed animals are front fermenters, equipped with a large fermenting tank of 100 L volume for cows, the ventricle. Hoofed animals are rear fermenters and their colon or caecum are the fermenting tanks. The fermenters are always microorganisms that are able to ferment cellulose. All fermenters ferment fibers not to glucose but to short saturated fatty acids of two to six carbon atoms length. Those short chain fatty acids (SCFA) are all water soluble and are thus easily transported in the blood mainly to the liver for further processing to longer chain saturated fatty acids. The fatty acids are then transported from the liver to fat cells for storage and later use. All blood glucose meters measure only glucose. So there is no easily available meter that measures the level of fructose or galactose in the blood. So by eating fructose containing food (fruit, plain sugar (saccharose) high fructose corn syrup (HFCS)) we have not got a clue about the levels of fructose or galactose. Most other simple sugars are in such a low amount that they are insignificant compared to glucose or fructose. So by replacing glucose by fructose we are actually stepping out of the ashes into the fire. More in Toxicity Physiology All mono-, di- and polysaccharides the human body needs are produced by the liver and other supporting cells in the body in enough amounts . It's been shown that the liver may produce up to 2 000 grams of glucose per day if necessary. Patients with diabetes have a higher production of glucose than healthy persons . Glucose is the major sugar in the body. Glycogen, branched animal polyglucose similar to vegetable starch, is available in the liver and in the muscles, about half a kg in toto. Most of all the other sugars are bound in different cellular organelles like glycated proteins, DNA and RNA to name a few. A human needs to have 3-6 mmol/L of glucose in the blood for red blood cells (RBC) anaerobic energy production as RBC lack mitochondria. Also, the specific RBC metabolic pathway (Rapoport Luebring Pathway) anaerobically converts glucose via 2,3-DPG to lactic acid. The 2,3-DPG is required for the release of oxygen from the haemoglobin molecule. All brain cells are equipped with mitochondria and are using solely AcCoA and oxygen as energy source. This means that the brain is not driven by glucose but the RBC do need glucose for oxygen release and intra-RBC energy production. So there is no need for a human to eat carbohydrates, children excepted. Just let us look at a breastfed newborn baby. Via the breast milk the baby gets about 8 percent of the total energy (E%) as animal protein which equals about 1 g protein/kg body weight. This is enough for the child to grow lengthwise by 50 % during the first year of life. Then the baby gets 36 E% of lactose, an animal carbohydrate, which is enough to increase the weight threefold during the first year. This equals to about 35-80 g of lactose per day. Finally, the baby gets 56 E% animal fat for the brain to grow normally and for energy production. It's about 0.1 g of animal fat per kcal which equals to about 200 g fat to an adult female or 300 g fat to an adult man. Also, the milk contains everything else for the survival of the child and the species. This diet for a child is time-proven to be the best food for a child below the age of one. Scientific reports from the 19th century and 20th century as well as the 21st century show that humans eating very low amounts of carbohydrates are very healthy and long living So a healthy human can have up to 100 g of carbohydrates per day to stay healthy but more carbohydrates increases the risk of metabolic diseases including death from carbohydrate intoxication. Forty percent of all patients with</p>	

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	<p>diabetes on a high carbohydrate diet are dead within five years after diagnosis Endocrinology An increase of blood glucose level above 6 mmol/L increase the release of insulin, an anabolic hormone produced in the pancreas. A decrease of blood glucose level increases the catabolic hormones like epinephrine, norepinephrine, ACTH, glucagons, TSH, T3, T4, vasopressin, growth hormones. This is like the anabolic insulin on one arm of a beam balance is counteracted by catabolic hormones on the other arm of the balance. By increasing glucose the insulin increases and the catabolic hormones are decreased . This is as a part of the body's response to an increased glucose level. • First of all, insulin slows down the intestinal movements and glucose uptake. • Next, insulin blocks fat and protein degradation to AcCoA as those sources of AcCoA is competing with the degradation of glucose to AcCoA and glucose level normalization is of highest priority. Too high a level of glucose is lethal, see Toxicity. AcCoA is produced from glucose to be burned in the mitochondria. • The body increases the body temperature to increase fuel consumption • The excess of AcCoA produced is polymerized to saturated fat of up to 18 carbon atoms length. That fat is transported to the fat cells and is stored and locked in by the insulin. • Insulin via insulin receptors make the cells take up more glucose in an attempt so normalize the blood glucose level. But every glucose molecule is surrounded by 190 water molecules which increases the volume 19-fold. This limits the glucose uptake and then the cell gets swollen at the brink of cell membrane rupture. Then the cell withdraw its insulin receptors to protect itself from cell membrane rupture. • We call it Insulin resistance and we punish the selfprotecting cell by injecting much more insulin. • The insulin tries to direct more glucose to make more glycogen, the storage form of glucose. But we can not store more than about 0,5 g glycogen in liver and scelettal muscles. • If the glucose level in the blood exceeds 10-15 mmol/L the glucose reuptake from primary urine is overloaded and glucose is leaving by urine, you get thirsty and drink more water. • If this is not enough, the body pulls the emergency brake. This means that the body has to burn glucose anaerobically (without oxygen) to make ATP, the intracellular energy produced normally from AcCoA in the mitochondria. One glucose molecule gives rise to 38 molecules ATP in the Embden-Meyerhof and Kreb's cycle pathways in the mitochondira. By burning glucose anaerobically the body is using 19 times more glucose to obtain the same amount of ATP. There is one major drawback, this metabolic pathway produces two molecules of lactic acid which makes the body more acid. This is called ketoacidosis, usually only patients with diabetes type 1 are affected. This condition can be lethal if not treated properly by more insulin and liquids without any more carbohydrates. The cause is insulin deficiency and carbohydrate overload. The body uses all these different mechanisms to normalize the glucose level to the normal range of 3-6 mmol/L. As shown above an increased glucose level increases the insulin level which increases conversion of glucose to fatty acids that are stored in the fat cells. Eat 100 g of carbohydrates and they will give energy for approximately 4 hours (h). for a normal human. The glucose level increases fast which increases the insulin level that converts about 50 g of glucose to about 10 g of fat. This means that the remaining 50 g glucose are used up after 2 h, the blood glucose level will decrease craving more carbohydrates to restore the blood glucose level up to normal level of 3-6 mmol/L. So the person eats another 100 g glucose and the process is repeated, 50 g carbohydrates</p>	

Organisation/ Company	General and detailed comments	WG response and actions
	<p>converted to 10 g fat that is stored. This means the person has overeaten by 100 percent and stored an additional 20 g fat in the body. If the same person had had 44 g of fat (equal to 55 g of 80 % butter) the same person had been satisfied the whole four hours, not getting hungry and having no tendency to overeat. This means that the carbohydrates are forcing persons to overeat for endocrinological reasons. So by restricting the carbohydrates to less than 100 g of carbohydrates most persons will have a normal glucose level and thus keep a normal insulin level. Persons with a metabolic disease has to lower the carbohydrates in food to even lower amounts of carbohydrates to normalize the glucose and insulin levels. Nutritional classification Carbohydrates are classified as a macronutrient as they are energy contributors. They are correctly called “Empty calories” by Livsmedelsverket in Sweden. Carbohydrates have no other nutritional value. Vegetables and fruit usually have less or much minerals and vitamins than food of animal origin per 100 g and even less per 100 kcal Toxicology Glucose is toxic in higher concentrations than 6 mmol/L which corresponds to 3 g glucose in the total blood volume of a normal 70 kg person. Glucose is easily nonenzymatically chemically bound to proteins. It is called Advanced Glycosylation Endproducts or AGE. The most well-known AGE is glycosylated haemoglobin called HbA1c. HbA1c has a glucose or fructose molecule attached to the amino acid valine in the N-terminal of the β-chain of the haemoglobin molecule . This addition of sugar to haemoglobin will sterically block the oxygen molecule from attaching to the haemoglobin which means the haemoglobin can not transport oxygen any longer. The sugar is irreversibly bound to the protein and the haemoglobin stays in the circulation up to 180 days when the RBC is taken out of service and recycled. A glucose level above 30-50 mmol/L is lethal. This means that the total blood volume of a 70 kg person contains more than 15-25 g glucose. A blood glucose meter measures only glucose. There are no freely available blood fructose meters so there is no easy method to measure fructose in the blood.. We know that fructose is five to ten times more potent than glucose to nonenzymatically bind to proteins and thus causing AGE . Also, the major fructose pathway is detoxification of the aldehyde fructose to AcCoA and then to fatty acids . Fructose is not glucose. Fructose does not suppress ghrelin. Acute fructose does not stimulate insulin (or leptin). Chronic fructose exposure promotes the Metabolic Syndrome Fructose increases de novo lipogenesis . Drugs and carbohydrates. There are quite a few drugs that can manipulate the level of glucose. Most catabolic hormones increase the level of glucose and insulin restores a high level of glucose. Statins decrease the release of insulin from the pancreatic beta cells as insulin needs large local concentrations of cholesterol in lipid rafts to release insulin from the beta cells Energy content The energy content of soluble carbohydrates reduced to monosaccharides are about 4 kcal per gram or 17 kJ per gram when the monosaccharides are metabolized via the Embden Meyerhof pathway to AcCoA and the AcCoA is running through the Krebs cycle. Any other parallel pathway generates less energy. Anaerobic digestion of glucose generates 0.9 kJ/g glucose or 0.2 kcal/g glucose Fibers per definition contain no energy at all. There are some rat studies hypothesizing contending that as rats are able to digest some fibers then humans have the same ability. This is quickly disproven by eating only fiber and measuring the hydrogen production measured in exhaled air. There is no hydrogen production when</p>	

Organisation/ Company	General and detailed comments	WG response and actions
	<p>humans eat only fibers. Vegetables contain carbohydrates and other nutrients encapsulated within cell walls of cellulose. As humans have no way to digest cellulose the nutrients are locked in by the cellulose. By cooking or freezing or mechanically breaking down the cellulose walls humans can reach the intracellular content of vegetables. But vegetables do have very water-diluted macro- and micronutrient content. You have to eat about 20-30 kg of cucumber to have 2 500 to 3 200 kcal per day. Requirement There is no requirement of carbohydrates. Carbohydrates in excess may be transformed to saturated fat. Carbohydrates in excess of insulin response is toxic and may be lethal if not enough insulin is present. Recommended intake The recommend intake of digestable carbohydrates for healthy persons is less than 100 grams per day. Restrict the amount of fructose to less than ten grams per day. Cellulose in vegetables can be eaten freely, roots and tubers may be eaten in moderation to limit the carbohydrates to less than 100 grams per day. Fruit may be eaten less than a fruit per day because of the high fructose content Bran and other grain fibres are nonessential and increase the risk of intestinal cancer and should be avoided. Persons with a carbohydrate metabolic disease are recommended to recognize their carbohydrate limit below 100 g carbohydrates per day and they should not eat fructose containing food.</p> <p>Adding a formatted and readable copy via ordinary e-mail to the scientific secretary</p>	

Public consultation NNR 2012: Chapter 12 (Protein)

Org/ Company	General & detailed comments	WG response and actions
University of Bergen, Norway	<p>Detailed and general comments are given below</p> <p>In general the chapter on protein in the NNR 2012 gives a good background for the given recommendations. Here are some few comments and suggested changes: There term “good-quality protein” is used repeatedly throughout the document. It seems like the background for this is presented in line 115-127. However, I have some comments to this section and the use of the term. As mentioned, it is preferable to have a good distribution of essential amino acids and also a high protein digestibility-corrected amino acid score. The protein digestibility-corrected amino acid scores are very similar for many animal food sources and plant sources. In fact, many soy products have a similar and in many cases higher scores than several animal food sources. A preferable distribution of amino acids can be achieved through an intake of e.g. both grains and legumes combined. To avoid confusion, it could be reasonable to avoid the use of “good-quality”. The paragraph in line 115-127, could also be slightly modified to e.g. “Dietary proteins of animal origin (meat, fish, milk, and egg) or a combination of plant protein from e.g. legumes and grains, will give a good distribution of essential amino acids. Most proteins are also reasonably well digested though with slightly lower digestibility of grains. In practice, the differences in quality between proteins may be less important in diets containing a mixture of proteins (8).” Accordingly, the sentences in line 58-61 it is written “Dietary proteins are found</p>	<p>Good-quality protein is a term used as basis for the WHO/FAO/UNU recommendations and accordingly also in other recommendations (e.g. IoM).</p> <p>We have now introduced the protein digestibility-corrected amino acid score in the text and rewritten the sentences</p>

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	<p>in almost all foods of animal and plant origin. Meat, fish, milk and eggs have a high content of protein of high quality. Pulses, nuts and seeds also have high protein content. That makes them important sources of proteins in vegetarian diets, especially for vegans who also exclude milk and eggs from their diet." I suggest that this is shortened to: "Dietary proteins are found in almost all foods of animal and plant origin. Meat, fish, milk and eggs, legumes, nuts and seeds all have high protein content."</p> <p>In line 619, there is a small spelling error "diseases" which could be changed to "diseases".</p> <p>In line 769-772, it is written with reference to the review by Hörnell et al that "The protein source seems to be important as there was suggestive evidence that intake of animal protein, especially from dairy, have a stronger association with growth than vegetable protein has."</p> <p>Based on my interpretation of the systematic review by Hörnell et al., increased growth seems to be related to weight/increased BMI except a cross-sectional study among 90 children by Hoppe et al. which also suggests a length difference. However, figure 1 and 2 in the article by Hoppe et al. shows substantial spread in the measurements indicating very low precision. Thus, it could be reasonable to change the sentence in line 769-772 to e.g. "A high intake of animal protein, especially from dairy, has been suggested to be association with increased body weight." (Hoppe C, Udam TR, Lauritzen L, Molgaard C, Juul A, Michaelsen KF. Animal protein intake, serum insulin-like growth factor I, and growth in healthy 2.5-y-old Danish children. Am J Clin Nutr 2004; 80: 447-52.) (Hörnell A, Lagström H, Lande B, Thorsdottir I. Protein intake from 0 to 18 years of age and 1039 its relation to health: a systematic literature review for the 5th Nordic Nutrition Recommendations. Food Nutr Res 2013; 57. doi: 10.3402/fnr.v57i0.21083.)</p>	<p>Corrected</p> <p>We have emphasized the importance of weight (see line 791..) and Hoppe et al is included in the SR by Hörnell et al.</p>
LRF Dairy Sweden	<p>General comments:</p> <p>LRF Dairy Sweden is concerned about the time frame for commenting the fifth batch of nutrition recommendations. In total 807 pages are published in batch 5 to be read and commented on by 18 July the latest, in the Swedish holiday season. This is not respectful to the scientific community and stakeholders giving feedback on the recommendations. This procedure lowers significantly the reliability of the final recommendations and also on the process and transparency. If not the background material is open for comments, it is of no use to comment on the chapters on recommendations based on the background material. Several of the systematic reviews are not submitted to Food & Nutrition Research yet, and have therefore not gone through the review process. The order of the total process of establishing nutrition recommendations is questionable. The references should be updated.</p> <p>Detailed comments:</p> <p>What is the reason for pointing out associations that is inconclusive? Example page 8, lines 364-368: "Vegetable protein intake was found (inconclusively) associated with decreased fracture risk (Pedersen et al 2013), but this finding was not supported by older studies...It is misleading to the readers and consumers</p>	<p>Generally the time schedule of the NNR5 revision has been postponed due to the overwhelming burden of work. We also regret the timing and the deadline</p> <p>Sometimes it may be appropriate to mention NS results in the overall relationship. But we shall consider whether the sentence needs to be clarified/ reworded</p>

Org/ Company	General & detailed comments	WG response and actions
Norwegian Directorate of Health	<p>General comments:</p> <p>We endorse the main conclusions and recommendations in this chapter. However, we have some comments that we ask the NNR-project group to take into account when revising the draft. You may improve the explanations about how you arrive from adequate protein intake expressed as g/kg BW to recommended protein levels expressed as E%. If the thinking round these transitions and recommendations is not changed since last edition, this was adequately explained in NNR 2004.</p> <p>You suggest raising the recommended intake of protein from 0.83 g for adults to 1.2-1.4 g/kg BW among the elderly, and that the target for protein intake among elderly should be 18 E%. This is quite different from the conclusions and recommendations in NNR 2004, see e.g. page 204. Please clarify if the increased need for protein among elderly (65+ years) is a consequence of normal aging or mainly a consequence of increased prevalence of illnesses. We doubt that an average active and fairly healthy person aged 65-75 years need a protein intake of 18 E% to cover protein requirements. However, when this is said, an Acceptable Macronutrient Distribution Range for protein of 10-20 E% seems realistic in relation to current dietary habits and protein intake in the Nordic populations.</p> <p>UL – you should consider including a comment regarding UL for protein in the introduction, and in chapter 1. In order to be precautionary in relation to potential adverse effects of high protein intakes you may introduce a similar wording as have been used for polyunsaturated fatty acids in several editions of NNR. The average protein intake among small children and adults are in large groups of our populations now close to the upper range of recommended intake. This may have been further discussed in this chapter.</p> <p>Detailed comments</p> <p>I. 28. There are some inconsistencies in the recommendations given in the box versus figures in</p>	<p>The recommendation is not changed from the NNR 2004, but we found it appropriate also to “translate” to the corresponding g/kg BW, since the systematic reviews also deal with g/ kg BW. As mentioned p 13 line 634 we used reference energy intakes for healthy adults with a PAL of 1.6 to express E% as g/kg BW as well.</p> <p>The NNR 2004 recommendation for adults and the elderly was not 0.83 g / kg BW, but it was expressed in E% and in the range of 10-20 E% and with a population goal of 15 E%, which should be used for planning purposes as well. In the present recommendation (NNR 2012) for elderly the range is narrowed to 15-20 E%. The background is that with the age-related decrease in energy intake elderly it may not result in a sufficient protein intake in the range of 10-15 E%. The NNR 2012 do not include population goals anymore (only ranges), but they do deal with diet planning. The target for planning purposes is changed for the elderly from 15 E% to 18 E%. The increase in the recommendation for the elderly are based on the assumption that the requirement based on N-balance studies is the same for elderly and other adults, but the optimal intake based on outcomes like mortality, morbidity and functional outcomes point at a higher intake.</p> <p>It is not possible to establish an UL for protein and that is why we only mention it in the protein chapter</p>

Org/ Company	General & detailed comments	WG response and actions
	<p>the general text line 29-37, as well as figures given in chapter 1. It is appreciated that you give figures concerning recommended intake of protein as gram per kg body weight to cover requirements both in the box and elsewhere in the chapter. However, you may give a better explanation how the figures for absolute intake g/kg BW and E% correspond and differs. In our understanding NNR on line 634-7 NNR concludes that a protein intake of 0.83 g per kg BW and day cover requirements for protein (amino acids) for adults. And as you mention here this amount corresponds to a dietary protein content of approximately 10 E% for adults with common physical activity in the Nordic countries.</p> <p>However, you recommend a protein intake in the range 10-20 E%. We assume that the upper level of this range is primarily set out of practical reasons. In NNR 2004 (page 14) this was well explained: "Protein might provide less than 10 E%, but in order to achieve a varied diet according to Nordic dietary habits the recommendation is 15 E% for planning purposes. This intake of protein should more than adequately met the requirements for essential amino acids." You should consider using this expression again both in the protein chapter and in chapter 1. If not you should explain why the new NNR in this context differ from NNR 2004.</p> <p>UL – you should consider including a comment regarding UL for protein in the introduction, and in chapter 1. As you conclude on line 858 a UL cannot be established. However, you on line 812-63 report potential adverse effects of high protein intakes, and on line 854-8 you mention "potential harmful effects of a long-term dietary protein intake above 20-23 E% the UL for protein intake for adults of 20 E% is unchanged."</p> <p>In order to be precautionary in relation to potential adverse effects of high protein intakes you may introduce a similar wording as have been used for polyunsaturated fatty acids in several editions of NNR. "An intake of polyunsaturated fatty acids exceeding 10 E% is not recommended, because very high intakes may theoretically increase the risk of adverse health effects."</p> <p>The time line for age in the heading of this box is a bit awkward. You may change the order and follow increasing age and start with 6-11 months from left to right in the heading. 30-42. The wording of this section may be improved. It is a bit awkward that you here translate protein recommendations backwards from E% to g/kg BW. Line 31 here you recommend 0.9 g/kg BW for age group 2-17 year, and on line the recommendation is 1.1-1.3 g/kg BW for adults.</p> <p>On the next line you state that those with energy intakes below 8 MJ/d shall increase their protein intake, but you give the same figure 1.1-1.3 g/kg BW, as for adults in general. We suggest that you describe and discuss figures for absolute and relative protein intake for the different age groups separately in these sections.</p> <p>64. You may add Norway after Iceland. According to Norkost 2010-11 the mean protein intake was 18 E% (SD 4 E%). Thus a considerable proportion of the adult population had protein intake above 20 E%.</p> <p>146-60. Wording may be improved. 149. "differences between" – what?</p>	<p>Yes, the box includes some inconsistencies compared to the text. We shall clarify this. The numbers in g/kg BW reflects the recommendation for planning purposes, 15 E% and 18 % for adults and elderly, respectively, and not the recommended range.</p> <p>According to our assumptions regarding the connection between E% and g/kg BW a protein intake less than 10 E% may not be sufficient to cover the requirement (and the subsequent recommendation based on N-balance studies) among persons with a PAL less than 1.6. That is why we have modified the expression</p> <p>In line 857 we state that: the upper range for protein intake of adults of 20 E% is unchanged Thus the range is unchanged, but we cannot establish a upper intake level (UL)</p> <p>We have changed the wording so that it includes that the recommendation takes into account the potential harmful effects.</p> <p>The numbers refer to different age groups, this is clarified in final version.</p> <p>In line 39-42 we mention the elderly with energy intakes below 8 MJ</p> <p>Yes</p> <p>'estimated requirements' is added</p>

Org/ Company	General & detailed comments	WG response and actions
	<p>432. It is a bit surprising that only one RCT has body composition as outcome in relation to protein intake. In the extensive section concerning Protein and physical exercise (489-590) you refer details from lots of different studies. You may consider putting these two sections closer to each other.</p> <p>425-70. You may add a conclusion/summary at the end of this section.</p> <p>489-90. This section is long and difficult to read and comprehend. Wording may be improved and the text could preferably be shortened.</p> <p>512-3. here you quote that muscle mass increased whereas fat-free mass decreased –is the quote correct?</p> <p>634-50. These paragraphs contain important information, however, the wording may be improved. See comments above.</p> <p>634-7. You may use a broader pen here, as a protein intake of 0.83 g/kg BW corresponds to approximately 9-11 E% among adult men and women with PAL at 1.6 and 1.8.</p> <p>636. Here you in a way claim that 10 E% represent the lower intake level for adequate protein intake among adults with PAL of 1.6. This wording may be misleading. See earlier comments.</p> <p>638.</p> <p>Based on the available evidence, This sentence should be reformulated. As we understand it the upper range of the recommendation 20 E% protein to the general population is based more on common sense and practical considerations than on hard evidence.</p> <p>647-50. Here you state: "In relation to physical activity" "no special considerations is given to physical activity levels in the present dietary recommendations." The paragraph should be reformulated and made clearer. First, NNR recommendation take physical activity into consideration, and this had been repeatedly discussed in relation to levels of energy intake and PAL in earlier sections. Second, if you want to make a statement concerning protein intake in relation to exercise and body building you may do this more explicit. E.g. use the conclusions from sections regarding Muscle mass, strength and function and Protein and physical exercise.</p> <p>653. Please define elderly.</p> <p>690. You suggest raising the recommended intake of protein from 0.83 g for adults to 1.2-1.4 g/kg BW among the elderly (an increase of approximately 50 %), and that the target for protein intake among elderly should be 18 E%. Please clarify, discuss or comment if the increased need</p>	<p>According to the eligibility criteria for the systematic review only one study was included among adults, while other studies are included in the discussion part regarding exercise.</p> <p>No change</p> <p>Shortened in final version</p> <p>It is more correct to mention that it was muscle hypertrophy (expressed as an increased mid-thigh muscle area) but if is correct that fat-free mass decreased.</p> <p>We have reformulated to: At reference energy intakes (see the chapter on Energy), a protein intake of 0.8 g/kg BW/day corresponds to approximately 10 E% from protein provided a moderate physical activity level (PAL) of 1.6.</p> <p>Thus, 10 E% protein might represent the lower intake range for healthy adults with a PAL of 1.6.</p> <p>The upper range of the recommendation is <i>both</i> based on the available evidence, <i>and</i> according to the Nordic dietary habits.</p> <p>Reformulated in final version</p> <p>In the SR and at line 684 we have defined elderly as > 65 years</p> <p>See comments above regarding general comments</p>

Org/ Company	General & detailed comments	WG response and actions
	<p>for protein among elderly (65+ years) is a consequence of normal aging or mainly by increased prevalence of illnesses. Does an average active and fairly healthy person aged 65-75 years need a protein intake of 18 E% to cover protein requirements? These recommendations are quite different from NNR 2004 page 204: "A protein intake corresponding to the intake for adults, i.e. 10-20 E%, is recommended since this amount also seems abundant for the elderly. For food planning purposes for elderly people with very low energy intake (<6.5 MJ), the protein intake should be at least 1.0 gram protein per kg body weight per day." The NNR-SLR by Pedersen et al. conclude: "... that the evidence is assessed probable regarding estimated average requirement of 0.65 g protein of high quality per kg body weight per day based on N-balance studies and the subsequent recommended dietary allowance of 0.83 g good-quality protein per kg BW and day represent the minimum dietary protein intake of virtually all healthy elderly persons. This corresponds to an average intake of approximately 10 E% from protein. The estimation of optimal level of protein intake based on the evidence of ranging from suggestive to inconclusive, but altogether pointing in the direction of beneficial effects from a somewhat higher intake than the minimum need (page 10)" "Although in particular some long term prospective studies indicate that an intake of up to at least 1.2-1.5 g protein/kg BW/day (i.e. approximately 15-20 E%) is safe and may have beneficial effects, adequate enough data do not exist to estimate this optimal intake of protein based on the main physiological end points in the elderly (page15)" We doubt that an average active and fairly healthy person aged 65-75 years need a protein intake of 18 E% to cover protein requirements. You may reconsider the suggested changes in the protein recommendations, and they should be commented in an additional section "Reasoning behind the recommendations" in the main chapter on protein as in most other chapters. However, when this is said, an Acceptable Macronutrient Distribution Range for protein of 10-20 E% seems realistic in relation to current dietary habits and protein intake in the Nordic populations.</p> <p>736. Table 13.1 Here you introduce the concept "Safe level" of protein intake. The expression safe level often is used in relation to UL and adverse effects of high intakes. You should consider using "adequate" as in NNR 2004.</p> <p>For age 12-23 the figure for E% is 12-15. It should be 10-15 E%.</p> <p>752-81. Before you conclude with the recommendations at the end of these paragraphs you should consider commenting the high protein intakes among in 1 and 2 year old children in Nordic countries in relation to the NNR-SLR conclusion that the evidence was convincing that higher protein intake in infancy and early childhood contributes to increased risk for later obesity. Norwegian 2 year old children had a mean protein intake of 14.8 (SD 2.0) E% in a nation-wide dietary survey 2007. Icelandic dietary surveys reports mean intakes of protein of approximately 15 E% among 1 year old children. Thus the mean intake of protein s close to the upper range of 15 E%, and a great proportion of them is above 15 E%.</p> <p>793-97. These sentences are difficult to read. Please reformulate.</p>	<p>This is the term used by FAO/WHO 2007, http://whqlibdoc.who.int/trs/WHO_TRS_935_en_g.pdf</p> <p>Corrected</p> <p>This has now been underlined in the chapter, in two places in the sub-chapter "Infants and children" (lines 774-6 and 784-7)</p> <p>No change</p> <p>No change</p> <p>No change</p>

Org/ Company	General & detailed comments	WG response and actions
	<p>834-42. If possible please conclude at the end of this paragraph.</p> <p>863. Please give a conclusion at the end of this section.</p> <p>864. Please add the section "Reasoning behind the recommendations". This has been very useful in other chapters.</p>	Has been included in final draft
<p>Dept. of Renal Medicine, Aarhus University Hospital, Denmark</p>	<p>General comments</p> <p>Comments on suggested increase of guideline of protein intake for elderly people (from intake as in the general population (15% energy as protein) to 18% of energy or 1.2-1.4 g/kg body wt: The literature review of Pedersen and Cederholm is of a great help to get an overview, and as the authors state the evidence is not more than suggestive, that recommendations should be changed. It is likely that the same possible benefit inhibiting sarcopenia and bone loss could be achieved through optimising energy intake and spreading meals over the day – all including some protein. Such strategy (page 12) will be without the possible side effects of a general increase of protein intake in the elderly. Although we agree that side effects of elevating protein intake are not clear from short term studies in humans, a high protein diet has shown deleterious in rodents as well as pigs, and if general suggestions for protein intake are elevated the following negative impacts may follow: 1) renal lesions in people with a normal glomerular filtration rate (GFR), particularly persons with adipositas and diabetes mellitus 2) progression of chronic kidney disease (CKD), 3) more hypertension (sodium follows protein) 4) high protein intake may be associated to higher cardiovascular and cancer mortality 5) more gout related to uric acid 6) more stones in the urinary tract 7) loss of appetite due to accumulation of uremic toxins in persons with unknown CKD 8) reduced quality of life related to more rules to follow and expenses to food supplements Thus, side effects may likely overshadow possible advantages, and we believe more evidence should be gained before changing recommendations for the public on protein intake.</p> <p>Detailed comments</p> <p>Ad 1) As stated on page 5 increasing protein intake causes an acute increase in GFR1 (hyperfiltration) opposite what is seen when initiating blockade of the renin-angiotensin system. Hyperfiltration and the likely associated increase in intraglomerular pressure may cause lesions as shown in the healthy pig within 4 months. Ad 2). In addition, ignoring the acute effect of protein intake on GFR when analyzing long term study effects of changes in protein intake will obviously blur the results. In accordance with the pig study, one study in obese and otherwise healthy humans3 demonstrated enlarged kidneys after 6 months on a moderately high protein diet. Ad 2) 10-15% of populations like the Nordic suffer from different degrees of CKD, most of them without knowing it. In the elderly, a much higher fraction of "the healthy population" will have CKD, since GFR decreases with age and more so in hypertensives. Protein restriction has been recommended in CKD4, but most nephrologists recommend a normal diet to improve quality of life and avoid malnutrition. A high protein diet is advised against in CKD. Actually, protein load may accelerate human aging, which is however rather speculative at the</p>	<p>The protein recommendation for the elderly is only increased slightly from the NNR 2004, i.e. the range is narrowed to 15-20 E%. The background is that with the age-related decrease in energy intake elderly it may not result in a sufficient protein intake in the range of 10-15 E%. For planning purposes we have increased the recommendation from 15 E% to 18 E% (an increase of 20 % compared to the NNR 2004) and it is mainly based the SR pointing at a higher optimal intake regarding morbidity and functional outcomes. Just to mention – the Danish recommendation for sick people (Sygehuskosten) is also 18 E%.</p> <p>We are not recommending a high protein diet (> 25 E%) and the SR did not find evidence for harmful effects when the protein E% was less than 20.</p> <p>Since we do not recommend a high-protein diet we refer to our comments above.</p>

Org/ Company	General & detailed comments	WG response and actions
	<p>moment5. Ad 3) It is indicated on page 9 that in the Rotterdam prospective cohort study persons 70+ with 19% of energy as protein had more hypertension than those with 14% of energy as protein at 6 years follow up. A higher sodium intake related to intake of more meat may cause hypertension as well as calcium loss (page 12). Ad 4) Associations between intake of red meat and total, cardiovascular and cancer mortality were found6. Ad 5) Gout is a very common problem in the elderly, likely associated with CKD and intake of diuretics. Increasing protein intake will aggravate these problems. Ad 6) Urolithiasis increases with protein intake, it is painful and associated with urinary tract infections and renal failure. Altogether, we agree that the evidence for a change of guidelines on protein to the elderly ranging from suggestive to inconclusive (page 10) is very sparse, and since evidence on possible long term side effects of such change of guidelines are not included, we are clearly against such change of guidelines, that can show to be deleterious to the individual and of a very negative economical impact for society (future expenses to dialysis and treatment of the mentioned side effect morbidity). We fully appreciate that the new suggestion is only minor, but pushing the average may still be deleterious. For that reason we also don't recommend very low protein diets to elderly people to delay dialysis needs, although such diet was well tolerated in a high compliance population7. Peter Marckmann, MD, DMSci Chief consultant Department of Nephrology Roskilde Hospital peter.marckmann@dadlnet.dk Bente Jespersen, MD, DMSci Professor, Chief consultant Department of Nephrology Aarhus University Hospital, Skejby Bente.Jespersen@KL.AU.DK 1) Effect of short-term high-protein compared with normal-protein diets on renal hemodynamics and associated variables in healthy young men. Frank H, Graf J, Amann-Gassner U, Bratke R, Daniel H, Heemann U, Hauner H. Am J Clin Nutr. 2009 Dec;90(6):1509-16. Erratum in: Am J Clin Nutr. 2010 Feb;91(2):494 2) Long-term high intake of whole proteins results in renal damage in pigs. Jia Y, Hwang SY, House JD, Ogborn MR, Weiler HA, O K, Aukema HM. J Nutr. 2010 Sep;140(9):1646-52. 3) Changes in renal function during weight loss induced by high vs low-protein low-fat diets in overweight subjects.Skov AR, Toubro S, Bülow J, Krabbe K, Parving HH, Astrup A.Int J Obes Relat Metab Disord. 1999 Nov;23(11):1170-7. 4) Effect of a very low-protein diet on outcomes: long-term follow-up of the Modification of Diet in Renal Disease (MDRD) Study Menon V, Kopple JD, Wang X, et al. Am J Kidney Dis 2009; 53:208. 5) Klotho to treat kidney fibrosis.Sanchez-Niño MD, Sanz AB, Ortiz A. J Am Soc Nephrol. 2013 Apr;24(5):687-9. 6) Red Meat Consumption and Mortality: results from 2 prospective cohort studies. Pan A, Sun Q, Bernstein AM, Schulze MB, Manson JE, Stampfer MJ, Willett WC, Hu FB. Arch Intern Med 2012; ;172(7):555-63. 7) Efficacy and safety of a very-low-protein diet when postponing dialysis in the elderly: a prospective randomized multicenter controlled study.Brunori G, Viola BF, Parrinello G, De Biase V, Como G, Franco V, Garibotto G, Zubani R, Cancarini GC. Am J Kidney Dis. 2007 May;49(5):569-80.</p>	

Org/ Company	General & detailed comments	WG response and actions
Danish Cancer Society	General comments only I just want to congratulate the authors on an excellent thorough work and the careful SRs. If anything is missing it is recommendations for timing of protein intake in relation to physical activity and training. The SRs underline the need for future research, to support optimal intake of protein in different physiological situations.	There are only short term studies, and thus it is too preliminary to make recommendations for timing, but we mention these studies in line 532-539 The SRs underline the need for future research, to support optimal intake of protein in different physiological situations.
Swedish Food Federation	Detailed comments only Page 2, lines 85-88: Mistake in sentence "Thus the conversion factor can vary from 5.30 (sunflower) and 5.83 (wheat) in foods that contain protein with high nitrogen content to 6.38 (milk) in foods containing proteins with lower nitrogen content." This sentence should read "Thus the conversion factor can vary from 5.30 (sunflower) and 5.83 (wheat) in foods that contain protein with low nitrogen content to 6.38 (milk) in foods containing proteins with high nitrogen content."	The sentence has been changed

NNR 2012: Public consultation – Chapter 13 (Alcohol)

Organisation/ Company	Alcohol General and detailed comments	WG observations and actions
Dept Endo, Aalborg Hospital, DK	General comments The alcohol chapter focuses very much on CVD with an addendum on cancer. I suggest to include brief comments on the much lower incidence of some autoimmune diseases associated with alcohol intake. See PubMed PMID:23170908 and PMID:22802427 where a dose dependent reduction in risk up to around 4 fold was observed for both Graves hyperthyroidism and overt autoimmune hypothyroidism.	Acknowledged. However, NNR and the background review focused on common diet-related diseases. The evidence seem to be too limited for inclusion, 2 case-controls studies.
National Institute for Health and Welfare, Finland	General comments p 7 lines 283-284 'In Finland it is generally considered that occasional, moderate alcohol use is not a barrier to breastfeeding.' This is not correct and should be changed as: 'Abstinence from alcohol during pregnancy and lactation is recommended in Finland.' - this is direct translation from the official recommendation from the book written by: Kaija Hasunen, Marja Kalavainen, Hilka Keinonen, Hanna Lagström, Arja Lyytikäinen, Annika Nurtila, Terttu Peltola, Sanna Talvia. The Child, Family and Food. Nutrition recommendations for infants and young children as well as pregnant and breastfeeding mothers. Helsinki 2004. 'Healthy mothers with a balanced diet do not need to make any specific changes in their diet during pregnancy and breastfeeding. Abstinence from alcohol during pregnancy and lactation is recommended in Finland.' This is in Finnish as follows (and found also in the following web-page): Alkoholi ja odotus- ja imetysajan ravitsemus: Alkoholin käyttöä vältetään raskauden ja	Text on advice in Nordic countries removed.

Organisation/ Company	Alcohol General and detailed comments	WG observations and actions
	<p>imetyksen aikana alkoholin aiheuttamien sikiöhaittojen ehkäisemiseksi. http://www.ravitsemusneuvottelukunta.fi/attachment/vrn/lapsi.perhe.ruoka.pdf* (see comment in the end of the document)</p> <p>OTHER; MORE GENERAL COMMENTS: The weight control part is at a very general level compared to the cardiovascular part. Could it be that the association between alcohol consumption and weight is different for each alcohol group (light, moderate or heavy drinkers)? What are the definitions for “abstainers, light and moderate” drinkers in this review? They are somewhat different through the publications. For example, are abstainers lifetime abstainers, last-year abstainers or can they drink alcohol small amounts? What about the difficulties in measuring alcohol consumption? It would be nice to have a brief discussion on that; usually we catch 30-50% of alcohol consumption in epidemiological studies/surveys compared to national alcohol selling statistics and heavy drinkers poorly participate in studies. Furthermore, abstainers often have a very heterogeneous history with alcohol or various reasons leading them not to drink. This is relevant when the final recommendations are planned. For example, the sentence “...alcohol accounts for about 3-6% of energy intake in adults” is based on the study results but it is clearly an underestimation of the real situation due to the difficulty of measuring alcohol consumption. In Finland, those who used alcohol had twice the recommended alcohol energy in their diet (and again that is underestimation). Is red wine better than other alcohol types according to the current state of the art? Or is the main factor the amount of ethanol consumed? What is the situation of the French Paradox? What about older persons. Should the recommended amounts be smaller for them? It has also been said that the current recommendations do not work because subjects do not know how much is one alcohol portion. The amount of alcohol per one portion is bigger at home than in restaurants. Perhaps, in general, we need some explanations why women have the same risk compared to the men regardless of much lower alcohol consumption.</p> <p>Detailed comments lines 283-284 ‘In Finland it is generally considered that occasional, moderate alcohol use is not a barrier to breastfeeding.’ This is not correct and should be changed as: ‘Abstinence from alcohol during pregnancy and lactation is recommended in Finland.’</p>	<p>The NNR has evaluated associations between intake of alcohol and alcoholic beverages and various health outcomes. There is uncertainty in reported consumption in most epidemiological studies, which has to be accounted for. The issue with abstainers is also can confound associations, since this category may include previous high-consumers.</p> <p>The chapter focuses on total alcohol intake, but associations with various types of alcoholic beverages are mentioned in the section on cancer, weight maintenance, and lactation.</p> <p>Examples of portions of beer, wine and spirits corresponding to units of alcohol are given as a footnote prior the reference list. Advice and information on alcohol consumption has to be adapted to the target groups and is outside the scope of NNR</p> <p>Text on advice in Nordic countries removed.</p>
Helsedirektora tet, Norway	<p>Generell kommentar.</p> <p>Til orientering: Norske anbefalinger for ernæring og fysisk aktivitet som ble utgitt i 2005 baserte seg på NNR 2004. I avsnittet om anbefalinger om alkohol var imidlertid følgende setning tilføyd: «Sosial- og helsedirektoratet vil utforme anbefalinger om bruk av alkohol ut fra en samlet vurdering av helsemessige og sosiale konsekvenser av bruk av alkohol.» Direktoratet bruker nå i 2013 følgende anbefaling om bruk av alkohol: «Drikk ikke alkohol for helsens skyld. Unngå beruselse, og tenk over de situasjonene der du inntar alkohol. Barn og unge under 18 år anbefales helt å avstå fra alkohol. Gravide anbefales helt å avstå fra alkohol.»</p>	<p>Acknowledged. The advice “Drikk icke alkohol for helsens skyld” is an implementation of the recommendations, although implicit. Adverse effects of binge drinking is also highlighted.</p>

Organisation/ Company	Alcohol General and detailed comments	WG observations and actions
	<p>Detailed comments</p> <p>31. Tallene 3-6 kan endres til 2-6. I den nasjonale kostholdsundersøkelsen blant voksne Norkost 2010-11 bidro alkohol i gjennomsnitt med henholdsvis 2,1 og 2,5 prosent av energiinntaket hos kvinner og menn. I forbruksundersøkelser i privathusholdninger 2007-9 utgjorde alkohol i gjennomsnitt 2 prosent av kostens energiinnhold.</p> <p>194. WCRF brukte begrepet «convincing» og ikke «sufficient».</p> <p>241. Det er vel tvilsomt om dokumentasjonen om sammenhengen mellom inntak av alkohol og kreft er i tråd med anbefalingen i NNR. I konklusjonen linje 238 nevner dere at det ikke er en «safe limit of intake» i forhold til kreftisiko. Nasjonalt råd for ernæring kommenterte råd om alkohol på følgende måte i rapporten «Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer. Metodologi og vitenskapelig kunnskapsgrunnlag. Oslo: Nasjonalt råd for ernæring, 2011» (side 311): «Inntak av alkohol anbefales ikke i de norske kostrådene. WCRF konkluderer i sin rapport at det ikke finnes en sikker nedre grense hvor risiko for kreftsykdommer ikke øker. Dersom man kun skulle basert seg på evidens i forhold til kreftsykdommer, så burde ethvert inntak av alkohol unngås. Siden et moderat inntak av alkohol kan redusere risiko for koronar hjertesykdom, velger WCRF følgende formulering: "Hvis alkohol benyttes, bør man begrense inntaket til to enheter for menn og en enhet for kvinner per dag". En reduksjon i risiko for koronar hjertesykdom kan imidlertid oppnås ved å endre mange andre levevaner. Et høyt inntak av alkohol øker risiko for ulykker og selvmord, og fører til mange sosiale problemer. Det er imidlertid urealistisk å forvente at man skal ha et nullforbruk av alkohol i Norge, og det er viktig at kostrådene balanseres mot matkultur og matglede. Som en helhetlig vurdering er det derfor i de norske kostrådene valgt å bruke ordlyden "Inntak av alkohol anbefales ikke" siden man ikke ønsker aktivt å anbefale at folk skal drikke alkohol. Dersom alkohol benyttes, bør man begrense inntaket til to enheter for menn og en enhet for kvinner per dag.» Man bør for øvrig være oppmerksom på at et inntak av 1-2 enheter alkohol om dagen avviker fra hva som er vanlig drikkemønster i Norge. Medianforbruket av alkohol i den norske befolkningen er lavere enn dette.</p> <p>305. formuleringen «Lactating women should also have a limited intake» anbefales endret i tråd med språkbruken på linje 298 «...is recommended to limit alcohol intake».</p>	<p>Corrected</p> <p>Corrected</p> <p>Acknowledged. However, NNR arrives at the same advice and stresses that alcohol use among adolescents and young adults has negative health consequences in the long-term.</p> <p>Accepted</p>
National Food Agency, Sweden	<p>Detailed comments</p> <p>Could the recommendations from lines 298-308 be included in a box or summarized in other way?</p> <p>78 binge- shouldn't this be binge drinking when standing alone?</p>	<p>Included</p> <p>Corrected</p>

* From Lapsi, perhe ja ruoka

Vähäinen, satunnainen alkoholijuomien nauttiminen, kuten lasillinen viiniä aterialla tai olut saunan jälkeen, ei estä imetystä. Näissä tilanteissa voi odottaa pari tuntia ennen imetystä. Alkoholit poistuu rintamaidosta samalla nopeudella kuin äidin verestä (eli noin 7 g tunnissa). Humalatilaa jälkeen on oltava imettämättä niin kauan

kunnes äiti on täysin selvä. Alkoholia on rintamaidossa lähes yhtä paljon kuin äidin veressä. Imeväisikäisellä alkoholin palaminen on hidasta, koska maksa kykenee hajottamaan alkoholia täydellisesti vasta 5-7 vuoden iässä. Lapsi on herkkä alkoholin haitallisille vaikutuksille (mm. hypoglykemia, velttous ja uneliaisuus). Äidin runsas alkoholin käyttö haittaa lasta eniten välillisesti esimerkiksi tapaturmien ja hoidon laiminlyönnin vuoksi.

In a summary it is said that alcohol should be avoided during pregnancy and lactation but in this text it is said that an occasional beer or a glass of wine is OK (the mother should wait for an hour or two before breastfeeding though).

There seems not to be any scientific evidence for this, though.

NNR 2012: Public consultation- Chapter 14 (Dietary antioxidants)

Organisation/ Company	Comments	WG comments and observations
University of East Anglia, UK	<p>General comments</p> <p>1. The Recommendations are concerned with antioxidant supplements (lines 298-299). Should the chapter be renamed? If so, then it would only be necessary provide a brief introduction on possible antioxidant mechanisms (but not an extensive description of free radical scavenging and in vitro work as this is now generally accepted as not predictive of the in vivo situation) and then describe supplementation trials. 2. According to Leitzmann (Essential of Human Nutrition (2nd ed), Mann & Truswell 2002) phytochemicals can be classified into the following groups in relation to their chemical structure: Polyphenols Flavonoids Tannins Lignans Stilbenes Phenolic acids Carotenoids Protease-inhibitors Phytosterols Phyto-oestrogen Phytic acid Monoterpenes Saponins Sulphides Glucosinolates It would be helpful if the chapter contained the above (or an alternative) scheme. 2. The chapter would be easier to follow if evidence for antioxidant effects of each of the classes of compounds and/or foods that are a rich source of the proposed active phytochemical were described and discussed in separate paragraphs / sections. For example lines 179 onwards contains a mixture of phytochemicals and foods and effects. Can these be separated?</p> <p>Detailed comments</p> <p>1. Lines 30-33. The sentences referring to 'compelling' evidence on the pathophysiology of many diseases and oxidative stress making a 'significant' contribution to all inflammatory diseases....is rather over-stated. Is there substantive evidence to support the involvement of oxidative stress in all of the diseases and disorders listed? Presumably this is the conclusion reached by the 3 authors cited, but</p>	<p>Comments are relevant and appreciated. Chapter revised, compound groups and properties more clearly described.</p> <p>Sentence modified accordingly.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>there are others who conclude differently.</p> <p>2. Line 61. REplace 'several' with many.</p> <p>3. Lines 66 onwards, also in other parts of the chapter. The action of phytochemicals (at least in cell culture and animal models) is dose-dependent.</p> <p>4. Line 165. Replace 'it is not always clear' with uncertain. The general view is that cell culture findings cannot be extrapolated to humans. It is also important to point out that it is the effect of metabolites (of which there may be very many) that should be measured, not the parent compound, unless this is the form that is presented to the tissues.</p> <p>5. Lines 179 onwards. This is a long paragraph covering many classes of compounds / foods. Can it be divided up and the potential actions of each class of phytochemicals discussed separately?</p> <p>6. Line 227. 'Disappointingly' is an emotive term. Is it appropriate here?</p> <p>7. Line 265. The 'high-dosage' vitamin E is half that of the CHAOS study (with positive findings). How do these doses relate to Nordic intakes?</p> <p>8. Line 275. Insert by implication/extrapolation at the end of the line, before 'oxidative stress related diseases...'</p>	<p>OK.</p> <p>OK.</p> <p>OK.</p> <p>Partly revised and clarified</p> <p>Deleted.</p> <p>Reworded</p> <p>Sentence removed, new section on Dietary antioxidants and health</p>
University of Helsinki, Finland	<p>General comments</p> <p>I cannot see any rationale for the NNR “antioxidant” chapter as it is. We know that the human body needs, eg, vitamins C and E. If we will have Nordic Nutrition Recommendations, we must summarize knowledge about vitamins C and E and we must give some reference intake levels for them. However, to my knowledge there is no evidence that human body needs carotenoids or plant phenolic compounds etc. Given that the conclusion of the antioxidant chapter is “the human studies published so far do not support a beneficial effect of antioxidant supplements”, I cannot see why the antioxidant chapter would be needed at all in the NNR. Human beings ingest a large number of substances. It is not reasonable to extend nutritional recommendations to cover all substances that are ingested. Limitation of NNR to essential nutrients would seem reasonable. Good science is focused on specific issues. If we want to understand what is the effect of beta-carotene on human health, then we must study the effects of beta-carotene. Instead, the antioxidant chapter is fuzzy</p>	<p>Comments appreciated. The WG has decided to include the chapter, since there is still much research in the area and that other aspects than those purely related to essential functions of vitamins and mineral play role for human health.</p> <p>Chapter revised and restructured with a focus on dietary sources.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>in its approach with many comments on plants, even though we do not know to what extent the effects of plants are caused by antioxidant vs. non-antioxidant substances. Furthermore, it is possible that many of the assumed benefits of plants are explained by residual confounding. In such respect the discussion in the antioxidant chapter is not properly focused on antioxidants per se. The author is uncritical in his comments on plants and in many parts of the text he assumes that all effects of plants are effects of antioxidants, which obviously is not the case. Science consists of experimental findings, theories, and logical argumentation. This texts is largely about beliefs, speculations and assumptions. NNR draft Chapter 3 states that systematic literature search was done on some topics but not on antioxidants. I cannot understand such policy in the preparation of NNR. A systematic literature search probably would have forced the author to look at relevant studies. The issues about vitamin E, vitamin C and selenium in the “antioxidant” chapter should be moved to the respective chapters. There should not be duplication so that those antioxidants are discussed both in their own chapters and also in the antioxidant chapter. If an “antioxidant” chapter shall be maintained, it should focus on other antioxidants (i.e. mainly beta-carotene) or on the combinations of several antioxidants. In such a case a separate “antioxidant” chapter might be reasonable. However, I suggest that it would be best of NNR to focus on essential nutrients.</p> <p>Detailed comments: Lines 15 to 72 INTRODUCTION NNR should focus on health effects of nutrients. This introduction goes too deep to the biology and chemistry of free radicals. In comparison, the introduction section of the vitamin C chapter is 5 lines and of the vitamin E chapter is 22 lines. The introduction of the antioxidant chapter is 58 lines, which is some 40-50 lines too much for the purpose. The author should select 1 or 2 reviews to lead the more-interested reader to detailed discussions about free radicals. Lines 24 to 26 “Many substances are evidenced to possess antioxidative properties in vitro but they do not necessarily act in the same manner in vivo.” This comment is important in challenging the relevance of the antioxidant chapter. This comment means that under in vitro conditions, lots of compounds are “antioxidants” because they react with, for example, hydroxyl radical. However, the same antioxidant substances may not react with less reactive oxidants that are more</p>	<p>See comments above</p>

Organisation/ Company	Comments	WG comments and observations
	<p>common in vivo. Therefore, many in vitro studies are biologically irrelevant and many “in-vitro antioxidants” are not “biologically-relevant antioxidants”. Lines 33 to 40 I cannot see any rationale to use 8 lines to list diseases for which free radicals have been proposed to have a role. In comparison, vitamin C and E chapters do not extensively list diseases in which those nutrients have been proposed to have a role (there are many). Such lists are irrelevant in this kind of text on nutritional recommendations. Furthermore, even if free radicals may have a role in a given disease, that does not imply that antioxidants would necessarily influence the disease.</p> <p>Lines 56 to 64 The antioxidant chapter should focus on antioxidants. It is not relevant to use space for listing organizations that consider fruit and vegetables good for health. That is irrelevant in the context. Besides, fruit and vegetables contain a large number of substances that are not antioxidants and we do not know to which extent the benefits are caused by antioxidants vs. non-antioxidants. Furthermore, this comment on the health benefit of fruit and vegetables is scientifically naïve since there are numerous life style variables that vary strongly with fruit and vegetables. A good illustration of this problem was published by Davey Smith's group in Plos Medicine: http://www.ncbi.nlm.nih.gov/pubmed/18076282 They summarized: “We demonstrate that behavioural, socioeconomic, and physiological factors are strongly interrelated, with 45% of all possible pairwise associations between 96 nongenetic characteristics (n = 4,560 correlations) being significant at the $p < 0.01$ level (the ratio of observed to expected significant associations was 45; p-value for difference between observed and expected < 0.000001).” Thus, the implication of that PLOS paper is that even if we adjust for smoking, alcohol, sex, age and a few other standard confounding variables, there can still be many dozens or hundreds of other variables that are important residual confounders. We do not know how much the health differences are explained by fruit and vegetables per se compared with the numerous life style variables that strongly correlate with those foods. Therefore, the comments on the health effects of fruit and vegetables are simplistic. Introduction should be cut to 10 lines or less so that 1 or 2 reviews are cited for further information.</p> <p>COMPOUNDS WITH ABILITY TO SCAVENGE OR NEUTRALIZE FREE RADICALS Lines 75 to 90 The author does not give any evidence that carotenoids or phenolic compounds have relevance as antioxidants in the human body. Frei et al. analyzed the most</p>	<p>Text moved and discussed in section Dietary oxidants and health.</p> <p>This is described in a subsequent section, Effects of antioxidant-rich diets in experimental animals and</p>

Organisation/ Company	Comments	WG comments and observations
	<p>important antioxidants in human plasma and they found that, in their model systems, vitamin C was oxidized earlier than other antioxidants: http://www.pnas.org/content/86/16/6377 http://www.pnas.org/content/85/24/9748 If the author of the antioxidant chapter considers that carotenoids and phenolic compounds have biological relevance as antioxidants in the human body, the author should give scientific references to support their role by references to studies comparable to those above. If there is no evidence that carotenoids and phenolic compounds have biological relevance in the human body, why should NNR waste space for discussing them? Where is the science indicating that carotenoids or plant phenolic compounds are biologically relevant antioxidants? Line 83 "Their major role in plants..." NNR is about human nutritional recommendations. What is their major role in humans? If there is no role in humans, why are they included in NNR? Lines 92 to 95 "glutathione ... is not absorbed" Given that glutathione is broken in the guts, I cannot see any rationale to these lines in NNR. Lines 97 to 104 Comments on vitamins C and E should be deleted as there is a separate chapter for them. The author refers to in vitro studies but does not give any data to suggest that the interactions have biological relevance in the human body. NNR should focus on the human body, and the text should not consist of speculations based only on in vitro findings. See also below.</p> <p>Lines 102 to 104 "Thus, the total amount or appropriate balance of antioxidants in the diet may be a better concept than individual dietary antioxidants." The author does not give any reference for this comment and it seems pure speculation to me. As mentioned above, Frei et al found that there is much difference between the major antioxidants in their oxidation in plasma by a model system: http://www.pnas.org/content/86/16/6377 http://www.pnas.org/content/85/24/9748 Frei's reports are is not consistent with author's speculations. Difference in the rate of oxidation between vitamin C and other oxidants means that the "total amount" of antioxidants is not a relevant concept. "Total amount" is relevant only if the reaction rate of all antioxidants with all oxidants is the same, which obviously cannot be the case for all antioxidants and oxidants. Furthermore, the Frei study shows that vitamin E is oxidized to the oxidized form only after vitamin C is first consumed. Thus, vitamin E can serve as an antioxidant even though there is no remaining vitamin C. Thus, there is no basis for the author's</p>	<p>humans.</p> <p>Included since they have antioxidant properties</p> <p>A general statement, comparable to the concept of dietary patterns vs single foods/nutrients.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>speculation about some mystic “balance of antioxidants”. Vitamin C is antioxidant without vitamin E, and vitamin E is antioxidant without vitamin C. If the author has some empirical evidence for his speculations on lines 102 to 104, the author should refer to them. If the author does not have any empirical evidence, then my view is that the Frei studies (as one example) refute author's speculations.</p> <p>Lines 107 to 108 “The antioxidant defense capacity in a plant may or may not be relevant to human health” Good point, but given such a lack of knowledge, why is space used for speculations on plant antioxidants?</p> <p>Lines 114 to 117 Given that the author does not refer to any studies that would indicate that high level of plant “total antioxidants” would have any relevance for health, this sentence is irrelevant. Lines 117 to 120 “groups of individuals eating high amounts of such antioxidant-rich foods...” The author ignores the problem of residual confounders, see above. We do not know whether it is fruit and vegetables, or some of the dozens of life style factors that are strongly correlated with diets, that explain variation in health by variation in food intake. Even if it is fruit and vegetables per se, it is not obvious that it is the antioxidants of fruit and vegetables instead of the non-antioxidants that explain the benefit.</p> <p>COMPOUNDS WITH ABILITY TO INDUCE ANTIOXIDANT ENZYMES Lines 128 to 137 Evidently, plants contain lots of substances that can influence enzyme functions in the human body. However, changes in metabolism can be adaptations without any net effects on long term health. The author does not refer to any evidence that the described effects would influence human health. Therefore, this text has no relevance in a nutritional recommendation.</p> <p>Line 134 to 137 “It is believed that...” Science consists of experimental findings, theories, and logical argumentation. Writing that someone believes something is not scientific argumentation. What is the empirical evidence? In my view, NNR should produce scientific texts instead of writings about beliefs and speculations.</p> <p>Furthermore, if we consider the theory of evolution and its biochemical implications we can reason: If the optimal level of some enzyme would be much higher, evidently evolution would have led to permanently higher level of that enzyme, because that would give survival benefit. In physiology there are lots of adaptations. When environment or food changes, some genes become activated and some inactivated and some enzyme levels go up and some go down. Such adaptive changes no not</p>	<p>Relevant in the context.</p> <p>This section describes methodological problems.</p> <p>Section describes classes of compounds. See section, Effects of antioxidant-rich diets in experimental animals and humans</p> <p>A general statement</p>

Organisation/ Company	Comments	WG comments and observations
	<p>mean that long term health changes. Author's proposal to eat "benign or non-damaging plant compounds [to] induce the phase 2 enzymes" in order "to 'neutralize' toxic agents such as free radicals and other toxic electrophiles when they appear " is as silly as proposing someone to eat penicillin each day to protect against pneumococci when they appear.</p> <p>Line 141 "it is believed.." See above.</p> <p>Lines 144 to 145 I cannot see the purpose of this kind of list. Does the author propose that readers should increase their intake of broccoli, etc. What is the purpose of such a list given that the author does not give any evidence that induction of phase 2 detoxification enzymes might influence health. Such induction can be simply an adaptation without any net benefit on long-term health, see above. Lines 147 to 148 "the importance of this defence mechanism in the overall protection against oxidative damage is still uncertain." Important point, but why should NNR waste space for speculations on topics that are so uncertain.</p> <p>Lines 152 to 154 Changes in the level of glutathione peroxidase can be an adaptation without any net effect on long term health. The author does not give any evidence that the increase in glutathione peroxidase level might influence human health. We cannot assume that all increases in enzyme levels are beneficial in long term. If the optimal levels of some enzyme would be much higher, obviously evolution would have led to permanently higher level of that enzyme, because it would have given survival benefit. If the author believes that increase in glutathione peroxidase level is beneficial, the author should give empirical basis for such a statement. Science consists of experimental findings, theories, and logical argumentation. Where is the science of lines 152 to 154. This text has no relevance in a nutritional recommendation. Lines 156 to 161 There is a separate chapter on selenium and therefore comments on selenium are not relevant here.</p> <p>PROTECTIVE EFFECTS OF... Lines 164 to 167 "The suggested protective role of dietary antioxidants against oxidative damage is often based on extensive studies in cell culture systems. It is not always clear whether the effects in cell cultures, often observed with high doses of single compounds, can be readily extrapolated to humans." As the second sentence admits, effects in a cell culture do not teach us anything about long term health effects in humans. Therefore, conclusions about health effects of dietary antioxidants cannot be based on cell culture studies. Lines</p>	<p>These are examples of foods and a statement of the current evidence</p> <p>A general statement</p> <p>This section describes studies and the current evidence.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>169 to 170 "Antioxidant-rich diets also contain a lot of compounds which are not redox active, so other mechanisms may also exist." This is obvious. However, why does the author in many other parts of the chapter assume that it is the antioxidants that are relevant in plants? Thus, the argumentation is not consistent.</p> <p>Lines 172 to 175 "Such direct proof would only come from studies where different and well-defined preparations of antioxidants would be shown to affect a specific molecular target by protecting it from oxidation and where this target could be shown to have health beneficial effects." This is fundamentally incorrect comment in two ways: First, even if a preparation might have an effect on "a specific molecular target" in a short term study with cell culture, that does not teach us anything about long-term effects in intact body. It is possible that the whole human body has lots of other targets that are not revealed in a simplistic short term cell culture study. Second, concluding from the above sentence (lines 172 to 175), the author does not understand what "antioxidant" means. "Antioxidant" means that the substance reacts with reactive oxygen compounds that are generated by biochemical reactions and released to the surrounding medium in the body. Thus, an antioxidant can protect cell surfaces and soluble biomolecules (DNA, Proteins, Lipids) against the reactive oxygen compounds. Such a phenomenon is non-specific. Oxidants do not react with some specific gene or some single enzyme, etc. Instead they attack biomolecules largely by random. By reacting with the oxygen compounds, antioxidants can protect numerous different kinds of biomolecules, but the effects are nonspecific. Thus, there cannot be specific targets for "antioxidants" in the same sense as there are specific targets for ordinary drugs (and hormones etc). Therefore the concept of "a specific molecular target" in the above sentence reveals that the author does not understand what "antioxidant" means. If an effect of a substance is specific on a target, then the effect is not an "antioxidant effect" in the sense that term is commonly used. On lines 19 to 21, the author writes: "Antioxidants are any substance that delays or prevents (quenche) oxidation of various oxidisable substrates, such as lipids, carbohydrates, proteins and DNA ". This sentence is correct and it means that the effects of antioxidants are non-specific. The discrepancy between this sentence (lines 19 and 21) and the sentence stating that antioxidants can be specific (lines 172 and 175) is puzzling. Lines 176 to 177 "suitable gold standard biomarkers of oxidative stress in vivo are needed in such studies to clarify</p>	<p>This is a somewhat complicated statement, but the compounds with potential antioxidant properties are activated through different mechanisms. It also focuses on need of controlled studies.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>the health beneficial effects of antioxidants.” This is incorrect. For example, ATBC Study and CARET study showed that beta-carotene increased mortality and PHS found no effect on health. That kind of studies can teach us about “health beneficial effects of antioxidants” or lack of them. “Biomarkers of oxidative stress” do not teach us anything about long term effects of antioxidants on clinically relevant outcomes. Another example: in many short term laboratory studies, vitamin E has positively influenced biomarkers, but those effects did not translate to health benefits in long term trials with clinically relevant outcomes.</p> <p>Lines 192 to 193 “a substantial variation of plasma carotenoid levels was observed between 16 different regions in 9 European countries in the EPIC study” The author does not give any evidence to suggest that this variation explains variation in health in the 16 regions. There are lots of cultural variation between European countries, but most of the variation does not have cause-effect relation to health. It makes no sense to list variations between EU countries in the NNR if there is no relevant conclusion from the variation.</p> <p>Lines 200 to 203 “new biochemical technologies...” Evidence-based medicine emphasizes that conclusions about intervention effects should be based on clinically relevant outcomes and not on “surrogates” such as laboratory markers. There are lots of examples of sound biochemical explanations for pathological processes, which did not materialize at the level of health. About the problems with surrogates, see for example: http://www.ncbi.nlm.nih.gov/pubmed/8815760 http://www.ncbi.nlm.nih.gov/pubmed/22711298 “New biochemical technologies” are interesting, but they cannot “confirm” any clinically relevant effects. Thus the sentence is unsound. The author does not understand the problem of surrogates.</p> <p>INTERVENTION TRIALS WITH ANTIOXIDANT SUPPLEMENTS Bjelakovic et al wrote Cochrane review about the effects of antioxidants on mortality. Why is that review not included in this section? http://www.ncbi.nlm.nih.gov/pubmed/22419320 Bjelakovic et al write: “78 RCTs with 296,707 participants were included. ... We found no evidence to support antioxidant supplements for primary or secondary prevention.” First, the review itself is important. Second, given that the author did not search the literature, he could have picked a large number of relevant RCTs from the Bjelakovic review. Lines 211 to 212 and Lines 219 to 220 The same problem as above on Lines 200 to 203 Lack of effect on a surrogate does not prove that there is no</p>	<p>This text is deleted.</p> <p>The examples are complementary indicators that might be used to identify causal relations with health outcomes.</p> <p>Acknowledged. This and other relevant studies are added.</p> <p>Acknowledged. The study</p>

Organisation/ Company	Comments	WG comments and observations
	<p>clinically relevant effect. Observed effect on a surrogate does not prove that there is a clinically relevant effect. Thus, using space to discuss surrogates is not optimal use of space, when the same space can be used to discuss the effects on clinically relevant outcomes, e.g. discussing the RCTs that are collected by Bjelakovic et al.</p> <p>Lines 224 to 240 In 1990s, three large RCTs on beta-carotene were published. This text describes two RCTs but ignores the third RCT. ATBC and CARET found strong evidence of harm of beta-carotene. The third trial, PHS, is not mentioned in this chapter; it found no benefit or harm of beta-carotene:</p> <p>http://www.ncbi.nlm.nih.gov/pubmed/8602179 Only 11% of the participants of the PHS trial were current smokers, whereas all ATBC Study participants were current smokers and CARET participants were either current smokers or had exposure to asbestos. Thus, to explain the divergence between the three trials, it is possible that smoking (+asbestos) modified the effect of beta-carotene. PHS should be included in the discussion of beta-carotene, and smoking/asbestos exposure should be mentioned as a probable/possible explanation for the divergence between the ATBC and CARET studies and the PHS study. Evidence of beta-carotene effect modification by smoking was also found in a study of common cold incidence in the ATBC Study: http://dx.doi.org/10.2147/NDS.S13299 "Among participants older than 70 years, the extent of smoking modified the effect so that β-carotene increased the incidence of colds in those who started smoking at an early age and smoked heavily: RR = 1.16 (95%CI: 1.02–1.33), but decreased the incidence in those who started smoking at a later age and smoked less: RR = 0.76 (95% CI: 0.61–0.94)." These CI:s are far from each other. The effect of beta-carotene on pneumonia incidence in the ATBC Study was also modified by smoking, by the variable "age of smoking initiation" (P = 0.004): http://dx.doi.org/10.1378/chest.125.2.557 Thus there is direct evidence of cigarette smoking exposure as a modifier of beta-carotene effect on the incidence of the common cold and pneumonia. Thus, these within-ATBC analyses give further support to the explanation that the definite harm of beta-carotene on the primary endpoints in the ATBC and CARET studies may be explained by smoking/asbestos, when PHS found no harm. Referring only to the ATBC and CARET makes the text biased. The reader gets an impression that all published large RCTs have found significant harm of beta-carotene. Possibly the harm is restricted to smokers. Lines 246 "Potential adverse effects" What are they? The potential adverse effects should be described</p>	<p>included in Bjelakovic. Also two more recent papers are included (PHSII).</p>

Organisation/ Company	Comments	WG comments and observations
	<p>explicitly and not so that the reader must go to the original paper to find out what “adverse effect” means. Lines 248 to 251 “Graat studied the effect of antioxidant supplementation on immune response...” That is not correct. “Immune response” means measuring immunoglobulins, leukocyte levels or their functions, or cytokines etc. Graat measured clinical effects on respiratory infections. That is not correctly described as immune responses. Infections are clinically relevant outcomes, immune responses are laboratory outcomes, i.e. surrogates. Furthermore, the Graat study should be described in the vitamin E chapter and not here. Lines 253 to 269 As the text mentions, the Miller meta-analysis included 19 vitamin E trials and there are more trials published after 2005. Selection of studies to lines from 253 to 269 seems fully arbitrary. As described above, the vitamin E trials should be described in the vitamin E chapter. HPS and ASAP trials may be relevant here if the chapter on antioxidants is limited to studies other than pure vitamin C and E.</p> <p>Lines 276 to 283 Is the purpose of the NNR to independently write down own recommendations, or is the purpose of NNR to search for some kind of consensus/average of the recommendations of other organizations? If the purpose is to carry out independent assessment of evidence, then the opinions of other organizations are not essential. Obviously, the author should check other recommendations for their references and arguments, but this kind of listing of other organizations is irrelevant. English is a second language for me and I am usually cautious in commenting the language of other authors. However, this draft seems to be particularly clumsy in many places. A few examples: Lines 24 to 27 “Many substances are evidenced to possess antioxidative properties in vitro but they do not necessarily act in the same manner in vivo.” In my view that could be written as a more easy-to-read version: “Many substances are antioxidants in vitro, but not in vivo.” The point of the author's comment is that: many in vitro tests use e.g. hydroxyl radicals, which reacts with very many substances and a substance can be labeled an “antioxidant” if it reacts with hydroxyl radical. However, the same antioxidant substances may not react with the less reactive oxidants that are more common in vivo. Therefore, many in vitro studies are biologically irrelevant. Line 42 “A complex endogenous antioxidant defence system has evolved in all living organisms ...” This gives an impression that the defense system has evolved independently in different organisms, whereas it evolved in very old ancestors of all of the current organisms. It</p>	<p>Section on Recommendations is deleted</p> <p>Section revised.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>is possible that the author also considers that the antioxidant system evolved in very old ancestors, but that is not what he is writing.</p> <p>Lines 276 to 277 “the human studies published so far do not support a beneficial effect of antioxidant supplements.” Lines 297 to 298 “the human studies published so far do not support a beneficial effect of antioxidant supplements.” What is the point of repeating the same sentence after 20 lines? Lines 288 to 289 “there is insufficient scientific evidence to show that the antioxidative mechanisms are specifically involved in the protective effects of fruits and vegetables.” Lines 294 to 295 “There is insufficient scientific evidence to show that specific antioxidative mechanisms are involved in the protective effects of fruits and vegetables.” What is the point of repeating the same sentence after 3 lines? REFERENCES As noted above, the Bjelakovic et al. review about antioxidants and mortality found 78 RCTs. Obviously, there are many more RCTs on other clinically relevant outcomes. This reference list contains numerous papers about berries, juices, tea, etc. see below. Scientifically relevant discussion about health effects of antioxidants should focus on RCTs with specific antioxidants (not berries and juices) and clinically relevant effects (not surrogates). Good science is specific. If the topic is “antioxidants” then the focus should be on antioxidants and not on berries and juices and teas etc. Also, the reference list contains lots of reviews. 1 or 2 reviews are reasonable for guiding the reader to more detailed texts. However, listing over a dozen reviews in this kind of chapter is unreasonable. 18. Broccoli sprouts: 19. induction of phase 2 enzymes 20. phytochemicals 21. blueberry, spinach or strawberry dietary supplementation. 22. black raspberries 23. berry extracts. 24. Walnut polyphenolics 25. Nut consumption, vegetarian diets, 26. Pomegranate juice 27. Pomegranate juice 28. Brussels sprouts 29. brussels sprouts. 30. Dietary flavonols 31. onion meal 32. vegetables and fruits 33. green or black tea 34. Green tea extract</p>	
Riksförbundet Hälsöfrämjandet, Sweden	<p>General comments:</p> <p>Line 285-291. It is a pity not to take into consideration the quality of the fruits and vegetables in the recommendations. Although there is no study that clearly show that organic has better nutritional balance than conventionally grown products, pesticides in large amount can cause oxidative stress. Many vegetables are treated with a cocktail of pesticides instead of using high amount of one pesticide and there is no research of the cocktail effect of chemicals that we use in our society. This section of</p>	<p>Comment appreciated.</p> <p>However, this aspect was not the focus of the revision.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>scientific support is that the literature is inconclusive, so supplemental use cannot be recommended or un-recommended. The vast majority of current evidence is based on high dosage intakes of a few vitamins or minerals, which is very different from the most commonly consumed food supplement: a multivitamin and mineral containing RDA-like dosages for most of the essential micronutrients. Physicians' health study is the only long term RCT on a healthy population investigating the impact of a multivitamin-mineral on chronic diseases. Recently two of the four major end-points of Physicians Health Study II have been published and the two remaining end-points: cognitive decline and eye health are expected to be published in 2012. We strongly encourage you to wait for those results and include them in the relevant chapters, as this is the kind of supplement taken by aprox 50% of the adult population (at least in Denmark).</p> <p>Detailed comments: Line 298: This statement is not scientifically supported. There is both pretty good evidence in favour (listed above for selenium primarily) a lot of neutral results, the results with un-beneficial outcomes are primarily coming from studies using dosages well above what is commonly consumed as a supplement and in many cases even above the upper tolerable levels (CARET, ABTC...). The only long term RCT on a healthy population investigating reduction of chronic diseases has recently been published: Physicians health study II (Gaziano, 2012) shows a modest, but significant, reduction in incidence of all cause cancer. The used intervention was a commonly used multivitamin and mineral supplements containing RDA-like dosages of beta-carotene, selenium, vitamin C, Vitamin E – this kind of supplement that is taken by aprox 50% of the adult Danish population. Gaziano J. 2012. Multivitamins in the prevention of cancer in men: The physicians' health study ii randomized controlled trial. JAMA: The Journal of the American Medical Association 1-10.</p>	<p>"There is a large body of evidence suggesting that elevated intakes of certain supplements, mainly vitamins with antioxidative properties, might increase the risk of certain adverse health effects, including mortality. Thus, there is no scientific justification for using supplements as a tool for adjusting an unbalanced diet.</p> <p>See above.</p>
Funktionaalisen lääketieteen yhdistys, Finland	<p>General comments: Funktionaalisen lääketieteen yhdistys ry Functional Medicine Society in Finland www.fms.fi info.fms.fi Nordic Council of Ministers For health and Social Affairs Nordic Nutrition Recommendations (NNR) 23.11.2012 Dear Sirs, We suggest the following changes in the NNR recommendations : Dietary antioxidants Suggestion to recommendations Intake of supplemental antioxidants does not seem to be justified</p>	<p>Chapter has been revised. Heading "Recommendations" deleted. A concluding section states: "There is a large body of</p>

Organisation/ Company	Comments	WG comments and observations
	<p>for a healthy population in large. Specified recommendations are given separately for the individual antioxidants according to EU commissions regulation 432/2012. The recommendation is based on limited numbers of studies s 5. For example the WHO Monica study has since 1985 generated a great number of studies related to cardiovascular diseases. Some new follow-up studies inspired by the WHO Monica project have showed the benefit of food supplements (for example in preventing oxidative stress related diseases). This report, follow-up studies and adjacent studies have been neglected. All studies should include reports of the following studies. Newest published criticism of flawed results and their conclusions should be cited. It seems that different methodological approaches of meta analysis may yield contradictory results (Gerss et al 2009). In large food supplement population studies, the results are compiled and reported based on individual self-reports and limited laboratory determinations. It is a well-known fact that self-medication can have an impact on the final reporting and results. In the introduction it is stated that the NNR guidelines are given for healthy populations and should not be implicated on individual persons or individual intake of food supplements. This should be mentioned in every individual recommendation. Obviously the situation is totally different when it comes to different diseases. For that reason it should be underlined that these recommendations are given for a healthy Scandinavian large population with no diagnosed underlying diseases and their medications.</p> <p>Rows 293-299: Reasoning behind the recommendation is over simplification of conclusions ignoring huge amount of contradictory scientifically proved facts in specified populations. For antioxidants such as vitamin C, vitamin E and beta-carotene, EU has stipulated regulations which should be uniform and evaluated in all dietary recommendations. Vitamin E Rows 264-267: A more recent paper shows that different methodological approaches of meta analysis yield contradictory results (Gerss et al 2009). NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations includes only healthy large populations not individuals. Vitamin C NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations includes only healthy large populations not individuals.</p>	<p>evidence suggesting that elevated intakes of certain supplements, mainly vitamins with antioxidative properties, might increase the risk of certain adverse health effects, including mortality. Thus, there is no scientific justification for using supplements as a tool for adjusting an unbalanced diet.</p> <p>See above.</p>

Organisation/ Company	Comments	WG comments and observations
	<p>Selenium NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations includes only healthy large populations not individuals. Gerss, J., Köpcke, W. The questionable association of vitamin E supplementation and mortality--inconsistent results of different meta-analytic approaches. Cell Mol Biol (Noisy-le-grand). 2009, 55 suppl: OL1111-1120.</p> <p>Yours sincerely, Erkki Antila President , PhD, Med.Lic. Funktionaalisen lääketieteen yhdistys (FLY) ry - Functional Medicine Society in Finland</p>	
Health Product Wholesalers' Association, Finland	<p>General comments:</p> <p>Dear Sirs, Our comments to your draft for Nordic Nutrition Recommendations, Dietary antioxidants Our suggestion is that no recommendations for antioxidants should be made because the results are controversial. There are very few scientific references from the European area that support these recommendations. The large intervention trials are poorly referred to and they should be referred to largely with sub results. It is not clear if these draft recommendations concern healthy population or individuals. No references are made to European regulations and health claims from the European Commission (432/2012) or for example to the Committee of Experts on Nutrition P-SC-NU RD 8 (14 REV 2 Nutrition in care homes and home care report and recommendations: from recommendation to action 5.12.2008). The suggestion is controversial in the antioxidant support (rows 298-299) because you have separate recommendations for single vitamins C and E In the protective effects of antioxidant-rich diets in experimental animal and human studies (rows 163-190) the positive effects have not been taken into account. In the draft form the recommendations confuse consumers, sellers and manufacturers. Vitamin C, E and Selenium The recommendations do not take in consideration EFSA's scientific studies and references or European Commission's regulations for health claims 16.5.2012 (432/2012). In the draft form the recommendations confuse consumers, sellers and manufacturers. The EU regulation 432/2012 is new but it should be taken as a part of the recommendations. Best regards, Luontaistuotteiden Tukku kaupan Liitto ry Health Product Wholesalers' Association</p>	<p>Chapter has been revised.</p> <p>Heading "Recommendations" deleted. A concluding section states:</p> <p>"There is a large body of evidence suggesting that elevated intakes of certain supplements, mainly vitamins with antioxidative properties, might increase the risk of certain adverse health effects, including mortality. Thus, there is no scientific justification for using supplements as a tool for adjusting an unbalanced diet.</p>

Organisation/ Company	Vitamin A General & detailed comments	WG observations and actions
Selskab for Ernæringsforskning, Denmark	<p>General comments</p> <p>Line 143, 145, 159-162, 170,171,173, 183, 292, 316, 319, 371: Replace "/day" with "/d"</p> <p>Detailed comments</p> <p>Line 220: Replace "Anintervention" with " An intervention"</p> <p>Line 378: Replace "don't" with "do not"</p> <p>Line 385 (and line 9): Replace "Referenser" with "References"</p>	Amended
National Food Agency, Sweden	<p>Detailed comments</p> <p>11 In the box a definition of RE is missed</p> <p>23 These are not metabolites, they are ester forms</p> <p>45-48 Clarification of how the factors should be used</p> <p>96-97 Are other data about absorption e.g. carotenoids available?</p> <p>340 We suggest a sentence that summarize and clarify the paragraph about retinol and osteoporosis</p> <p>361 We suggest a sentence that summarize and clarify the paragraph about retinol and fractures</p> <p>323: main component of a meal instead of main course of a meal</p> <p>376, 380 and 382: Is µg right? RE?</p>	<p>Explained in the text</p> <p>Corrected</p> <p>Used when calculating REs from food composition data</p> <p>Examples are given in previous sections</p> <p>Added</p> <p>Added</p> <p>Whole liver</p> <p>µg, refer to retinol = RE</p>

Organisation/ Company	Vitamin A General & detailed comments	WG observations and actions
Helsedirektoratet, Norway	<p>Detailed comments</p> <p>12. Note to table #: is the UL for post-menopausal women 1500 also as pre-formed retinol?</p> <p>49. Why is RE not exchanged to RAE? This was done in the Norwegian nutrition recommendations when they were revised in 2005.</p> <p>69. I suggest that you update intake data to the most recent national dietary surveys. It is nice that you give information about the intake of preformed vitamin A; you may also add information about total vitamin A intake. Please also include data about the main sources for total and preformed vitamin A, respectively. E.g. as proportion of total intake from food (animal and plant origin), supplements, fortified foods and other sources as cosmetics, respectively.</p> <p>51 and 243. Sources and intake of vitamin A: Cosmetics as a source for vitamin A may be mentioned. The Norwegian Scientific Committee for Food Safety has in 2012 published an assessment of the risk related to the use of retinol and retinyl esters in cosmetic products. They concluded that the most important sources of vitamin A in the population is diet, followed by food supplements and then cosmetics. Topical application of cosmetic products as estimated in standard scenarios (0.05% in body lotions and 0.3% in face and hand cream), increases the total exposure to vitamin A (retinol and retinyl esters) in all age groups. For further information see: http://www.vkm.no/dav/afdc8a9688.pdf You may also mention the role of vitamin A added to animal feed. Ref:Consequences for the consumer of the use of vitamin A in animal nutrition. The EFSA Journal (2008) 873, 1-81. http://www.efsa.europa.eu/en/efsajournal/doc/873.pdf</p> <p>163. The expression "recent studies" does not seem proper when the reference is IoM 2001.</p> <p>322. The recommendation that pregnant women should avoid eating liver as main course of a meal may be further commented or explained to help the reader</p> <p>349. Should "1.426 µg/d" be read 1426 µg/d or 1.426 mg/d?</p>	<p>Added</p> <p>Updated</p> <p>?</p> <p>Deleted</p> <p>Clarified</p> <p>1,429 µg/d (1.429 g/d)</p>

NNR 2012: Public consultation – Chapter 16 (Vitamin D)

Organisation/ Company	Vitamin D comments	WG response & actions
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Organisation/ Company	Vitamin D comments	WG response & actions
Närhälsan Skövde, Sweden	General comments only Hi! You don't write anything about HOW we are going to achieve getting 10 mcg vitamin D/day from food or HOW we will get it otherwise. You have to eat oily fish 3 times a week for dinner plus enriched milk and margarine plus eggs and herring for breakfast some days to get 70 mcg D-vitamin/week. If you don't think that's realistic you should come up with other suggestions I think. Isn't it called "Nordic Nutrition RECOMMENDATIONS"?	Strategies to achieve RIs for different population groups are primarily a management issue. However, this is an important issue, but should be managed at the national level, accounting for e.g. EU regulation, Codex guidelines etc.
Ministry of Social Affairs and Health, Finland	General comments only Lines 747-750 The scientific base behind these age-limits needs to be properly explained, now it is very unclear where they come from. Moreover, the recommendation for the age-group 61-74 is difficult to understand. What is "little sun-exposure" need to be properly described, based on best evidence available (with potential seasonal variation etc.) If these are not more clear we'll end up having very different interpretations in different countries.	Age categories are the same as in NNR 2004. Little sun-exposure could of course be better defined but the term used reflect the fact that the studies used for setting the RIs are derived from studies conducted in the winter season in the Nordic countries where sun-exposure is limited.
LRF Mjölkl/LRF Dairy Sweden	General comments only How have the reviewers dealt with the insecurities in measurements of vitamin D status and the large variations between laboratories when comparing results from different studies? How have the reviewers dealt with the fact that it now will be even more difficult to reach the new recommendation? A discussion regarding this should be added.	The reviewers have been aware of the insecurities in measurements of vitamin D status and large variations between laboratories and have added this into their systematic review.

Organisation/ Company	Vitamin D comments	WG response & actions
Sodexo	<p>General comments only</p> <p>Sodexo Sweden prepares and delivers 120 000 meals every day and has therefore a major impact on many Swedes daily diet. We serve food to school children, working adults, as well as to patients and elderly. Regarding the new recommended intake of vitamin D (10 µg for children and 20 µg for people over 75 years), proposed in NNR 2012, we believe that it will be difficult to achieve these recommendations with the products available on the market today. In many of our contracts, our customers require that the lunch should cover 30% of the recommended daily intake of vitamin D for children (7.5 g / day) respectively 20-25% of the recommended daily intake of vitamin D for the people over 75 years (10 mg / day). It is already a challenge to reach these recommendations, especially when it comes to vegetarian diet, and we therefore expect that the new recommendations will make the meal planning even more challenging. To facilitate reaching the new proposed recommendations in practice, we suggest that more products on the market are fortified with vitamin D or that products already fortified are further enriched. We would also appreciate clearer practical examples on how to reach the new recommendations when it comes to meal composition, especially for vegetarian diets. Furthermore, we wish that the concerned authorities would make a statement regarding how much of recommended daily intake of vitamin D a lunch meal in the public sector can be expected to cover. This to increase the understanding of the customers that today require that lunch will cover 30%, and 20-25% of the recommended daily intake of all nutrients, including vitamin D.</p>	<p>Strategies to achieve RIs for different population groups are primarily a management issue. However, this is an important issue, and will be managed at the national level, accounting for e.g. EU regulation, Codex guidelines etc.</p>
University of Bergen, Norway	<p>General comments only</p> <p>In general the NNR 2012 on vitamin D is well written. In the section on dietary sources and intake, it could be worthy to also add plant milk products to the list (as many readers may interpret milk as cow's milk and as plant milk products from soy, oat and rice often are enriched with vitamin D).</p>	<p>Some plant-based milk drinks contains added vitamin D. However, the fortification strategies in the various Nordic countries may differ.</p>
THL, Finland	<p>General comments</p> <p>The structure of this chapter is messy. It may be more logical to start with Requirement and recommended intake. There are also too many subtitles and further subtitles to subtitles. Decreasing and/or numbering them may help. It would be useful to state the differences between Nordic countries in enriching foods with vitamin D and that this have an effect on supplementation recommendations</p>	<p>The WG considers the general structure to follow a logical order, from physiology, health outcomes and derivation of dietary reference values. However, the layout will be improved in the final, printed version.</p> <p>The issue of national policies on fortification and</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>It is difficult to understand why NNR5-vitamin D group is not following the same policy with IOM regarding sun light exposure to vitamin D. Especially, because the high latitudes of the Nordic countries make the exposure period short and due to the relatively large variation in latitude between the regions, also varying. One should also remember that sun light exposure is carcinogenic. The effect of sun light exposure is taken into account differently for different population groups (children and adults; older adults and elderly; pregnancy and lactation). E.g., for children and adults it is stated on page 15, rows 697-699 "A higher intake may be necessary for some parts of the population due to limited sun-exposure..." (no statement on the amount). Whereas for older adults and elderly it is stated on page 16 rows 747-748 "An intake of 10 ug/d is recommended for the age-group 61-74 years. For people with little or no sun-exposure, an intake of 20 ug/d is recommended". After that on page 17 under the title "Reasoning behind the recommendation" it is stated on the rows 779-780 "For people with little or no sun-exposure, an intake of 20 ug/d is recommended". It is unclear to whom all this refers to. - It is not clear, why the recommendation is different for the age groups 61-74 and over 75 years. - Just to confirm, is the sufficient serum 25OHD concentration > 50 nmol/L or > 50 nmol/L?</p> <p>Detailed comments Page 2, rows 68-70, for the sake of cohesion one should talk about vitamin D3 in connection of fishes – On page 16 row 753 there should be a reference to the sentence "There is a marked increase in 1,25-dihydroxyvitamin D in plasma during pregnancy"</p>	<p>supplementation is actually a management issue, but could perhaps be mentioned.</p> <p>Regarding sun-exposure, NNR includes recommendations on regular physical activity which can be combined with various outdoor activities, which is also common habit. For those with little or no outdoor activities or who for cultural and other expose limited skin surface RI is higher.</p> <p>The ability to synthesise vitamin D decreases with age, less outdoor activities and sun-exposure tend to decrease. Also, evidence support a role for adequate vitamin D status (>50 nmol/L) and decreased risk of fractures, muscle function and CVD.</p> <p>Included in the final version.</p> <p>Included</p>

Organisation/ Company	Vitamin D comments	WG response & actions
Norwegian Directorate of Health	<p>General comments only</p> <p>L. 69 You here mention: "Certain freshwater fish may contain appreciable quantities of vitamin D." This might be confusing for the reader, and you should rephrase this sentence. Is this because of the fat content of freshwater fish or has freshwater fish generally a high content of vitamin D?</p> <p>L. 71 ... higher biopotency (than what?)</p> <p>L. 87 here you just give acronyms for analytical methods. Please first give the full name of the method and the acronym in brackets.</p> <p>line. 97-98 "Normally, serum 25OHD concentrations <30 nmol/L are regarded as deficient." a) It is common to use <25 nmol/l to define deficiency. b) Normally? Usually? By whom? We believe that criteria to define deficient and sufficient levels of vitamin D status have been debated the last decades. Thus you may give some more information concerning definitions and criteria for status, reflect current discussions, and give your conclusions to the reader.</p> <p>L 99. To make it easier for the reader please give some information about latitudes in relation to well-known geographical spots (e.g. capitals, polar circle etc.). For examples see the SLR under Discussion/UV-exposure.</p> <p>Line 109-47. Finnish data concerning vitamin D status is lacking in this section.</p> <p>Line 692. NNR5 suggest a recommended intake of vitamin D of 10 micrograms per day for 2-74 years of age. This is done without comments in relation to the conclusion made in the SLR: "In conclusion, if 97,5% of the population up to 75 years of age is to maintain the target 50 nmol/L concentration of 25(OH)D, the corresponding intake of vitamin D would be 15 micrograms/day."</p> <p>Line 769. Reasoning behind the recommendation. Here you may add a short discussion or comments concerning differences regarding the recommendations made by NNR and other expert groups as DRI by IoM, USA. You may also integrate additional text from the SLR, Authors conclusions.</p>	<p>Rephrased: Certain lean freshwater fish may contain high concentrations</p> <p>25OHD have higher biopotency <i>than vit D</i>.</p> <p>Relevant acronyms are spelled out.</p> <p>References to cut-offs has been added.</p> <p>Latitudes covering Nordic countries have been added in the introductory part.</p> <p>Section on intake and status is updated in final version.</p> <p>The conclusions of the SR will be commented in the final version.</p> <p>Discussed in the final version.</p>
Danish Cancer Society Research Center	<p>General comments</p> <p>Thank you for this very detailed and thorough literature review. From the Danish Cancer Society we have a considerable interest in the debate regarding vitamin D and naturally in the connected official recommendations. Our main concern regards whether the existing literature provides sufficient causality to recommend supplementation for the entire Nordic population. The recommended 10 ug/d will only be possible to reach through dietary supplements for a very high fraction of the population. A universal recommendation for dietary supplementation is a shift in the Nordic tradition which we believe deserves broad discussion. Especially taking the study mentioned in line 340 (Durup et al. 2012) into account, showing</p>	<p>The WG acknowledges the comments. The concern that the RI would require supplementation for the entire Nordic population is perhaps an overdue interpretation. Strategies to achieve RIs for different population groups are a management issue, not the primary subject of NNR. However, micronutrient inadequacies have previously been addressed in various ways, and are still an important strategy in several Nordic</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>higher mortality with higher serum levels in a large group of Danes. Our relatively strong objections towards the revised recommendations (see also the specific comments in the next section) are based on the long history of over-positive interpretations of vitamin-micronutrient associations (vitamin C, betacaroten, folate etc.). We believe that the potential negative effects of supplementation are not taken into considerable account with regard to vitamin D. Further, general supplementation with vitamin D will probably lead to use of combination products and thereby also to supplementation with other vitamins and/or minerals. The most relevant probably being calcium. Supplementation with calcium has already been related to increased incidence of heart disease (though not in combination with vitamin D), and there is emerging evidence for that calcium supplementation may increase incidence of prostate cancer. These issues need to be considered.</p> <p>Detailed comments</p> <p>L 59: There is, as far as we know, no evidence that vitamin D insufficiency is a greater problem at the Nordic latitudes compared to Southern Europe.</p> <p>L 97: Why is deficiency defined as 30 nmol/l, normally 25 nmol/l is used?</p> <p>L 96 – L 147: Focus is almost exclusively on 50 nmol/l – why not stronger focus on the fraction with deficiency?</p> <p>L 253-255: Focus is on a biological association between BMI and vitamin D, but maybe they are merely determinants of the same lifestyle.</p> <p>L 278-494: Considering that 10 ug/d is almost impossible to reach through the diet, evidence for raising the vitamin D recommendation can only be based on trials studying vitamin D supplementation. The only endpoints where any such evidence can be found are total mortality and fractures. As one of the most important methodological issues in intervention studies is external generalisability, the exact composition of the intervened populations needs to be considered. With few deviations, we believe that the reported interventions are based on relatively old and/or disabled persons. The youngest is, we believe, the WHI study – which is postmenopausal women. We fail to see the evidence for premenopausal women and men below app 70 years. We consequently find it</p>	<p>countries. Examples are food fortification, which includes e.g. vitamin D and iodide, advice on supplementation aimed at specific groups, etc. cod liver oil, providing vitamin D, has been and is still an integrated part of the diet in Iceland and Norway, and previously used in other countries, too.</p> <p>The question of risk of adverse effects due to proposed increases in RI from 7.5 to 10 µg/d is most likely minimal, since the aim is to keep S25OHD concentrations at or above 50 nmol/L also during the winter season. Moderate sun exposure during summer will generally raise concentrations up to 80-100 nmol/L with limited influence of dietary intake at that level. Effective monitoring of dietary intake, food fortification and supplement use is important, as for several other nutrients.</p> <p>Acknowledged, but dietary factors including fortification and use of cod liver oil, sun exposure play a role.</p> <p>The choice of cut-off of 30 nmol/l is based on the IoM report 2011. This cut-off of 50 nmol/l is used to indicate sufficient vit D-status. Agree.</p> <p>As mentioned above, strategies to achieve RIs is<u>should be</u> a management task. However, the aim of the <u>recommended intakes</u> re- is to maintain a <u>optimal sufficient</u> vit D status (<u>i.e. serum concentration</u>) throughout the year in the general population. Sufficient status is associated with reduced risk of fractures, falls, mortality and CVD. These refer to RCTs and cohort studies. There is also some evidence of an inverse association with</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	problematic to use these studies to recommend supplementation for much younger age groups. In addition, the lack of dose-response (L 305) speaks against a strong causal effect of vitamin D on mortality.	risk of colorectal and breast cancer. An intake of 10 µg per day during wintertime in the Nordic countries has shown not to result in high 25OHD concentrations.
Children's Hospital, University of Helsinki, Finland	<p>General comments</p> <p>As indicated in our detailed comments, the literature review has been inadequate. Several studies in pregnant women, infants, children and adolescents indicate that a total intake of 10 µg of vitamin D daily will be inadequate at least in Finland. The use of very old references and biased sources in the systematic review (and totally ignoring several good quality cross-sectional studies) is not a scientifically solid background for the new guidelines.</p> <p>Detailed comments</p> <p>Page 3, line 97: Normally, serum 25OHD concentrations <30 nmol/L are regarded as deficient, 30-50 are 97 insufficient and >50 nmol/L are sufficient for the majority of the population. COMMENT: References are missing, this is a far too general statement. An expert panel behind RDA concluded that 25-OHD concentration above 50 nmol/L is required for normal growth and function of body.</p> <p>Page 3, line 103. In a review of studies on population groups in dietary transition in the Nordic countries published in 1990-2011. COMMENT: Latest Nordic Nutrition Recommendations were updated in 2004. It is arguable if references before year 2004 should be used in this systematic review. Firstly, because of delay in publishing. Typically it takes about 2 years to publish a study. Thus, publication in 2004 illustrates the situation in 2002 or even earlier. Secondly, recommendations for vitamin D were revised in 2004 from 5 to 7.5 µg. It is thus important to look the current situation, not that in the beginning of 2000! Thirdly, since 1990 laboratory techniques have evolved significantly, it is not sensible to compare results from 90's with findings in 2010 or later. The focus should be on the newest data if we really want to improve guidelines and move forward.</p> <p>Page 3, line 120→ COMMENT: Cross-sectional studies describing vitamin D status and intake of vitamin D in Finland are missing. Several studies have evaluated this but none have been included in the report! e.g. Viljakainen HT, Pekkinen M, Saarnio E, Karp H, Lamberg-Allardt C, Mäkitie O. Dual effect of adipose tissue on bone health during growth. Bone. 2011 Feb;48(2):212-7. Median total intakes in school children were 8.8 µg for girls and 10.5 µg for boys. These included intake from dietary sources by food frequency questionnaire and from habitual</p>	<p>The SR focuses on SRs and meta-analyses, but additional studies relevant for the Nordic setting have been included. Cross-sectional studies are not adequate to assess health outcomes. They can be used for describing the current situation with respect to dietary intake and status.</p> <p>References to cut-offs are given. However, >50 nmol/L is the cut-off for sufficient status and has been used as a criterion, so this is in line with the proposal.</p> <p>The choice of timeframe for the SR can off-course be discussed. The WG considered it valuable to include some overlap from NNR 2004, since that was not based on a SR.</p> <p>The issue of analytical techniques for assessing 25OHD is discussed and the limitations acknowledged.</p> <p>Section on intake and status is updated in final version.</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>supplements. Mean serum 25-OHD concentration in this group was 42 nmol/l, measured with HPLC. This indicates that the recommended 10 µg intake of vitamin D will be insufficient! Viljakainen HT, Korhonen T, Hytinantti T, Laitinen EK, Andersson S, Mäkitie O, Lamberg-Allardt C. Maternal vitamin D status affects bone growth in early childhood--a prospective cohort study. Osteoporos Int. 2011 Mar;22(3):883-91. In this study total intake of vitamin D and S-25-OHD (with IDS EIA) were determined in 87 Finnish infants aged 14 months. Mean dietary intake was 12.3 (±3.0) µg calculated from 3-day food records and S-25-OHD 65(±20) nmol/L measured with IDS EIA. Holmlund-Suila E, Viljakainen H, Hytinantti T, Lamberg-Allardt C, Andersson S, Mäkitie O. High-dose vitamin d intervention in infants--effects on vitamin d status, calcium homeostasis, and bone strength. J Clin Endocrinol Metab. 2012 Nov;97(11):4139-47. This paper describes the 25-OHD concentration at birth (median, 53 nmol/l) in 113 newborn infants. At 3 months, the mean S-25-OHD values were 88, 124, and 153 nmol/liter, and the minimum values were 46, 57, and 86 nmol/liter in the groups receiving vitamin D supplementation 10, 30, and 40 µg per day.</p> <p>Page 4, line 136 COMMENT: There is a Finnish study describing the intake and vitamin D status in pregnant women (Viljakainen HT, Saarnio E, Hytinantti T, Miettinen M, Surcel H, Mäkitie O, Andersson S, Laitinen K, Lamberg-Allardt C. Maternal vitamin D status determines bone variables in the newborn. J Clin Endocrinol Metab. 2010 Apr;95(4):1749-57.). In this study vitamin D status was measured twice during pregnancy at first trimester and 2 days post partum; 41.0 (13.6) and 45.1 (11.9). Maternal intake of vitamin D was calculated at 3rd trimester 14.3 (5.8) µg and it correlated with S-25-OHD 2 days post partum.</p> <p>Page 6, 231: There are no straightforward explanations, but factors such as prior vitamin D status, compliance, latitude, and season might have played a role. COMMENT: We agree on this, but we wonder why randomized controlled trials are highlighted in the review. One should acknowledge the fact that baseline habitual intake has not been estimated in every study and intakes may vary between groups. Thus, if one adds 5 µg to habitual intake of 2 µg or 10 µg it will give a totally different response. Furthermore, intervention may interfere with the subject's behavior. Cross-sectional studies are more reliable when the aim is to describe intake and vitamin D status. Therefore much more emphasis should be placed on reliable good quality cross-sectional studies that evaluate habitual total intake of vitamin D and simultaneous S-25-OHD concentration.</p> <p>Comments to the systematic review The following electronic databases were searched: MEDLINE, and Swemed. The search was conducted using medical</p>	<p>RCTs are used to assess relation between graded intakes of vit D and S25OHD, including studies carried out during the winter season. Dietary vitamin D-intakes are included. Studies also account for variability with respect to age, gender, latitude, baseline vit D status, etc. Cross-sectional studies cannot be used as primary data sources.</p> <p>See response above.</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	subject heading terms (MESH) (see appendix I). The search was done in two batches, the first covering 2000-March 2010 and the second March 2009-February 2011. In To be published in Food & Nutrition Research the first search the investigators focused only systematic literature reviews (SLRs) and in the second on systematic reviews and randomized control trials (RCTs) published after March 2009. Furthermore we used snowballing for SLRs and RCTs published after that and until May 2012. COMMENT: Panel should have chosen studies focusing on Nordic countries. Affluence, genetics and lifestyle are more similar within Nordic countries than between US or Australia or China. Why collecting data from systematic reviews? As a researcher I would prefer looking at original articles (and making my own conclusions rather than relying on others). Two systematic reviews might include the same original articles - how was this controlled? We strongly argue against using systematic reviews as primary source of data !	
National Food Agency	General comments only Line 78 In an article by Schöttker et al (http://ajcn.nutrition.org/content/97/4/782.long) two immunoassays (Diasorin-Liaison and IDS-iSYS) for 25(OH)D measurements were standardized retrospectively to LC-MS/MS. Maybe this article should be mentioned and more discussion about different assays.	Will be considered, although published after SR literature search.
National Food Agency	General comments The chapter provides a good overview of the Vitamin D area. One general comment: In NNR2012 a serum 25OHD concentrations of >50 nmol/L is used as an indicator of sufficiency. However, serum 25OHD concentrations differ by analytical method and proportions below a certain cut-off differ depending on what analytical method that has been used. It would be useful to mention and discuss analytical methods when summarizing the review results and when setting the cut-off for sufficiency. Detailed comments Lines 56-58 in the Introduction are a bit confusing and was difficult to follow. The first sentence starts with the very general comment that vitamin D3 is present in some foods. The following sentence continues with the more specific information that Vitamin D2 can be formed by UV irradiation of ergosterol in mushrooms and yeast. I suggest you make a more general sentence in the Introduction (that vit D3 and also D2 are present in some foods) and then leave the details on vit D2 formation in mushrooms to the paragraph on dietary sources.	Accepted. Amended in the final version.

Feltkode ændret

Organisation/ Company	Vitamin D comments	WG response & actions
Private person	<p>General comments only:</p> <p>Line 54: The genes in the human is the same as in the older Stone Age. It would be fine if some words tell something about how much vitamin D the people in the Stone Age get from the sun, summer resp winter.</p> <p>Humble, Mats 2007: http://larkiv.lakartidningen.se/2007/temp/pda33136.pdf</p> <p>The heading Källor för D-vitamin: The sun can give _250 ug/d summer-time. References are provided at the bottom of the page.</p> <p>Line 203, 587, 784: The total D-vitamin supply is dominated of the sun. The food only give a fraction. We should do a distinction in the recommendations between summer and winter, and so</p> <p>On. The oral supply should be higher during the periods the person only is _indoors. The oral supply should be higher if the sun has a low angle, which it have in the Nordic countries during the winter. Persons with dark skin living in the Nordic countries can require higher oral supply. Persons who is wearing veil can require higher oral supply.</p> <p>Another method is to say: The oral supply should be limited, when our skin is exposed for sun, that have a high angel.</p>	<p>The issue of the human genome is relevant. However, estimates derived from calculated maximal sun-exposure derived skin synthesis during summer during stone age cannot be used as a basis for setting DRIs.</p> <p>The WG acknowledge that sun-exposure during summer is a major vit D-source, given that people have enough sun-exposure, and that dietary supply generally is less important. However, from a public health point of view, it is prudent to set RIs that apply for the general population over the seasons, since some parts of the population may not obtain sufficient sun-exposure even during the summer season.</p>
Danish Endocrinology Society	<p>Only general comments:</p> <p>Comments on effects of calcium and vitamin D (CaD) supplementation on risk of fracture</p> <p>p. 8. In the chapter on calcium, it is stated that “calcium supplementation in combination with vitamin D may be effective in reducing fractures in institutionalized populations although the effect in the general population is unclear”. Similarly, in the section on vitamin D it is stated that CaD “has been shown to reduce the risk of total fracture and hip fracture in institutionalized elderly, whereas supplementation with vitamin D alone has not shown a significant effect (Lamberg-Allardt et al. 2013)”. It should be noted that most of the studies included in the cited systematic reviews have been performed non-institutionalized elderly. Accordingly, the summary risk estimates, showing a reduced fracture risk in subjects randomized to CaD, are based on data, which to a large extent are derived for findings in non-institutionalized elderly. It may be considered to draw a conclusion saying that “SRs of randomized controlled trials on supplementation with calcium and vitamin D in combination have in general shown a reduced risk of fractures in response to supplementation in the elderly. The effect is apparently most pronounced in institutionalized elderly and/or in subjects with low vitamin D levels prior to the intervention. Supplementation with</p>	<p>Amended in final version.</p>

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>vitamin D alone has not shown significant effects".</p> <p>Comments to "Lower intake level" of vitamin D p.14 A number of good arguments for defining the Upper intake level of vitamin D are stated. However, there are almost no reasons mentioned for defining the threshold for the lower intake level. One paper is cited concerning newborn Chinese infants in whom no clinical signs of rickets were evident at six months of age, following a minimum supplementation of 2.5 microgram/d. However, with an average 25OHD level (geometric mean) of approximately 30 nmol/l within this group, a notable proportion of the population had levels in the range known to be associated with an increased risk of rickets. The small study population and short study duration (6 mo.) are obvious limitations to this study. As the lower intake level (LI) is defined as a cut-off intake value below which an intake could lead to clinical deficiency symptoms in most individuals, this threshold is of specific importance to those with no or very limited sun-exposure. In adults (and, most likely also in children) without/with very limited sun-exposure, a daily intake of 2.5 microgram/d is unlikely to be sufficient to maintain a 25OHD concentration > 27.5 nmol/l and is likely, in the long run, to result in rickets/osteomalacia in "several" individuals. Accordingly, it may be considered to increase LI to 7.5 (or at least 5) microgram/d – a footnote may be added saying that this is considered to be the minimum requirement in persons with very limited sun exposure. Comments to "Body weight" p. 12. It is stated that "an association between vitamin D intake and body weight is inconclusive", but from several recent RCT's it is clear that the results may be described as conclusive – there are no effects (if all negative results should be stated as inconclusive nothing will never be conclusive).</p>	<p>In NNR 2004, the LI was mainly relevant for elderly. Results from the RCTs using various doses of vit D supplements indicate that an intake of 2.5-3 µg/d would maintain serum 25OHD of about 30 nmol/L during wintertime, provided sufficient sun-exposure during the summer season (see figures). However, for individuals with no or little sun exposure this intake may be insufficient to maintain this level. There is insufficient data to set a LI for e.g. infants under these circumstances. The present LI can be used to assess the probability of insufficient vit D status, but additional information is needed, e.g. living conditions, and eventually serum 25OHD, to verify this.</p> <p>The NNR SR identified one SR with covering BW, which included 3 RCTs with vit D only and 2 with vit D+Ca. They were negative, although the latter 2 studies showed minor reductions in BW. One could consider using the term "unlikely" instead of inconclusive to describe the evidence.</p>

Organisation/ Company	Vitamin D comments	WG response & actions																														
Minivent AB, Sweden	<p>General comments only</p> <p>Vitamin D 50 Line 51-52 should be replaced by the following table</p> <table><tr><th>Recommendation table</th><th>Vitamin D3</th><th>Age group</th><th>Minimum µg/d</th><th>Maximum µg/d</th></tr><tr><td>Recommended intake RI</td><td>Children below age 10</td><td>75 µg</td><td>250 µg</td><td>Children above age 10</td></tr><tr><td>125 µg</td><td>500 µg</td><td>Adults</td><td>125 µg</td><td>500 µg</td></tr><tr><td>Pregnancy and lactation</td><td>250 µg</td><td>500 µg</td><td>Elderly</td><td>125 µg</td></tr><tr><td>500 µg</td><td>Average requirements AR</td><td>125 µg</td><td>500 µg</td><td>Lower intake level adult LI</td></tr><tr><td>125 µg</td><td>Upper intake level</td><td>500 µg</td><td colspan="2">Line 53 to 64 should be replaced by the following text</td></tr></table> <p>Introduction Vitamin D3, a seco-steroid hormone, is a prehormone essential to animals including man. The existence of a substance now named vitamin D is reported to have been known since early antiquity. The first scientific description of rickets as a deficiency was reported in the 17th century by Dr. Daniel Whistler (1645) and Professor Francis Glisson (1650). Nutrition was acknowledged as an experimental science in the period 1910th – 1930th and one of the major breakthroughs in the science of nutrition was the understanding of the causative factors of rickets and the development and the appreciation of the existence of vitamins . Traditionally, vitamin D3 is regarded as an anti rickets vitamin that is used exclusively to prevent rickets and osteoporosis. Recent research has shown that most cells in the body has Vitamin D Receptors (VDR) in the plasma membrane as well as in the cellular nuclei and that there are several hundreds of cellular reactions that are vitamin D3 dependent . Also, vitamin D3 has a profound involvement in the immune systems, the endocrinological systems and a lot of other vital systems in the body . Vitamin D deficiency has been linked to inflammatory and long latency diseases, such as multiple sclerosis, rheumatoid arthritis, lupus, tuberculosis, diabetes, cardiovascular disease, and various cancers, to name just a few. The view of regarding vitamin D3 as a calcium regulator and rickets preventer only is an oversimplification and disregarding of true and modern science. The authorities have to have a better knowledge basis otherwise we are unable to make any decisions. Line 67 to line 76 should be replaced by the following text</p> <p>Normal levels of vitamin D3 in the blood. There has been much debate about the normal level of the vitamin D3. Clinical chemical laboratories in Sweden usually defines less than 25 nmol/L as vitamin D3 deficiency, 25-75 nmol/L as vitamin D3 insufficiency, 75-250 nmol/L as optimal level of vitamin D3 and more than 250 nmol/L as potentially toxic level of vitamin D3 . Line 77 should be replaced by the following text: Physiological level of vitamin D3 There has been large debates over the physiological level of vitamin D3. It is quite simple in reality. There are several reports supporting that there is a linear relationship between intake of vitamin D3 and level of vitamin D3 in the blood. When you increase the</p>	Recommendation table	Vitamin D3	Age group	Minimum µg/d	Maximum µg/d	Recommended intake RI	Children below age 10	75 µg	250 µg	Children above age 10	125 µg	500 µg	Adults	125 µg	500 µg	Pregnancy and lactation	250 µg	500 µg	Elderly	125 µg	500 µg	Average requirements AR	125 µg	500 µg	Lower intake level adult LI	125 µg	Upper intake level	500 µg	Line 53 to 64 should be replaced by the following text		<p>The WG thanks for comments. However, the recommended intakes proposed are not supported by available scientific evidence.</p>
Recommendation table	Vitamin D3	Age group	Minimum µg/d	Maximum µg/d																												
Recommended intake RI	Children below age 10	75 µg	250 µg	Children above age 10																												
125 µg	500 µg	Adults	125 µg	500 µg																												
Pregnancy and lactation	250 µg	500 µg	Elderly	125 µg																												
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125 µg	Upper intake level	500 µg	Line 53 to 64 should be replaced by the following text																													

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>intake by 10 µg then the level of vitamin D3 will increase by 10 nmol/L, there is a linear relationship . Also, lactating women need to have 125 nmol/L to give enough vitamin D3 in the breast milk to her baby. Parathyroid hormones are elevated when the vitamin D3 level is below 125 nmol/L. Darker skinned people living in the sun of Africa usually have a vitamin D3 level of 125-250 nmol/L This shows that the physiological level of vitamin D3 is more than 125 nmol/L and that levels below 125 nmol/L must be considered as deficiency. This means that the clinical chemical laboratories have to change their normal values as well. Toxicity Brohult and Jonsson showed in the beginning of 1970th that 2 500 µg vitamin D2/day for one year was without any toxic symptoms . All toxic incidents but one is showing very large doses of vitamin D2, not vitamin D3 1 000 µg daily to infants gave toxicity in one to four months . That is equal to 25 000 µg/day (1 000 000 IU) to an adult. In Finland in 1950th up to 1964 they recommended giving 125 µg to newborns without any symptoms of toxicity . 125 µg to a newborn of 2,8 kg is equivalent to 3 125 µg to a 70 kg adult per day. In USA there was an accidental oversupplementation of Vitamin D in milk in the middle of 1990. The dose was up to 6 000 µg/day for half a year. The researcher found no signs of toxicity There are differences between vitamin D3 (animal vitamin D3 or cholecalciferol) and vitamin D2 (vegetable vitamin D2 or ergocalciferol) In 2008 G Jones showed that an upper level of 750 nmol/L is safe. In Poland the recommendation is vitamin D3 at 2,5 µg/kg bodyweight, equals an adult dose of 175 µg to an adult . In Sweden NNR4 recommends infants to have 10µg/day which equals an adult dose of 250 µg/day. There is scientific information by R Vieth from 1999 where the recommendation is at least 125 µg per day and a toxic dose is estimated to more than 1250 µg per day. This means that the tolerable upper level of vitamin D3 intake is safe by a factor of at least 2, maybe a safety factor of 8, when the daily intake is below 3 500 µg/day or 50 µg*kg-1*day-1 (or 140 000 IU per day or 2 000 IU per kg and day). Line 96-98 should be replaced by the following text Vitamin D3 status in Nordic populations According to various reports the majority of the populations in many countries have levels of vitamin D3 below the “optimal” level of minimum 75 nmol/L , also found in Norway by NNR5 and in Sweden . But there are no reports of vitamin D3 levels below the higher physiological level of 125 nmol/L. This is embarrassing as there must be an even larger proportion of the populations that are deficient in vitamin D3. Also, the levels of vitamin D in Norway is shown to be too low, just 40 nmol/L A Finnish study giving 50 µg to newborns in 1966 reduced the incidence of diabetes type 1 by 78 % up to 30 years after birth . 50 µg to a newborn of 2,8 kg is equivalent to 1 250 µg (or 50 000 IU) vitamin D3 per day to a 70 kg adult. It should</p>	

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>be remembered that the Finnish authorities in 1964 had changed the previous recommendations of 125 µg vitamin D3 from 1950th down to 50 µg per day. The recommendations previous to 1964 is equivalent to 3 125 µg (or 125 000 IU) vitamin D3 per day to an adult. Now, since the Finnish recommendation was reduced to 10 µg in 1996, the number of patients with diabetes type 1 has soared in Finland. Line 148 should be replaced by the following text Summary In most of the recent studies the results are indicating that the majority of the Nordic populations are severely deficient in vitamin 3. The results of the present deficiency we are starting to see in increasing rates of diabetes type 1, multiple sclerosis, rheumatoid arthritis, lupus, tuberculosis, diabetes type 2, cardiovascular disease, and various cancers, to name just a few. Lines 154-155 should be replaced by the following text factors such as skin surface exposed, season, latitude. Dermal production of vitamin D3 is not affected by pigmentation. . But the production of vitamin D3 is negatively correlated to the baseline vitamin D3 level so those with a low vitamin D3 level synthesises more vitamin D3 than those with a higher vitamin D3 level. Those with a higher S-cholesterol had a higher synthesis rate than those with a lower S-cholesterol level. 24 % body surface irradiated with 1.1 J/m² 10 minutes every two to three days with a total irradiation time of 40 minutes gave a mean increase of 25 nmol/L. Taking in account that the half life of vitamin D3 is one to two months then the level of vitamin D3 should be well above the physiological 125 nmol/L at the beginning of autumn/fall. Otherwise the supplies formed during summer sun exposure will be insufficient during winter and spring until the summer sun can replenish the supplies. Lines 203-204 should be replaced by the following text Supplements The recommended level of vitamin D3 as more than 125 nmol/L is difficult to reach on food alone. The best source of vitamin D3 is the UVB-rays of the sun. The problem with the sun's UVB-rays is that the sun has to be at least 35° above the horizon for the UVB to penetrate the dense atmosphere at sea level to reach the skin. At a lower inclination angle the UVB-rays are absorbed by the atmosphere and can not induce the synthesis of vitamin D3 from cholesterol. So the UVB is producing vitamin D3 only about 2-3 months before and after the summer solstice here in the Northern latitudes. The rest of the year the population has to rely on enough vitamin D3 acquired during the summer or on enough vitamin D3 in food and vitamin D3 supplementation. The amount of vitamin D3 in fortified milk is 0.45 µg/dL. This means that you have to drink 277 dL milk per day. This will mean 12 500 kcal per day to obtain 125 µg vitamin D3 per day. Also eating fish, salmon, then it's necessary to eat 10 hg salmon per day to reach 125 µg This means that you have to be out in the sun at</p>	

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>least 1 h every day during summer without sunscreen or covering clothes or have a vitamin D3 supplement with at least 125 µg vitamin D3 per day year around. Line 211 should be replaced by the following text with 50 µ/d. This indicates that also children of any age must have supplement of at least 125 µg of vitamin D3 to reach the physiological level of at least 125 nmol/L. Line 258 should be replaced by the following text There seem to be a nice linear relationship between supplements of the number of µg giving about the same number of nmol/L in serum/plasma. It should be remembered that the level of vitamin D3 on serum/plasma does not mirror the total amount of vitamin D3 in the storage volume of fat as we do not measure the concentration of vitamin D3 in fat or other tissues. This implies that obese persons will need a higher intake than lean persons until the fat tissue is saturated with vitamin D3. Then the obese person can revert to normal amounts of supplements or sunshine. We should always remember that the homeostasis in the body always will keep the body within normal limits unless we disturb the homeostasis due to sheer ignorance and maltreatment. Lines 273-276 should be replaced by the following text osteomalacia in adults. By solely focussing on vitamin D3 and bone health is a way to simplify and disregard the last decades of scientific research. We now know that vitamin D3 is indeed vital to maintain general health and that a physiological level of at least 125 nmol/L is important to breast feeding and to prevent and even treat diseases like diabetes type 1, multiple sclerosis, rheumatoid arthritis, lupus, tuberculosis, diabetes type 2, cardiovascular disease, muscle strength and various cancers, to name just a few. We now know that most cells are having VDRs both at membrane levels as well as nuclear levels. This means that we do need vitamin D3 in much higher concentrations and doses than previously assumed. Lines 279-340 should be replaced by the following text Most studies regarding vitamin D3 and mortality compares placebo (no vitamin D3 supplement) with a tiny supplement (5-40 µg) of vitamin D3 and extra calcium. You can not see any differences when you compare almost nothing with almost nothing. Previously it is shown that the physiological level of vitamin D3 is at least 125 nmol/L which means that the minimal supplement amount should be at least 125 µg of vitamin D3. Most studies regarding vitamin D are using vitamin D2 (ergocalciferol). Erocalciferol is a vegetable vitamin D analogue that is not working in animals except competitively blocking the VDR thus preventing vitaminD3 (cholecalciferol) reacting with the VDR. Also, vitamin D3 can react with sulphate thus improving the transport of vitamin D3-sulfate through cellular membranes by a factor of about 300 times . Ergocalciferol can (luckily) not react with sulfate. The effect of ergocalciferol on</p>	

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>bone health is described a hundred years ago when the knowledge was less than now. But ergocalciferol is toxic at lower levels than vitamin D3 . What is shown is that low levels of vitamin D3 increases the mortality rate. Lines 350-420 should be replaced by the following text A too low serum 25(OH)D3 concentration increase the risk of rickets and increase the level of parathyroid hormone. As long as the level of parathyroid hormone is above normal range is just one of several indicators of vitamin D3 deficiency. This is just one more indicator of the physiological level of 125 nmol/L o vitamin D3. To try to establish a specific “fracture prone level” is just another attempt to disregard the power of homeostasis. Just follow the physiological level of vitamin D3 and there are no worries. Falls are decreased and muscle strength is improved when the level of vitamin D3 is above the physiological level of 125 nmol/L. Lines 497-502 should be replaced by the following text In Finland it is shown that an intake of 125-50 µg per day to infants was giving a very low incidence of diabetes type 1 . When the vitamin D3-supplementation decreased in 1975 to 25 µg and in 1992 to a meagre 10 µg then diabetes type 1 has soared Lines 505-517 should be replaced by the following text Vitamin D deficiency impairs insulin secretion and induces glucose intolerance. Several vitamin D related genes are associated with different pathogenetic traits of the disease . Lines 526-550 should be replaced by the following text A previously known already known in 1947 Obermer recommended: “Until further experimental evidence, adequate and incontrovertible, is made available, I submit that we should play for safety. In a climate like that of England every pregnant woman should be given a supplement of vitamin D in doses of not less than 10,000 IU per day in the first 7 months, and 20,000 IU (per day) during the 8th and 9th months.” Hiherto, there are no experimental evidence, adequate and incontrovertible, is published. The conclusion is that pregnant women should have 250-500 µg per day to have the physiological level of at least 125 nmol/L. This secures that the child will have enough vitamin D3 via the mother’s breast milk. Lines 552-576 should be deleted. Duplicated information in lines 350-420 Lines 580-585 should be deleted. Duplicated information in lines 273-276 Lines 593-767 should be replaced by the following text The NNR5 finds it easy to establish a physiological level of at least 125 nmol/L of vitamin D3 as shown above. Pregnancy and lactation E. Obermere, a British gynaecologist, issued in 1947 the following statement : “Until further experimental evidence, adequate and incontrovertible, is made available, I submit that we should play for safety. In a climate like that of England every pregnant woman should be given a supplement of vitamin D in doses of not less than 10,000 IU per day in the first 7</p>	

Organisation/ Company	Vitamin D comments	WG response & actions
	<p>months, and 20,000 IU (per day) during the 8th and 9th months.” This is equal to 250 µg per day during the first 7 months of pregnancy and 500 µg per day the last two months of pregnancy. No experimental evidence, adequate and incontrovertible, is made available hitherto to change the recommendations from 1947. Rather, the Finnish experiences from 1950th to 1975 had shown that children should have between 125 and 50 µg vitamin D3 is not only safe but helps preventing diabetes type 1 . This is approximately 4 to 8 µg vitamin D3 per day and kg bodyweight. During lactation the mother’s viamin D3 level should be maintained at or more than 125 nmol/L during lactation to ensure the vitamin D3 transfer via breast milk to he child. Children The information above shows incontrovertible that children of age less than 10 years of age are recommended to have at least 125 µg Vitamin D3 as a supplement to maintain an adequate level of at least 125 nmol/L. Adults Children above 10 years age can use the same recommendations as the adults or at least 125 µg vitamin D3 per day. The safe upper limit is at least 3 500 µg per day. Elderly Elderly above 65 years of age can use the same recommendations as the adults or at least 125 µg vitamin D3 per day. The safe upper limit is at least 3 500 µg per day. This will prevent a majority of falls and bone fractures as the physiological level of vitamin D3 is maintained throughout the whole life. Lines 772-782 should be replaced by the following text and falls and CVD outcomes. The level of vitamin D3 in NNR2004 of more than 50 nmol/L is inadequate according to both old and especially new knowledge. Many studies have shown that the physiological level of vitamin D3 is above 125 nmol/L. Also, many studies have shown that vitamin D3 levels below the physiological level of 125 nmol/L increases the risk of disease. The sun induced production of vitamin D3 is limited by physical laws to about four months during summer in the Nordic countries. Another limitation is the use of sunblock (contains cancer causing benzene derivates to absorb UVA and UVB) and covering clothes that blocks the synthesis of vitamin D3. These sun blocking advice are not protecting against skin cancer , . The supplementation level is thus set to at least 125 µg per day. Lines 884-820 should be replaced by the following text Upper limit of vitamin D3 intake The American IOM (Institute of Medicine) and EFSA has established an upper limit of 100 µg vitamin D3 to adults. These ULs settled by IOM and EFSA seem to be too low as there are a lot of toxicity and physiological knowledge recommending a much higher Tolerable Upper Limit of vitamin D3 . See the part on toxicity. Also, EFSA have recommends an upper limit intake of vitamin D3 to newborn of 25 µg per day. That is equal to 625 µg or 25 000 IU per day to an adult . This mean that EFSA has settled two different upper limits, one that is 25 times higher than the</p>	

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	<p>next. The proposed upper level of vitamin D3 intake of 500 µg is safe by a factor of at least 2, probably a safety factor of 8, when the daily intake is below 3 500 µg/day or 50 µg*kg⁻¹*day⁻¹ (or 140 000 IU per day or 2 000 IU per kg and day). Line 822 should be replaced by the following text Summary Man seems, according to old and modern scientific research, to need a physiological level of at least 125 nmol/L vitamin D3 to stay healthy. As sun induced synthesis of vitamin D3 has decreased due to the recommendations of the Swedish Irradiation safety authority to avoid vitamin D3 producing sunshine the proportion of individuals with vitamin D3 deficiency have increased compared to 1992. The information presented here shows that the physiological level of vitamin D3 should be maintained at more than 125 nmol/L and that levels below 125 nmol/L must be considered as deficiency. The sources of vitamin D3 in food are scarce so we are forced to rely on supplements of vitamin D3. It is well known and established that there is a direct linear relationship between intake in µg per day and level of vitamin D3 in plasma. The recommendation is therefore according to the table below to reach the population level of at least 125 nmol/L. The safe upper limit of vitamin D3 intake is more than 3 125 µg per day or more than 50 µg/kg/day. The recommendations to the population should thus be as shown below:</p> <p>Recommendation table Vitamin D3 Age group Minimum µg/d Maximum µg/d Recommended intake RI Children below age 10 75 µg 250 µg Children above age 10 125 µg 500 µg Adults 125 µg 500 µg Pregnancy and lactation 250 µg 500 µg Elderly 125 µg 500 µg Average requirements AR 125 µg 500 µg Lower intake level adult LI 125 µg Upper intake level 500 µg</p> <p>Also sending a formatted file for the consultation to be readable. Sending it to livsmedelsverket@slv.se</p>	

NNR 2012: Public consultation – Chapter 17 (Vitamin E)

General and detailed comment	WG observations and actions
<p>University of Helsinki, Finland</p> <p>General comments</p> <p>Literature on vitamin E is extensive and I understand that NNR cannot cover all interesting studies on vitamin E. Nevertheless, NNR should cover the most informative studies on vitamin E and health, but that is not the case with the draft. NNR Chapter 3 states that systematic literature search was done on some topics but not on vitamin E. I cannot understand such policy in the preparation of NNR. There are important new studies on vitamin E after the previous NNR</p>	<p>The WG acknowledge the comments highlighting the complexity of interpreting effects of vitamin E supplementation on various health outcomes.</p>

General and detailed comment	WG observations and actions
<p>version. As the result of the lack of systematic literature search, the vitamin E chapter misses many important recent studies. I am sure that a systematic literature search would have identified many or all of them. NNR draft Chapter 3, page 13, second paragraph states: "Studies on Nordic population groups have been included where available" This is not true for the vitamin E chapter.</p> <p>The section on Safety is inconsistent with NNR Chapter 3. Pages 6 to 7 and Fig. 3.3 of Chapter 3 describe the reasoning behind the Upper intake limit (UL) but that reasoning is not followed in the vitamin E draft. See specific problems below. The Safety section is severely biased: Graat trial is not mentioned, Leppälä study is mentioned but incorrectly cited and improperly described, reports of increased risk of heart failure are not mentioned in the Safety section, and several recent analyses of the large Finnish ATBC Study are ignored. I cannot see the rationale of NNR to copy the UL from a 10 year old EU-report and claim that the "revised up-to-date NNR" is based on currently available evidence about vitamin E. There are several recent studies that challenge the validity of the UL. After the publication of the previous NNR, several studies have reported adverse effects with vitamin E doses lower than the proposed UL of 300 alpha-TE. According to Chapter 3, the UL should be lower than LOAEL so that there is UF (uncertainly factor, safety margin) between. That is not the case with the vitamin E draft. Below, I give specific comments in the order that the most important issues are first and then I go towards issues that are less fundamentally important. The order is: SAFETY CHRONIC DISEASES RECOMMENDED INTAKES INTRODUCTION PHYSIOLOGY DIETARY SOURCES AND INTAKES</p> <p>Detailed comments</p> <p>SAFETY: Upper intake levels and toxicity</p> <p>Lines 298-301 This text refers to Hathcock review in AJCN (2005) for the claim that vitamin E doses up to 1600 IU per day (1073 mg/d) are safe. I pointed out that the Hathcock review was biased: http://ajcn.nutrition.org/content/82/5/1141.2</p> <p>In that letter, I wrote: "[Hathcock's excerpt first:] "At present, the evidence is not convincing that vitamin E supplementation up to the UL [ie, the tolerable upper intake level, or 1000 mg/d] increases the risk of death due to CVD or other causes." However, to focus only on the effect on mortality is a very narrow view of safety. A recent double-blind, placebo-controlled trial in 652 Dutch persons aged ≥ 60 y found greater severity of respiratory infections among participants supplemented with 200 mg vitamin E/d than among those not given vitamin E (3). During respiratory episodes, the presence of fever (P = 0.009) and the restriction of activity (P = 0.02) were more common, the number of symptoms was higher (P = 0.03), and the total illness duration was longer (P = 0.02) among the vitamin E-supplemented participants than among those who received no vitamin E. These findings directly point out that some population groups may be harmed by vitamin E supplementation. Thus, when assessing the safety of vitamin E, it is not reasonable to focus only on mortality when there is evidence of aggravation of a disease that is very common." Thus, Hathcock ignored the Graat et al trial (JAMA 2002) which found significantly increased severity of respiratory infections when participants were administered 200 mg vitamin E per day. http://www.ncbi.nlm.nih.gov/pubmed/12169075 200 mg/d is much less than the 1000 mg/d which Hathcock claimed safe and which is uncritically reiterated in the vitamin E chapter. Increase in</p>	<p>Results from the frequently cited ATBC-study showed increased risk of hemorrhagic stroke, but no. of cases were few. A reduced risk of cerebral infarction (ischemic stroke) was also observed. Analyses combined groups receiving vit E alone and vit E+beta-carotene, which complicates the interpretation. In sub-analyses vit E supplementation was associated with either increased or decreased total mortality, depending on intake of vit C, age and smoking. In the meta-analyses of Bjelakovic et al. (2007, 2008) interventions included either vit E alone or in combination with other nutrients. As stated in the Draft Chapter "Generalization of these findings, largely based on studies in patients with chronic diseases to healthy adults is, however, uncertain". Dose-response for associations is not available. This is given for total mortality in the analysis by Miller et al. (2005), with a cut-of about 270 mg/d. A meta-analysis of vit E supplementation on stroke risk show no association for total stroke, a reduced risk for ischaemic and an increased risk for hemorrhagic stroke (Schürks et al. 2010). Doses range from 50 to 300 mg/d. Effects were seen in men, with no association in one study among women. This study is included in the final version.</p> <p>The section on Upper levels has been</p>

General and detailed comment	WG observations and actions
<p>the severity of respiratory infections is a relevant adverse effect. It should not be ignored in the Hathcock review nor in the NNR. The Graat study is not mentioned in the Safety section of the NNR vitamin E chapter. Why is it not mentioned? Lines 308 to 310 and Lines 143 to 145 In scientific texts, transparency is important and explicit references should be given. The ATBC study on stroke was carried out by Dr. Leppälä at our department and it was published in 2000: http://www.ncbi.nlm.nih.gov/pubmed/11030804 Leppälä wrote: "Vitamin E supplementation increased the risk of subarachnoid hemorrhage (RR, 2.45; 95% CI, 1.08-5.55) in hypertensive men but had no effect among normotensive men." It is unacceptable to refer to the original NEJM (1994) paper as a citation to the ATBC stroke findings (2000) since the stroke findings were published 6 years later (on year 2000, see the above link). A reader should be given the correct references for clear statement of facts, and not a paper which reported only the primary endpoint findings of the ATBC Study. Also, the restriction of this harm to hypertensive men should be mentioned. The dose of vitamin E in the ATBC Study should be mentioned. It was 50 mg/day dl-α-tocopheryl acetate corresponding to 22 a-TE/day. Given that 22 a-TE/day increases mortality due to hemorrhagic stroke by 330% (95% CI: 19% to 2300%) and the incidence of stroke by 145% (above) in hypertensive men on the basis of the Leppälä 2000 study, setting the upper limit of vitamin E safety to 300 a-TE is inconsistent with NNR Chapter 3. Thus, lines 308 to 310 and and also lines 143 to 145 do not tell to the reader who found what and when and with which vitamin E dose. Therefore the text is not transparent. Good science is transparent. Description of the Leppälä study needs rewriting.</p> <p>Lines 296 to 333 There are relevant findings about the toxicity of vitamin E published after the Hathcock review in 2005, yet many essential studies are ignored in the NNR draft on vitamin E. In a detailed analyses of the ATBC Study data set, I found that there is strong evidence that the effect of 50 mg/day vitamin E on mortality was heterogeneous in 6 subgroups formed by baseline age and dietary vitamin C intake ($P=0.0005$; $\chi^2(5 \text{ df}) = 22.2$) http://dx.doi.org/10.1093/aje/kwn413 In ATBC participants with baseline age 50-62 years and dietary vitamin C intake above the median ($N=11000$), 50 mg/day vitamin E increased mortality by 19% (95% CI: 5% to 35%). Furthermore, the harm of vitamin E appeared only after 3.3 years of supplementation. Thus, for the early supplementation period between 0.0 to 3.2 years, vitamin E was safe (no increase in mortality), but thereafter vitamin E increased mortality by 38% (95% CI: 17% to 63%)(i.e. for the supplementation period between 3.3 to 8 years). Cox regression model improved significantly when separate vitamin E effects were added to the early and late supplementation phases ($P = 0.007$) so that random variation is no reasonable explanation for the time-variation in vitamin E effect. Why is the 38% increase in mortality by long-term low-dose vitamin E supplementation ignored in the safety section of the NNR? The ATBC Study was carried out in Finland and therefore it is a particularly relevant data set for understanding the potential harm of vitamin E in Nordic countries.</p> <p>Lines 296 to 297 "Toxicity of vitamin E is low apparently due to efficient inbuilt control which prevents any excess accumulation of vitamin in the body" This statement is refuted by the increase in mortality after the 3 year lag period, see above. We do not know the mechanism of toxicity, but the 3-year lag indicates that there is some kind of accumulation. It can be accumulation of vitamin E itself or it can be accumulation of some metabolites generated by</p>	<p>revised and the conclusion regarding vit E supplementation has been modified: "Taken together, the available scientific data suggest that there are no overall benefits of prolonged high intakes of supplemental vitamin E in the general population. The UL established by EFSA for vitamin E as supplement, 300 mg/d, was included in NNR 2004. In the absence of clear risks associated with long-term high supplemental intakes of vitamin E this UL is maintained in NNR 2012. However, additional, long-term studies are warranted."</p> <p>See above</p> <p>Modified.</p>

General and detailed comment	WG observations and actions
<p>higher vitamin E levels in the body. We do not know, but the above sentence (lines 296-7) is not justified.</p> <p>Lines 308 to 327 The harm of vitamin E in the ATBC Study is not limited to stroke and mortality. In my criticism of the Hathcock review (above), I mentioned an earlier ATBC analysis which found non-significant, but worrying, increase in pneumonia risk in participants who started to smoke at young age (<20 yr). Pneumonia is the most common severe infection and, given the popular interest in vitamin E supplements, increase in pneumonia risk by 50 mg/day vitamin E would have public health importance. Therefore I analyzed the effect of vitamin E on pneumonia further. In 2008, I published a paper in which I showed that the above mentioned increase in pneumonia risk was restricted to low body-weight and high body-weight ATBC study participants who had high dietary vitamin C intake. In the low body-weight (<60 kg) participants who had dietary vitamin C intake above the median, 50 mg/day vitamin E supplementation increased pneumonia risk by 248% (95% CI: 61% to 650%; only 9 pneumonia cases in the no-vitamin E group but 26 cases in the vitamin E groups leads to a wide CI). In the high body-weight (>100 kg) participants who had dietary vitamin C intake above the median, vitamin E supplementation increased pneumonia risk by 1350% (95% CI: 84% to 11000%; only 1 pneumonia case in the no-vitamin E group but 12 cases in the vitamin E groups leads to a very wide CI). http://dx.doi.org/10.1186/1475-2891-7-33 In 2011, I published a second subgroup analysis of the ATBC pneumonia cases using a different rationalization. I showed that there is strong evidence that the effect of vitamin E on pneumonia incidence is heterogeneous in 8 subgroups that were based on the level of smoking and the level of exercise ($P=0.0004$; $\chi^2(7 \text{ df}) = 26.6$) Among ATBC Study participants who started smoking at <20 yr and smoked a pack of cigarettes per day or more and did not carry out leisure time exercise, 50 mg/day vitamin E increased pneumonia risk by 79% (95% CI: 27% to 150%). http://dx.doi.org/10.2147/CLEP.S16114 In the 2011 paper I also showed that the two above subgroup analysis approaches (2008 and 2011) were not inconsistent, although they identified different sets (somewhat overlapping) of people who had increased risk of pneumonia because of being administered 50 mg/day vitamin E. In 2009, I published a subgroup analysis of the ATBC Study in which I showed that 50 mg/day vitamin E supplementation increased tuberculosis risk by 72% (95% CI: 4% to 185%) in participants who had dietary vitamin C intake above the median (i.e. 9half of the ATBC participants). http://dx.doi.org/10.1017/S0007114508923709 Tuberculosis is currently rare in Scandinavia and in that respect the increased TB risk has less public health importance than the increased risk of pneumonia. However, vitamin E is being promoted for “improving the immune system” (eg. the Wu and Meydani review cited in the vitamin E chapter); however, the increase in TB risk shows that the effect can be opposite in some subpopulations. In 2006, ATBC Study reported that 50 mg/day vitamin E increased the incidence of the common cold in elderly heavy smokers who lived away of cities by 58% (95% CI: 23% to 101%) and in elderly heavy smokers who lived in a city by 35% (95% CI: 3% to 76%). http://www.jacn.org/content/25/4/332.full.pdf Thus, increase in the incidence of the common cold in old heavy smokers is also an adverse effect of vitamin E. The dose of vitamin E in the ATBC Study was rather low, 50 mg/day, and therefore all the above findings are concerning. Many of the ATBC subgroups that showed harm of vitamin E are not small in their size. In the study on mortality, about 40% of all the ATBC Study population (11000 of the total of 29000) fell to the subgroup that had the 38% increase in mortality after 3.3 years of vitamin E</p>	<p>See comments on the ATBC-study above</p>

General and detailed comment	WG observations and actions
<p>supplementation. Why doesn't the reviewer of the vitamin E chapter refer to these recent analyses of the ATBC Study, although the ATBC trial was carried out in a Nordic country? Since the ATBC study was carried out in Finland, it is directly relevant to vitamin E toxicity for some groups of middle-aged Scandinavian male smokers. Thus, the ATBC Study has found that vitamin E supplementation increased: 1) total mortality and 2) mortality due to hemorrhagic stroke and 3) incidence of pneumonia and 4) incidence of TB and 5) incidence of the common cold in some subpopulations of the ATBC Study. The ATBC Study used 50 mg/day dl-α-tocopheryl acetate corresponding to 22 a-TE/day. In addition to the ATBC Study, two other studies have found adverse effects of vitamin E with doses lower than proposed UL of 300 a-TE/day: The Graat trial (JAMA 2002) found that 200 mg/day vitamin E increased the severity of respiratory infections, see the findings at the top of this commentary. Graat writes that vitamin E was as "α-tocopheryl acetate". Assuming that this means dl-α-tocopheryl acetate, this corresponds to 90 a-TE/day. http://www.ncbi.nlm.nih.gov/pubmed/12169075 Sesso (2008) found 74% (95% CI: 4% to 191%) increase in hemorrhagic stroke. They administered 400 IU all-rac-alpha-tocopheryl acetate corresponding to 270 a-TE every other day which further corresponds to 135 a-TE/day. http://www.ncbi.nlm.nih.gov/pubmed/18997197</p> <p>DECIDING THE UL -LEVEL FOR VITAMIN E The section on vitamin E Safety is inconsistent with the NNR Chapter 3. Pages 6 and 7 and Fig. 3.3 of NNR Chapter 3 describe the reasoning that should be used for the Upper intake limit (UL). LOAEL means the experimental observation of the lowest [in this case vitamin E] dose that has caused harm. Furthermore, as Fig. 3.3 of Chapter shows, there should be uncertainty factor UF between the LOAEL and UL. Thus, UL should be somewhat smaller than the LOAEL and not equal, given that the LOAEL has been proven to cause harm. On the basis of the 5 clinically relevant outcomes (see the list above), the ATBC Study gives LOAEL of 22 a-TE. The ATBC Study was carried out with a Nordic population group and therefore it is particularly important for the NNR. Chapter 3 explicitly states that one goal of NNR is to look at studies with Nordic populations in particular (page 13, second paragraph). Given such a goal, why are the ATBC Study findings ignored? Even if the ATBC Study findings might be ignored (for what reason - that should be explicit in the NNR), the Graat (JAMA 2002) study found harm by 90 a-TE/day in their population and Sesso (JAMA 2008) found harm by 135 a-TE/day. They too are substantially lower than the UL of 300 a-TE. The Graat study is ignored in the Safety section as mentioned above. The Sesso study is mentioned in the Safety section but the discrepancy between its dose causing harm and the UL is ignored. Thus, even if the author might have some justification to ignore the randomized, double-blind, placebo-controlled long-term ATBC Study, there are two other RCTs that also have found significant harm by doses below the proposed UL. All this data was ignored when the UL has been decided. Lines 303 to 304 "The [EU] scientific committee on food has proposed an upper level of vitamin E of 300 mg/day for adults" Is the purpose of the NNR to independently write down own recommendations, or is the purpose of the NNR to copy the levels from other sources? If the purpose is to carry out independent assessment of evidence, then the opinions of other organizations are not essential. Obviously, the vitamin E author should check other recommendations for their references and arguments, but the kind of copying of UL is unreasonable. NNR does not provide any own argumentation to support the UL of 300 a-TE for vitamin E. Furthermore, the EU report was published in 2003. Did the</p>	<p>The evidence for establishing a LOAL is insufficient.</p>

General and detailed comment	WG observations and actions
<p>EU report consider the harm of 200 mg/day in the Graat (JAMA 2002) study? I dont have a copy of the EU report. Often official reports take a year or more from acceptance to publication so the Graat RCT might be missing. The vitamin E draft writes that the limit of the EU report was based on “effects of ... vitamin E supplementation on blood clotting and includes an uncertainty factor.” Thus, it seems that the Graat (2002) study was not yet included in the 2003 report. In any case, the 2003 EU report cannot include the findings of the ATBC Study published after 2003. Nor can it include the HOPE trial results on HF published in 2005 or the GISSI trial results on HF published in 2006. Nor can it include the Sesso (2008) findings. http://www.ncbi.nlm.nih.gov/pubmed/15769967 http://www.ncbi.nlm.nih.gov/pubmed/16645413 http://www.ncbi.nlm.nih.gov/pubmed/18997197</p> <p>Why doesn't the vitamin E author discuss in the Safety section the trials which found increase in heart failure by vitamin E supplementation? The HOPE study is mentioned on line 143 (Lonn), but not in the Safety section, the GISSI study (middle link above) is not mentioned at all in the vitamin E chapter. The Sesso (2008) study is mentioned in the Safety section, but the actual adverse effect findings are not described (74% increase in hemorrhagic stroke). I cannot understand the lack of consistency, when the Safety section mentions Leppälä (2000) and Sesso (2008) studies, and both of them indicate that LOAEL is less than 200 a-TE, yet the NNR vitamin E chapter still copies the old EU (2003) limit even though this approach is fully inconsistent with the NNR approach described in Chapter 3: pages 6 and 7 and Fig. 3.3.</p> <p>Lines 301 to 302 The same problem as above, but the FNB report is even older.</p> <p>Line 306 “this UL is also used in NNR 2012” I cannot see the rationale of NNR to copy the UL from a 10 year old EU-report and claim that the “revised up-to-date NNR” are based on currently available evidence about vitamin E. There are several recent studies that challenge the validity of the UL as described above. If the NNR organization claims that there is scientific basis for the selected UL levels, then the science should be described transparently. What is the justification for the UL of 300 a-TE. There is no description about the studies on which it is based. Thus, there are severe omissions in the presentation of studies in the Safety section. In addition, the section on Safety is not transparent. For example, as described above, the Leppälä 2000 study is mentioned, but the reference is incorrect and the dose and the size of the harm are not mentioned in the Safety section. Good science is transparent. The section on Safety should be fully rewritten.</p> <p>Lines 319 to 327 First, referring to the Miller study, the author of the vitamin E chapter suggests that the harm of vitamin E is primarily a dose-effect question. The strong heterogeneity in the effect of low-dose vitamin E (22 a-TE/d) on mortality and pneumonia in the ATBC Study refutes the dose-response as the primary issue when we consider vitamin E toxicity. Instead, the harm of vitamin E seems to be more defined by the characteristics of the persons, e.g. age, smoking, dietary vitamin C intake, level of exercise (see above); ATBC included only males, we do not know about females. Second, “causal relationship between vitamin E supplementation and increased mortality remains to be unclear”. As described above, the difference in mortality between the ATBC vitamin E and no-vitamin E participants is so great in the P-scale so that it is not reasonable to describe the status of the causal relation “unclear”: $P=0.0005$ for the heterogeneity over the 6 subgroups and simultaneously $P=0.007$ for the time-modification of the vitamin E harm. How</p>	<p>This statement is based on that several studies include people with diseases or high risk of disease.</p>

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<p>small P-values does the vitamin E reviewer require before drawing cause-effect conclusions?</p> <p>Lines 314 to 317 This text discusses possible benefits of “decreased blood clotting”. It is possible that there are some benefits for some people. However, the heading of this section is “Upper intake levels and toxicity” Possible benefits should not be discussed under that heading, but under a different heading.</p> <p>Lines 329 to 330 This text also discusses possible benefits. The heading of this section is “Upper intake levels and toxicity” Possible benefits or lack of them are not relevant under that heading.</p> <p>CHRONIC DISEASES</p> <p>Lines 123 to 146 There are numerous meta-analyses on vitamin E and mortality and two of them are cited on lines 320-321 elsewhere in the vitamin E chapter. Mortality of old people is primarily dependent on chronic diseases. However, in this section on chronic diseases, the meta-analyses on mortality are ignored. Although few reports (lines 136-140) suggest that vitamin E supplementation might be beneficial for some people, the lack of overall benefit in the mortality studies should be mentioned so that the reader is not misled by the only-a-few positive findings. For example the Miller and the Bjelakovic meta-analyses should be mentioned to give perspective to the few positive reports (as they are already in the ref-list). Given that there are dozens of RCTs on vitamin E, the multiple-comparison problem means that we should expect several false positive findings just because of random variation. Furthermore, over 1 false positive finding is expected per 20 RCTs because essentially all of the RCTs used several outcomes and in many studies subgroup analyses were don. Therefore the overall negative picture should be discussed together with the few selected positive findings. If only the few positive findings are mentioned, that leads to bias and misleads readers. It is fine to summarize some positive studies in the vitamin E chapter and I do not suggest deleting them; still the overall negative picture should be shown more clearly e.g. through the meta-analyses mentioned above. Furthermore, since mortality of old people depends on chronic diseases, the heterogeneity in the vitamin E effect on mortality in the ATBC Study participants is also relevant under the heading of chronic diseases: The 2009 ATBC study found that vitamin E decreased mortality among those aged 66–69 years who had high vitamin C intake by 41% (95% CI: –56% to –21%): http://dx.doi.org/10.1093/aje/kwn413 In 2011, I showed that addition of separate vitamin E effects for the 65–70 years age range and for the ages over 71 significantly improved the statistical model ($\chi^2(1 \text{ df}) = 5.0$, $P = 0.03$) indicating that the effect of vitamin E was different for ATBC participants older than 71 yr. Among ATBC participants who had high dietary vitamin C intake and smoked less than a pack per day, vitamin E extended life span by 2 years at the upper limit of the life span. http://dx.doi.org/10.1093/ageing/afq178 These findings are relevant to the question about potential beneficial vitamin E effects on chronic diseases of elderly males in Nordic countries, since most of old-age mortality is caused by chronic diseases.</p> <p>Lines 144 to 146 This kind of report of harm would be better in the Safety section and numerical data should also be given. How much was the adverse effect increased?</p> <p>Lines 173 to 177 On the basis of laboratory studies on the immune system, there has been speculation that vitamin E supplementation might be beneficial against respiratory and other infections. However, no consistent benefit has been</p>	<p>OK, deleted.</p> <p>Not justified.</p>

General and detailed comment	WG observations and actions
<p>found and some studies on infection have found statistically significant harm of vitamin E supplementation. The NNR draft refers to the speculation by Wu and Meydani, whereas it should put much more weight on the RCTs. Graat et al (JAMA 2002) found significantly more severe respiratory infections in old people administered 0.2 g/day vitamin E (see also the top of this commentary). http://www.ncbi.nlm.nih.gov/pubmed/12169075 Why is the Graat trial not cited? In the ATBC Study, 50 mg/day vitamin E increased the risk of tuberculosis and pneumonia in some subgroups, see above. ATBC Study did not identify any subgroup in which vitamin E might reduce the risk of TB: http://dx.doi.org/10.1017/S0007114508923709 In the ATBC Study, there was a group of participants in which vitamin E decreased the incidence of pneumonia by 69% (95% CI: -43% to -83%): participants who were least exposed to smoking and exercised during leisure: http://dx.doi.org/10.2147/CLEP.S16114 Thus, it is possible that some pneumonia cases might be prevented by vitamin E if supplementation is focused on right people. In 2006, ATBC Study reported that the effect of vitamin E on common cold incidence was modified by age and the level of smoking. Old and least smoking citydwellers had statistically highly significant benefit of vitamin E, as it decreased the incidence of colds by 46% (95% CI: -20% to -63%): http://www.jacn.org/content/25/4/332.full.pdf This ATBC study analysis was based on 55,770 common cold episodes recorded during 69,094 person-years of active follow-up. In comparison, the Graat (JAMA 2002) trial recorded only 1024 common cold episodes. Thus, if NNR will comment on vitamin E effect on respiratory infections, the 2006 ATBC paper with clinical acute respiratory infections is much more informative than the Wu and Meydani review. Respiratory infections are not chronic diseases and therefore the author of the vitamin E chapter should either delete lines 173 to 177 or add a new heading “vitamin E and infections”. The latter would seem to be a better choice because there has been lots of interest in the possible immune system effects of vitamin E. In any case lines 173 to 177 are not under a correct heading in the NNR draft.</p> <p>RECOMMENDED INTAKES</p> <p>Lines 238 to 241 “Among older smoking men lowest mortality during 19 years was reported for those whose serum alpha-tocopherol values adjusted for cholesterol at baseline were between 13-14 mg/L (30.2-32.5 µmol/L) after which no further benefit was noted (Wright et al 2006).” With Dr. Miller, I pointed out that the reasoning of the Wright (2006) paper was flawed: http://ajcn.nutrition.org/content/86/1/261.full If vitamin E intakes below 13-14 mg/day would be harmful, then we should expect that vitamin E supplementation would decrease mortality in participants with low dietary vitamin E intakes. However, that was not observed in the Wright study. We wrote, for example: “Recommendations for vitamin E supplementation are not supported by findings from the trial period of the ATBC Study. In subjects in the lowest quintile of plasma α-tocopherol concentration, the similar mortality in the groups with supplement intakes of 50 and 0 mg α-tocopherol (n = 1628 and 1610, respectively; see Table 3 in reference 2) refutes the notions that a low α-tocopherol intake—ie, 9.4 mg/d—is the specific cause of high mortality and that correction of this “deficiency” with 50 mg α-tocopherol/d would affect mortality in this high-risk quintile.” The above mortality figures are taken from Wrights Table 3: http://ajcn.nutrition.org/content/84/5/1200/T3.expansion.html Thus, Dr. Miller and myself challenged the reasoning of Wright et al. No one has challenged the reasoning of the above excerpt either in the</p>	<p>The Wright study is cited, but not used for the derivation of DRVs.</p>

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<p>AJCN or privately, and therefore the above excerpt refutes lines 238 to 241. Observational studies on nutrition have severe problems since people who have low dietary vitamin E intake have simultaneously dozens or hundreds of other factors that differ from people who have high vitamin E intake. A good illustration of this problem was published by Davey Smith's group in Plos Medicine: http://www.ncbi.nlm.nih.gov/pubmed/18076282 They summarized: "We demonstrate that behavioural, socioeconomic, and physiological factors are strongly interrelated, with 45% of all possible pairwise associations between 96 nongenetic characteristics (n = 4,560 correlations) being significant at the p < 0.01 level (the ratio of observed to expected significant associations was 45; p-value for difference between observed and expected < 0.000001)." Thus, the implication of that paper is that even if we adjust for smoking, alcohol, sex, age and a few other standard confounding variables, there can still be many dozens or hundreds of other variables that are important residual confounders. Thus, when Wright found that low dietary vitamin E intake was associated with higher mortality - but 50 mg/day vitamin E supplementation did not influence mortality at all among the low dietary vitamin E participants - then Dr. Wright should have concluded that the higher mortality of people getting low dietary vitamin E intake cannot be explained by vitamin E per se, but by some of the dozens or hundreds of other "behavioural, socioeconomic, and physiological factors" mentioned in the Davey Smith's paper. Thus, the Wright paper is irrelevant when the NNR goal is to consider the health effects of vitamin E.</p> <p>Lines 258 to 259 "8" and "10" does not have a unit. I can guess that the unit is a-TE, but that is not written. Furthermore, the a-TE is not defined in the Introduction, i.e. how many milligrams it is. Scientific text should be clear and transparent. NNR should not leave it to the reader to guess what the probable units are.</p> <p>INTRODUCTION Lines 16 to 38 The unit "a-TE" is introduced, but there is no description how many milligrams it is. The definition of a-TE is not clear.</p> <p>Line 36 "Conversion factor" The same problem. The text does not describe which units are converted to which other units or what does the conversion factor mean.</p> <p>Line 23 "Previously vitamin E ... alpha-TE" What does this mean? Does that mean that in the old literature a-TE was used, but current literature uses some other unit? What is the new unit, if a-TE is limited to the "previous literature"? Why is a-TE unit used in the Table, if it is a "previous" unit? Should there be "currently vitamin E ... a-TE"? Previously and currently are very different.</p> <p>Line 28 "all-rac-alpha-tocopherol or dl-alpha-tocopherol" This statement is not clear. Do the two terms mean two different chemicals or two different names for a single chemical? I know the answer, but the text should be so clear that the ordinary reader does not need to guess which of the two alternative interpretations of the sentence is correct.</p> <p>Lines 298 to 301 The same unit should be used consistently in the vitamin E chapter. "1600 IU (1073 mg)" should be changed to a-TE if that is the main unit in the chapter.</p> <p>Line 312 The same unit should be used consistently in the vitamin E chapter. "400 IU (270 mg)" should be changed to TE if that is the main unit in the chapter.</p> <p>Line 316 The same unit should be used consistently in the vitamin E chapter. "600 IU (400 mg)" should be changed to TE</p>	<p>This is defined in the subsequent paragraph. See above</p> <p>Clarified in final version.</p> <p>These terms are equal.</p> <p>Units stated in original articles stated with amounts in mg where available. See above</p> <p>See above</p>

General and detailed comment	WG observations and actions
<p>if that is the main unit in the chapter.</p> <p>Line 323 The same unit should be used consistently in the vitamin E chapter. "150 IU (100 mg)" should be changed to TE if that is the main unit in the chapter.</p> <p>PHYSIOLOGY Line 72-73 "large individual variation in human responses to vitamin E" This is not clear. Does the author refer to clinically relevant responses, or laboratory outcomes (surrogates), or levels of vitamin E in blood/tissues after changes in intake levels? What does "response" mean in the context?</p> <p>Lines 113-114 and Lines 176 to 177 "differences in the individual responses to alpha-tocopherol are also suggested to arise due to genetic factors" Evidently, genes explain some part of the variation in the effects of vitamin E. However, the evidence of heterogeneity in the Belisle paper is very weak: http://ajcn.nutrition.org/content/92/1/106.full Abstract "The effect of vitamin E on lower RI depended on sex and the SNP at IL-10 -819G→A (P = 0.03 for interaction for lower RI)." P = 0.03 is very weak evidence in genetics because lots of comparisons are done and only the "positive" findings are published. There is a severe problem with multiple comparisons in genetics. See for example the paper by Ioannidis, in which he used genetics as an example: http://www.ncbi.nlm.nih.gov/pubmed/16060722 As described above, in the ATBC Study (a Nordic RCT), age, level of smoking, dietary vitamin C intake, and leisure-time exercise explained large part of variation in the vitamin E effect on total mortality (P = 0.0005 for the test of heterogeneity over 6 subgroups) and incidence of pneumonia (P = 0.0004 for the test of heterogeneity over 8 subgroups). The effect of vitamin E on the incidence of the common cold was also significantly heterogeneous (P = 0.000000004 for the test of heterogeneity over age in ATBC participants who smoked 5-14 cigarettes per day and lived in a city and P = 0.0000002 for the test of heterogeneity over age in those who smoked >15 cigarettes per day and lived away of cities). Thus, on the P-scale, the evidence that life-style factors modify vitamin E effect is very much stronger than the evidence that genes modify vitamin E effect. Thus the life-style and demographic factors should also be mentioned as a cause of individual variation in vitamin E responses, in particular since the much stronger evidence is from a Nordic population.</p> <p>DIETARY SOURCES AND INTAKES</p> <p>Lines 52 to 54 The references presented are official reports and very difficult to reach, and 4 of the 5 are non-English reports. Why doesn't the chapter refer to papers that are easily available and in English language. For example, the distribution of the vitamin E levels of 27000 ATBC study participants (Finnish males) is published in a paper that is openly available simply by clicking the link below (vitamin E distribution is in table 2): http://dx.doi.org/10.1378/chest.125.2.557 The NNR report should be user friendly so that it prefers references that are easy-to-reach in contrast to very-difficult-to-reach non-English reports. Obviously, there are many more easy-to-reach papers in addition to the one above. In any case, they should be preferred in favor of non-English difficult-to-reach reports.</p>	<p>See above</p> <p>Reworded</p> <p>No further comment added</p> <p>Data from national dietary surveys in the Nordic countries are used, some which may only be available in national language.</p>
<p>General comments</p> <p>In the table shown before the text, vitamin E is still presented in alpha-TE although according to present knowledge alpha-tocopherol is the only form that is recognized to meet human requirement. I also suggest that either Greek</p>	<p>Most comments/suggested amendments accepted.</p>

General and detailed comment	WG observations and actions
<p>alphabet α- or word alpha- written in English should be used uniformly through the text. See detailed comments on the table. Since vitamin E is still presented in IUs especially in USA, factors for converting IUs of vitamin E to alpha-tocopherol (mg) should be added in the introduction. These factors are different for natural and synthetic alpha-tocopherol and they also differ from the USP conversion factors. Sentence "In older literature 1 alpha-TE corresponds 1.49 IU" is unnecessary and should be removed. See detailed comments on line 38. I suggest that in the text concerning studies which presented vitamin E in IUs no calculations are made to convert IUs to milligrams of alpha-tocopherol. When conversion factors from vitamin E in IUs to alpha-tocopherol in mgs are given and the type of alpha-tocopherol used is specified in the text, it is possible for a reader to make these calculations himself. See comments on lines 134, 140-141, 145, 154, 312, 316, 322, 324. In my mind it is appropriate to present correspondence between IUs and milligrams in the text if these figures are already given in the original publication, e.g. in Hatchock et al 2005, cited in the text on line 300. It appears that the section "Reasoning behind the recommendation" only repeats which is already said in the former sections "Adults", "Children", "Pregnancy and lactation". I suggest that this section is deleted and the section "Requirement and recommended intake" is renamed "Recommended intake and reasoning behind recommendation". See detailed comments on lines 5, 9, 179 and 280-294. The text of recommendations for children and pregnancy and lactation are directly based on the previous recommendations where alpha-TEs were used. In my mind in this NNR5 recommendation alpha-tocopherol should be used. See detailed comments on lines 271, 275, 277. In addition few minor comments, mainly printing errors are included, see detailed comments on lines 150-151, 161, 217, 258, 302-303, 309. In my mind presentation of references in the text should be standardized and given either by numbers or by writers' names through all the chapters of NNR5.</p> <p>Detailed comments</p> <p>Table of vitamin E: Vitamin E should be given as alpha-tocopherol not as alpha-TE. I also suggest that either Greek alphabet α- or word alpha- written in English should be used uniformly through the text.</p> <p>Line 5 should be: Recommended intake and reasoning behind recommendation</p> <p>Line 9 should be removed.</p> <p>Line 38 should be: acetate (Burton et al 1998, Horwitt 1999). In older literature vitamin E activity was expressed as IUs. One IU is equivalent to 0.67 mg of the natural form and 0.45 mg of the synthetic form of the vitamin (Office of Dietary Supplements 2011).</p> <p>Line 38: Sentence "In older literature 1 alpha-TE corresponds 1.49 IU." should be removed.</p> <p>Line 134 should be: supplementation of healthy women with 600 IU of a natural form of vitamin E.</p> <p>Lines 140-141 should be: to benefit from 600 IU natural source vitamin E supplementation every other day (Cook et al 2007). In another study of high risk population daily supplementation with 400 IU natural alpha-tocopherol did.</p> <p>Line 145 should be: reported among healthy physicians who received 400 IU synthetic alpha-tocopherol every</p>	<p>Not accepted.</p> <p>Heading "Reasoning behind recommendation" is used in all chapters providing a short summary. Accepted</p> <p>Deleted Accepted. Sentences revised.</p> <p>OK</p>

General and detailed comment	WG observations and actions
<p>Lines 150-151 should be: 2011). The decreased prostate cancer risk associated with 50 mg daily dose of synthetic alpha-tocopherol among middle-aged Finnish male smokers (Heinonen et al 1998) has not been</p> <p>Line 154 should be: had received a daily supplement of 400 IU synthetic alpha-tocopherol on</p> <p>Line 161 should be: al 2009, Lin et al 2009, Lippman et al 2009). Results from observational studies of alpha-</p> <p>Line 179 should be: Recommended intake and reasoning behind recommendation</p> <p>Line 217 should be: studies by Horwitt et al (1963) showed an increased haemolytic tendency in subjects</p> <p>Line 258 should be: maintained. The RI of vitamin E is set to 8 mg alpha-tocopherol/d and 10 mg alpha-</p> <p>Line 271 should be: In NNR 2012, the recommended intakes are based on a ratio of at least 0.6 mg alpha-tocopherol/g</p> <p>Line 275 should be: The recommended intake value for pregnancy is set to 10 mg alpha-tocopherol/d, which is applicable in the</p> <p>Line 277 should be: intake during lactation (11 mg alpha-tocopherol/d) also includes the extra need to cover secretion in</p> <p>Lines 280-294 should be removed.</p> <p>Lines 302-303 should be: tocopherol per day in the US recommendations (Food and Nutrition Board 2000). The Scientific Committee on Food (Scientific Committee on Food 2003) has proposed an</p> <p>Line 309 should be: ATBC study suggesting increased mortality due to hemorrhagic stroke (The Alpha-</p> <p>Line 312 should be: 400 IU synthetic alpha-tocopherol every other day (Sesso et al 2008). Thus high</p> <p>Line 316 should be: healthy women given 600 IU supplement of a natural form of alpha-tocopherol</p> <p>Line 322 should be: supplemented with doses of 400 IU per day or higher, and a dose-response analysis</p> <p>Line 324 should be: IU/day (Miller et al 2005). Generalization of these findings, largely based on studies</p>	<p>OK</p> <p>OK</p> <p>Retained</p> <p>OK</p> <p>Alpha-TE</p> <p>Alpha-TE</p> <p>Alpha-TE</p> <p>Alpha-TE</p> <p>Shortened</p> <p>Reference adjusted in final version</p> <p>See above</p> <p>See above</p>
<p>Svensk Mjöl</p> <p>General comments</p> <p>The criteria for selection of papers for the scientific reviews are not shown. Publications chosen for evaluation based on the criteria are not listed. The authors of the chapters of draft proposals are not included. The connection between systematic review and draft proposal are not transparent. In general, old references are used. This indicates that references from new literature searches not are used, making the transparency of the process hard to rely on. The chapters are not updated with dietary sources of nutrient based on the results from Riksmaten 2010-11. It is important to use the latest information on these results, to justify the nutrition recommendation. Recommendations from other countries/organizations should be included in every chapter in a consistent way. The bioavailability of a nutrient from the major contributing food categories in the Nordic countries should be included. The influence of a nutrient in food sources should be distinguished from the same nutrient added to the diet as supplement.</p> <p>Detailed comments</p> <p>Page 7, line 23: What cheeses? Different cheeses have very divergent roles in the diet. Hard cheeses are often used on a daily basis, whereas for example blue cheese is consumed less often. There is thus a difference between cheeses as</p>	<p>The literature search will be available.</p> <p>The expert and peer reviewer responsible for the update are listed in connection with the preface to the final publication.</p>

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<p>sources for vitamin K, which might be of concern. In the past 20 years, additional support for vitamin K functions in the nervous system has emerged. Aspects regarding the role of vitamin K for the nervous system and cognitive function should thus be added.</p>	
<p>Nutrition Unit, National Institute for Health and Welfare</p> <p>On lines 58-60, you compare children's vitamin E intake to that of adults. Please consider including the results on children's vitamin E intake from Kyttälä et. al, Food consumption and nutrient intake in Finnish 1–6-year-old children, Public Health Nutrition 2010, 13(6A), 947–95. The corresponding values are presented in the report 'The diet of Finnish preschoolers' (Publications of the National Public Health Institute B32/2008) adjusted for energy. Serum vitamin E concentrations of healthy Finnish children can be found in Uusitalo L, Nevalainen J, Niinistö S, Alfthan G, Sundvall J, Korhonen T, Kenward MG, Oja H, Veijola R, Simell O, Ilonen J, Knip M, Virtanen SM. Serum alpha- and gamma-tocopherol concentrations and risk of advanced beta cell autoimmunity in Children With HLA-conferred susceptibility to type 1 diabetes mellitus. Diabetologia 2008;51:773-80.</p>	<p>Intake data are updated in the final version.</p>
<p>Danish Food and Drink Federation</p> <p>General comments</p> <p>In the chapters for Vitamin E and dietary antioxidants it is stated that supplemental intakes are not recommended due to current evidence. The strongest sentence with scientific support is that the literature is inconclusive, so supplemental use cannot be recommended or un-recommended. The vast majority of current evidence is based on high dosage intakes of a few vitamins or minerals, which is very different from the most commonly consumed food supplement: a multivitamin and mineral containing RDA-like dosages for most of the essential micronutrients. Physicians' health study is the only long term RCT on a healthy population investigating the impact of a multivitamin-mineral on chronic diseases. Recently two of the four major end-points of Physicians Health Study II have been published and the two remaining end-points: cognitive decline and eye health are expected to be published in 2012. We strongly encourage you to wait for those results and include them in the relevant chapters, as this is the kind of supplement taken by aprox 50% of the adult population (at least in Denmark).</p> <p>Detailed comments</p> <p>Line 198 Please discuss the differences from the IoM/US Food and Nutrition board recommendations, where RI is 15 mg for adults and AR is 12 mg – it's a difference of a 100% in the AR – and that deserves to be justified</p> <p>Line 329-330 should be a bit more precise. No benefits of very high (>100mg) intakes of vitamin</p> <p>Line 331-333 Please delete this sentence or specify the amount of supplemental Vitamin E that cannot be justified (>100mg).</p> <p>The first sentence line 329-331 is scientifically justified and could stand alone.</p>	<p>This is done in the final version.</p> <p>Included in final version.</p>

General and detailed comment	WG observations and actions
<p>Helsedirektoratet, Norway</p> <p>Detailed comments</p> <p>27. Explain the acronym RRR-</p> <p>53-4. Please add intake data from Norkost 2010-11.</p> <p>129-32 and 133-7. These sentences are difficult to understand. Please rephrase.</p> <p>137. Those – not clear what those refers to?</p> <p>139were suggested ... not clear, may use another expression.</p> <p>295-333. UL The conclusion at line331-33 seems adequate. However, it may seem as the first and the third section in this chapter contradicts each other regarding the UL of 300 mg/d. The references quoted on line 321 and 324 indicate side effects as increased mortality at intakes of vitamin E from supplements already at intake levels of 100 and 270 mg/d. Thus the UL seems to high. You may reconsider the level of your UL.</p>	<p>Basic chemistry</p> <p>Intake data are updated in the final version.</p> <p>Clarified</p> <p>Clarified</p> <p>Rephrased</p> <p>Acknowledged. The section is partly reworded.</p>

NNR 2012: Public consultation – Chapter 18 (Vitamin K)

General and detailed comments	WG observations and actions
<p>General comments</p> <p>Most chapters start with a conclusion/recommendation. This is very convenient for the reader. Thus this chapter also should start with the conclusions/recommendations concerning vitamin K. Even if you do not give a recommendations concerning intake of vitamin K this is relevant information for the reader. Anyhow you give a clear conclusion regarding newborns.</p> <p>Detailed comments</p> <p>24. You here mention menaquinone-7. “-7” thus it for the reader seems to be several varieties of this compound, and this may be explained.</p> <p>111-2. Bacterial synthesis is mentioned for the first time here. This could be expanded and the importance of bacterial synthesis could be explained under the section about Physiology.</p>	<p>NNR 2012 does not set RI for vit K, but a provisional reference value. However, a box will be added before the introduction and the text in the section “Requirement and recommended intake” will be clarified.</p> <p>A few other MK-compounds mentioned</p> <p>Mentioned in the introduction.</p>

General and detailed comments	WG observations and actions
<p>VitaK at Maastricht University, The Netherlands</p> <p>General comments</p> <p>For requirement it is regrettable that the Triage theory (Ames) has not been mentioned. Triage explains why a sustained increase of vitamin K intake may prevent age-related diseases.</p> <p>The question how much extra vitamin K should be taken can be addressed by looking at uncarboxylated Gla-proteins osteocalcin and MGP. A significant decrease of circulating ucOC and ucMGP is seen in healthy subjects at 45-90 mcg/day, with a maximal effect at 180 mcg/day of MK-7 (Theuwissen et al).</p> <p>For other K vitamers these intakes may be different, but it surely is an indication. What I am missing is the differentiation between K1 and K2 in the recommendations. For sure the number of intervention studies with clinical endpoints is too small. But until we have sufficient data (which may take another 10 years) I think it would be wise to recommend intakes as large as the highest quartile in the normal, non-supplemented population. This would be around 45 mcg/day. Together with the observed K1 intakes (between 70 and 100 mcg/day) this would result in about 120 mcg/day of total vitamin K, with a good proportion between K1 and K2. The total intake would then be around the American recommendations. I know this is only a rough estimate, but I don't have a better one for now.</p> <p>Detailed comments</p> <p>Line 51: The spelling should be Gijsbers, not Gijsbergs</p> <p>Line 60: The longer half-life times only concern the long-chain menaquinones, not MK-4 for instance.</p> <p>Line 73: the problem of Bolton-Smith was not that she could not find protective effect at certain sites, but that the control group did not lose bone at these sites. So it was basically impossible to measure a protective effect. At the site where bone loss was found in the controls (ultradistal radius) a significant effect was found of a combined vitamin D+K intervention.</p> <p>Lines 74/84: My comment is that if effects are found, this is rather on BMC and bone strength than on BMD. Indeed there are also studies showing no effect, but in general they are too short. We have just completed our third long-term intervention study (after K1 and MK-4, it was now MK-7) and in all three studies a significant effect is only seen in the third intervention year. Most negative studies you refer to (Binkley, Emaus) are only one or two years. An exception to this is the study by Kanellakis et al, who incorporated MK-7 treatment in a change of life-style. They saw an effect after one year, but this study is lacking in your review.</p> <p>Line 94: A protective effect against cardiovascular disease and mortality. References should be completed with: Geleijnse et al and Beulens et al.</p>	<p>Triage theory cited in final version.</p> <p>WG considers data are insufficient to establish a RI, both for vit K1 and K2.</p> <p>The U.S. reference intakes are "Adequate Intakes" based on observed intakes of phylloquinone (only). There is limited information on typical intakes of vit K in the Nordic countries, although the available studies report average intakes among adults ranging from 90-120 µg/d K1 and 15-20 µg/d K2.</p> <p>In the final version references are updated.</p> <p>The section "Requirement and recommended intake" is elaborated.</p> <p>References replaced by numbers..</p> <p>Corrected.</p> <p>Acknowledged.</p> <p>Difficult to interpret results of combined vit D+K supplement.</p> <p>Reference to Kanellakis added.</p> <p>The Geleijnse study is included. Beulens et al. is a cross-sectional study and</p>

General and detailed comments	WG observations and actions
	therefore not included.
<p>MTT Agrifood research Finland</p> <p>General comments</p> <p>A real or relative vitamin K deficiency has been connected with coronary artery calcification (Beulens et al. 2008, Shea et al. 2009), cardiovascular diseases (Gast et al. 2008) and bone health (Koitaya 2009, Iwamoto et al. 2009). However, results of epidemiological and clinical studies are unanimous. Reasons for divergent results may be variations found in genes of vitamin K-dependent enzymes or proteins. According to Nimptsch et al. (2009) the association between dietary vitamin K and serum undercarboxylated osteocalcin is modulated by vitamin K epoxide reductase (VKORC1) genotype. Only the +2255 GG genotype seems to be efficient enough to reduce the ratio of serum undercarboxylated osteocalcin/total intact osteocalcin. Elevated serum osteocalcin concentrations have been associated with increased hip fracture risk and reduced bone mineral density. VKORC1 genotype (2255 non-TT (rs 2359612) is associated with cardiovascular events in patients with drug-eluting stent (DES) implantation, suggesting the role of coagulation system (Suh et al. 2009). Polymorphisms of VKORC1 seem to affect the risk of cardiovascular diseases (Wang et al. 2006, 2012). GAS-6 gene variant c.834+7G>A may increase risk of CVD (Jiang et al. 2009) or stroke (Munoz et al. (2004). Suh et al. (2009), Jiang et al. (2009), Munoz et al. (2004) and Wang et al. (2006) have not determined vitamin K2 status of their patients. Polymorphism of γ-glutamyl carboxylase has been suggested to affect the correlation between serum menaquinone-7 concentrations and γ-carboxylation of osteocalcin (Sogabe et al. 2007).</p> <p>Recent reviews (Ferland 2012a,b) propose a significant role for vitamin K in the nervous system. Nimptsch et al. (2010) have reported relation of vitamin K intake with cancer risk.</p> <p>McCann and Ames (2009) have linked vitamin K inadequacy especially with diseases of aging. As the aged population in Northern countries increases rapidly, the costs of lifestyle diseases/diseases of aging will also increase. Vitamin K might be one possibility to lessen the health care costs.</p> <p>To get to the bottom of the effects of vitamin K large-scale population studies are needed. The known</p>	<p>Comments appreciated. Several of the references screened and some included, although not useful for setting dietary reference intakes.</p>

General and detailed comments	WG observations and actions
(and unknown) polymorphisms in the genes of vitamin K metabolizing enzymes should be notified as well as the bioavailability of different vitamin K forms and the possible impact of gut microbiome (and dietary fiber). *	
<p>NIFES, Norway</p> <p>General comments</p> <p>It is somewhat difficult to find the suggested recommendations for vitamin K in this chapter. Many of the other vitamins have tabulated values, but I can't find similar tables for vitamin K. The recommendations might very well be written within the text, but is it possible to highlight the recommended values for vitamin K for 2012 a bit more?</p>	There is no RI established.

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NNR 2012: Public consultation – Chapter 19 (Thiamin)

Organisation / Company	Thiamin General and detailed comments	WG observations and action
Selskab for Ernæringsforskning, Denmark	Detailed comments Line 58: replace “indicators” with “biomarkers” Line 59: replace “In NNR” with “The NNR” Line 66: replace “indicator” with “biomarker”	Replaced Replaced

Organisation / Company	Thiamin General and detailed comments	WG observations and action
	Line 100-101 & 112: : The recommendations are not unchanged when LI for men in 2004 was 0.5 while in 2012 it is 0.6.	No change
Minivent AB, Sweden	<p>General comments</p> <p>Line 107: MANIFEST BERIBERI DOES EXIST IN SWEDEN WITH THE ELDERLY! Contact me if you want a case description, which I am wellinformed on. This 83 year old person was non-smoker and total abstainer, the whole life. He was thinking much about his health, and ordered health-advise from National Institute of Public Health in the 1970:th. On the whole, he was eating healthy food. He was normally _not_ eating coffee, buns and cakes. In spite of this, he was stricken with thiamin deficiency in manifest degree./ No line: Search-words on Internet: Spelling: beriberi, beri-beri, beri beri, Kakke. English spelling: thiamin, thiamine, aneurin. German spelling: thiamin.</p> <p>Detailed comments</p> <p>Line 94: Two hyphens have disappeared. (There is different types of hyphens (dashes) in computers)./ Line 107:/ HOW MANY people have thiamin deficiency in i-countries? WHO: 23 RESP 43 % WITH THE ELDERLY HAD THIAMIN DEFICIENCY according to studies. More research with the elderly about B-vitamins is needed: http://whqlibdoc.who.int/publications/2004/9241546123_chap9.pdf > Page 168, 185 in the document. (Not the page according to the application). Quotations from the document:/ "HOORN et al. (26) reported that _23%_ of 153 patients aged _65–93 years_ were deemed _deficient_ based on a transketolase activation coefficient greater than 1.27, which was normalized after thiamine administration. NICHOLS and BASU (27) found that only _57%_ of 60 adults aged _65–74 years_ had TPP effects of less than 14% and suggested that ageing may increase thiamine requirements.". "9.9 Recommendations for future research" ... "Actual requirements of B-complex vitamins are LEAST CERTAIN FOR children, adolescents, pregnant and lactating women, and the ELDERLY, and as such, deserve further study."/ 100 % – 57 % = _43 %_. WHY does the elderly get thiamin deficiency?: http://www.svenskmjolk.se/Global/Dokument/Epi-tradet/Mjolk,%20smör%20och%20ost/Näring%20och%20hälsa/Vitamin-%20och%20mineralguiden/Vitamin_och_mineralguiden_07.pdf > Page 2: Authors of the document: BRANTH, Stefan & HAMBRAEUS, Leif. > Page 21: THE TRANSPORT PROTEIN that is helping the thiamin to get resorbed, IS DECREASING BY THE INCREASING AGE. > 76: References./ (Here is the webpage which is placed above: http://www.svenskmjolk.se/Mjolk-smor-och-ost/Naring/Vitaminer-och-mineraler/#.UOzqSI6dUZM</p>	<p>A few case-reports published in the Nordic medical journals during the recent decade. Mainly in connection with alcohol abuse.</p> <p>Corrected</p>
LRF Mjolk, Sweden	<p>General comments</p> <p>The text is not very well structured, and does thus not match the other chapters.</p>	Some editorial changes made

Organisation / Company	Thiamin General and detailed comments	WG observations and action
	Detailed comments Line 23: Is there possibly a more recent knowledge?	Few new relevant studies published
National Food Agency, Sweden	Detailed comments 11 Box with recommendations: add 1.0 mg for elderly people at intakes < 8 MJ/d UL abbreviation ND is not clear, please use - as for other nutrients with no UL 108 add "for elderly" after ...planning diets to clarify the difference in recommendations for low energy diets for adults and for elderly	Added Added
Helsedirektoratet, Norway	Detailed comments 98 and 108. In these two sections you recommend that when planning diets with energy levels below 8 MJ/d, the thiamin content should be at least 0.8 (line 99) and 1.0 mg/d (line 109). You may clarify if the recommendation regarding 1 mg/d is for elderly subjects.	Clarified

NNR 2012: Public consultation – Chapter 20 (Riboflavin)

Organisation/ Company	General and detailed comments	WG observations and actions
Selskab for Ernæringsforskning, Denmark	General comments Line 125-132 and 108-117 are identical Detailed comments Line 26: Replace "Riboflavin occurs in foods free or" with "Riboflavin occurs in foods in the free form or" OR "Riboflavin occurs in foods unbound or"	Revised Corrected
National Food Agency, Sweden	General comments Add names in B series for all B vitamins, i.e. B1---B12 Detailed comments 57, 64, 91 please change the numbered references into name-date format	OK References corrected in final version

NNR 2012: Public consultation – Chapter 21 (Niacin)

Organisation/ Company	Niacin General and detailed comments	WG observations and actions
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SNF Swedish Nutrition Foundation	Detailed comments Line numbers: 47-52: "In the absence of new scientific data the reference values for niacin given in NNR 2004 are kept unchanged."... "This corresponds to an intake of 17-19 NE/d for adult men and 14-15 NE/d for adult women." In NNR 2012, RI for men is set to 18, while it was 19 in NNR 2004. For women, the recommendation is unchanged at 15. In NNR 2004, the RI thus corresponded to the upper limit of the quoted interval for both men and women, while in the NNR 2012 draft the RI corresponds to the upper limit of the interval for women and the mean of the interval for men. What is the reason for this very minor change?	RIs have been adjusted to the revised reference values for energy intake
Selskab for Ernæringsforskning, Denmark	General comments Line 63-64: Comment: The recommendations are not unchanged when RI for men in 2004 was 19 while in 2012 it is 18.	RIs have been adjusted to the revised reference values for energy intake
National Food Agency, Sweden	General comments Add names in B series for all B vitamins i.e. B1---B12. Detailed comments 11 Add 8 NE/d at intakes < 8 MJ/d in box, as for Thiamin	For relevant B-vitamins Added

NNR 2012 Public consultation – Chapter 22 (Vitamin B6)

Organisation/ Company	General and detailed comments	WG responses and actions
University of Bergen, Norway	In general the NNR 2012 on vitamin B6 is well written. Here are some few minor comments: In the section on dietary sources and intake, it could be worthy to add "banana and avocado" to the list of important sources (http://ndb.nal.usda.gov/ndb/foods/show/2237). In line 149, there is a minor misspelling: "ifve studies" instead of "five studies". References: USDA National Nutrient Database for Standard Reference: http://ndb.nal.usda.gov/ndb/foods/show/2237?fg=&man=&lfacet=&count=&max=&sort=&qlookup=&offset=&format=Full&new=&measureby= Detailed comments: See general comments	Foods and food groups mentioned are major dietary sources according to national dietary surveys. Corrected
Takeda Nycomed, Norway	Detailed comments: Line 17, table. The RI for vitamin B6 is 1,6 mg for men, not 1,5.	The RI is 1.5 mg/d.

Organisation/ Company	General and detailed comments	WG responses and actions
Danish Cancer Society	<p>It looks as if the chapter is not quite finished, because of the changes in references.</p> <p>Detailed comments: line 28 reference to chapter xx</p> <p>line 136 reference into brackets line 149 misspelling of the word five line 195 half a bracket line 197 missing dot at the end</p>	<p>References reorganized in final version</p> <p>Chapter numbering given in final version. Corrected Corrected Corrected Corrected</p>
Valio Ltd	<p>Detailed comments: Row 27: Important sources of vitamin B6: add milk to the list</p>	Dairy products added.
LRF Mjölks/Dairy Sweden	<p>Detailed comments. Line 20: Please clarify the relation between pyridoxine, pyridoxal and pyridoxamine in relation to PLP. Line 27: The criteria for including foods as important contributors of vitamin B6 in the diet are not consistent. In Riksmaten 2010-11 fish and fish dishes contributes with 9 %, offal with 0 %, meat and meat dishes with 13 % and potatoes with 10 %. Dairy products are not mentioned as important contributors of vitamin B6 in the diet although they contribute with 7 %. Please correct this matter.</p> <p>Line 37: What does the word “vitamers” mean? Please clarify or choose another word.</p> <p>Line 201: Please clarify what the figure “0.06 mg/L” refers to. Line 202: Does this mean 0,5 mg vitamin B6 per day? Please clarify this. Line 259: What are the natures of these relations? Positive, negative, U-shaped? Please clarify this statement. Line 260: Please declare the actual recommendations in the text.</p>	<p>This is explained in Physiology and metabolism. Food and food groups mentioned are major dietary sources according to national dietary surveys. Revised. The term “vitamers” refers to chemical compounds with a similar molecular structure that provide vitamin activity. This is a common term used. Vitamin B6 added. Per day. Clarified.</p> <p>The RIs are given in the box in the introductory section.</p>

Organisation/ Company	General and detailed comments	WG responses and actions
National Food Agency, Sweden	Detailed comments: Vitamin B6 / Physiology and metabolism/ line 40-42 In most fruits, vegetables and grains, a part of the pyridoxine occurs as a glucoside, which is considered to be less bioavailable than other non-glucoside forms (2, 4) There are now methods for determination of free and bound vitamin B6 in vegetables, legumes and cereals, see reference Determination of total vitamin B6 in foods by isocratic HPLC: a comparison with microbiological analysis. M Kall Food Chemistry 82 (2003) 315-327 DOI 10:1016/S0308-8146(02)00568-X Some examples on glucoside-bound pyridoxin data (available but not published in Sweden, Livsmedelssystemet) average and range in % vegetables 22 (2 - 55) legumes 49 (34-65) bread 24 (6-44) pasta 26 (9-44 for whole grain products) quinoa, bulgur, couscous 37 (21-55). In general the average is larger than 15 % as mentioned in lines 42-43. It would be valuable if these data were available, and more data determined, e g to enable evaluation on vegetarian diets.	Comments appreciated, but further amendments.

NNR 2012: Public consultation – Chapter 23 (Folate)

Organisation /Company	Folate General and detailed comments	WG observations and comments
Danish Cancer Society	General comments: lines 140-182: Important to mention the possible adverse effect of high intakes of folate on certain cancers, to prevent people from taking megadoses. Detailed comments: line 26: Format issue in men, smaller than the rest of the table lines 122-123: Reporting one study on the possible association between maternal folate status and childhood hyperactivity is suitable to raise counterproductive speculations among lay people. lines 140 and 147: choose the right wording suggestive or probable many or most ? Double expressions is not helpful to the reader	A concern has been raised that high intakes of folic acid from supplements or fortification potentially may cause increases in cancer rates. However, this concern does not apply to folate naturally occurring in foods. Line 140-14: The text has been edited as suggested.
Norwegian Directorate of Health	General comments: We suggest that you add comments/conclusions regarding a) fortification and b) 400 microgram supplemental folic acid before and during early pregnancy. See NNR-SLR by Witthöft et al page 17 and 18, respectively. The Norwegian recommendations 2005 differed from those given in NNR 2004 concerning folate. We recommended 400 microgram folate per day for women in reproductive age and also to pregnant and lactating women. However, we added a foot note to these groups: "g) Folat reduserer risikoen for nevrallrørsdefekt. Kvinner som planlegger graviditet anbefales derfor et folattilskudd på 400 mikrogram daglig fra siste måned før befruktning og i svangerskapets første tre måneder (3).» (3) refers to: Anbefalinger og virkemidler for økt folatinntak blant kvinner i fertil alder. Rapport 1/1998. Statens ernæringsråd, Oslo 1998. We intend to keep this recommendation also in the next edition of our recommendations. (This comment is also submitted in relation to chapter 1)	Issuing recommendations regarding supplementation is a management issue and as such not included in the tasks of the NNR revision.

Organisation /Company	Folate General and detailed comments	WG observations and comments
	<p>Detailed comments: 21. Please include a comment concerning UL in the box. 128. You write: “homocysteine status has been associated to higher school grades”. This may be rephrased. 133 and 140. Here you conclude the evidence as suggestive/probable. Please differ between these two grades. 110-210. Health outcomes: If possible please add a conclusion wrapping up the evidence for each health outcome. This has been done in many other chapters and is very useful for the reader. 236 “improvements” do you really mean improvement or the more neutral increased levels of ...? 239-41. un-metabolized folic acid was determined – please explain/comment if this is good or bad. 277-79. Recommendations concerning folic acids supplements may be included here. See NNR-SLR by Witthöft et al page 18; 5 authors’ conclusions. Actually the wording of the first line of this conclusion is difficult to understand. (“There is no evidence in evaluated references because of which the folate expert group recommends alteration of the current recommendations” 284. You may here add Norwegian data. In Norwegian surveys mean folate intake was 220 microgram/d (SD 90 microgram/d) among women aged 18-39 years (Totland et al. Norkost 2010-11). 314-“scientific evidence indicates” – if possible use the more often used terms possible; conclusive etc. 332. UL 1000 microgram/d. Please clarify if this is in the form of supplements and fortification only, as in the foot note used on page 25 in NNR 2004.</p>	<p>A comment regarding UL has been added</p> <p>The wording has been changed.</p> <p>We mean improvements as for some indicators the levels are increased and for some they are decreased (e.g. homocystein)</p> <p>The text has been edited; detected instead of determined. “No studies regarding implications of un-metabolised folic acid on human health are available” has been added.</p> <p>The dietary intake data is in the beginning of the chapter and in a separate table in the end of the book (for all Nordic countries).</p> <p>The sentence “scientific evidence...” has been revised.</p> <p>It is clearly stated in the text that the UL is only set for folic acid. A note regarding UL has been added into the table in the beginning of the chapter.</p>
SNF Swedish Nutrition Foundation 	<p>General comments only</p> <p>General • It would be helpful if different levels of folate status (low, high) were defined. • It would be helpful if different levels of folate intake (low, high, “mega doses”) were defined. Page 7-8, pregnancy and lactation. • The text states that the extra need during lactation is 100 ug/d (line 302). It would be helpful if the text also stated the extra need during pregnancy. • The recommendations during pregnancy is 500 ug/d. It would be helpful if it was explicitly stated if this recommendation is valid for the first, second and/or the third trimester, and also during lactation. • In the NNR SR by Witthöft et al, it is concluded that fertile women are recommended a supplemental dose of 400 ug folic acid before and during early pregnancy. However, the question of supplementation is not discussed in the chapter. When comparing the conclusion by Witthöft et al with the chapter it is not clear if the supplemental 400 ug is part of the recommended 500 ug, or not. It would be helpful if the chapter was completed with a comment on the need of supplemental folic acid before and during pregnancy, according to the conclusions in the NNR SR.</p>	<p>These have not always been defined, and recommendations often differ between studies. Adequate intake was defined as “according to the -recommendations”.</p> <p>The intake recommendation of folate during pregnancy and lactation is 500 µg per day. This is clearly stated in the beginning of the chapter.</p> <p>Issuing recommendations regarding supplementation is a management issue and as such not a task included in the NNR revision.</p>

Organisation /Company	Folate General and detailed comments	WG observations and comments
University of Oslo, Norway	<p>General comments: Thank you for the opportunity to comment. I suggest that you consider to make some references to our study of folate status in Norwegian infants and toddlers. See detailed comments/suggestions below. Hay et al. Folate and cobalamin status in relation to breastfeeding and weaning in healthy infants. Am J Clin Nutr 2008;88:105-14. Hay et al. Maternal folate and cobalamin status predicts vitamin status in newborns and 6-mo-old infants. J Nutr. 2010;140(3):557-64. Hay et al. Folate and cobalamin status in relation to diet in healthy 2-y-old children. Am J Clin Nutr 2011;93:727-35. Yours sincerely Gry Hay Department of Nutrition University of Oslo gry.hay@medisin.uio.no Phone +47 41 50 50 41</p> <p>Detailed comments: Line 91. In an unfortified toddler population in the Nordic setting, serum folate was positively associated with intakes of fruit and berries and grain products (Hay et al. Am J Clin Nutr 2011; 93:727-35). Line 130 or line 288: In a Norwegian study, maternal serum folate in pregnancy predicted infant serum folate at birth, which, in turn was positively associated with serum folate at 6 mo. (Hay et al. J Nutr. 2010;140(3):557-64). Line 300: At age 6 mo, serum folate was positively correlated with duration of exclusive breastfeeding, in a group of Norwegian infants (Hay et al 2008; 88:105-14). Line 302. The folate content in breastmilk is relatively unaffected by maternal intake or status, including supplementation (US women) (Allen 2012; Adv Nutr;3(3):362-9). Results from a Norwegian study supports the view that exclusive breastfeeding for 6 mo will maintain sufficient folate status (Hay et al. 2008;88:105-14). Line 308. In NNR 2004 the recommended intake in children 2-5 y was 80 ug/d. In a study of Norwegian toddlers, only 6% had low serum folate (<10nmol/L), in spite of a relatively low dietary folate intake; median (Q1-Q3) = 87 ug/d (74-106) ug folate/d. The serum folate concentration, even in the lowest quartile intake category, was sufficient to maintain low tHcy concentrations. (Hay et al. Am J Clin Nutr 2011;93:727-35). References: Hay et al. Folate and cobalamin status in relation to breastfeeding and weaning in healthy infants. Am J Clin Nutr 2008;88:105-14. Hay et al. Maternal folate and cobalamin status predicts vitamin status in newborns and 6-mo-old infants. J Nutr. 2010;140(3):557-64. Hay et al. Folate and cobalamin status in relation to diet in healthy 2-y-old children. Am J Clin Nutr 2011;93:727-35. Line 158. Smith AD et al. Is folic acid good for everyone? Am J Clin Nutr 2008;87(3):517-33. Line 158. Refsum H & Smith AD. Are we ready for mandatory fortification with vitamin B12. Am J Clin Nutr 2008;88(2):253-4. Line 302. Allen L. B vitamins in breast milk: relative importance of maternal status and intake, and effects on infant status and function. Adv Nutr 2012;3(3):362-9.</p>	The literature search identified two articles by Hay et al. However, these did not address the specific research question defined by the SR, and could therefore not be included.

Organisation /Company	Folate General and detailed comments	WG observations and comments
SLU, Sweden	föreslår strykning av raderna 179-182, dvs Ulrika Ericsson studier på polymorfism och cancer.	The text has been edited as suggested.

NNR 2012 Public consultation – Chapter 24 (Vitamin B12)

Organisation/ Company	General and detailed comments	WG responses and actions
University of Bergen, Norway	<p>In general the NNR 2012 on vitamin B12 is well written. Here are some comments and suggestions (see detailed comments below):</p> <p>Detailed comments: In the section on dietary sources and intake, it could be worthy to add a sentence to clarify that only bacteria can synthesise vitamin B12: “Vitamin B12 is synthesised in bacteria [and Archea] (Raux et al.), and are obtained by herbivorous animals through rumination or coprophagia.” It could also be important to add that enriched plant milk products to the list of sources, as enriched plant milk in Nordic countries usually have equivalent concentration of B12 compared to cow's milk and as these are important sources of B12 for vegetarians. Thus, the following sentence: “Plant milk (e.g. soy milk, oat milk and rice milk) enriched B12 are also an important source of vitamin B12 in vegetarians. “ could be added after the sentence “Liver, meat, fish, shellfish, milk and cheese are particularly good sources.”</p> <p>As none of the participants in reference 13 (Rauma et al.) used any vitamin B12 supplementation, the sentence “Deficiency caused by inadequate dietary intake is only observed in adults who have been eating vegan diets for many years” could benefit from adding “without taking vitamin B12 supplements or including products enriched with vitamin B12.”. References: Raux E, Schubert HL, Warren MJ: Biosynthesis of cobalamin (vitamin B12): a bacterial conundrum. Cell Mol Life Sci 2000, 57(13-14):1880-1893 Ruth Lawson. The Gut and Digestion. Anatomy and Physiology of Animals. Wikibooks 2011. http://en.wikibooks.org/wiki/Anatomy_and_Physiology_of_Animals/The_Gut_and_Digestion Content of soy milk, Alpro used as an example: http://www.alpro.com/uk/soya-plain Rauma A-L, Törrönen R, Hänninen O, Mykkänen H. Vitamin B-12 Status of Long-Term Adherents of a Strict Uncooked Vegan Diet (“Living Food Diet”) Is Compromised. J Nutr 1995;125:2511-5. http://www.ncbi.nlm.nih.gov/pubmed/7562085</p>	<p>Some plant milk products (e.g. soy milk, oat milk and rice milk) may be enriched with B12 and thereby also an important source of vitamin B12 in vegetarians</p> <p>Added in the text</p>
Danish Cancer Society	<p>General comments: not yet implemented changes in references, make the chapter appear less integrated</p> <p>Detailed comments: line 40 changes i reference line 139 misspelling of the word associated line 204, 216-222, 227-229, 235-236, 239-240 and 250 and onwards changes in references and new ones.</p>	<p>References reorganized in final version</p> <p>Corrected See above</p>

Organisation/ Company	General and detailed comments	WG responses and actions
The Norwegian Vegan Society /Norsk vegansamfunn	<p>Detailed comments:</p> <p>1 Section in question This commentary is about section “3. Dietary sources and intake”, and more specifically this line: 25 ... Seaweed provides some biologically active vitamin B12 ... References to scientific journals and reports are provided throughout this text.</p> <p>2 Problem description The wording of line 25 is problematic, as it may lead some people to believe that they can improve their vitamin B12 status by using seaweeds. This can cause serious harm, as discussed in section 3 below. The mentioned line refers to a 1995 study by Rauma et. al. (your reference 13). There are some shortcomings to this study which makes it unsuited as the basis of a general statement like line 25. The study found that vegans who had eaten the seaweeds nori and/or chlorella had higher levels of vitamin B12 than those who did not. However, seaweeds have been shown in many studies to contain inactive vitamin B12 analogues [13] that can be mistaken for vitamin B12 in blood tests [14]. Moreover, these inactive vitamin B12 analogues might interfere with vitamin B12 metabolism [15]. A more accurate way to determine if a food can improve vitamin B12 status is to measure methylmalonic acid (MMA) levels before and after feeding. Methylmalonic acid increases in vitamin B12 deficiency, and it’s the only metabolite in humans that requires only vitamin B12 for its clearance (homocysteine can be reduced using vitamin B12, folate, or vitamin B6), and is the most sensitive indicator of vitamin B12 status [16]. Of the seaweeds, only nori has been tested to see if it lowers MMA levels and it failed to do so [12]. Also, the study is not taking known variations into consideration. - Vitamin B12-content in plants is known to vary depending on the local environment the plants grow in, and the plants do not synthesize the vitamin themselves [6]. To algae, the local presence of cobalt and bacteria producing biologically active vitamin B12 is necessary for the plant to contain the vitamin [7]. - In addition, the packaging, storage, transportation, and preparation methods of plants that originally contained vitamin B12 can differ and may affect the amount of biologically active vitamin B12. For example, a significant difference between raw and dried nori in this regard has been observed [5]. Finally, the study is small. There are only 9 test subjects in the relevant group.</p> <p>3 Status of the current literature and recommendations for B12-intake on a plant based diet According to a broad 2013 literature review by Norris et. al. [10], there are at this time no studies that show that a particular plant food, including Nori and Chlorella seaweed, obtained from multiple regions, consistently improve vitamin B12 status via the lowering of MMA-levels. Because of the potentially severe effects of vitamin B12 deficiency, especially in infants, books on plant-based nutrition [8, 9] recommend supplements and fortified foods as the only reliable source of the vitamin, as well as testing, to detect those few that have low absorption of the vitamin.</p> <p>4 Recommended changes Line 25 should be replaced. It should be clear that seaweeds and other plant foods are not considered reliable sources of vitamin B12 even though some plants contain varying levels of biologically active vitamin B12. 25 .. Large scale studies on vitamin B12 absorption from particular plant foods must be performed in order to determine if these can be reliable sources of the vitamin. Optionally, the line could be deleted.</p> <p>5 Sources and references 1. Watanabe, F., Yabuta, Y., Tanioka, Y., & Bito, T. (2013). Biologically Active</p>	Text revised in line with comment.

Organisation/ Company	General and detailed comments	WG responses and actions
	<p>Vitamin B12 Compounds in Foods for Preventing Deficiency among Vegetarians and Elderly Subjects: a Review. Journal of Agricultural and Food Chemistry. 2. Van den Berg, H., Daqnelie, P. C., & Van Staveren, W. A. (1988). Vitamin B12 and seaweed. Lancet, 1(8579), 242. 3. Carmel, R. A. L. P. H., Karnaze, D. S., & Weiner, J. M. (1988). Neurologic abnormalities in cobalamin deficiency are associated with higher cobalamin “analogue” values than are hematologic abnormalities. J Lab Clin Med, 111(1), 57-62. 4. Oh, R., & Brown, D. L. (2003). Vitamin B12 deficiency. American family physician, 67(5), 979-986. 5. Yamada, K., Yamada, Y., Fukuda, M., & Yamada, S. (1999). Bioavailability of dried asakusanori (Porphyra tenera) as a source of cobalamin (vitamin B12). International journal for vitamin and nutrition research, 69(6), 412-418. 6. Warren, M. J., Raux, E., Schubert, H. L., & Escalante-Semerena, J. C. (2002). The biosynthesis of adenosylcobalamin (vitamin B12). Natural product reports, 19(4), 390-412. 7. Croft, M. T., Lawrence, A. D., Raux-Deery, E., Warren, M. J., & Smith, A. G. (2005). Algae acquire vitamin B12 through a symbiotic relationship with bacteria. Nature, 438(7064), 90-93. 8. Davis, Brenda & Melina, Vesanto (2000). Becoming Vegan: The Complete Guide to Adopting a Healthy Plant-based Diet. Book Publishing Company (TN). - Brenda Davis is a Registered Dietitian, Nutritionist and past chair of the Vegetarian Nutrition Dietetic Practice Group of the American Dietetic Association - Vesanto Melina has a Master of Science degree in Nutrition and is a Registered Dietitian 9. Norris, Jack & Messina, Virginia (2011). Vegan for Life. Lifelong publishing. - Virginia Messina has a Master’s degree in public health, with a focus on nutrition and is a Registered Dietitian - Jack Norris is a Registered Dietitian with a focus on plant based nutrition 10. R.D. Norris, Jack (2013). B12 in Vegan Foods. Vegan Health, a project of Vegan Outreach. Published at http://veganhealth.org/b12/plant</p>	
LRF Mjölkdairy Sweden	<p>Detailed comments.</p> <p>Line 25: Please insert an updated and correct evaluation of the vitamin B12 activity from seaweed. A recently published reference is: “Biologically Active Vitamin B12 Compounds in Foods for Preventing Deficiency among Vegetarians and Elderly Subjects. Watanabe F et al. J Agric Food Chem. 2013;61(28):6769-6775.</p> <p>Line 26: Reference 13 is rather old. Is it still valid? If not, please include more recent information.</p>	<p>Reference added. However, the adequacy of seaweed as a vitamin B12 source is uncertain</p> <p>There are no recent relevant Nordic studies</p>

NNR 2012: Public consultation – Chapter 25 (Biotin)

Organisation / Company	Biotin General & detailed comments	WG observations and actions
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LRF Mjöl, Sweden	Detailed comments Lines 17-20: According to the text “liver, kidney, egg yolk, rolled oats and wheat bran are as rich sources”. However 70% of intake is said to come from bread, cereal products, dairy products and eggs. Should not the sources that stands for 70% of the intake not be mentioned as “rich sources”? Line 18: If there are figures for intakes in the other Nordic countries this should preferably be mentioned here.	As there are few data on which food groups contribute to dietary intake, some individual foods are mentioned
National Food Agency, Sweden	General comments Add names in B series for all B vitamins i.e. B1---B12 Detailed comments 18 Only Danish adults are included and the reference is quite old. Is there more recent literature on intakes in Nordic countries?	Not necessary here No intake data from other Nordic countries available.

NNR 2012: Public consultation – Chapter 26 (Pantothenic acid)

Organisation/ Company	Pantothenic acid General and detailed comments	WG observations and actions
Selskab for Ernæringsforskning, Denmark	Line 26: replace “availability” with “bioavailability”	Corrected

NNR 2012: Public consultation – Chapter 27 (Vitamin C)

Organisation/ Company	Comments	Response(s) to comments
Helsedirektoratet, Norway	18-19. It is not so clear what the intention of these lines is. 24-28. Should be rephrased. This is better explained on lines 85-89. 71. You may add information about levels of normal and/or optimal plasma levels of vitamin C.	The relationships between plasma AA and health is explained in the paragraph on lines 43—54.

Organisation/ Company	Comments	Response(s) to comments
Linus Pauling Institute, USA	The vitamin C article on your Web site is well written and provides a balanced view of the evidence. It is overall on the conservative side, but scientifically defensible. My conclusion is the 200 mg/d is reasonable as it will maximize vitamin C body pool and hence antioxidant capacity, and the (potential) benefit to risk ratio is very large. I am attaching the article FYI. You may want to cite it in your vitamin C article: Balz Frei, Ines Birlouez-Aragon & Jens Lykkesfeldt (2012): Authors' Perspective: What is the Optimum Intake of Vitamin C in Humans?, Critical Reviews in Food Science and Nutrition, 52:9, 815-829 Sincerely, Balz Frei, Ph.D. Director and Endowed Chair Distinguished Professor of Biochemistry and Biophysics Linus Pauling Institute Oregon State University 307 Linus Pauling Science Center Corvallis, OR 97331 phone 541 737-5078 fax 541 737-5077 email balz.frei@oregonstate.edu http://lpi.oregonstate.edu	Thank you for the interesting comment. We share your view on the approach to vitamin C recommendations (referring to your paper in Crit Rev Food Sci Nutr). However, we still maintain our view that the plasma levels compatible with prevention of particularly cardiovascular diseases are slightly lower than you suggested. We admit that higher levels than those observed in the 30 th percentile are in some studies related to still decreased morbidity. The evidence was, however, not considered convincing enough and therefore we maintain our present recommendation of 75 mg/day.
Sykehuset Innlandet Kongsvinger, Norway	The recommended intake of vitamin C should always be higher (with some margin) than that required to reach the threshold for no renal reabsorption, because the existence of an energy costing apparatus for reabsorption implies an evolutionary advantage for levels higher than that.	We based the recommendations mainly on the relationship between plasma AA and chronic diseases (cardiovascular diseases, in particular). This approach was used also in NNR4 and the update did not show any clear reasons to increase the recommendations.
Institute for Orthomolecular Medicine, Denmark	Vitamin C mg/d Recommended intake RI 300 Average requirement AR 90 Lower level of intake LI 20 Upper level of intake UL -	It is unfortunately difficult to give a response to this comment, since the reasons for these recommendations (e.g. RI 300 mg) are not clarified. Therefore, we maintain our view that by starting from plasma AA and morbidity, and calculating from these data to estimated dietary intakes, there are no clear indications for changing the present recommendation for vitamin C.

Organisation/ Company	Comments	Response(s) to comments
University of Helsinki, Finland	<p>I was a reviewer for the NNR chapter on vitamin C. I commented the draft and the author properly took into account my comments. I will not get back to those previous comments. However, now I read the NNR Chapter 3: Nutrition recommendations – background and principles. I realized that the NNR chapter on vitamin C is inconsistent with NNR Chapter 3. NNR Chapter 3, page 3, paragraph at the bottom states: “The recommendations are intended for healthy individuals. Generally, the recommendations cover increased requirements for example during short-term mild infections” The current vitamin C chapter is not consistent with the second sentence of the above excerpt. I do not remember whether I was, or was not, instructed to read Chapter 3 as relevant background for the review job, but I did not read it. I had assumed that the recommendations are intended for “healthy individuals” as the first sentence states. However, the second sentence claims that the recommendations cover also short-term mild infections, which is not the same as restricting to “healthy people”. When people are sick, then they are sick and they are not healthy individuals. In the case of vitamin C, those two sentences make a big difference. The common cold is the most common “short-term mild infection” and there are a few dozen RCTs on vitamin C for the common cold. Vitamin C does not have prophylactic effects against the common cold in the ordinary population, which is properly stated in the vitamin C chapter. Thus, on the basis of numerous RCTs, regular vitamin C doses above 75 mg/day are not useful for “healthy individuals” for the purpose of preventing short-term mild infections. However, the second sentence claims that the recommended intake will also “cover increased requirements during short-term mild infections”, but that is not true with 75 mg/day of vitamin C. First, RCTs: Regular high dose vitamin C shortens the duration and alleviates the symptoms of the common cold, which is the most common short-term mild infection. In our Cochrane review, we calculated that regular (each day over the trial) ≥ 0.2 g/day vitamin C administration shortened the colds of adults by 7.7% (95% CI: 3% to 13%, $P = 0.00018$) and of children by 13.2% (95% CI: 5% to 22%, $P = 0.00025$). http://www.ncbi.nlm.nih.gov/pubmed/17636648 We used inclusion limit of 0.2 g/day, but most of the trials administered 1 g/day vitamin C. Thus, the above figures mainly measure the effect of regular 1 g/day vitamin C supplementation. Most of the trials did not examine the initial dietary vitamin C intake but there is no basis to assume that the dietary intake of the ordinary people was much lower than 75 mg/day, so that an increase from the assumed low level to 75 mg/day level could explain the observed benefit. Some RCTs examined plasma vitamin C levels and they were not low.</p>	<p>Thank you for these comments. We will add the main conclusions from the Cochrane review in the chapter. However, these results do not give a reason to change the recommendations. We admit that the definition of a healthy individual in chapter 3 has not been written very well. This will be changed to clarify that the NNR’s are meant for individuals with no physiological needs for changed requirements of nutrients. As we see it, there are no reasons to believe that the effects of vitamin C on the duration of common cold would be caused by acute changes in vitamin C status (e.g. decrease in body pool).</p>

Organisation/ Company	Comments	Response(s) to comments
Dept. of Medical Biochemistry, St. Olav Hospital, Trondheim, Norway	line 224 Upper intake levels and toxicity. Vitamin C increases intestinal iron absorption. Therefore, it should be considered to add a sentence following the end of line 228, e.g. "Since vitamin C increases intestinal absorption of inorganic iron, it may also be prudent to advice a limit of 500 mg/d of supplemental vitamin C in individuals with hemochromatosis." Reference JC Barton, SM McDonnell, PC Adams, P Brissot, L Powell, CQ Edwards, JD Cook, KK Kowdley and the Hemochromatosis Management Working Group. "Management of hemochromatosis", Ch. 31, pp. 329-39 in Hemochromatosis: Genetics, pathophysiology, diagnosis and treatment. James C Barton, Corwin Q Edwards (eds.) Cambridge Univeristy Press, 2000.	This is in principle a highly relevant comment. However, we decided not to include hemochromatosis in the text, since we feel this is too detailed information on specific diseases.
Riksförbundet Hälsofrämjandet, Sweden	Line 226, "High intake may cause diarrhoea and other gastrointestinal disturbances." There are forms of vitamin C sold that are bound to Magnesium or Calcium as Magnesium ascorbate or Calcium ascorbate. In clinical use these forms have been found to cause less symtoms from the gastrointestinal tract. This aspect could be considered regarding available studies.	Thank you for the clarification. However, we feel that the word "may" in the text covers supplements causing fewer symptoms. It would be too detailed to describe potential effects of different types of supplements.
Danish Food and Drink Federation, Denmark	Line 177 and Line 220 – please insert references that relied on the same data	Lines 214—222 are a summary and therefore we have left it without references.
Funtionaalisen lääketieteen yhdistys ry, Finland	Funktionaalisen lääketieteen yhdistys ry Functional Medicine Society in Finland www.fms.fi info.fms.fi Nordic Council of Ministers For health and Social Affairs Nordic Nutrition Recommendations (NNR) 23.11.2012 Dear Sirs, We suggest the following changes in the NNR recommendations : Dietary antioxidants Suggestion to recommendations Intake of supplemental antioxidants does not seem to be justified for a healthy population in large. Specified recommendations are given separately for the individual antioxidants according to EU commissions regulation	Thank you for these interesting comments. We admit that the definition of a healthy individual in chapter 3 has not been written very well. This will be changed to clarify that the NNR's are meant for individuals with no physiological needs for changed requirements of nutrients. We

Organisation/ Company	Comments	Response(s) to comments
	<p>432/2012. The recommendation is based on limited numbers of studies s 5. For example the WHO Monica study has since 1985 generated a great number of studies related to cardiovascular diseases. Some new follow-up studies inspired by the WHO Monica project have showed the benefit of food supplements (for example in preventing oxidative stress related diseases). This report, follow-up studies and adjacent studies have been neglected. All studies should include reports of the following studies. Newest published criticism of flawed results and their conclusions should be cited. It seems that different methodological approaches of meta analysis may yield contradictory results (Gerss et al 2009). In large food supplement population studies, the results are compiled and reported based on individual self-reports and limited laboratory determinations. It is a well-known fact that self-medication can have an impact on the final reporting and results. In the introduction it is stated that the NNR guidelines are given for healthy populations and should not be implicated on individual persons or individual intake of food supplements. This should be mentioned in every individual recommendation. Obviously the situation is totally different when it comes to different diseases. For that reason it should be underlined that these recommendations are given for a healthy Scandinavian large population with no diagnosed underlying diseases and their medications. Rows 293-299: Reasoning behind the recommendation is over simplification of conclusions ignoring huge amount of contradictory scientifically proved facts in specified populations. For antioxidants such as vitamin C, vitamin E and beta-carotene, EU has stipulated regulations which should be uniform and evaluated in all dietary recommendations. Vitamin E Rows 264-267: A more recent paper shows that different methodological approaches of meta analysis yield contradictory results (Gerss et al 2009). NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations includes only healthy large populations not individuals. Vitamin C NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations include only healthy large populations not individuals. Selenium NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations include only healthy large populations not individuals. Gerss, J., Köpcke, W. The questionable association of vitamin E</p>	<p>feel this should be enough and that the definition of what is meant by a healthy individual does not need to be repeated in every chapter.</p>

Organisation/ Company	Comments	Response(s) to comments
	supplementation and mortality--inconsistent results of different meta-analytic approaches. Cell Mol Biol (Noisy-le-grand). 2009, 55 suppl: OL1111-1120.	

NNR 2012 Public consultation – Chapter 28 (Calcium)

Organisation Company	Comments	WG response & actions
University of East Anglia, UK	<p>General comments</p> <p>1. The report provides figures for Recommended Intake but no Average Requirements for different population groups. Can you explain the basis for setting RIs, given that there are no ARs. Are the RIs intended to be equivalent to an AI (adequate intake)?</p> <p>2. The summary table provides figures for children 2-5y, 6-9y, 10-13y and then women and men. What are the figures for children aged 14-18y? In the text there is reference to ages 10-20y having an RI of 900 mg/d (line 532). Also, are the RIs the same for elderly people? It would help if pregnant and lactating women were included in the summary table.</p> <p>3. On page 3, lines 104-106, it is stated that the varying degree of absorption makes it impossible to use figures for fractional absorption - these are required to calculate requirements using the factorial approach. On page 12, lines 505, 508 and page 13, line 527, reference is made to assumptions about absorption, which were used to derive NNR 2004 figures. Bone calcium accretion data are given and then an RI is given - how was this calculated? What assumptions were made about % absorption and were endogenous losses taken into account? It could be argued that where data exist for efficiency of absorption in children adapted to dietary intakes equivalent to recommended levels, these could be used to derive requirements since the composition of the diet is not an important variable when Nordic diets are being considered.</p> <p>4. On page 13, line 526, one of the references (26) to efficient calcium absorption during puberty refers to a study in which girls were given either 250 or 1500 mg calcium/d. The study was designed to examine acute adaptation to insufficient calcium supply and is not representative of the Nordic diet. Incidentally, ref 26 is incorrectly cited (O'Brien not O'Brian, and the title of the paper is wrongly cited).</p> <p>5. On page 13, line 528 onwards, an argument is presented about adverse effects of calcium on iron absorption, yet very many studies have clearly shown that the impact of calcium on iron absorption</p>	<p>AR is set for adults and for children.</p> <p>Has been updated in the final draft.</p> <p>The sentence has been modified in the final draft. In previous NNR editions various assumptions were made. The section on DRVs for children have been revised</p> <p>Section revised.</p> <p>Section revised, new references added.</p>

Organisation Company	Comments	WG response & actions
	<p>is short-term (from single meals) and is not supported in studies of a longer duration are undertaken or in epidemiological data. Therefore this is not a convincing argument to support the selected RI.</p> <p>Detailed comments</p> <p>1. Line 72. It would be helpful to mention the fact that it is the presence of oxalate that reduces calcium absorption, which explains why calcium in kale, a low oxalate green vegetable, is well absorbed.</p> <p>2. Page 12, line 509. Why is the study of Vatanparast et al classified as 'low-quality'?</p> <p>3. Page 12, lines 521 onwards, and page 2, lines 66 onwards. Since mean intakes are above RIs in Nordic countries, the argument for using a conservative approach to the RIs for children is relatively weak. Would it not be more appropriate to take a cautious approach and take into account the evidence that higher intakes of calcium are associated with small (but significant) increases in BMD? The arguments for adaptive changes in efficiency of absorption when calcium intakes are low are not totally convincing given the fact that intakes of calcium are much higher in the Nordic countries than in these old metabolic studies. The premise upon which RIs for children are derived are not transparent and they are unconvincing in the chapter as it stands. Pages 12-14 would greatly benefit from a major rewrite to explain more clearly how the figures were reached. In contrast, the description of the systematic review on calcium and health endpoints is well described, but this information is not used for setting requirements!</p>	<p>Details on factors affecting absorption have been moved to Physiology. QA was made by the experts based on the QA tools. Comment acknowledged. However, intakes vary widely among population groups nowadays. Intakes are more close to RI among older school children.</p> <p>Section on Req & rec revised.</p>
Närhälsan Skövde, Sweden	<p>General comments:</p> <p>Hi! I don't see that you have seen the calcium study on women in the Swedish mammography cohort, published in BMJ 2013. Unfortunately I can't see the reference on Medscape: http://www.medscape.com/viewarticle/779600_1, but you can get it from one of the authors here: karl.michaelsson@surgsci.uu.se. And in Stroke, published online febr 28 2013, there were another study: http://www.medscape.com/viewarticle/780125</p>	<p>The SR covers papers published up to December 2011. The WG has evaluated several papers published until spring 2013. The study by Michaëlsson et al. showed that the lowest mortality occurred in the category with a median intake of 840 mg/d, also used as the reference category. This is close to the suggested RI. Regarding UL, it is based</p>

Organisation Company	Comments	WG response & actions
		on the EFSA opinion 2012.
Närhälsan Skövde, Sweden	General comments: If you don't take into consideration the new studies referred to previously - Karl Michaelssen et al for instance - I get really, really disappointed and feel that the coming recommendations are already old, when they come out.	See response above.
Danish Cancer Society	General comments Overall a fine chapter - my comments are primarily detailed comments with regards to grammar and wording. Detailed comments line 57: comma after "bone" and "found" after "exclusively" line 75: comma after "intestine" line 108: Do you mean 1000 mg/day? line 124-126: I do not understand the meaning of this sentence - could you clarify? Both gramatically and with regards to content I think there is something rong, but I can't figure out, what it is you mean to say. line 145: "of" after "doses" line 164-165: You postulate that adequate vitamin D is a crucial factor when assessing the role of calcium intake, and I agree with this, but maybe you could briefly expand on it for readers not knowing about the association between these two micronutrients line 179: the sentence should read: between calcium intake during pregnancy and bone health in the offspring line 190-: This is a repetition of the very first sentence line 560: you lack a "with" after "associated"	Corrected. Corrected. Corrected. A comment is added. Corrected. OK, but retained. Paragraph has been revised

Organisation Company	Comments	WG response & actions
LRF Mjolk/LRF Dairy Sweden	<p>General comments:</p> <p>This chapter should focus on DIETARY calcium. Calcium supplements could however be mentioned, but be as a central part as it is now. Effects on calcium from diet and supplements must be separated in this chapter. Now the sources of calcium are mixed throughout the text. Please include the health outcomes for persons not having dairy products. Why is the text on calcium in relation to prostate cancer not identical between this chapter and the systematic review? Check the correct wording in the report from WCRF/AICR 2007, page 129.</p> <p>Detailed comments:</p> <p>Page 2, line 65. Include references of the dietary intake of calcium.</p> <p>Page 2, line 65. Include the results from Riksmaten 2010-11.</p> <p>Page 2, line 72. Include reference of the absorption of calcium from vegetables.</p> <p>Page 9, line369. Include the conclusions form the report from WCRF/AICR 2007.</p> <p>Page 9, line 387. Use the exact wording from the report from WCRF/AICR 2007 on the association between calcium and prostate cancer.</p> <p>Page 13, line529. The references are often very old. Update the chapter with current papers. If there are no more recent papers, state that. Regarding calcium inhibiting the absorption of iron an expert meeting in collaboration with Swedish Dairy Association and Swedish Nutrition Foundation was hold in 2006.Experts participation was e.g. Lena Hultén, Nils-Georg Asp and Bo Lönnerdal participated in the meeting. For information: http://www.svenskmjolk.se/Mjolk-smor-och-ost/Naring/Vitaminer-och-mineraler/Mjolk-fungerar-med-jarnrik-maltid--rapport-fran-forskarmote/#Oförändrad_järnstatus_av_högt_kalciumintag The conclusion from the meeting was that a high intake of dairy products didn't affects the iron intake in the long run. This conclusion was also informed on in Scandinavian Journal on Food and Nutrition 2006, 50, 1. Calcium does not impair iron status in practice. Include the text of the chapter on iron that in the long term there is no importance of the inhibition of calcium on the iron intake. Further, the Swedish National Food Administration write specifically in their dietary advices for pre school (Råd för mat i Förskolan) that there are no longer any scientific evidence that milk inhibits iron absorption.</p>	<p>Acknowledged. However, the focus is on health effects of calcium, thus studies using supplemental calcium are included. There are also studies on effects of dietary sources, e.g. epidemiological studies.</p> <p>Section on sources and intake updated.</p> <p>Section on Physiology revised.</p> <p>The SR by Chung is cited as the most recent</p> <p>Revised.</p>

Organisation Company	Comments	WG response & actions
The Vegan Society (Norway) and The Norwegian Vegetarian Society	<p>General comments</p> <p>Regarding "3. Dietary sources and intake". We believe it is important to mention more non-dairy sources of calcium. This information applies to everyone allergic/intolerant for casein or lactose or that for health, environmental or other reasons consume little or no dairy products. More details follow in the "Detailed comments" section.</p>	Examples added
	<p>Detailed comments</p> <p>P. 3. Dietary sources and intake", lines number 71-72. We believe it is important to mention more non-dairy sources of calcium. This information applies to everyone allergic/intolerant for casein or lactose or that for health, environmental or other reasons consume little or no dairy products. In particular, calcium-enriched plant milks, e.g. soy- and oatmilk, should be mentioned as a good source of calcium. Calcium enriched soy- and oatmilk products have become easily available over the last few years, at least in the cities, and most of them *by design* have the exact same calcium content as cow's milk. The calcium absorption rate for soymilk is equivalent to that of cow's milk [3, 7] . Current content: 71 Green vegetables have a variable content of calcium which is mostly not very well 72 absorbed – with a few exceptions such as kale. Our suggestion: 71 Plant milks enriched with calcium, for example soymilk and oatmilk, commonly contain 72 calcium levels equal to that of cow's milk. 73 Vegetables high in calcium include kale, mustard greens, bok choy and turnip greens. 74 Note that the calcium absorption from most other green vegetables, including spinach and rhubarb, is low because of their high oxalate contents. 75 In addition to these high-calcium foods, many plant foods, including almonds, sesame seeds with shell, navy beans and refried beans, provide some calcium.</p> <p>Sources and references: The amount of calcium found in different foods is listed in the Norwegian food composition table [1], as well as the larger USDA Nutrient database [2]. A table of plant based sources of calcium, based on [2, 3, 4, 5, 7, 8], with their calcium and oxalate contents can be found in [6]. Line 75 is based based on [4], while lines 73-74 are based on [5, 8, 9]. [1] The Norwegian Directorate of Health, the Norwegian Institute of Public Health & al. 2013. The Norwegian Food Composition Table. http://www.matvaretabellen.no [2] U.S. Department of Agriculture, Agricultural Research Service. 2012. USDA National Nutrient Database for Standard Reference, Release 25. Nutrient Data Laboratory. Home Page, http://www.ars.usda.gov/ba/bhnrc/ndl [3] Zhao Y, Martin BR, Weaver CM. Calcium bioavailability of calcium carbonate fortified soymilk is equivalent to cow's milk in young women. J Nutr. 2005 Oct;135(10):2379-82. http://www.ncbi.nlm.nih.gov/pubmed/16177199 [4] Weaver CM, Plawecki KL. Dietary calcium: adequacy of a vegetarian diet. Am J Clin Nutr. 1994 May;59(5 Suppl):1238S-1241S. http://www.ncbi.nlm.nih.gov/pubmed/8172128 [5] Weaver CM, Heaney RP, Nickel KP, Packard PI. Calcium Bioavailability from High Oxalate Vegetables: Chinese Vegetables, Sweet Potatoes and Rhubarb. Journal of Food Science. 1997 May;62(3):524-5.</p>	Some examples added

Organisation Company	Comments	WG response & actions
	<p>http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2621.1997.tb04421.x/abstract [6] Calcium Absorption from Plant Foods, Jack Norris. 2013.</p> <p>http://www.veganhealth.org/articles/bones#caabsor [7] Tang AL, Walker KZ, Wilcox G, Strauss BJ, Ashton JF, Stojanovska L. Calcium absorption in Australian osteopenic post-menopausal women: an acute comparative study of fortified soymilk to cows' milk. Asia Pac J Clin Nutr. 2010;19(2):243-9.</p> <p>http://www.ncbi.nlm.nih.gov/pubmed/20460239 [8] Heaney RP, Weaver CM. Calcium absorption from kale. Am J Clin Nutr. 1990 Apr;51(4):656-7. http://www.ncbi.nlm.nih.gov/pubmed/2321572 [9] Heaney RP, Weaver CM, Recker RR. Calcium absorbability from spinach. Am J Clin Nutr. 1988 Apr;47(4):707-9. http://www.ncbi.nlm.nih.gov/pubmed/3354496</p>	
University of Bergen, Norway	<p>General comments:</p> <p>In general the NNR 2012 on calcium is well written. In the section on dietary sources and intake, it could be worthy to add plant milk products to the list of sources, as these usually have equivalent calcium concentration compared to cow's milk (as they are usually supplemented with calcium). The first sentence could then read "Milk, milk products in addition to plant milk products (e.g. supplemented soy milk, oat milk and rice milk) are good sources of calcium."</p>	Examples added
European lifelong learning program "Eating Together"	<p>General comments</p> <p>I write on behalf of the Lifelong learning program n° 2011-1-BE3-GRU06-03638 (http://www.dietethics.eu/en/nutrition/national-guidelines.php). The Danish Animal Alliance takes part in this European group, collecting official dietary guidelines for vegan diets around the world to help to protect people who choose to live on vegan diets. The Nordic Nutrition Recommendations are very interesting. We have requested an in depth study of your very good drafts and previous editions by Stephen Walsh (coordinator of the International Vegetarian Union's science committee). His suggestions on the Calcium chapter focuses from line 64 onwards, to suggest several seemingly omitted good calcium sources for vegan diets, as well as a more positive evaluation of its absorption through common green vegetables.</p> <p>Detailed comments</p> <p>Dietary sources and intake (starting line 64) The statement on green vegetables is unduly negative as the majority of common green vegetables have well absorbed calcium (http://ajcn.nutrition.org/content/70/3/543s.full.pdf+html). The main exception is spinach. For people who follow a plant based diet, good sources of calcium include fortified plant milk or juice, tofu (prepared with calcium-sulfate) and low-oxalate green vegetables.</p>	<p>The WG acknowledge the comments and suggestions. The section on dietary sources and intake will be amended with some more examples.</p> <p>Many of the issues will be covered in the chapter on vegetarian diets</p>

Organisation Company	Comments	WG response & actions
National Institute for Health and Welfare (THL), Finland	<p>General comments:</p> <p>Comments to the chapters of Iron, Vitamin D, Calcium and Phosphorus.</p> <p>GENERAL: In all chapters concerning the micronutrients and vitamins there should first be the table of what is the new recommendation for different age groups and pregnant/lactating. If there are changes from the previous recommendation, then this should be in a table and reasoning for the change. Right after the table, the paragraph should begin with the chapter 'Reasoning behind the recommendation'. The chapters should be in following order: NUTRIENT X Table 1 : new recommendation for the nutrient X Table 2 : upper intake level and toxicity for the nutrient x Reasoning behind the recommendation Physiology and metabolism (briefly, referring) Requirements and recommended intakes for: - infants - children and adolescents - adults - pregnancy and lactation - older adults and elderly - special needs when relevant (cancer, DM, CVD, Obesity, etc). Also these chapters could be more focused and a lot shorter, not including all the facts like a teaching manual, but referring to the other actual texts like the IOM recommendations. All the comment rounds have included very incoherent chapters, as if the writers wouldn't have the same writing guidelines and wouldn't write into the same book. Various age groups should be named in the same way in all chapters. There should be an own chapter about supplementation; why different practices in different countries, where do these come from, etc. For instance where comes the new age limit in vitamin D recommendation? Why did it change? If countries don't agree, then it is important for experts to know why and be able to rationalize himself.</p>	<p>WG acknowledge the suggestions. Some restructuring will be made in the final version.</p> <p>The chapters include short summaries and conclusions from the SRs, where applicable. This is needed to give the scientific background to the recommendations.</p> <p>The WG does not consider the chapters unduly lengthy and detailed. Cf e.g. reports by IoM and WCRF.</p> <p>Regarding supplementation this is covered in Ch. "Use of NNR"</p>
Norwegian Directorate of Health	<p>Detailed comments:</p> <p>Line 65-7. This sentence may be improved. What do you mean with " , of the order of". The NNR recommendations are below 900 mg/d.</p> <p>Line 71-72. Is it possible to attain calcium intakes at recommended levels by a vegetarian diet? You may add information about this question. This may also be discussed in the chapter concerning vegetarian diets.</p> <p>Line 76-77. If it is possible please give information concerning the relative significance of passive diffusion and active transport of calcium during absorption?</p> <p>Line 386-7. If possible please add your grading of the evidence concerning calcium and prostate cancer in this sentence.</p> <p>Line 513-4. This sentence is incomplete, and better phrased in other sections e.g. 617-8 and 620-1.</p> <p>Line 619-21. Reasoning behind the recommendation. You may add a short discussion or comments concerning differences regarding the recommendations made by NNR and other expert groups as DRI by IoM, USA. You may also in this section integrate text from the SLR, Authors conclusions page 40-1.</p>	<p>Section on dietary sources revised.</p> <p>No exact data available.</p> <p>Evidence is limited – suggestive Amended. Added in final version</p>

Organisation Company	Comments	WG response & actions
The Norwegian Dairy Council	<p>General comments</p> <p>General comment on health effects of calcium. When talking about the potential health effects of calcium, it does not clearly state if the health effects are related to calcium intake from dietary sources or from supplements in addition to the dietary sources. This should be specified in this section when referring to the studies, as the calcium intake when using dietary supplements may be slightly higher than recommended. General comment on recommended intake and mean intake in children and adolescents It would be reasonable to mention the mean daily intake in different age groups, in a similar manner as when talking in general for women and men (mean intake for men and women). Data from the Norwegian dietary survey Ungkost shows that 70 % of the 13 years old girls do not meet the recommended intake of calcium. The mean intake for boys and girls in different age groups should be shown. Possible challenges Although the mean intake of calcium is according to the recommendations, it is important to specify and point out the groups of the population that may have challenges in meeting the recommended daily intake of calcium. Typically, it is at the crucial period of maximal skeletal accretion that adolescents, girls especially, decrease their intake of dairy products and replace milk with for instance soft drinks. Nonetheless, subgroups of individuals, particularly those with poor nutrition, should be mentioned. References: Norkost 3. En landsomfattende kostholdsundersøkelse blant menn og kvinner i Norge i alderen 18 – 70 år, 2010 – 11. Weaver CM, Plawecki KL. Dietary calcium adequacy of a vegetarian diet. Am J Clin Nutr59: 1238S–1241S, 1994. Andersen og Øverby. Ungkost 2000. Landsomfattende kostholdsundersøkelse blant elever i 4.-og 8. klasse i Norge. Helsedirektoratet, 2002.</p> <p>Detailed comments</p> <p>Dietary sources and intake Page 2, line 64, regarding dietary sources and intake: Calcium is classically associated with dairy products; milk, yoghurt and cheese are good sources of calcium, providing the major share of calcium from foods in the general diet. When talking about dietary sources of calcium, it should be referred to milk and dairy's contribution to the intake of calcium in the diet, not to that the consumption of milk and milk products is high in the Nordic countries. For instance dairy foods, including milk, provide 67 % of the calcium in the Norwegian diet. The majority of the remaining calcium in the diet comes from bread (6 %), vegetables (4%), fruit and berries (4 %) and meat and meat products (3 %). These dietary data from Norway shows that certain vegetables and grains provide calcium, but the quantities required to replace the amount of calcium from milk and dairy practically limit the role of these foods in achieving adequate calcium intake. If intake of dairy products is limited or avoided, achieving adequate calcium intake requires regular use of fortified foods and supplements.</p> <p>Line 67: The mean intake of calcium for Norwegian women is 811 mg calcium per day, and the mean intake should therefore be in order of 800 – 1200 mg for women (not 900 – 1200 mg as it says in the draft proposal). As mentioned, there may be groups of people in which the mean intake</p>	<p>Health effects associated with Ca from foods and supplements are described more clearly, where possible. However, not all SRs or studies give sufficient detail.</p> <p>Amended in the final version.</p>

Organisation Company	Comments	WG response & actions
	<p>does not meet the recommendation. Dietary data from Norway shows that women in the age groups of 50–59 and 60–70 years have a mean intake of 784 and 768 mg of calcium respectively, which is below the recommended intake of 800 mg. It should also be specified whether the mean intake is referring to total intake (calcium from natural food sources and supplements) or only natural food sources. References: Norkost 3. En landsomfattende kostholdsundersøkelse blant menn og kvinner i Norge i alderen 18 – 70 år, 2010 – 11. Weaver CM, Plawewski KL. Dietary calcium adequacy of a vegetarian diet. Am J Clin Nutr 59: 1238S–1241S, 1994. Andersen og Øverby. Ungkost 2000. Landsomfattende kostholdsundersøkelse blant elever i 4.-og 8. klasse i Norge. Helsedirektoratet, 2002.</p>	
Minivent AB, Sweden	<p>General comments</p> <p>Introduction 42 Lines 50- 55 should be replaced with the following text. half is bound to albumin. Parathyroid hormone and 1,25(OH)2D are the most important hormones in the regulation of calcium homeostasis. They contribute to maintenance of constant calcium concentration in plasma by regulating the influx and efflux of calcium in intestine, bone and kidney. Maintenance of a constant concentration of ionised calcium is of vital importance and calcium homeostasis is probably the most tightly regulated homeostatic mechanism in the body</p> <p>Lines 77- 79 should be replaced with the following text. or by an active energy requiring process. The latter is dependent on the action of 1,25-dihydroxyvitamin D3 (1,25(OH)2D), the hormonal form of vitamin D. Calcium absorption is thus decreased in vitamin D deficiency. This means that we can not increase the uptake of calcium unless we have at least the physiological level of at least 125 nmol/L. See the Public consultation answer on Vitamin D3. The difference between dietary calcium and that</p> <p>Lines 100- 462 should be replaced with the following text. (Guéguen et al. 2000). The net calcium absorption in infants is reported to range from about 30 % to 60 %, and between 25-40 % in older children, depending on absolute intake (Abrams 2010). The net absorption is relatively high during puberty (found to be about 34 % on an intake of 925 mg/d (5)) and then declines to 25-20 % in adulthood and even lower at advanced age (Guéguen et al. 2000; Schaafsma 1997). This indicates that the amount of net absorption is about 300 mg per day independent of the oral intake. Calcium is lost from the body via faeces, urine and skin. Non-absorbed calcium is lost with faeces. In adults on intakes of about 1000 mg the loss amounts to about 70 to 80 % of the intake. This indicates that the amount of net absorption is about 300 mg per day independent of the oral intake. An appreciable amount is excreted via faeces as calcium soaps. Loss via skin and sweat is generally small, about 20-50 mg/d (6,7). Under warm conditions or high physical activity the loss may be appreciably greater. Loss via urine may vary appreciably from person to person, generally between 100 and 400 mg/d in adults, but is relatively constant within individuals even if the intake varies. In the balance study by Malm (1) the urinary loss decreased from 231 to 201 mg/d (not significant) upon reduction of the intake from 940 mg/d to 450 mg/d. This experiment shows that a decrease of</p>	<p>Identical text in draft.</p> <p>The stated requirement of >125 nmol (25OHD?) is not scientifically substantiated</p> <p>Not accepted</p>

Organisation Company	Comments	WG response & actions
	<p>calcium intake is independent of the intake as long as the intake is higher than the uptake/urinary excretion and that the level of vitamin D3 is above 125 nmol/L. The calcium balance is very tightly regulated as long as the intake exceeds urinary excretion. The intestinal uptake is limited to 100-400 mg per day and the blood level of calcium is very tightly regulated by a free urinary excretion and a regulated uptake from the primary urine, again with a little help from my friends vitamin D3 derivatives. So the intestines regulate the uptake of calcium and serum level is very tightly regulated by the kidneys as any excess is excreted.</p> <p>Lines 464- 618 should be replaced with the following text. Requirement and recommended intake The old fashioned physiological, biochemical and endocrinological knowledge shows us that the calcium intake, uptake and excretion is strictly regulated in the body. By eating more calcium than excreted in the urine and that the level of vitamin D3 is above the physiological level of 125 nmol/L the body will maintain calcium homeostasis. The minimum intake of 500 mg calcium applies to all ages and sexes and is then ample for most individuals as long as the level of vitamin D3 is above the physiological level of 125 nmol/L.</p> <p>Lines 619- 621 should be replaced with the following text. Reasoning behind the recommendation The recommendations for calcium are maintained and can be decreased in NNR 2012 since no strong scientific evidence to change has emerged. This is under the condition that the level of vitamin D3 is above the physiological level of 125 nmol/L.</p>	

NNR 2012: Public consultation – Chapter 29 (Phosphorus)

Organisation / Company	Phosphorus general & detailed comments	WG response and actions
Valio Ltd	<p>General comments only:</p> <p>144: Based on the calculations of the recommended dietary Ca and P intakes, the optimal dietary calcium-to-phosphorus molar ratio (Ca:P molar ratio) is suggested to be 1. Concerning foods in adult diets, only dairy products have Ca:P ratios equal or close to the suggested dietary Ca:P weight ratio of 1.3.</p> <p>146: milk is one of the biggest sources of phosphorus, but it doesn't digest so well than e.g. food additive phosphorus.</p>	Acknowledged

NNR 2012: Public consultation – Chapter 30 (Magnesium)

Organisation/ Company	Magnesium General and detailed comments	WG responses and actions
SNF Swedish Nutrition Foundation	<p>Detailed comments</p> <p>Line numbers: 46-49 “The physiological manifestations of severe magnesium depletion are the following: hypokalaemia and hypercalcaemia, neuromuscular hyperexcitability, electrocardiographic abnormalities and cardiac arrhythmias.” The wording is kept unchanged from NNR 2004 (except for the word following, which has been corrected from “followings” in NNR 2004). The clinical presentation in this condition, which is seen in e.g. intestinal failure patients with large intestinal losses, rather includes hypocalcaemia, due to a combined calcium and magnesium depletion, which can be quite resistant to calcium supplementation until the magnesium deficiency has been corrected. This has been attributed to unresponsiveness in PTH secretion in magnesium deficiency (Andersson et al 1986). Common textbooks in nutrition also mention the association hypomagnesaemia and hypocalcaemia (Garrow et al, Human nutrition and dietetics, p. 173 in the 10th edition). What is the link to hypercalcaemia? Ref: Andersson H, Bosaeus I, Brummer R-J et al: Nutritional and metabolic consequences of extensive bowel resection. Digestive Diseases 1986;4:193-202.</p>	<p>Not relevant. Physiological manifestations are only discussed very briefly here. It is not relevant to include more detailed information related to certain clinical conditions, as it outside the scope of the NNR.</p>
Selskab for Ernæringsforskning, Denmark	<p>General comments</p> <p>Line 22-23: For the other nutrients (e.g. thiamin, riboflavin, zinc) the average dietary intake is also mentioned here and then the reader is referred to the chapter on dietary intake in Nordic countries.</p>	<p>Added in final version.</p>
National Food Agency, Sweden	<p>Detailed comments</p> <p>113 please make a clear conclusion that no UL is set in NNR 5 in order to avoid confusion with UL from other organizations</p>	<p>No UL has been set for Mg. The text refers to ULs set by US FNB and EFSA for Mg in dietary supplements.</p>
DI Fødevarer, Denmark	<p>General comments</p> <p>The Danish Food and Drink Federation hereby wants to provide comments to the draft chapter on magnesium. Comments relating to Upper intake levels and toxicity (I 104-112) It should be emphasized that adverse effects of magnesium does not relate to non-dissociable sources of magnesium like magnesium bound to amino acids. This means the UL does not relate to all sources of magnesium found in food supplements. Summary box (I 9) The UL for magnesium is not included in the summary box on the first page. Please provide justification in line with how other summary boxes for other vitamins or minerals are made. In this case it would be relevant to comment in line with the available data.</p>	<p>The section on ULs cite ULs set by US FNB and EFSA for Mg in dietary supplements without going into details about the individual studies. UL values have been added where relevant.</p>

General comment Detailed comment	WG observations and actions
<p>Danish Nutrition Society</p> <p>The references need to be updated:</p> <p>1) The references 40-56 are not mentioned in the text, but are in the reference list. On page 7, line 322, ref. 39 is mentioned, the next reference mentioned in the text is ref. 57 on page 7, line 332.</p> <p>2) There is no references mentioned in section on 'Children', page 10, line 452-460</p> <p>3) There is no references mentioned in section on 'Pregnancy and lactation', page 10, line 462-467</p> <p>4) References 83-93 plus the 2 extra unnumbered are not mentioned in the text, but are in the reference list. The last reference mentioned in the text is ref. 82, page 11, line 480 The last sentence (page 11, line 487-488) "Thus, the long-term recommendation in NNR 2004 is maintained" should be considered to be deleted or changed to "The long-term recommendation in NNR 2004 is maintained, 2-2.3 g/d (5-6 g salt)", in order to distinguish more clearly between the recommendations (which are changed/reduced compared to NNR 2004) and the long-term recommendations (which are not changed – and now similar to the present recommendations).</p>	<p>References updated in final version</p>
<p>The Finnish Heart Association</p> <p>lines 444-445 We suggest that the sodium intake should be less than 2.0 g/day (5.0 g salt/day) for adults.</p> <p>lines 459-460 We suggest that the sodium intake should be less than 0.2 g/1000 kJ (0.5 g salt/1000 kJ). The recommendation for daily intake should also be set to less than 2.0 g/day (5.0 g salt/day) in case of higher energy intakes.</p>	<p>Not accepted 0.2 g Na/MJ for children</p>
<p>Norwegian Institute of Public Health and the University of Oslo</p> <p>This is a comprehensive overview on the relation between salt, hypertension and cardiovascular disease. I have two suggestions: As systematic reviews are emphasized in NNR5, they could have a more prominent role in the text. Although many relevant studies are referred to, a controversial new studies which have gained attention and which have been further discussed and criticized in the scientific literature is missing (Stolarz-Skrzypek K et al. JAMA 2011;305:1777-85) and so is O'Donnell MJ et al. JAMA 2011;306:2229-38. The interesting follow-up of the TOHP study could also be referred to (Cook NR et al. BMJ 2007; 334:885-8).</p>	<p>Relevant comments. Studies will be commented in the final draft.</p>
<p>Danish Food and Drink Federation</p> <p>Line 440-449 The document states any recommendation on sodium intake have to be based on practical and public</p>	<p>This is based on e.g.</p>

General comment Detailed comment	WG observations and actions
<p>health considerations, rather than on precise estimates of an optional physiological intake. And then based on a pragmatic evaluation of available data a sodium intake of less than 100 mmol would be feasible at population level. We need substantiation of this claim. If the substantiation is based on dietary habits and content of salt in the food supply in one Nordic country, we need substantiation why this is relevant to the broader Nordic context. What is meant by the feasible in this context? (feasible for who?)</p>	<p>“translation” of nutrient recommendations to diets, and a reduction of salt intake in processed foods, see e.g. SNO-report</p>
<p>Helsedirektoratet, Norway</p> <p>General comments</p> <p>Most chapters start with a conclusion or recommendation. This is very convenient for the reader. Thus this chapter about salt should also start with the recommendations concerning both salt and sodium. Both salt and sodium should be mentioned in this conclusion as the text in this chapter concerns both salt and sodium. Reference intakes for sodium intake and UL should be given, and requirements for sodium should be mentioned in the introduction.</p> <p>Detailed comments</p> <p>24. In the introduction, or later, it could be explained that all sources of sodium in the foods are included when total sodium and salt content is calculated.</p> <p>38. Salt intake reported in the most recent Norwegian national dietary survey Norkost 2010-11 may be reported here, as well as the recent Swedish Riksmaten 2011.</p> <p>40-44. As sodium excretion in urine is an established method to measure sodium intake, both 24-hour and spot urine measurements should be described shortly.</p> <p>41-43. It is here said that dietary survey methods give similar levels of sodium intake as 24-hour urinary sodium. This should be further commented because most of the dietary surveys do not report use of discretionary salt at the table and at home. Furthermore urinary sodium does not include sodium excreted through other channels as sweat and faeces (5-10 % of total excreted sodium). Thus both methods may underestimate sodium intake but in different ways.</p> <p>79-420. This chapter gives a comprehensive overview on the relation between salt, hypertension and cardiovascular disease. As systematic reviews are emphasized in NNR5, they could have a more prominent role in the text. Although many relevant studies are referred to, a few new studies which have gained attention and which have been further discussed in the scientific literature is missing as Stolarz-Skrzypek K et al. JAMA 2011;305:1777-85 and O'Donnell MJ et al. JAMA 2011;306:2229-38. The follow-up of the TOHP study could also be referred to e.g. Cook NR et al. BMJ 2007; 334:885-8. Hypertension is a diagnosis in itself and is given great attention in medical services. A section concerning salt and hypertension may be included in addition to the sections concerning morbidity and cardiovascular risk. 438. A recent Danish report regarding costs could be mentioned: Konsekvensberegninger for reduktion af danskernes saltindtag Forskningscenter for Forebyggelse og Sundhed Marts 2012</p>	<p>Included in final version</p> <p>OK</p> <p>Included in final version</p> <p>Added</p> <p>Further elaborated in final version</p> <p>Further elaborated in final version</p>

General comment Detailed comment	WG observations and actions
<p>http://www.altomkost.dk/NR/rdonlyres/D63CA843-75F4-43FA-BC22-DAB2EDAE02A4/0/Konsekvensberegningerforreduktionafdanskernessaltindtag.pdf 441-449. Text regarding sodium requirements should be included, as it is done in the section concerning pregnancy, 462-7.</p> <p>452 second sentence: Do you mean SBP levels observed “among adults” in low?</p> <p>459. The recommended sodium intake for children should correspond to the text that will be used in the main summary recommendation in chapter 1.</p> <p>470-1. It may be explained that the 5 g/d recommendation from WHO is a population based recommendation, and that this is somewhat different than recommendations for individuals as those from IoM.</p> <p>482-8. Requirements for sodium should be mentioned.</p>	<p>Clarified Amended</p> <p>OK</p> <p>Added</p>
<p>National Institute for Health and Welfare, Finland</p> <p>lines 459-460 In line with the suggestions made in chapter 1 I would like to correct the sodium recommendation for children: The sodium intake should be less than 0.2 g/1 MJ (0.5 g salt/1 MJ) and should not exceed 4-5 g/d.</p>	<p>Accepted</p>
<p>Swedish Dairy Association</p> <p>General comments</p> <p>Most of the studies included support a lowering of the salt intake. How come these studies have been selected? Which studies have been excluded?</p> <p>Detailed comments</p> <p>In the chapter of dietary sources and intake (row 30, 31), there is only one reference telling the sources of sodium. Why is this only one selected? The referred study is Finnish and eight years old. Are there no studies from the other Nordic countries? Why is not for example Riksmaten 2011/2012 included? At rows 255, 256 you refer to the DASH-trials as clearly showing an effect of sodium restriction on blood pressure, independent of other dietary factors. But the DASH-trials was all about other dietary factors, for example a high intake in potassium, calcium and fibre.</p>	<p>Additional references added</p> <p>Section updated in final version</p>
<p>Copenhagen University Hospital, Rigshospitalet, Denmark</p> <p>General comments</p> <p>Jeg har læst NNR begrundelserne for salt reduktion. Jeg har tidligere læst Institute of Medicine’s begrundelser, som danner grundlag for FDA’s beslutning. IOM’s grundlag er karakteriseret ved at IOM overhovedet ikke citerer studier, der er kritiske over for saltreduktion – ikke et eneste. En så enøjet fremstilling er videnskabeligt upålidelig. NNR fremstillingen nævner kritisk litteratur, men karakteristisk er, at svaghederne ved disse studier fremhæves og styrkerne bortforklares. Modsat de positive studier (som er klart i overtal blandt de nævnte referencer). Her</p>	<p>Relevance and implications of the Intersalt study is questioned as well as association between sodium intake, blood pressure and CVD morbidity and mortality. WG acknowledge the</p>

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<p>fremhæves styrkerne, mens svaghederne enten slet ikke nævnes eller bortforklares. Det værste er at fordelingen mellem lovprisende og kritiske studier er udtalt skævvreden. NNR skriftet er således generelt tendentiøst og for useriøst til at jeg kan bruge mere tid på det . Det burde skrives fuldstændig om og gerne af en der har forstand på emnet. Enkelte specifikke eksempler kommenterer jeg nedenfor.</p> <p>Detailed comments</p> <p>Vedr Intersalt, side 2-3: I løbet af det 20. århundrede er blodtrykket i den vestlige verden støt faldet med i alt 15 mm Hg, formentlig pga den stigende generelle sundhed. For hver undersøgt fødsels kohorte op gennem det 20. århundrede er blodtrykket faldet. De yngre kohorter har lavere blodtryk end de ældre, selv om blodtrykkene er målt ved samme alder. De forskellige kohorter er derfor usammenlignelige og Intersalt's "age-blood pressure" diskussion derfor meningsløs.</p> <p>Side 10: "A lower sodium intake is associated with decreased risk of CVD morbidity and mortality". Det er forkert. En samlet analyse af alle befolkningsstudier viser at befolkningsgrupper der indtager 6 g salt eller mindre har en højere mortalitet end befolkningsgrupper der indtager mellem 6 og 12 g. De befolkningsstudier, der viser denne sammenhæng er ikke citeret i NNR skriftet.</p> <p>Side 11: "There is a progressive dose-response relationship between sodium intake and blood pressure": Denne påstand er baseret på He og MacGregor's selektive meta-analyse. Punktsværmen, som denne analyse er baseret på er nærmest U-formet og bestemt ikke normal fordelt. Der er således ingen dokumentation for " a progressive dose-response relationship". Dermed falder den væsentligste begrundelse for salt reduktion bort.</p>	<p>methodological problems in assessing sodium intake as a source of confounding in many observational studies. However, the evidence from meta-analyses of sodium intake, blood pressure and health outcomes such as CVD and gastric cancer supports a limitation of the current intake.</p>
<p>Minivent AB, Sweden</p> <p>General comments</p> <p>The text in the Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 the part of Sodium as salt is definitely wrong from a scientific standpoint and must be replaced. My draft proposal is the following text with lines number 1001-. Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 Sodium as salt Answer to the Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 the part of Sodium as salt. Key words Sodium, blood pressure, essential nutrient, tolerable lower intake level, tolerable upper intake level, food safety Summary Sodium is an essential nutrient involved in fluid and electrolyte balance and is required at a very closely controlled extracellular concentration of 137-145 mmol/L for normal cellular function . The main function of sodium in the body is to maintain the transmembrane electrical potential with sodium on the outside of the (cell) membrane and potassium on the inside. This is crucial for the survival of all cells. Salt is excreted totally passively by the glomeruli when the blood is filtrated in the kidneys. The excretion capacity is practically unlimited with 1 000 grams to 2 000 grams of salt</p>	<p>The comments and suggested revisions regarding recommended intakes have a number of shortcomings and are biased with respect to cited references concerning health outcomes. References [1] to [36] are copied from a commentary published 2005 on the website www.second-opinions.co.uk/salt-and-</p>

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<p>per day. The major problem for the body and the kidneys is to reabsorb enough sodium (usually more than 99 % but less than 100 % of excreted sodium in glomeruli) from the primary urine to stabilize and maintain the normal level of sodium in blood and extracellular fluid at the precise level of 137-145 mmol/L . We are therefore unable to manipulate the blood pressure by manipulating the amount of sodium in the food. All excess of sodium intake is immediately excreted in the renal glomeruli and not reabsorbed in the renal tubuli. Any deficiency in sodium intake versus sodium excretion is almost immediately life threatening. It is totally safe to let us be guided by our gustatory system when we add salt and water to our food. We do have multiple sodium sensors and volume sensors in our body including a central processing unit closely controlling both sodium and water levels in the body. There is no scientific relationship between salt intake and blood pressure/hypertension. There is no way to manipulate the blood pressure by manipulating the salt intake. Introduction Sodium is an essential nutrient involved in fluid and electrolyte balance and is required at a closely controlled extracellular concentration of 137-145 mmol/L for normal cellular function . The main function of sodium in the body is to maintain the transmembrane electrical potential with sodium on the outside of the (cell) membrane and potassium on the inside. This is crucial for the survival of all cells. Sodium is present in foods as a normal constituent at a normal level around 12 to 25 mmol/100 g of food (0,5-1,5 g salt/100 g of food). Sodium is also added to foods, mainly as sodium chloride (commonly known as salt) during processing, cooking and immediately prior to consumption, but also in lesser amounts in other forms, for example as sodium nitrate, sodium phosphate or sodium glutamate. The main reasons for the addition of salt during the processing of foods are for taste, flavour, texture and preservation . Dietary sources and intake The main sources of sodium in the diet are foods e.g. diet preparations, sauces, bread, cheese, spreads, meat and fish products . The contribution of sodium from added salt in cooking and at the table varies but in average it constitutes approximately 10 to 20 % of the total salt intake. Data on the total dietary intake of sodium in Nordic populations are scarce. According to national clinical chemistry laboratories the availability of salt in the Nordic countries is estimated to be 10-25 g per capita and day . Estimations of the sodium intake from national dietary surveys among adults generally show lower values. This means that survey estimations are generally underestimating the salt intake. Physiology and metabolism Sodium is an essential nutrient involved in fluid and electrolyte balance and is required at a closely controlled extracellular concentration of 137-145 mmol/L for normal cellular function . The main function of sodium in the body is to maintain the transmembrane electrical potential with sodium on the outside of the (cell) membranes and potassium on the inside. This is crucial for the survival of all cells. Mean daily sodium intakes of populations in Sweden range from about 150 to more than 300 mmol (4-10 g sodium or about 9-25 g salt) and are in an appropriate excess of dietary minimum needs (about 3 g sodium/day in adults). The main source of sodium in the diet is from all kinds of foods (about 70-75% of the total intake), with about 10-15% from naturally occurring sodium in unprocessed foods and about 10-15% from discretionary sodium added during cooking and at the table . The body has to maintain a very accurate level of extracellular sodium with a very stable concentration of 141 mmol/L $\pm$4 mmol/L which is</p>	<p>hypertension.html Comments on physiology</p> <p>Some minor amendments will be included in final draft</p>

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<p>equal to almost 9 grams of salt per liter serum or 3.6 grams of sodium per liter serum. So the total amount in a 70 kg person's blood is about 10 grams or 430 mmols of sodium or 25 grams of salt. The same concentration of 141 mmol/L must be upheld extracellularly as well. This means that the total amount of extracellular sodium is approximately 150 grams of salt or 60 grams or 2.5 mols of sodium Sodium is passively excreted in the glomeruli of the kidneys. As the total blood volume of 5.6 liters passes the kidneys several times per 24 hours the blood is filtrating out 1 000 to 2 000 grams of salt (400 to 800 grams or 17-34 moles of sodium). Recalculated to milligrams per minute it will be 700 to 1 400 milligrams salt per minute which is equal to 280 mg sodium to 560 mg sodium (12-25 millimoles) each and every minute of the day . So there is never a problem for the body of getting rid of any excess salt as long as you drink enough water. The body's major problem is to reabsorb up to 99.5 % of the salt excreted in the glomeruli. This is possible by active transport of sodium back from the tubuli into the kidney's capillary vessels. To be able to actively reabsorb sodium from primary urine the renal tubuli need the blood pressure rising hormones renin, aldosteron and angiotensin from the pituitary gland. Then the tubuli, by active transport, reabsorb 990-1990 grams of salt or 390 to 800 grams of sodium or 17 moles to 34 moles of sodium via the urine per day thus excreting only the fractional of more than 3 grams (more than 130 mmol) of sodium per day. The very small, compared to the great amount of filtrated sodium, excretion of 10-25 grams of salt has to be replaced each and every day as we do not have any infinitive sources of salt in the body like a salt mine or a nuclear plant converting other atoms to sodium atoms.</p> <p>Requirement The conclusion of the normal physiology of the kidney is that an adult human of 70 kg has to eat at least seven and a half grams of salt per day to reassure that the body always has enough salt or sodium in the blood and then the rest of the body. Man can also eat at least 100 grams of salt per day without any problems as long as you can drink enough pure water and you obey your thirst. Acute deficiency can develop in connection with heavy sweating in combination with large fluid intakes devoid of sodium, or in connection with prolonged vomiting and diarrhoea without salt supply. Clinical symptoms include muscle seizures, loss of appetite and circulation disturbances. Severe deficiency can result in coma and death . Salt and blood pressure From a public health perspective the role of sodium as dietary salt in the regulation of blood pressure has received most interest. The relationship between salt and blood pressure has been discussed for more than 100 years and there are no physiological, biochemical and endocrinological evidence that the blood pressure can be manipulated by manipulating the sodium intake except that severe deficiency can result in coma and death . All manipulative experiments on humans follow the same protocol like the DASH trial: One arm with an extremely low sodium intake like 3 g salt, one arm with a low intake of 6-9 g salt and then the blood pressure is compared between the arms , . Usually, the extremely low arm has 1-4 mm Hg lower blood pressure due to lower extracellular volume including lower blood volume due to the body's hemostasis trying to keep up the concentration of sodium between 137-145 mmol/L. The problem with these studies is that the third arm with 20-30 g salt is always missing. That third arm would show no difference between the low salt arm and the high salt arm and thus disproving the beautiful</p>	

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<p>hypothesis which is in opposition to the old fashioned physiology knowledge from the previous centuries. Other dietary factors and blood pressure A number of dietary factors and physical activity have been associated with but not the cause of blood pressure. These include e.g. alcohol, potassium, calcium, magnesium, and fatty acid composition (see respective chapter). But NNRS have not included carbohydrates as blood pressure increasing agent. Salt and morbidity and mortality There are only few studies that have investigated the relationship between sodium intake and morbidity and mortality. National Health and Nutrition Examination Survey, NHANES I, is a project Center of Disease Control and Prevention in USA that started already in 1971. In 1997 Dr Helen Whalley wrote in Lancet that the analysis of NHANES I showed that the rate of CVD increased with 20 % in those on a low salt diet than those on a regular salt diet . In May 2006 Dr Hillel W Cohen showed that data from NHANES II and 2008 in NHANES III that eating less than 5 g salt per day increased the risk for heart disease with more than 50 %. The results are in opposition to most authorities recommendation about maximum 5 g salt per day . Dr Cohen said “We are of the opinion that these results are not supportive of the present recommendations. We urge those issuing the guidelines to go back to their original data and check with more results before issuing general recommendations. And it s totally obvious that the results do not support current recommendations.” Recommended intake There is no scientific or physiological support of a restriction of the salt intake . There is no scientific or physiological support for a relationship between salt intake and blood pressure. Sodium is essential and we have to ingest the same amount of sodium that we loose every day to avoid disease and premature death. Any ingested excess of sodium is immediately excreted without changing neither the blood concentration of sodium nor the blood pressure. There is very old knowledge about salt deficiency and premature death, it is, among many synonyms, called the fire engineers disease after the demise of steam engine engineers shovelling coal in the hot environment of a steam engine furnace. It is also well known that a heat stroke, circulation collapse, sodium deficiency, water intoxication, Syndrome of Inappropriate ADH excretion (SIADH) and Syndrome of Inappropriate Salt and Water Intake (SISWI) is due to excess excretion of salt or too small a salt intake or too much water intake . It is well known in France 2003 that there were several elderly persons that died prematurely during a heat wave despite getting enough water. But they did not get enough salt so they died from sodium deficiency. Now the French authorities have changed the instructions, staff should give enough salt together with water to the elderly for them to survive. In Sweden presently 30 % of all medicine patients at Skånes Universitetssjukhus (SUS) are hospitalized due to sodium deficiency and 19 % of these patients have a premature death in the hospital. This means that 6 % of all medicine patients in Sweden suffer a premature death due to hyponatremia. That article in Läkartidningen is indicating a higher risk of premature intrahospital salt deficiency death in Sweden for approximately 60 patients per day. Also, EFSA issued a report in 2005 where EFSA concluded on page 18: “Conclusions and recommendations Derivation of a tolerable upper intake level (UL) The available data are not sufficient to establish an upper level for sodium from dietary sources.” Adults Based on a pragmatic evaluation of the available data, a sodium intake of minimum 130 mmol (3 g) per day (7.5 g salt) would be prudent at the</p>	

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<p>population level. The current average sodium intake in the Nordic countries can be estimated at 4-10 grams (170-425 mmol per day, 10-25 g salt). The proposed population targets would therefore conform to the actual intake of salt on a population level and even include the necessity of an increased intake of salt in risk prone individuals as elderly and sick individuals. Children Small children are very able to regulate the salt intake as long as they have enough water intake. Breast milk is the perfect example with relatively low macronutrient concentration in a diluted solution with 70 mmol/L sodium, corresponding to about 50 g salt to an adult of 70 kg weight, during the first day of life. Within 14 days the amount has decreased to 10 mmol/L corresponding to 8 g salt per day to a 70 kg person. Based on a pragmatic evaluation of the available data, recommended sodium intake for children up to about 12 years of age is minimum 8 mmol/kg (20 mg per kg, salt 50 mg per kg) per day is prudent at the population level. The recommended sodium intake for children from about 13 years of age is set to 1 g per 1000 kJ, which is based on the energy-adjusted recommended levels for adult women. Pregnancy and lactation Pregnancy as well as lactation are associated with an increase in the physiological requirements for sodium, i.e. daily requirement during pregnancy will be increased to more than 10 g or 450 mmol sodium per day or more than 25 g salt per day to minimize the risk of pre-eclampsia and increased to more than 10 g or 450 mmol per day or more than 25 g salt per day to minimize the risk of hyponatremia during lactation. These amounts are normal and can apparently be handled by the homeostatic system of the body. There is evidence to suggest that sodium requirements during pregnancy and lactation are increased from that of non-pregnant women. Reasoning behind the recommendation Sodium is an essential nutrient involved in fluid and electrolyte balance and is required at a closely controlled extracellular concentration of 137-145 mmol/L for normal cellular function . The main function of sodium in the body is to maintain the transmembrane electrical potential with sodium on the outside of the (cell) membrane and potassium on the inside. This is crucial for the survival of all cells. There is no dose-response relationship between sodium intake and blood pressure. Any recommendations on the sodium intake thus have to be based on estimate of an optimal physiological intake. Based on a pragmatic evaluation of the available data, a sodium intake of more than 150 mmol (3.5 g) per day (9 g salt) would be prudent at the population level. Thus, the long term recommendation in NNR 2004 is increased to a prudent value. Discussion Mean daily sodium intakes of populations in Sweden range from about 150 to more than 300 mmol (4-10 g sodium or about 9-25 g salt) and are in an appropriate excess of dietary minimum needs (about 3 g sodium/day in adults). The main source of sodium in the diet is from all kinds of foods (about 70-75% of the total intake), with about 10-15% from naturally occurring sodium in unprocessed foods and about 10-15% from discretionary sodium added during cooking and at the table . There are no major adverse effects of increased sodium intake when ingested in normal tasting food according to EFSA . A sodium level below 135 mmol/L is usually due to a too low intake of salt (less than combined losses via urine, sweat and faeces) and can give rise to serious health hazards including premature death . A very serious problem is that the median value of P-Na in Dalecarlia, Sweden, has decreased from 141 to 139 mmol/L during the last three years . This means that on a population basis the sodium</p>	

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<p>intake is too low indicating a higher risk of premature death . There is no relationship between salt intake and blood pressure. All sodium ingested in excess to basic losses is immediately excreted via the glomeruli filtration. The kidney can excrete between 12 mmol and 25 mmol (280-560 mg sodium or 700-1 400 mg salt) per minute and is equal to 17 000 to 35 000 mmol of sodium per 24 hours or 1 000 000 to 2 000 000 milligrams (equal to 1-2 kg) of salt per day. Dietary deficiency of sodium is nowadays very common due to the widespread maladvice to eat less salt and to less occurrence of sodium in foods. According to the literature about 30 percent of all patients in a medical ward are hospitalized due to sodium deficiency and 19 % of those patients die prematurely in the hospital due to easily cured salt deficiency. Nutritional requirements and recommendations Human populations survive on wide extremes of habitual sodium consumption from 10 to 450 mmol/day. The ability to survive at low levels of consumption is dependent upon adaptive mechanisms which reduced losses in sweat, stool and urine. For most populations, the habitual levels of sodium consumption greatly exceed the physiological requirements, and there are few data which determine the minimal levels of sodium consumption required to maintain health in people who have adapted to low levels of sodium consumption over long periods of time For sodium, the acceptable range of intakes for adults is established by the normal excretion values of sodium in urine in clinical chemical laboratories of Sweden to 150-300 mmol/day and adding the normally small amount of losses via faeces and sweat of usually more than 50 mmol/day . Children have the same requirements of salt as adults per kg bodyweight. The children's kidneys do work perfectly from birth as breast milk contains enough of both salt and water for the survival of the newborn child. It is shown that a newborn baby gets extra salt the first fortnight corresponding to 60 grams salt (24 grams or 1 mol of sodium) to a 70 kg adult during the first day. After two weeks the amount of sodium has decreased to corresponding 10 grams of salt per day to an adult , . Conclusion The old fashioned physiology, biochemistry and endocrinology shows that salt is an essential micronutrient in the human body. Normal salt intake in the Swedish population is normally at least nine and more than eighteen grams of salt per day according to daily urine excretion. Manipulation of salt intake outside the normal physiological boundaries between ten and 100 grams of salt intake is shown to increase disease and premature death. Nordic Nutrition Recommendations regarding salt is that the population should eat an adequate amount of salt, normally at least 3 grams of sodium or 7,5 grams of sodium chloride also called salt. Requirement and recommended intake Adults Nordic Nutrition Recommendations regarding salt is that adults are advised to eat an adequate amount of salt, normally more than 3 grams of sodium or 7,5 grams of sodium chloride also called salt as long as the food is not too salty to your taste. Also, adults are advised to drink normally 1-3 liters of water accordingly to quench the thirst and facilitate excretion of excess salt. Children Children are advised to eat the same food with the same saltiness as parents do, as always have been the case. Based on a pragmatic evaluation of the available data, recommended sodium intake for children up to about 12 years of age is minimum 8 mmol/kg (20 mg per kg, salt 50 mg per kg) per day is prudent at the population level. The recommended sodium intake for children from about 13 years of age is set to 1 g per 1000 kJ, which is based on the energy-adjusted recommended</p>	

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<p>levels for adult women. Reasoning behind the recommendation Normal physiology has shown that salt is an essential micronutrient that has to be replenished in excess for the body's homeostasis. Also, enough pure water is recommended to support the excretion of excess sodium. Lower intake levels There are severe dangers of serious disease or even premature death of salt deficiency if the intake is lower than recommended above. Upper intake levels and toxicity As has been shown above there is no need to issue an upper intake level from dietary sources as salt is excreted passively and the level of salt in the body is maintained actively by the kidneys and hormones. There has not been shown any toxicity when sodium is ingested from dietary sources Mora November 25, 2012 Björn Hammarskjöld M.D., former consultant in Pediatrics Ph.D. in Biochemistry Independent scientist in Nutrition More References [1]. Spitzer S, et al. The influence of social situations on ambulatory blood pressure. Psychosom Med 1992; 54: 71. [2]. Singer, Adam J. and Hollander, Judd E. Blood pressure — Assessment of interarm differences. Arch Int Med 1996; 156: 2005- 08 [3]. Stallones RA. The Rise and Fall of Ischaemic Heart Disease. Scientific American 1980; 243 (5): 43-9. [4]. Nakatsuka H, et al. Effectiveness of attention to reduce salt in diet, as evidenced by reduced urinary excretion of salt. Ecol Food Nutr 1991; 26: 323-332. [5]. Gothberg G, et al. Response to slow graded bleeding in salt depleted rats. J Hypertension 1983; 1(Suppl 2): 24. [6]. Wassertheil-Smoller S, et al. Effect of antihypertensives on sexual function and quality of life: the TAIM Study. Ann Intern Med 1991; 114: 613-20. [7]. Medical Research Council Working Party: MRC trial of treatment of mild hypertension: principle results. Br Med J 1985; 291: 97 [8]. — Adverse reactions to bendrofluazide and propranolol. Lancet 1981; ii: 632. [9]. Editorial. More on hypertension labelling. Lancet 1985; i: 1138. [10]. Milne BJ, Logan AG, Flanagan PT. Alteration in health perception and life-style in treated hypertensives. J Chronic Dis 1985; 38: 37. [11]. Brown JJ, et al. Salt and hypertension. Lancet 1984; ii: 456. [12]. Swales J. Salt and High Blood Pressure: A Study in Education, Persuasion and Naïveté. A Diet of Reason. Social Affairs Unit, 1986 [13]. Lichtenstein MJ, et al. Heart rate, employment status and prevalent IHD confound relationship between cereal fibre intake and blood pressure. J Epid Comm Hlth 1986; 40: 330. [14]. Barker DJP, Godfrey KM, Osmond C, Bull A. The relation of fetal length, ponderal index and head circumference to blood pressure and the risk of hypertension in later life. Paed Perinat Epidem 1992; 6: 35. [15]. Doyle W, Crawford MA, Wynn AHA, Wynn SW. Maternal nutrient intake and birthweight. J Hum Nutr Dietet 1989; 2: 451-22. [16]. Alderman M N, Cohen H, Madhavan S. Dietary sodium intake and mortality: the National Health and Nutrition Examination Survey (NHANES I). Lancet 1998; 351: 781-85 [17]. Whalley H. Salt and Hypertension: consensus or controversy? Lancet 1997; 350: 1686. [18]. Graudal N A, Gallee A M, Garred P. Effects of Sodium Restriction on Blood Pressure, Renin, Aldosterone, Catecholamines, Cholesterols, and Triglyceride : A Meta-analysis. JAMA 1998; 279:1383-1391 [19]. Merlo J, et al. Incidence of myocardial infarction in elderly men being treated with antihypertensive drugs: population based cohort study. Br Med J 1996; 313: 457-61 [20]. Bloom W L. Inhibition of salt excretion by carbohydrate. Arch Int Med. 1962; 109: 26-32. [21]. Friedman G D, et al. Precursors of essential hypertension: body weight, alcohol and salt use, and parental history of hypertension. Prev Med 1988; 17: 387-400 [22]. Appel LJ, et al. Does supplementation of diet with</p>	

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<p>"fish oil" reduce blood pressure? Arch Int Med, 1993; 153: 1429-38 [23]. Connor W E. Diabetes, fish oil, and vascular disease. Ann Int Med 1995; 123: 950-52. [24]. Kenny D. The paradoxical effects of fish oil on blood pressure. Med Hypoth 1992; 37: 97. [25]. Moran J P, et al. Plasma ascorbic acid concentrations relate inversely to blood pressure in human subjects. Am J Clin Nutr 1993; 57: 213-17. [26]. Witteman JCM, et al. Reduction of blood pressure with oral magnesium supplementation in women with mild to moderate hypertension. Am J Clin Nutr, 1994; 60: 129-35 [27]. Dahl L K Heine M. Effects of chronic excess salt feeding: Enhanced hypertensogenic effect of sea salt over sodium chloride. J Exp Med 1961; 113: 1067-76 [28]. Reusser M E. and McCarron, D A. Micronutrient effects on blood pressure regulation. Nutr Rev 1994; 52: 367-75. [29]. Yen L-L, et al. Comparison of relaxation techniques, routine blood pressure measurements, and self-learning packages in hypertension control. Prev Med 1996; 25: 339-45 [30]. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med 1997;157:2413—46. [31]. Dwyer JH, Dwyer KM, Scribner RA, et al. Dietary calcium, calcium supplementation, and blood pressure in African American adolescents. Am J Clin Nutr 1998;68:648—55. [32]. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. N Engl J Med 1997; 336:1117—24. [33]. McCarron DA, Morris CD, Henry HJ, Stanton JL. Blood pressure and nutrient intake in the United States: an analysis of the Health and Nutrition Examination Survey I. Science 1984;224:1392—8. [34]. McCarron DA, Morris C, Cole C. Dietary calcium in human hypertension. Science 1982;217:267—9. [35]. Alderman M. Data linking sodium intake to subsequent morbid and fatal outcomes must be studied. Br Med J 1997; 315: 484-5 [36]. Le Fanu J. Cross cultural studies such as Intersalt study cannot be used to infer causality. Br Med J 1997; 315: 484 [37] Nadezda Koleganova,1 Grzegorz Piecha,1,2,3 Eberhard Ritz,2 Luis Eduardo Becker,2 Annett Müller,1 Monika Weckbach,1 Jens Randel Nyengaard,4 Peter Schirmacher,1 and Marie-Luise Gross-Weissmann1 Both</p> <p>Detailed comments</p> <p>Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 - Sodium as salt Comments to the Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 the part of Sodium as salt.</p> <p>The text in the Public consultation on the draft proposal for the first part of the Nordic Nutrition Recommendations 2012 the part of Sodium as salt is definitely wrong from a scientific standpoint and must be replaced. The parts in lines 24-488 needs to be rephrased to be correct from the points of views in physiology, biochemistry and endocrinology. So the proposed wordings are in the added proposition.</p> <p>Comments regarding the NNR5 about salt. It is interesting that the NNR5 experts are referring to an older EFSA report from 1993 (lines 492-4) instead of the EFSA report of 2005 in the reference list above. This clearly shows that the NNR5 experts seems to avoid modern and relevant references that oppose the experts hypothesis that salt is a health hazard It seems that the NNR5 experts looking into the salt issue seems to be lacking fundamental knowledge in</p>	

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<p>human physiology, biochemistry and endocrinology as they seem to be totally ignorant in these areas of knowledge. The NNR5 experts must also, if they had done their homework properly, know that the nutrition experts of the US Department of Agriculture already 1999 knew that salt reduction had no positive effect on blood pressure. This is according to a transcribed taped discussion readable on the Health departments web site Also, in an article Stolarz-Skrzypek K et al write in their Conclusions : “Conclusions In this population-based cohort, systolic blood pressure, but not diastolic pressure, changes over time aligned with change in sodium excretion, but this association did not translate into a higher risk of hypertension or CVD complications. Lower sodium excretion was associated with higher CVD mortality.” “Lower sodium excretion was associated with higher CVD mortality” is interpreted by associated professor Wulf Becker in Livsmedelsverkets Protokoll nr 51 (Minutes number 51) at a meeting with expertgruppen för Kost- och hälsofrågor on 2011-09-20 §7 page 3 “En annan meta-analys av Stolarz-Skrzypek K et al visar att högt saltintag är associerat med ökad risk för stroke och hjärt-kärlsjukdom. http://jama.ama-assn.org/content/305/17/1777.full.pdf+html?etoc ” ”(Another meta analysis by Stolarz-Skrzypek K et al shows that a high salt intake is associated to increased risk for stroke and cardio-vascular diseases)” Becker here seems to violate the Swedish Constitution 1st chapter 9th §, both the matter of factness and the objectivity criteria. I’d rather see Becker declare other reasons than infringement of the Swedish Constitution for his statement. But the facts remains, Becker told the expert group a conclusion contrary to the conclusions in the article. Also, there are quite a few articles stating that a low salt intake increases the CVD mortality, e.g. Alderman et al , Whalley and Cohen . Dr Helen Whalley wrote in Lancet that the analysis of NHANES I showed that eating a low salt diet showed a 20 % increase in CVD. In May 2006 Dr Hillel Cohen showed data from NHANES II and 2008 data from NHANES III describing that eating less than five grams of salt increased the risk of coronary disease more than 50 %. Again, some of the crucial articles regarding salt and hypertension are missing, e.g. the Cochrane review from 2011 . The conclusion in the Cochrane abstract is: “Sodium reduction resulted in a 1 % decrease in blood pressure in normotensives, a 3,5 % decrease in hypertensives, a significant increase in plasma renin, plasma aldosterone, plasma adrenalin and plasma noradrenalin, a 2,5 % increase in cholesterol and a 7 % increase in triglycerides. In general these effects were stable in studies lasting for 2 weeks or more.” This means that the decrease in blood pressure is nonsignificant but there are significant increase in hypertensive and also sodium resorption hormones like plasma renin, plasma aldosterone, plasma adrenalin and plasma noradrenalin as the sodium level in the blood decreases while eating too low amounts of salt. When the salt level in the blood decreases below 125 mmol/L the kidneys try to restore a higher salt concentration by decreasing the blood volume and then the extra cellular volume decreases as well thus lowering the blood pressure. The homeostasis of the body tries to restore the volume by increasing ADH which dilutes the blood again by lowering sodium concentration threatening the homeostasis and life of the patient. This condition is called SIADH or SISWI, easily corrected by letting the patient eat salt and drink water and the body will restore homeostasis. I.V. infusion of salts is another alternative but that usually takes longer time and is more dangerous than the oral</p>	

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<p>route. A third option is to use ADH receptor blockers like tolvaptan, a vasopressin antagonist. But there are not much of an effect as 60 % of the patients treated with tolvaptan had normal sodium excretion after 30 days while 26 % in the placebo group had normal sodium excretion. According to Läkartidningen 2012 about 19 % of all patients with hyponatremia die of sodium deficiency despite treatment with tolvaptan. But they do not get enough sodium in their diet. The first National Health and Nutrition Examination Survey (NHANES I) established baseline information during 1971-75 in a representative sample of 20,729 American adults aged twenty-five to seventy-five. Of these, 11,348 underwent medical and nutritional examination. They were rechecked in 1992. By then there had been 3,923 deaths, of which 1,970 were due to a cardiovascular disease. Comparing salt intakes, this study found that all-cause mortality was inversely related to salt intake. In other words, those who ate the most salt had the fewest deaths — from any cause. And the same was found for cardiovascular deaths. Dr Helen Whalley writing a feature in the Lancet, talks of the continuing debate on the supposed association between salt and hypertension. She points out that an analysis of the NHANES I survey shows that 'the heart attack fatality rate among those on low-sodium diets was 20% higher than those on normal diets.' She goes on to report a study on the Salt Institute's website on the impact of long-term salt reduction. It found 'a four-fold increase in heart attacks among those on low-salt diets'. In 1998 a large meta-analysis was published in an attempt to resolve the controversy . Fifty-eight trials published between 1966 and the end of 1997 were reviewed to estimate the effects of reduced sodium intake on systolic and diastolic blood pressure, particularly as in recent years the debate has been extended by studies indicating that reducing sodium intake has adverse effects. They found that reducing salt intake did reduce blood pressure slightly, but that it increased LDL cholesterol, the so-called 'bad' cholesterol. They conclude that 'These results do not support a general recommendation to reduce sodium intake. Salt restriction research is biased All those studies showing a decreased blood pressure with decreased salt intake are biased. They all have one arm with a low salt level like 6-9 gram salt per day and another arm with extremely low salt, around 3 grams per day. The findings are a slight decrease in blood pressure due to hypovolemia despite increase in the levels of hypertensive hormones like renin, aldosterone, adrenalin and noradrenalin. But all trials have seemingly actively forgotten the third arm, giving patients 20-30 g salt per day. That third arm will have the same blood pressure as the one with 6-9 g salt. This is because the glomeruli are filtering out about 1 g salt per minute and thus the blood pressure can not increase due to the fact that there is no salt retention, everything is filtered out. Still, the main problem for the kidneys is the reabsorption of sodium using at least three blood pressure increasing and sodium retention hormones. But it is also interesting that patients with diabetes having the highest intake of salt have less insulin resistance. According to physiological research from 1970th it is shown that newborn children in the colostrums gets very high concentrations of sodium during the first day of life corresponding to more than 60 grams salt for a 70 kg adult . Within a fortnight the breastmilk contains sodium according to 10 grams of salt to an adult. Conclusion The old fashioned physiology, biochemistry and endocrinology shows that salt is an essential micronutrient in the human body. Normal salt intake in the Swedish</p>	

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<p>population is normally at least nine and more than eighteen grams of salt per day according to daily urine excretion. Manipulation of salt intake outside the physiological normal boundaries between ten and 100 grams of salt intake is shown to increase disease and premature death.</p> <p>Requirement and recommended intake Adults Adults are advised to eat at least ten grams of salt as long as the food is not too salty to your taste. Also, drink normally 1-3 liters of water accordingly to quench the thirst. Children Children are advised to eat the same food as parents do, as always have been the case. Reasoning behind the recommendation Normal physiology has shown that salt is an essential micronutrient that has to be replenished in excess for the body's homeostasis. Also, enough pure water is recommended to support the excretion of excess sodium. Lower intake levels There are severe dangers of serious disease or even premature death of salt deficiency if the intake is lower than recommended above. Upper intake levels and toxicity As has been shown above there is no need to issue an upper intake level from dietary sources as salt is excreted passively and the level of salt in the body is maintained actively by the kidneys and hormones. There has not been shown any toxicity when sodium is ingested from dietary sources</p> <p>More References [1]. Spitzer S, et al. The influence of social situations on ambulatory blood pressure. Psychosom Med 1992; 54: 71. [2]. Singer, Adam J. and Hollander, Judd E. Blood pressure — Assessment of interarm differences. Arch Int Med 1996; 156: 2005- 08 [3]. Stallones RA. The Rise and Fall of Ischaemic Heart Disease. Scientific American 1980; 243 (5): 43-9. [4]. Nakatsuka H, et al. Effectiveness of attention to reduce salt in diet, as evidenced by reduced urinary excretion of salt. Ecol Food Nutr 1991; 26: 323-332. [5]. Gothberg G, et al. Response to slow graded bleeding in salt depleted rats. J Hypertension 1983; 1(Suppl 2): 24. [6]. Wassertheil-Smoller S, et al. Effect of antihypertensives on sexual function and quality of life: the TAIM Study. Ann Intern Med 1991; 114: 613-20. [7]. Medical Research Council Working Party: MRC trial of treatment of mild hypertension: principle results. Br Med J 1985; 291: 97 [8]. — Adverse reactions to bendrofluazide and propranolol. Lancet 1981; ii: 632. [9]. Editorial. More on hypertension labelling. Lancet 1985; i: 1138. [10]. Milne BJ, Logan AG, Flanagan PT. Alteration in health perception and life-style in treated hypertensives. J Chronic Dis 1985; 38: 37. [11]. Brown JJ, et al. Salt and hypertension. Lancet 1984; ii: 456. [12]. Swales J. Salt and High Blood Pressure: A Study in Education, Persuasion and Naïveté. A Diet of Reason. Social Affairs Unit, 1986 [13]. Lichtenstein MJ, et al. Heart rate, employment status and prevalent IHD confound relationship between cereal fibre intake and blood pressure. J Epid Comm Hlth 1986; 40: 330. [14]. Barker DJP, Godfrey KM, Osmond C, Bull A. The relation of fetal length, ponderal index and head circumference to blood pressure and the risk of hypertension in later life. Paed Perinat Epidem 1992; 6: 35. [15]. Doyle W, Crawford MA, Wynn AHA, Wynn SW. Maternal nutrient intake and birthweight. J Hum Nutr Dietet 1989; 2: 451-22. [16]. Alderman M N, Cohen H, Madhavan S. Dietary sodium intake and mortality: the National Health and Nutrition Examination Survey (NHANES I). Lancet 1998; 351: 781-85 [17]. Whalley H. Salt and Hypertension: consensus or controversy? Lancet 1997; 350: 1686.</p>	

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<p>in body weight and adiposity (Prada et al. 2000, Xavier et al. 2003, Okamoto et al. 2004) which were associated with a decrease in whole body glucose uptake evaluated with the euglycemic hyperinsulinemic clamp (Prada et al. 2000). In addition, other studies have shown that a severe salt restriction induces increased plasma triacylglycerol and total cholesterol concentration in humans as well as in rats (Sharma et al. 1990, Ruppert et al. 1991, Catanozi et al. 2001). Taking these reports together, it seems that a severe sodium restriction could dysregulate insulin sensitivity and increase adiposity. However, the molecular mechanisms by which a low salt diet can induce insulin resistance are not yet completely understood. Under such conditions, insulin resistance may be tissue-specific to the liver and muscle, whereas adipose tissue remains sensitive to insulin, as seen in a previous study (Hirata et al. 2003).</p>	

Minivent AB, Sweden, second comment

There is another cation that works together with sodium, potassium. Just by using the same layout and almost all of the same information NNR5 can use the potassium information as a template for sodium information. So here it is: Sodium – NNR 2012 1 (5) Sodium

Sodium Recommendations

Sodium g/d: Women and men Recommended intake RI:10-25

Lower intake level LI: More than 7,5

Upper intake level UL: No information

Sodium g/d. Children 0-6, 7-12, 13-46 y Recommended intake RI..3-15, 5-20, 7.5-20 Lower intake level LI: More than 3, 5, 6

Upper intake level UL: No information

Introduction

The major proportion of the sodium in the body (98 %) is found outside of the cells and sodium is the quantitatively most important extracellular cation. Intracellular sodium, which constitutes the remaining 2 %, is important for regulating the membrane potential of the cells, and thereby for nerve and muscle function, blood pressure regulation etc. Sodium also participates in the acid-base balance. 1 mmol sodium is equivalent to 23 mg.

Dietary sources and intake

Important sodium sources in the Nordic diets are ordinary normally salted food, and milk products. The average dietary intake ranges from 10 to 25 g per day (see Chapter XX Intake of Vitamins and minerals in Nordic countries).

Physiology and metabolism

The absorption of sodium is effective and about 90 % of the dietary sodium is normally absorbed from the gut. The sodium balance is primarily regulated by renal excretion in urine. A small proportion can be lost in sweat. Requirement and recommended intake Sodium deficiency can develop as a consequence of increasing losses from the gastrointestinal tract and kidneys, e.g. during prolonged diarrhoea or vomiting, and in connection with the use of laxatives or diuretics. Sodium deficiency due to low dietary intake alone is not uncommon, due to the widespread occurrence of low salt in foods propaganda. Treatment with diuretics without sodium compensation or sodium sparing diuretics can, however, lead to deficiency. Hyperaldosteronism, hereditary defects of renal salt transporters, such as Bartter's syndrome and Gitelman's syndrome, and excessive consumption of licorice increase sodium retention and potassium excretion and may lead to hyponatremia. Symptoms of sodium deficiency are associated with disturbed cell membrane function and include muscle weakness and disturbances in heart function, which can lead to arrhythmia and heart seizure. Mental disturbances, e.g. depression and confusion, can also develop. The losses of sodium via the gastrointestinal tract, urinary excretion and sweat comprise about 10 to 25 g/d (150-400 mmol), but 7,5 g/d (130 mmol) is needed to avoid low plasma levels and loss of total body sodium in adults. The sodium intake may affect sodium balance and sodium intakes of 10-30 mmol/d may induce water retention and an increase in blood pressure, both in normotensive and hypertensive subjects. An inverse relationship between cardiovascular death and sodium excretion was also observed. A number of

studies of both normotensive and hypertensive subjects indicate that an increased sodium intake as supplements can lower blood pressure and increase urinary sodium excretion. The lack of clear dose-response observed in the studies could be due to factors such as differences in duration of studies, initial blood pressure, sodium intake, habitual diet, race and age. An inverse association between sodium intake and the risk of stroke has been shown in most cohort studies

Reasoning behind the recommendation

The recommended intake of sodium in NNR 2004 was based on flawed data on the effect of sodium on blood pressure. Several clinical trials and population surveys published thereafter support the finding that a diet rich in sodium have a favourable effect on blood pressure. The reference values are increased compared to NNR 2004, since there are new scientific data to justify major changes. The recommended intakes are set at 10-25 g/d (170-400 mmol) for men and women. The figure for women also includes pregnant and lactating women. It should be pointed out that sodium intakes over and above these values might have further beneficial effects. The reference values for children and adolescents are extrapolated from adult values based on needs for growth and adjusted for body weight.

Lower intake levels

The lower limit is estimated to 7.5 g/d (130 mmol) for adults.

Upper intake levels and toxicity

Sodium chloride has been associated with acute poisoning in humans. Case reports have described heart failure, cyanosis and cardiac arrest after ingestion of high doses of sodium chloride tablets. Gastrointestinal effects have also been described after chronic ingestion of sodium chloride in case studies and supplementation studies. This is characterised by abdominal pain, nausea and vomiting, diarrhoea, and ulceration of the oesophagus, stomach and duodenum and ileum. The occurrence and severity of the effects depend on a number of factors of which formulation of the preparation, dose and gut transit time and of course enough water intake seem to be most important. Dietary sodium has not been associated with any negative effects in healthy subjects. Prolonged high sodium intakes from diet and sodium-containing salt may, however, cause hyponatraemia and affect heart function in subjects with renal insufficiency or impaired kidney function. Sea water without enough dilution with pure and salt free water is shown to be lethal due to the kidneys inability to excrete sodium without enough water. The available data are insufficient to set an upper level for dietary sodium. Supplemental intakes up to 50 g/d are generally not associated with overt adverse effects, but certain preparations may induce mild lesions of the gastrointestinal mucosa. It seems prudent to include sodium from sodium-containing salt in this figure.

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Detailed comments More References

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- [37]. Nadezda Koleganova,¹ Grzegorz Piecha,^{1,2,3} Eberhard Ritz,² Luis Eduardo Becker,² Annett Müller,¹ Monika Weckbach,¹ Jens Randel Nyengaard,⁴ Peter Schirmacher,¹ and Marie-Luise Gross-Weissmann¹ Both high and low maternal salt intake in pregnancy alter kidney development in the offspring <http://ajprenal.physiology.org/content/301/2/F344.full.pdf+html>
Low salt intake modulates insulin signalling <http://joe.endocrinology-journals.org/cgi/content/abstract/185/3/429>

Reduction in daily sodium intake has been recommended as a non-pharmacological approach to the treatment of hypertension (Chobanian & Hill 2000, Sacks et al. 2001, Vollmer et al. 2001, Chobanian et al. 2003). However, a severe restriction of sodium chloride intake has shown adverse effects on glucose and lipid metabolism (Iwaoka et al. 1988, Sharma et al. 1990, Egan et al. 1991, Ruppert et al. 1991, Catanozi et al. 2001). In previous reports, chronic salt restriction in Wistar rats induced increases in body weight and adiposity (Prada et al. 2000, Xavier et al. 2003, Okamoto et al. 2004) which were associated with a decrease in whole body glucose uptake evaluated with the euglycemic hyperinsulinemic clamp (Prada et al. 2000). In addition, other studies have shown that a severe salt restriction induces increased plasma triacylglycerol and total cholesterol concentration in humans as well as in rats (Sharma et al. 1990, Ruppert et al. 1991, Catanozi et al. 2001). Taking these reports together, it seems that a severe sodium restriction could dysregulate insulin sensitivity and increase adiposity. However, the molecular mechanisms by which a low salt diet can induce insulin resistance are not yet completely understood. Under such conditions, insulin resistance may be tissue-specific to the liver and muscle, whereas adipose tissue remains sensitive to insulin, as seen in a previous study (Hirata et al. 2003).

NNR 2012: Public consultation – Chapter 32 (Potassium)

General and detailed comments	WG observations and actions
<p>University of East Anglia, UK</p> <p>General comments</p> <p>The text is similar to the 2004 Recommendations. On lines 108-110 it is stated that clinical trials and surveys published since then support the findings of the 2004 report. It would be helpful for the reader if the 2004 report was summarised (i.e. details of studies were not repeated) and instead the studies and survey published since 2004 were described in detail.</p> <p>Detailed comments</p> <ol style="list-style-type: none"> 1. The references need attention e.g. lines 137-139 refer to Ref 24. 2. On line 24, the dietary intakes are the same as NNR4 2004. Are these taken from the same database or are there newer data? If so, it would be good to comment on the fact that intakes have not changed despite changes in dietary patterns and food composition, including the use of potassium as a salt substitute. 	<p>Previous studies cited in NNR 2004 will be summarized in final draft</p> <p>OK. Updated in final version</p>
<p>Danish Nutrition Society</p> <p>There seems to be missing a reference to the meta-analysis mentioned on page 2, line 73-79 (I assume it is reference 9).</p>	<p>OK</p>
<p>Helsedirektoratet, Norway</p> <p>23. Bread and cereals may be included in dietary sources.</p> <p>28-30. The section on physiology and metabolism is very short, and may be expanded. See chapter on sodium and salt.</p> <p>106-17. It may be explained why figures for an average requirement is not given.</p>	<p>OK</p> <p>Not further elaborated</p> <p>Included in final draft</p>
<p>National Institute for Health and Welfare, Finland</p> <p>Line 12 Recommended intake of potassium for children aged 10 - 13 years. The recommended intake should be 2.9/3.3 g/d for girls/boys (NOT FOR BOYS/GIRLS)</p>	<p>Corrected</p>

NNR 2012: Public consultation – Chapter 33 (Iron)

Organisation Company	General and detailed comments (WG observation and actions inserted)
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Organisation Company	General and detailed comments (WG observation and actions inserted)
University of East Anglia, UK	<p>General comments</p> <p>1. The summary table (line 20) appears to have some of the population groups missing, including older children, pregnant and lactating women.</p> <p><u>WG observation and action:</u> This is the accepted style in the NNR, all chapters are similarly introduced with this kind of table. 2. The RI, according to line 381, is the quantity of iron that meets the needs of 90% of the population, whereas the AR is the quantity that meets the needs of 50% of the population. The convention is for RIs (or RDAs, PRIs etc) to be a value that meets the needs of 97.5% of the population (AR + 2SDs). The Nordic approach is therefore different and it needs to be explained very early on in the chapter.</p> <p><u>WG observation and action:</u> We use the conventional method in most cases, see chapter 3 ("Nutrition recommendations – background and principles"): „The term <i>recommended intake (RI)</i> refers to the amount of a nutrient that according to present scientific knowledge and information on dietary patterns can meet the known requirement and maintain good nutritional status among practically all healthy individuals in a particular life stage and gender group. When the distribution of a requirement among individuals in a group can be assumed to be approximately normally distributed (or symmetrical) and a standard deviation (SD) can be determined, the <i>RI</i> can be set as follows (Fig. 3.2): $RI = AR + 2 \text{ SD AR}$. For other nutrients where data about the variability in requirements are insufficient to calculate a SD AR, an approximate coefficient of variation (CV) of 10 -15 percent may be used (see Fig 3.2).“</p> <p>For menstruating women and iron recommendations NNR 5 calculated the recommendation in a slightly different manner. Justification for use of 90th percentile was (former unrevised version) given in line 341-350. Explained in the text: “The 90th percentile of need represents the recommended intake, and is justified by the fact that those in the top 5th centile of iron need, probably have a higher absorption rate than 15 %”, and that explains „The iron need for about 90 % of women is satisfied by 15 mg/day“ as well as „The iron need of about 90 % of post-menarchal girls aged 10-17 years is satisfied by 15 mg/day“, both also shown in table 2. This is now noted in the beginning of the sub-chapter (Requirement and recommended intakes”) and the text is rewritten, improved for clarity.</p> <p>3. How is the lower level of intake (line 21) derived?</p> <p><u>WG observation and action:</u> Again we refer to chapter 3: “The <i>lower intake level (LI)</i> is defined as a cut-off intake value below which an intake could lead to clinical deficiency symptoms in most individuals. Establishment of a <i>LI</i> is thus based on observations of individuals, and is in many cases based on criteria other than the average requirement.”</p> <p>Considering the recommendations of iron in NNR 5, this is explained in the text, which includes, and bases on the SR done for NNR 5: “In the recent SR on health effects and iron intake it was concluded that there is no reason to alter the NNRs on iron intake from 2004...”. <i>LI</i> of iron for adults is at the same level in NNR 5 as SACN 2010 did, which is $AR - 2 \text{ SD AR}$. This is now also referred to in the beginning of the rewritten sub-chapter: “Requirement and recommended intakes”.</p> <p>4. Some of the references are quite old and not necessarily the best evidence to support statements in the chapter. For example, ref 6 (line 89) is a paper on iron absorption in hemochromatosis, not iron deficiency; ref 8 (line 93) is the IOM report whereas primary data on the effect of ascorbic acid on iron absorption would be preferable; ref 9 (line 95) is old, more recent studies on the meat factor have been published and could be cited; lines 101-102 refer to copper and zinc absorption and more recent papers have been published on</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>transporters since ref 14 (line 102).</p> <p><u>WG observation and action:</u> Thank you for these observations. The references on these issues are now corrected or added, by more appropriate publications with primary or more recent data.</p> <p>5. The section that begins on line 175 describes the effects of iron deficiency, rather than symptoms. Should the findings of the systematic review be summarised in this section? The statement that cognitive function is probably only affected with IDA has been challenged in more recent years - effects of ID without anemia have been reported - perhaps this should be mentioned? Also it would be good to cite John Beard's work on temperature regulation instead of a textbook (ref 38).</p> <p><u>WG observation and action:</u> The text describes the effect of deficiency, now correct in the sub-title. Yes, there are references showing association between lower cognitive scores and less developed iron deficiency than anemia, this is now mentioned here (as well as before later in the text). Beard is now referred. We do find it too much of a repetition to have the findings of the SLR summarised here.</p> <p>6. Lines 235 onwards. What is the prevalence of ID and IDA in pregnant women in Nordic countries?</p> <p><u>WG observation and action:</u> ID is 10- 22% in women of childbearing age and IDA is 0-17% in pregnant women, see the second and the last sentences in this sub-chapter: "Prevalence of iron deficiency and iron deficiency anaemia"</p> <p>7. In Table 2, figures are given to one decimal place, but in the text describing requirement and RI, the numbers are rounded - could the text be more consistent with the table?</p> <p><u>WG observation and action:</u> Thank you for this observation. We have altered the table and use rounded values for intake necessary to cover 50%, 90% and 95% of groups' iron needs, given a diet with 15% iron absorbed.</p> <p>8. Line 323 onwards. Is it sensible to combine requirements of infants aged 7m with children aged up to 5y? Do these two age groups really consume the same quantity of iron? Is the AR based on usual intake (of healthy children), in which case it would be more accurately described as an Adequate Intake?</p> <p><u>WG observation and action:</u> The intake varies between age groups and countries, and is 6-10 mg/d on average at 7 months - 6 years. It does not change the reasoning behind the recommendation, which is higher than the mean intake.</p> <p>9. The whole section on requirements and recommended intakes is not very easy to follow. The basis upon which the figures are derived is not totally transparent.</p> <p><u>WG observation and action:</u> The section is now restructured to make it easy to follow.</p> <p>10. Line 550. The quantitative UL is set as 10 mg/d - in addition to usual dietary intake. It is not clear how this number was obtained. If 60 mg/d taken regularly leads to 'biochemical iron overload', this means that it is assumed that the diet supplies 50 mg iron/d - not a sound argument!</p> <p><u>WG observation and action:</u> Thanks the text has now been rewritten.</p> <p>Detailed comments</p> <p>1. Line 39. Ref 147 is a paper on 6y old children, not women of childbearing age. Could the references be renumbered so they appear in order. This section was obviously cut and pasted from the end of the document.</p> <p><u>WG observation and action:</u> This has been corrected and the references renumbered.</p> <p>2. Line 42. Typo - flour.</p> <p>3. Line 84 and elsewhere - spelling of 'haeme' should be 'haem' (En) or 'heme' (US).</p> <p>4. Line 116. Spelling - change 'advice' to advise.</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p><u>WG observation and action:</u> Now corrected in the document (to haem).</p> <p>5. Line 161. Begin the sentence with 'When iron supply is inadequate...'</p> <p><u>WG observation and action:</u> Done.</p> <p>6. Table 2, footnote 1. Green's data is good quality but Hunt et al (2009) have published some more recent data which should be cited. Does this change the calculation of basal losses?</p> <p><u>WG observation and action:</u> Hunt's reference is now used in the text, but it did not change the calculations in table 2. Basal losses are discussed in the introduction of Hunt's paper but not in the results or discussion. One can however guess that there is very little or no difference in basal losses between the studies, as the excretion values for men are not different between the references of Hunt and of Green. We chose to use the calculation unchanged. Green is also used by e.g. SACN 2010, as a reference to basal losses. There is a small difference in total iron excretion values, which may be because one uses average and the other medians. According to Hunt et al average iron excretion in mg/d was for men:1.07; all menstruating women:1.69; women without those using hormonal contraceptive:1.89; postmenopausal women:1.08. Table 2 median iron excretion values in mg/d are, men:1.05; menstruating women:1.35 (90perc.:2.22); postmenopausal women:0.87. The revised text considers this and other possible different values in the literature, i.e. menstrual losses, total losses, body weight.</p> <p>7. Footnote 5. What are these reference weights? It is normal practice to use the WHO reference weights for children.</p> <p><u>WG observation and action:</u> Nordic reference weights chosen of NNR experts for the age groups 10-13years and 14-17 years are used in this table (Danish children, before the increasing frequency of overweight/obesity). Other reference, and why these were chosen for the calculations are now discussed in the text.</p> <p>8. Line 389. Of instead of 'on'. 9. Line 412. Regimen instead of 'regime'.</p> <p><u>WG observation and action:</u> Done.</p> <p>10. Line 471. According to Hunt & Zeng (2005) and Roe et al (2005) heterozygotes do not absorb more iron than wild type men.</p> <p><u>WG observation and action:</u> This is now mentioned and new reference added.</p> <p>11. Line 489. From instead of 'form'. 12. Line 535. With instead of 'to'.</p> <p><u>WG observation and action:</u> Done.</p>
University of Bergen, Norway	<p>General comments only:</p> <p>In general I think the NNR 2012 on iron is useful with a good basis on the current evidence. The introduction and the sections on dietary sources and intake, physiology and metabolism, absorption and bioavailability, iron deficiency and iron deficiency anaemia, are in my opinion well presented. In the requirement and recommended intakes (line 267), there is a sentence "Vegetarians and athletes, especially female athletes, are two groups that may deserve special attention with respect to their iron needs." According to Hunt (Bioavailability of iron, zinc, and other trace minerals from vegetarian diets. Am J Clin Nutr 2003) and the position of the American Dietetic Association from 2009 (Position of the American Dietetic Association: vegetarian diets. J Am Diet Assoc 2009), there might not be necessary to single out vegetarians in this context. The American Dietetic Association argues that "Whereas many studies of iron absorption have been short term, there is evidence that adaptation to low intakes takes place over the long term, and involves both increased absorption and decreased losses. Incidence of iron-deficiency anemia among vegetarians is similar to that of nonvegetarians. Although vegetarian adults have lower iron stores than nonvegetarians, their serum ferritin levels are usually within the normal range."</p> <p><u>WG observation and action:</u> Thank you for the comments. We now use the references you point out about iron needs of vegetarians.</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>The sentence did and does not indicate that there is a big risk i.e. the word “may” is used in the sentence: “...vegetarians.... may deserve special attention with respect to their iron needs...”</p> <p>In the section on iron and risk of diabetes, it is written in line 526-531 that “But the facts that heme iron intake is closely determined by the intake of red or processed meat and that subjects with high heme iron intake had significantly less healthy behavior (diet, physical activity, smoking, BMI) (140), and the absence of an effect of total iron intake, makes it likely that there are other, unknown dietary or lifestyle factors rather than iron that increase the risk of T2D. Heme iron intake is most likely only an indirect marker of T2D development.” In the meta-analysis published in Am J Clin Nutr 2011 by Pan, Sun, Bernstein, Schulze, Manson, Willett and Hu (Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis), it was found that both unprocessed and processed red meat intakes were positively associated with T2D risk after adjustment for age, BMI, and other lifestyle (including smoking) and dietary risk factors (based on 4,033,322 person-years of follow-up). Thus, it could be justified to write e.g. that “Based on the meta-analysis by Pan et al. from 2011, the association between heme iron and T2D could be related to increased risk of T2D associated with intake of red meat”. Even though it is written correctly that “There is no conclusive evidence for an association of iron intake and the development of type 1 diabetes (15)”, for those reading quickly, it is easy to miss that this sentence is on type 1 diabetes, while the rest of the section is on type 2 diabetes. To avoid confusion, I suggest starting the sentence with “Regarding type 1 diabetes, there is no conclusive evidence for an association of iron intake”.</p> <p><u>WG observation and action:</u> Thanks. We have added the reference suggested and the explanation concerning red meat. We have also made it clear when talking about T1DM and T2DM.</p>
LRF Mjolk/LRF Dairy Sweden	<p>The comments from LRF Dairy Sweden (LRF Mjolk, tidigare Svensk Mjolk) are given by Helena Lindmark-Månsson, adjunct professor and nutrition expert, Annika Smedman, nutritionist and PhD, and Maja Nordström, M Sc Agr. General comments: Please update the chapter with the result for Riksmaten 2010-11.</p> <p><u>WG observation and action:</u> Has been updated. Intake values are also collected in a specific chapter in NNR 5.</p> <p>The references are generally very old. Update the chapter with current papers. If there are no more recent papers, state that. Also regarding calcium inhibiting the absorption of iron, the references used are old. Calcium inhibiting the absorption of iron was the subject of an expert meeting in collaboration with Swedish Nutrition Foundation and Swedish Dairy Association, held in 2006. Experts participating were e.g. Lena Hultén, Nils-Georg Asp and Bo Lönnerdal participated in the meeting. For information: http://www.svenskmjolk.se/Mjolk-smor-och-ost/Naring/Vitaminer-och-mineraler/Mjolk-fungerar-med-jarnrik-maltid--rapport-fran-forskarmote/#Oförändrad_järnstatus_av_högt_kalciumintag The conclusion from the meeting was that a high intake of dairy products didn't affect the iron intake in the long run. This conclusion was also informed on in Scandinavian Journal on Food and Nutrition 2006, 50, 1. Calcium does not impair iron status in practice. Include the text of the chapter on iron that in the long term there is no importance of the inhibition of calcium on the iron intake. Further, the Swedish National Food Administration write specifically in their dietary advices for pre school (Råd för mat i förskolan, 2010) that there are no longer any scientific evidence that milk inhibits iron absorption. The inhibiting effects on iron uptake of for example phytates should also be discussed.</p> <p><u>WG observation and action:</u> References have been exchanged for more recent ones but original works (often older) are cited when appropriate. The information about enhancers and inhibitors of iron absorption (including a negative association between calcium intake and iron absorption) is valid, and so is also the information of less and sometimes no effect seen in whole diet studies, or studies</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>of longer duration. This is clearly explained in the chapter, see e.g. the second and third last paragraphs in the sub-chapter “Absorption and bioavailability”. This is now also made very clear in the second sub-chapter: “Dietary sources and intake”, in the later paragraph (see the last lines now added). Thank you for the references mentioned, but they are not suitable for the NNR 5 chapter. Phytates are mentioned 6 times in the text, see e.g. the second first and later paragraphs in the chapter “Absorption and bioavailability”, explaining their inhibit effect on iron absorption.</p> <p>Detailed comments Line 317-322: It should be mentioned that it however is important to introduce tasting portions to infants from 6 months, according the guide lines written by Swedish National Food Administration (Bra mat för spädbarn, 2011). <u>WG observation and action:</u> Thank you, this is pointed out and discussed in another chapter in NNR 5.</p>
National Institute for Health and Welfare (THL), Finland	<p>General comments only: Comments to the chapters of Iron, Vitamin D, Calcium and Phosphorus. GENERAL: In all chapters concerning the micronutrients and vitamins there should first be the table of what is the new recommendation for different age groups and pregnant/lactating. If there are changes from the previous recommendation, then this should be in a table and reasoning for the change. Right after the table, the paragraph should begin with the chapter ‘Reasoning behind the recommendation’. The chapters should be in following order: NUTRIENT X Table 1 : new recommendation for the nutrient X Table 2 : upper intake level and toxicity for the nutrient x Reasoning behind the recommendation Physiology and metabolism (briefly, referring) Requirements and recommended intakes for: - infants - children and adolescents - adults - pregnancy and lactation - older adults and elderly - special needs when relevant (cancer, DM, CVD, Obesity, etc) Also these chapters could be more focused and a lot shorter, not including all the facts like a teaching manual, but referring to the other actual texts like the IOM recommendations. All the comment rounds have included very incoherent chapters, as if the writers wouldn’t have the same writing guidelines and wouldn’t write into the same book. Various age groups should be named in the same way in all chapters. There should be an own chapter about supplementation; why different practices in different countries, where do these come from, etc. For instance where comes the new age limit in vitamin D recommendation? Why did it change? If countries don’t agree, then it is important for experts to know why and be able to rationalize himself. <u>WG observation and action:</u> Thank you. Your suggestions about a different construction of the chapters about nutrients are valid and will most likely be discussed later. The structures now used, based on the guidelines for writing the chapters in NNR 5, are as similar between chapters as we could do them for now. We hope we now mention clearly when evidence is not secure and we try to tell or explain when results/opinions differ. For specific problems it might be more relevant to look at the SLR associated with the NNR.</p>
Norwegian	General comments

Organisation Company	General and detailed comments (WG observation and actions inserted)
Directorate of Health	<p>Please add conclusions at the end of sections concerning intake and outcomes. See how this is done in the Calcium chapter. We strongly recommend that you include a section concerning “Reasoning behind the recommendation”. In such a chapter you may add a short discussion or comments concerning differences regarding the recommendations made by NNR and other expert groups as DRI by IoM, USA. You may also in this kind of section integrate text from the SLR, Authors conclusions.</p> <p><u>WG observation and action:</u> Thank you this is done now, underlining RI in the text (see also marked altered text in the chapter)</p> <p>Detailed comments</p> <p>21. Please add conclusions on UL from page 14.</p> <p><u>WG observation and action:</u> The UL is not added to the tables in the NNR 5.</p> <p>24. ... most common micronutrient deficiency – you may up-date reference 1</p> <p><u>WG observation and action:</u> References, especially in the introductory text, are now updated. We do not find it necessary to update this one, i.e. number 1.</p> <p>568. If you do not add a conclusion in relation to UL at 21 please add a conclusion here.</p> <p><u>WG observation and action:</u> Conclusions added.</p>
Animalia - The Norwegian Meat and Poultry Research Centre	<p>General comments only:</p> <p>Animalia – The Norwegian Meat and Poultry Research Centre appreciates the opportunity to comment on the chapter “Iron” in NNR5.</p> <p>Line 50 The draft sentence “For these vulnerable groups, consumption of foods inhibiting absorption should be limited or avoided with main meals containing iron, and if possible foods with enhancing factors, such as vitamin C, included” should also include an encouragement to choose iron containing foods with high bioavailability, like fish, poultry and meat.</p> <p><u>WG observation and action:</u> There is a sentence: “...iron rich foods such as variety of meat and lean meat” in the paragraph above the one you point out, approx. 4 lines above this sentence. Also e.g. in the sub-chapter “Absorption and bioavailability” see e.g. the first 4-5 lines in the second paragraph, also stating information about haem iron in meat. This is mentioned several places in the chapter, no more is needed.</p> <p>Line 54 The sentence “Meat, fish and poultry contain the MFP-factor, which also enhances iron absorption in the meal” is only focusing on the MFP factor, but should also include the fact that the iron in meat, fish and poultry has good bioavailability. Suggested reformulation: “Meat, fish and poultry contain haeme iron of high bioavailability, in addition to the MFP factor, which also enhances absorption of non-haeme iron in the meal”.</p> <p><u>WG observation and action:</u> Please see the answer above. As well as the sub-chapters and paragraphs mentioned above.</p> <p>Line 235 Iron status on two year old children in Norway should be included in the text and references. This study is the most comprehensive among the most recent ones (1). Since the data collection was conducted spring year 1999 and the iron supplementation of the Norwegian brown goat cheese was ceased in year 2000/2001, the iron status of the two year old children might even have been exacerbated since then. The brown goat cheese contributed with 9 % of the iron in the diet (1). 9 % of the children had serum ferritin < 10 microgram/Liter and 10 % had hemoglobin < 105 g/L. 6 % of the children had iron deficiency anemia, defined as hemoglobin < 110 g/L and serum ferritin < 12 microgram/L (1,2). References 1. Hovland E. Kosthold og jernstatus hos norske toåringer – en undersøkelse av friske norske toåringer i Oslo. Cand. Scient oppgave i klinisk ernæring. Johan Throne Holst Institutt for ernæringsforskning, Det medisinske fakultet, Universitetet i Oslo, 2002.</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>www.cristin.no/as/WebObjects/cristin.woa/wo/7.Profil.29.25.2.3.15.1.1.3 2. Hay G, et al. Iron status in a group of Norwegian children aged 6-24 months. Acta Paediatrica 2004; 93(5): 592-98 ISSN 0803-5253 www.ncbi.nlm.nih.gov/pubmed/15174778</p> <p><u>WG observation and action:</u> Thank you. We have added a sentence about the Norwegian two year olds. We also use reference Hay et al 2004 (formerly number 68, now with a new number).</p>
National Food Agency, Sweden	<p>General comments</p> <p>Clarity can be improved, especially concerning iron supplement during pregnancy and of T2D-associations with iron and the part on iron and cancer. A general table is needed on the content of haeme vs non-haeme iron in different cuts of different species of meat, and also on other food sources for iron.</p> <p><u>WG observation and action:</u> Thanks for all the wise comments. We have rephrased</p> <p><u>Detailed comments</u></p> <p>35 Recent dietary surveys... please revise long sentence to improve clarity 37 products where (??).</p> <p><u>WG observation and action:</u> Both done, thank you.</p> <p>40 ...higher in 6-year olds compared to which group?</p> <p><u>WG observation and action:</u> Clarified, thank you.</p> <p>46 Does comment on ceased fortification refer to flour fortification?</p> <p><u>WG observation and action:</u> Yes, mainly.</p> <p>47 “such as variety meats and lean meat” should it be “such as a variety of meats and lean meat” Comments the word meats does not exist, clarify what you mean by variety, different species (pigs, cattle, lambs, etc). Why lean meat, and compared to what? Is the fat content important for the iron content? Actually you should write about “meat and meat products from different species.”</p> <p><u>WG observation and action:</u> Thank you we have now included the word “of” in the sentence and deleted “s” from meat. In general lean meat has more iron per weight unit than fat meat. It is important to underline that lean meat gives iron (is a good source of iron in the diet). We do not find it relevant here to discuss meat from different species.</p> <p>49 Please consider to move this paragraph under the heading “absorption and bioavailability”</p> <p><u>WG observation and action:</u> This part is meant to be a short summary of these specific properties of iron, related to “Dietary sources and intake”, the paragraph has been improved now. 54 MFP factor is mentioned here, but defined on row.</p> <p><u>WG observation and action:</u> Changed now for improvement of the paragraph, here with “MFP-factor” in parenthesis.</p> <p>94. Better to explain the MFP the first time mentioned. Reference is missing about the MFP-factor on row</p> <p>54. <u>WG observation and action:</u> The reference is in in the sub-chapter “Absorption and bioavailability”, and that sentence is now depending on the first about the factor.</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>57 Please consider the word “staple food” for milk, could the sentence instead read : “...milk is an important source of several nutrients and should not be eliminated from the diet due to its effect on iron absorption”? <u>WG observation and action:</u> Done, thank you. 85 “10% of total iron in Nordic diet” Reference is missing. 85 “...where it accounts for about half the total iron.” what is the other half? Nonhaem iron, should be added. There is probably different percentgs of haemiron in different species (and cuts). It would be informative to have a table on iron content in different cuts of species, and the percentage of haem iron of total iron in pig, cattle, elk, deer and also in e.g. chicken compared to ostrich. It would also be informative to list some food containing iron other than meat, e.g. fish, egg, and some vegetables: cereals, spinach etc. <u>WG observation and action:</u> Non-haem is added. We do find it too detailed to discuss the iron amount in meat from different species.</p> <p>85 specify the components of haeme iron, i.e. myoglobin and cytochromes (red colour) Compare text on page 118 in this reference: www.wcrf.org/PDFs/WCRF%20International%20Conference%202010%20Abstract%20Book.pdf . <u>WG observation and action:</u> We do not find it important to discuss the components of haem iron. There is no page 118 in this reference you mention, the last page is number 71.</p> <p>93 “100 mg/day”. What is this number related to? Non-haeme iron in meals?? <u>WG observation and action:</u> Vitamin C. The text has been improved and this is clear now.</p> <p>112 Diminish? Or reduce? <u>WG observation and action:</u> Diminish replaced by reduce.</p> <p>115 “..individuals with marginal iron stores” What is meant by “ marginal iron stores”? <u>WG observation and action:</u> ID or close to the reference values of ferritin for ID. This is explained in the sub-chapter: “Iron deficiency and iron deficiency anaemia”, sub-heading: “Development”, and other places in the chapter.</p> <p>116 Suggested alternative: “There is no reason to advise against tea in conjunction with meals for the general population” <u>WG observation and action:</u> The suggested sentence has been added to the text.</p> <p>128 “one glass of milk (165 mg calcium) caused a 50 % reduction in iron absorption” is the reduction of iron independent of the level of iron in the diet? <u>WG observation and action:</u> Not of importance.</p> <p>156 “with poor iron status”, give a number what poor is 222 abbreviations in the table must be explained: MCV, TIBC, ZPP. Clarify MCV the sort fL <u>WG observation and action:</u> All these abbreviations are explained in text twice, e.g. in the same sub-chapter, 2 paragraphs above, they are also standard abbreviations. The measure unit for MCV, fL, is not explained, and that has now been added as a footnote to the table.</p> <p>243 “Iron status improves at the age of two..” consider separate from end of sentence “at one and two years of age poorer iron status... to improve clarity. <u>WG observation and action:</u> Done.</p> <p>255 “(s-ferritin>300 mg/L)” how high ca s-ferritin be? Is this extremely higt or too high? <u>WG observation and action:</u> It is high (the upper</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>level is about there, i.e. 300, but varies between laboratories, this is already explained in the text several times e.g. the last sub-chapter; "it seems clear that an s-ferritin level above 300 µg/l, which is often referred to as 'biochemical iron overload'). However, it is not easy to say what is too high for this percentage of this age group (see text) and should actually not be done here. For your information: High amount of iron stored in the body might mean haemochromatosis (and it varies if other symptoms are seen or not, but those could be heart failure, joint pains, liver cirrhosis, diabetes, sexual dysfunction, fatigue, and darkening of skin). High ferritin is also a secondary symptom to disease and could point to some other diseases e.g. liver disease, rheumatoid arthritis, or other inflammatory conditions.</p> <p>265 ...not normally distributed within the population group <u>WG observation and action</u>: Exactly, of course. Not added. 275The Hb concentration in infant blood (not to confuse with milk values as discussed before). <u>WG observation and action</u>: The word "birth" is right after in the sentence, but we changed it anyway and it now says: "Infants' blood Hb concentration falls rapidly after birth...."</p> <p>281 Present and past tense in sentence <u>WG observation and action</u>: Thanks, corrected and clarified.</p> <p>305 a metaanalysis <u>WG observation and action</u>: The "a" is added.</p> <p>319 "It therefore seems prudent to avoid such high intakes of cow's milk in infants and toddlers." What level is recommended? <u>WG observation and action</u>: There is old (not all referred to here) and new evidence that high levels, i.e. 300 ml of milk and above (according to the SR for NNR5) are too high for the age group in question (infant formula was developed in the former half of the last century because of this). There is no further specification of recommended level of intake of usual cow's milk considering iron.</p> <p>323 "The basic requirements... please revise into several sentences <u>WG observation and action</u>: Done.</p> <p>409-414 How does the basis of this recommendation on 40 mg iron relate to the background of the recently revised WHO guidelines on 30-60 mg iron daily, the 60 mg iron for women in areas where anemia and IDA is a serious public health problem? Do Nordic women need a higher dose as prophylaxis than the lowest dose recommended by WHO? Please note the adverse effects on pregnancy outcome and maternal health of supplementation and of high Hb during pregnancy as cited in the WHO guideline, 2012. From what gestational age is this prophylaxis recommended and for how many weeks? Are there any contra-indications of this prophylaxis? <u>WG observation and action</u>: The text has now improved and explains this, and does not use the word "recommended" but advised. (This is also answered in the SR, referred to in the chapter.)</p> <p>419 Menstruation and related iron losses starts in partial breast feeding, but iron losses due to milk production is also less when breast feeding partially? "Quite a long time" is relative to the population of comparison, maybe it is better to give a figure for comparison. <u>WG observation and action</u>: This is now more clear "women may have low iron stores postpartum and many women in the northern countries breastfeed their infants for quite a long time, i.e. several more months than half a year, and menstrual losses start within the partial breastfeeding period. Therefore recommendations for lactating women are the same as for women of childbearing age who are not pregnant, or 15 mg/d."</p> <p>438 Is 40 mg iron daily safe as prophylaxis during pregnancy when adding iron from dietary sources? What is known about the</p>

Organisation Company	General and detailed comments (WG observation and actions inserted)
	<p>regulation of iron homeostasis during pregnancy to support this prophylactic dose? <u>WG observation and action</u>: The text has now improved and explains this, and does not use the word “recommended” but advised. (This is also discussed in the SR, referred to in the chapter.)</p> <p>461 in heading ...effects of iron overload/excess iron? <u>WG observation and action</u>: This title will be: “Chronic effects of iron overload”. The title above is: “Acute effects of iron overload”.</p> <p>489 ..from high intake... <u>WG observation and action</u>: Ok now corrected.</p> <p>500 please clarify this suggestive effects at different alcohol intakes <u>WG observation and action</u>: We refer to the SR paper referred to in the chapter.</p> <p>506 This part is of great interest in relation to the debate on decreasing meat consumption in the population, which is discussed in the WCR book from 2010: www.wcrf.org/PDFs/WCRF%20International%20Conference%202010%20Abstract%20Book.pdf One hypothesis discussed, is that the iron (haem) content of food (i.e. meat iron) should relate to some cancers. It is important that NNR more evidently discuss a bit on this issue. <u>WG observation and action</u>: Yes it is true this is an interesting hypothesis. Thank you for the reference showing many abstracts, a few on iron or meat and cancer. We say as much as evidence allows us to do: “Several new studies have examined the possible relationship between dietary iron intake and various forms of cancer (129 - 138). However, no convincing evidence that dietary iron intake is associated with increased risk of colon cancer, lung cancer, breast cancer, esophageal cancer or other cancers, could be found (15).”</p> <p>519 examined <u>WG observation and action</u>: Has been corrected.</p> <p>530-537 Maybe “unknown” can be taken out? Refers to factors not properly measured or accounted for in analysis. This part is not clear and can probably be revised for clarity. What are the effects seen, why do authors not believe in the associations and what are the authors' conclusions? <u>WG observation and action</u>: This has now been made more clear: “...But the facts that haem iron intake is closely determined by the intake of red or processed meat and that subjects with high haem iron intake had significantly less healthy behavior (diet, physical activity, smoking, BMI) (140), and the absence of an effect of total iron intake, makes it possible that there are other dietary or lifestyle factors rather than iron that increase the risk of T2D. Haem iron intake might be an indirect marker of T2D development. However, a meta-analysis found intake of both unprocessed and processed red meat positively associated with T2D risk after adjustment for age, BMI, and lifestyle such as smoking (X14). The association between haem iron and T2D, could therefore be related to increased risk of T2D associated with intake of red meat.”</p> <p>548 Why can a level of iron that increase the risk of iron overload be recommended to all pregnant women? <u>WG observation and action</u>: This is actually not the case, see the rewritten UL chapter. (Also, actually WHO mentions the amount 60 mg/ several times a day to be the way to improve low ferritin in pregnancy.)</p>

Organisation/ Company	Zinc General and detailed comments	WG response and actions
	<p> http://www.ncbi.nlm.nih.gov/pubmed/4097279 http://www.ncbi.nlm.nih.gov/pubmed/4116227 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1991851/ http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1991648/ These papers did not report any important adverse effects of zinc in the doses used. If the NNR states that the “safe range” extends only to 25 mg/day (UL), then the reader thinks that this UL is based on evidence that some 50 to 100 mg/day of zinc would be harmful. However, the above reports indicate that people can take up to 150 mg/day zinc for long periods of time without any actual harm. Evidence-based medicine emphasizes that we should focus on clinically relevant outcomes, whereas the small changes in copper and blood lipids, mentioned in the NNR draft, are surrogates and do not necessarily mean any long term health effects. I do not know studies that would indicate that ordinary people would benefit of zinc supplementation above the usual diet. In that respect the UL is not a big issue. However, there is a risk that a very low UL would discourage testing zinc for some specific groups of patients such as those included in the papers listed above. Furthermore, there is strong evidence that zinc lozenges in doses over 75 mg/day can shorten the duration of colds by up to 42%: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3136969/ The relevant time scale in this case is 1-2 weeks of treatment. Given that 150 mg/day zinc is safe for months to years and given the lack of any relevant adverse effects in the zinc lozenge trials, there is no basis to discourage people from using zinc lozenges for treating the common cold in doses providing 75 to 100 mg/day of zinc. If the UL for zinc is set at 25 mg/day, then many readers would assume that 75-100 mg/day would be a harmful dose, whereas there is no such evidence. In my view the safety section should be rewritten so that the many papers reporting on the use of 150 mg/day of zinc for months are taken into account. In the current draft, all the above reports are ignored. In addition, the text should state that there is no evidence that zinc is not safe at doses up to 100 mg/day for a few week period. Than the NNR would not discourage people from using zinc lozenges for treating colds. Lines 231 to 233 I cannot see the rationale of NNR to copy the UL from a EU-report and pretend that the NNR is based on independent reasoning. If the purpose of NNR is to carry out independent assessment of evidence, then the opinions of other organizations are not essential. Obviously, the zinc author should check other documents for their references and arguments, but the kind of copying of UL is unreasonable. NNR does not provide any own argumentation to support the UL of 25 mg/day for zinc. </p>	

Organisation/ Company	Zinc General and detailed comments	WG response and actions
Vitenskapsko miteen for mattrygghet (VKM), Norway	<p>Deatiled comments</p> <p>Comments on Upper Intake Levels and toxicity (line 220-238) The EFSA opinion on UL for zinc is cited, and the derivation of UL for children in the EFSA opinion is explained as follows: "As there are no data on adverse effects of zinc intakes on children and adolescents, they extrapolated upper levels for children on a surface area basis." The NNR UL for zinc then concludes that UL for children is 12.5 mg/day - half of the UL for adults without any explanation for this conclusion or age groups of children. Is 12.5 mg/day valid for all agegroups from 1-18 years. The conclusion in NNR for UL children differs from the UL for children set by EFSA without any explanation for this difference. The following sentences might also be misunderstood: "The risk of excessive intake of zinc from food alone is very low. Symptoms of acute toxicity from excessive intake occur at intakes of gram quantities of zinc and are related to consumption of dietary supplements. At considerably lower intakes, there is a risk for negative effects on the metabolism of other trace elements, especially copper." At considerably lower intakes seems like an unprecise phrasing, and from the text it is also uncertain whether the risk of intakes interfering with copper from food alone is unlikely, or is toxic intakes from food alone unlikely.</p>	<p>The section on UL has been rewritten based on several comments from the public hearing.</p>
National Food Agency, Sweden	<p>Detailed comments</p> <p>9 Add UL - in box as for other nutrients</p> <p>30 We suggest that the sentence starting with "Foods with a high content of fat and...." is removed as it is generally true for several nutrients 237 Replace "it is recommended" with "EU SCF recommended" to avoid misunderstanding that this is a recommendation by NNR. Make a clear conclusion that no UL is set in NNR 5.</p>	<p>Included</p> <p>The section on UL has been rewritten based on several comments from the public hearing.</p>
Helsedirektora tet, Norway	<p>Detailed comments</p> <p>9-11. You may include figures or other information regarding UL in the table.</p>	<p>Included</p>

Organisation/ Company	Zinc General and detailed comments	WG response and actions
DI Fødevarer, Denmark	<p>General comments</p> <p>The Danish Food and Drink Federation hereby wants to provide comments to the draft chapter on zinc. Comments relating to Upper intake levels and toxicity (I 220-238) We believe it is important to indicate whether the UL's established are based on all sources or only supplements. Therefore it should be stressed that UL of 25 mg is for supplements only. It is evident from the quoted studies used by the Scientific Committee, but not stressed in the assessment itself. See risk assessment of Expert Group on Vitamins and Minerals (EMV, 2003). UL including all sources is established by CRN, IOM or FAO/WHO, all of which acknowledge a UL for zinc of 40 mg from all sources. A UL of 40 mg is also suggested in the Bonham study included in the review (Bonham et al., BMJ (2003), 695-703).</p>	<p>The section on UL has been rewritten based on several comments from the public hearing.</p>

NNR 2012: Public consultation – Chapter 35 (Iodine)

First name	Organisation/company	Comments	Response
Aileen Robertson	Metropolitan University College CPH and Danish Nutrition Society	Several editorial comments. Question if it could be useful to add few lines relating to the public health concerns about using salt for delivery of iodine. Inclusion of more specific recommendation for infants and children in the text.	We want to thank for a thorough overview and have made changes in the text according to the suggestions. Recommendations for infants and young children are now discussed in more detail. However, we did not include concerns about using salt for delivery of iodine. The main reason being that the chapter is dealing with recommendations on iodine intake, not how to reach the recommended intake level in each country. As not all the Nordic countries depend on salt as a source of iodine we did not find it appropriate to discuss iodized salt in more detail.
Anne Lise Brantsaeter	Norwegian Institute of Public Health	Comments related to the interpretation of results from the MoBa study. Suggestion to include historical data from Norway and Iceland as well as from Sweden, Finland and Denmark in the introduction section.	We appreciate very much your detailed and valuable comment, as well as suggestions how to correctly refer to the MoBa study. We have included suggested reference: Brantsaeter AL, Haugen M, Julshamn K, Alexander J, Meltzer HM. Evaluation of urinary iodine excretion as a biomarker for intake of milk and dairy products in pregnant women in the Norwegian Mother and Child Cohort Study (MoBa). Eur J Clin Nutr. 2009; 63:347-54, as well as a recent publication of relevance for the iodine chapter: Brantsæter AL, Abel MH, Haugen M, Meltzer HM. Risk of suboptimal iodine intake in pregnant norwegian women. Nutrients. 2013 Feb 6;5(2):424-40. doi: 10.3390/nu5020424. As suggested, historical information from Norway and Iceland have been included in the introduction.
Ewa Björkeson	Riksförbundet Hälsofrämjandet	Line 26-27. It might say that Iodine is especially high in Kelp.	As kelp is not part of a habitual diet in the Nordic countries, we don't find this suggestion relevant.
Björn Hammarskjöld	Minivent AB	Missing information about the goitrogenic effect of F in food.	It is acknowledged that high fluoride intake/exposure has been associated with effects on thyroid metabolism, However, at exposures necessary for caries prevention provided from tooth paste or drinking water has not been

First name	Organisation/company	Comments	Response
			shown to affect thyroid function. This is covered in the chapter on fluoride.
Iris Erlund	Nutrition	Comments about contradictory sentence related to iodine status in the Nordic countries.	The sentence has been removed, as we agree that the conclusion is not based on good data. We agree that there is need for better surveillance and more data on the level of iodine intake that ensures maternal and newborn euthyroidism, not least in the Nordic countries, as stated clearly in the revised chapter.
Helena Lindmark Månsson	Swedish Dairy Association	The iodine content in other foods than seaweed should be included, line 25-33. Iodine sources in the diet should be included.	This is a relevant comment. However, instead of including information about iodine content of different food, we excluded the information on the iodine content of seaweed, as the iodine content of different food might differ in the Nordic countries and the national nutrient databases should be used as source of such information.
Lars Johansson	Helsedirektoratet	Methods for measuring iodine excretion and interpretation of these data should be expanded in this chapter. 81-7. Here methods for measuring iodine excretion and interpretation of these data should be expanded. New knowledge regarding thyroglobulins from the last 5-10 years may also be included in this section/chapter.	As many high quality papers have been published in the past years related to methods for measuring iodine excretion and interpretation of these data, we don't find it appropriate to include this information in the chapter. However, we have included a systematic review covering methodology (Ristic-Medic et al. 2009) which will bring the reader to the right track in retrieving detailed information on the methodology.
		International cooperation has reduced iodine deficiency in many countries the last decades. This should be reflected in the introduction. Norway may here be added to Sweden and Finland. The details about Denmark are very important information, but they may be moved to the next section about dietary sources.	We have included historical information regarding Norway and Iceland in the introduction section, but decided to keep the Danish information in the introduction rather than moving the information into the section on dietary sources. The introduction is more balanced in the revised version, covering all the Nordic countries. As the recommendations are focused on the Nordic setting we don't find it appropriate to include the global aspect.

First name	Organisation/company	Comments	Response
		Please give information about the levels of iodine in salt in the different countries, and also add information about what is common levels globally.	We decided not to include detailed information regarding iodized salt in the chapter. The main reason being that the chapter is dealing with recommendations on iodine intake, not how to reach the recommended intake level in each country. As not all the Nordic countries depend on salt as a source of iodine we did not find it appropriate to discuss iodized salt in more detail.
		<p>37-8. Fodder; Iodine levels in milk seems to be at a similar level in Denmark, Norway and Sweden. This could be commented.</p> <p>55. ... increase metabolism ... please explain – do you mean regulate?</p> <p>62. Please explain what happens to the other 10 % of the iodine?</p>	<p>We decided not to include information on iodine content of different food in the chapter as it might differ somewhat in the Nordic countries. The national nutrient databases should be used as source of such information. Line 55. Yes, this is corrected, thank you.</p> <p>This is unabsorbed and excreted in the faeces.</p>
		The conclusion regarding differences in iodine status in the Nordic countries should be updated with reference to Andersson 2012. You may also add some of the few facts about iodine excretion (status) in the Nordic countries, especially for Denmark where health authorities has made efforts to reduce the level of goiter in the population the last ten years.	This sentence has been deleted, as it was based on insufficient data. The main conclusion is the lack of data and need for need for better surveillance in the Nordic countries.
		Please explain the interpretation of and relations between intake, UIC and UIE for the setting of recommendations in the text or/and in a table. 123-31. Please explain if and why NNR deviate from the WHO recommendation.	The interpretation of the MoBa study was not correct in the previous version of the chapter. This has also been pointed out by the scientists involved in the MoBa study and the text changed accordingly.

NNR 2012: Public consultation – Chapter 36 (Selenium)

	Selenium. General and detailed comments	WG observations and actions
University of East Anglia, UK	<p>General comments</p> <ol style="list-style-type: none"> 1. There have been a number of studies on selenium carried out and published since NNR4 2004, and the results have been used to argue for a change in the Recommendations. The use of selenoprotein P as the endpoint (instead of GSHPx) is an advancement, but it might perhaps be worth citing the conclusions of Combs et al 2011 (Combs GF Jr, Watts JC, Jackson MI, Johnson LK, Zeng H, Scheett AJ, Uthus EO, Schomburg L, Hoeg A, Hoefig CS, Davis CD, Milner JA. Determinants of selenium status in healthy adults. <i>Nutr J.</i> 2011 Jul 18;10:75) in order to present caveats for the use of selenoprotein P. 2. It is important that the evidence used to derive the new figures is described as comprehensively and transparently as possible. One of the problems with selenoprotein P is the fact that only a few centres in the world are able to measure it, which means that many studies still rely on serum/plasma selenium as an endpoint for examining the response to changes in selenium intake or cross-sectionally to explore the association between habitual intake and selenium status. The technical difficulties of measuring selenoprotein P (e.g. Ballihaut G, Kilpatrick LE, Kilpatrick EL, Davis WC. Multiple forms of selenoprotein P in a candidate human plasma standard reference material. <i>Metallomics.</i> 2012 Jun;4(6):533-8) should be mentioned in the chapter. This will give the reader an appreciation of why the data are so limited. 2. The section describing the reasoning behind the recommendation (lines 207-216) uses the results of a Chinese study to generate new recommendations. It is unfortunate that the population is not Western but it is probably the only study which can be used for the purpose of deriving Recommendations based on selenoprotein P. The intervention by Hurst et al 2010 (Reference 16) found that selenoprotein P in men and women reached a plateau at intakes of somewhere between 55 and 105 ug/d. The intakes in the Chinese study that optimise selenoprotein P appear to be lower. Is this worth mentioning? <p>Detailed comments</p> <ol style="list-style-type: none"> 1. Lines 8-9. The main biological functions of selenium are antioxidant activities and thyroid hormone metabolism. This statement could perhaps replace the 'thought to be mediated by glutathione peroxidase enzymes...' sentence. 2. Line 11. Replace 'kinetics' with metabolism. 3. Line 12. Delete 'for man'. 4. Line 23. the composition of foods varies according to the selenium content of the soil on which crops are grown and on which animals graze - it is not necessarily annual variation, nor is it dependent on the 'quality' of the crops. 5. Line 31. The references need renumbering. 6. There is some repetition in the paragraphs beginning on line 43 and on line 59. 7. Lines 69 and 70. Either remove 'organic' or provide evidence that the biomarkers cannot be used 	<p>The suggestion of including Combs et al article has been followed up.</p> <p>Ballihaut et al has been included as well as a description of the Chinese study which uses selenoprotein P as a biomarker of status.</p> <p>We think the discrepancy between the British and the Chinese intervention studies already has been covered: the British study has 50 ug Se as its lowest dose and thus it does not allow for conclusions of Se intake lower than this dose.</p> <p>All the comments here have been followed up. In some instances the phrasing or sentence has been deleted.</p>

	Selenium. General and detailed comments	WG observations and actions
	<p>for inorganic (as well as organic) selenium.</p> <p>8. Lines 75-78. This is not clear.</p> <p>9. Line 99. Suggest the sentence is changed to ...Three syndromes are associated with selenium deficiency, and that mention is made of viral infections in relation to Keshan disease.</p> <p>10. Line 128. Provide a reference for this statement.</p> <p>11. Line 130. Delete 'previous'.</p> <p>12. Line 133-134. Give evidence for the statement that intakes of 80 and 120 ug/d are required to maximise GSHPx activity in RBCs and platelets.</p> <p>13. Line 140. Provide a reference for this statement.</p> <p>14. Line 191. As pointed out by Hurst & Fairweather-Tait (Selenium and Vitamin E Supplementation for Cancer Prevention JAMA. 2009;301(18):1876-1877. doi:10.1001/jama.2009.626) in the SELECT study, median baseline serum selenium was relatively high at 135 µg/L. After selenomethionine supplementation it reached 250 µg/L, potentially above the beneficial range. A study by Bleys et al(2008) found a nonlinear relationship between serum selenium and risk of prostate cancer mortality; low serum selenium was associated with increased risk of mortality compared with the 120- to 150-µg/L range, and high serum selenium (>150 µg/L) was potentially associated with an increased risk of mortality. It might be worth expanding this section as the point about the effect of baseline selenium status is not widely appreciated. There is also a systematic analysis by Hurst et al 2012 (Hurst R, Hooper L, Norat T, Lau R, Aune D, Greenwood DC, Vieira R, Collings R, Harvey LJ, Sterne JA, Beynon R, Savović J, Fairweather-Tait SJ. Selenium and prostate cancer: systematic review and meta-analysis. Am J Clin Nutr. 2012 Jul;96(1):111-22) that might be worth citing.</p> <p>15. Lines 199-200. What duration? What endpoint? Cancer? By Western populations are North Americans excluded - what about the SELECT trial? The reference is Rayman 2000, which is old (see more recent review by Rayman, Lancet 2012;379:1256-68). Assuming this paragraph does not relate just to clinical trials on cancer (mention is made of cardiomyopathy so it must be a general paragraph), there are quite a number that could be described. This section needs updating - perhaps separate into two paragraphs, one on cancer (or add new information to the paragraph describing the NPC and SELECT trials) and one on non-cancer end-points. There is also the more recent research on differences in response to selenium in relation to genotype that might be worth citing.</p>	
Claus Hancke	<p>General comments</p> <p>Selenium µg/d Recommended intake RI 200 Average requirement AR 100 Lower level of intake LI 40 Upper level of intake UL 800</p>	Comments acknowledged. DRVs maintained.

	Selenium. General and detailed comments	WG observations and actions
Minivent AB	<p>General comments</p> <p>According to NNR5 own wording 145 Information on selenium requirements for children and pregnant and lactating women 146 is incomplete. During continued lactation, the selenium concentration of mother's 147 milk is reduced over time when selenium intake is less than 45-60 µg/day, but remains 148 unchanged at intakes of 80-100 µg/day. 149 150 This indicates that pregnant and lactating women should have at least 100 µg Se per day If the women get less Se they are depleting their own normal supplies of Se. Obey the physiology!</p>	<p>The recommendation for pregnant and lactating women of 60 µg/d, accounts for the increased needs for tissue growth and lactation. This accounts for typical selenium content in breast milk from women in Nordic countries.</p>
Nutrition Unit/National Institute for Health and Welfare (THL), Finland	<p>Detailed comments</p> <p>We would like to suggest that consumers of organic foods in Finland are mentioned as a possible risk group concerning the intake of selenium in NNR5. Since 1985 selenium as selenate has been added to artificial fertilizers in Finland and as a result plant food selenium has increased 5-10 fold. Indirectly also the selenium concentrations of foods of animal origin have increased significantly. However, plant foods cultivated without selenium-containing fertilizers have a selenium concentration comparable to that of the pre-fertilizer levels. The meat selenium concentration of animals fed biologically grown feeds is also much lower than meat of animals conventionally fed except for milk, which contains half of the conventional amount. As 70% of the total mean intake of selenium comprises milk and meat, the selenium intake of people who regularly consume organically grown products will be much lower than the recommended level. This is also true for vegetarians and vegans since foods of plant origin except cereals contain very little selenium.</p> <p>References: Eurola M, Alfthan G, Ekholm P, Levonmäki M, Root T, Venäläinen ER, Ylivainio K. Report of Selenium Working Group 2008. http://www.mtt.fi/met/pdf/met132.pdf (Finnish). Alfthan G, Aspila P, Ekholm P, Eurola M, Hartikainen H, Hero H, Hietaniemi V, Root T, Salminen P, Venäläinen ER, Aro A. Nationwide supplementation of sodium selenate to commercial fertilizers. History and 25-year results from the Finnish selenium monitoring program. in: Thompson B, Amoroso L, eds. Combating micronutrient deficiencies: Food-based approaches. Rome:FAO/CAB International 2010. http://www.fao.org/docrep/013/am027e/am027e.pdf Eurola M, Alfthan G, Ekholm P, Root T, Suoniitty T, Venäläinen E-R, Ylivainio K. Report of Selenium Working Group 2011. http://www.mtt.fi/mttraportti/pdf/mttraportti (Finnish).</p>	<p>A paragraph covering aspects on organic foods and vegetarian diets have been included.</p>

	Selenium. General and detailed comments	WG observations and actions
Swedish Dairy Association	<p>General comments</p> <p>During development of NNR5 (New Nordic Nutrition Recommendations) a table has been published as a "suggestion to NNR5 in practice" although the focus of the work is to develop recommendations and not dietary guidelines. In the "limit column" sugar sweetened dairy products are mentioned. Which references support the view that sugar in dairy products is harmful, but not sugar in other products? Correctly it should thus rather be written "sugar sweetened food products", which aims at reducing to the intake of added sugar in the diet. The criteria for selection of papers for the scientific reviews are not shown. Publications chosen for evaluation based on the criteria are not listed. The authors of the chapters of draft proposals are not included. The connection between systematic review and draft proposal are not transparent. In general, old references are used. This indicates that references from new literature searches are not used, making the transparency of the process hard to rely on. The chapters are not always updated with dietary sources of nutrient based on the results from Riksmaten 2010-11. It is important to use the latest information on these results, to justify the nutrition recommendation. Recommendations from other countries/organizations should be included in every chapter in a consistent way. The bioavailability of a nutrient from the major contributing food categories in the Nordic countries should be included. The influence of a nutrient in food sources should be distinguished from the same nutrient added to the diet as supplement.</p> <p>Detailed comments</p> <p>The text of the draft proposal on selenium is almost identical with the selenium chapter in NNR 2004. It is therefore difficult to identify the impact of the scientific review and the resulting interpretation. Very old references are used despite those in the only new paragraph, line 230-243. The contribution of selenium from major food categories in each Nordic country should be included. It is important to include the bioavailability from other food sources than fish, e.g. meat and dairy as these food categories contribute significantly to the selenium intake in Sweden, line 72-75. References should be included in the old paragraph under the heading "Physiology and metabolism", line 59-70, should be included. A justification to include the last paragraph, line 230-243, under the heading "Upper intake levels and toxicity" should be included.</p>	<p>The text has been considerably extended as to contribution of selenium from different food sources.</p> <p>Bioavailability is also handled more thoroughly.</p> <p>The last paragraph, line 230 – 243, has been deleted.</p>

	Selenium. General and detailed comments	WG observations and actions
Danish Food and Drink Federation	<p>General comments</p> <p>Selenium page 5 Line 199: you cannot use a 13 year old publication to say that there is no evidence. Please review Bjelakovic (Bjelakovic et al., 2008), Bardia (Bardia et al., 2008) and the following for relevant information: (Brinkman et al., 2006;Dagnelie et al., 2004;Etminan et al., 2005). Viñas et al shows that a considerable part of the Nordic population has a selenium intake below the estimated average requirement (Vinas et al., 2011) Literature Cited Bardia A, Tleyjeh IM, Cerhan JR, Sood AK, Limburg PJ, Erwin PJ, and Montori VM. 2008. Efficacy of antioxidant supplementation in reducing primary cancer incidence and mortality: systematic review and meta-analysis. Mayo Clin Proc 83:23-34. Bjelakovic G, Nikolova D, Simonetti RG, and Gluud C. 2008. Antioxidant supplements for preventing gastrointestinal cancers. Cochrane Database Syst RevCD004183. Brinkman M, Reulen RC, Kellen E, Buntinx F, and Zeegers MP. 2006. Are men with low selenium levels at increased risk of prostate cancer? Eur J Cancer 2006/09/02:2463-2471. Dagnelie PC, Schuurman AG, Goldbohm RA, and van den Brandt PA. 2004. Diet, anthropometric measures and prostate cancer risk: a review of prospective cohort and intervention studies. Ann Intern Med 145:1139BJU Int-1150. Etminan M, FitzGerald JM, Gleave M, and Chambers K. 2005. Intake of selenium in the prevention of prostate cancer: a systematic review and meta-analysis. Cancer Causes Control 16:1125-1131. Vinas BR, Barba LR, Ngo J, Gurinovic M, Novakovic R, Cavelaars A, de Groot LC, van't Veer P, Matthys C, and Majem LS. 2011. Projected prevalence of inadequate nutrient intakes in Europe. Ann Nutr Metab 59:84-95.</p>	The paragraphs have been updated.
Health Product Wholesalers' Association, Finland	<p>General comments</p> <p>Vitamin C, E and Selenium The recommendations do not take in consideration EFSA's scientific studies and references or European Commission's regulations for health claims 16.5.2012 (432/2012). In the draft form the recommendations confuse consumers, sellers and manufacturers. The EU regulation 432/2012 is new but it should be taken as a part of the recommendations.</p>	The EFSA opinions cover conditions for a specific claim and is not directly applicable for setting dietary reference values.
Funktionaalisen lääketieteen yhdistys ry Functional Medicine Society in Finland	<p>General comments</p> <p>Nordic Council of Ministers For health and Social Affairs Nordic Nutrition Recommendations (NNR) 23.11.2012</p> <p>Dear Sirs, We suggest the following changes in the NNR recommendations : Dietary antioxidants Suggestion to recommendations Intake of supplemental antioxidants does not seem to be justified for a healthy population in large. Specified recommendations are given separately for the individual antioxidants according to EU commission's regulation 432/2012. The recommendation is based on limited numbers of studies s 5. For example the WHO Monica study has since 1985 generated a great number of studies related to cardiovascular diseases. Some new follow-up studies inspired by the WHO Monica project have showed the benefit of food supplements (for example in preventing</p>	Comments appreciated. This topic is covered in the chapters on e.g. Antioxidants and vitamin E.

	Selenium. General and detailed comments	WG observations and actions
	<p>oxidative stress related diseases). This report, follow-up studies and adjacent studies have been neglected. All studies should include reports of the following studies. Newest published criticism of flawed results and their conclusions should be cited. It seems that different methodological approaches of meta analysis may yield contradictory results (Gerss et al 2009). In large food supplement population studies, the results are compiled and reported based on individual self-reports and limited laboratory determinations. It is a well-known fact that self-medication can have an impact on the final reporting and results. In the introduction it is stated that the NNR guidelines are given for healthy populations and should not be implicated on individual persons or individual intake of food supplements. This should be mentioned in every individual recommendation. Obviously the situation is totally different when it comes to different diseases. For that reason it should be underlined that these recommendations are given for a healthy Scandinavian large population with no diagnosed underlying diseases and their medications. Rows 293-299: Reasoning behind the recommendation is over simplification of conclusions ignoring huge amount of contradictory scientifically proved facts in specified populations. For antioxidants such as vitamin C, vitamin E and beta-carotene, EU has stipulated regulations which should be uniform and evaluated in all dietary recommendations. Vitamin E Rows 264-267: A more recent paper shows that different methodological approaches of meta analysis yield contradictory results (Gerss et al 2009). NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations includes only healthy large populations not individuals. Vitamin C NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations include only healthy large populations not individuals. Selenium NNR recommendations and their conclusions with references should be evaluated against the EU commission regulation 432/2012 including the scientific references behind the regulations. The NNR recommendations include only healthy large populations not individuals. Gerss, J., Köpcke, W. The questionable association of vitamin E supplementation and mortality--inconsistent results of different meta-analytic approaches. Cell Mol Biol (Noisy-le-grand). 2009, 55 suppl: OL1111-1120.</p>	<p>The EFSA opinions cover conditions for a specific claim and is not directly applicable for setting dietary reference values.</p>
Danish Cancer Society	<p>General comments</p> <p>The authors should mention the sources of selenium in the Danish and Swedish diet also (Norway, Finland and Iceland are mentioned briefly). Furthermore, an argument for the possible public health impact of the new recommendation could be made.</p>	<p>Acknowledged. Some text added.</p>

	Selenium. General and detailed comments	WG observations and actions
NIFES, Norway	<p>General comments</p> <p>As far as I have understood from the accompanying letter to this draft proposal, the recommendation for selenium is one of the few which has been changed in this revised version of NNR. From my point of view this change could have been more clearly described in the selenium chapter, both why it is increased and how much. Both aspects are somewhat covered in the text, but could preferably been emphasized a little more.</p>	Has been done.

NNR 2012: Public consultation – Chapter 37 (Copper)

General and detailed comment	WG observations and actions
<p>Metropolitan University College, Denmark</p> <p>General comments</p> <p>In the 'Introduction', I would consider it useful to mention the fact that copper cations in their free form can catalyse the generation of free radicals within the Fenton reaction, but that this reaction is abolished if copper is chelated by proteins. In 'Physiology and Metabolism', the role of caeruloplasmin should be mentioned, especially as it is mentioned repeatedly in the subsequent chapters.</p> <p>Detailed comments</p> <p>I. 21-25: Indication of two up to three concentrations/concentration ranges of copper in food might help the reader to get oriented on how much of a food category someone has to eat in order to cover copper needs.</p> <p>I. 31-32: It does not become clear why Zn-induced increase of metallothionein inhibits Cu absorption.</p> <p>I. 36: Add comma after liver</p> <p>I. 40: Maybe 'transports' or 'traffics' might be a better term than 'guides'?</p> <p>I. 41-42: The construction of this sentence with the 'and' extension looks awkward to me. What about '... but also through excretion via bile, which can account for approximately 0.5 to 1.5 mg of copper/d.'</p> <p>I. 45: ... copper of an adult ...</p> <p>I. 49: ... but it has been found ...</p> <p>I. 51: Please clarify whether copper deficiency has been observed in premature infants who recover from malnutrition and who are fed with cow's milk in parallel to diarrhoea or whether copper deficiency occurs also after consumption of cow's milk without diarrhoea (unclear in the current form).</p> <p>I. 55: delete 'the' I. 65. cytotoxicity of what?</p> <p>I. 78 add blank before 'all' (end of line)</p> <p>I. 79 replace ... none of these have ... with ... none of these indicators have ...</p>	<p>WG acknowledge the valuable comments. Final version is updated and language checked.</p>

General and detailed comment	WG observations and actions
<p>I. 81-82: It does not become clear whether the CCS concentration parallels copper availability or whether it declines with increasing copper absorption.</p> <p>I. 88-90: Please complete the sentence after the semicolon (verb is missing).</p> <p>I. 94: replace 'e.g' with 'such as'</p> <p>I. 96: 'immune function' (instead of 'immunefunction')</p> <p>I. 97: remove full stop after 'approximately'</p> <p>I. 98: 'caeruloplasmin, and SOD' (comma) I. 101: 'concentration, and erythrocyte' (comma)</p> <p>I. 104: in the NNR</p> <p>I. 113: '... older than one year ...' instead of '... more than one year old ...'</p> <p>I. 129: 'disorders' might be the better term than 'disturbance'</p> <p>I. 131/132: Please clarify: 'association': with what? and 'role of copper': for what? (If it relates to diarrhoea in the previous sentence, this should be made clear.)</p> <p>I. 147-149: repletion of I. 120-122 (redundancy?)</p> <p>I. 151-152: repletion of I. 113-114 (redundancy?)</p>	
<p>Swedish Dairy Association</p> <p>General comments</p> <p>Rows 50-51: Please specify if the children had been fed exclusively milk formula and cow's milk and therefore had a copper deficient diet. There is now a room for misinterpretation where worried parents may think that milk formula and cow's milk is the very cause of copper deficiency.</p>	Statement is clarified in the final version.
<p>Danish Food and Drink Federation</p> <p>page 4 line 136: Please mention IOM UL of 10mg and difference in assessment.</p>	<p>The SCF UL is cited as it is used in the European context. The U.S. UL is largely based on the same data, but IOM chose to apply an uncertainty factor of 1.0. SCF applied an uncertainty factor of 2, to allow for potential variability within the normal population. In addition SCF noted that the UL is not applicable during pregnancy and lactation due to inadequate data.</p>

NNR 2012: Public consultation – Chapter 38 (Chromium)

Organisation/ Company	Chromium General and detailed comments	WG observations and actions
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Organisation/ Company	Chromium General and detailed comments	WG observations and actions
Selskab for Ernæringsforsk ning, Denmark	Line 28, 56, 79, 96, 98,102, 114, 124: Replace “µg/day” with “µg/d” Detailed comments Line 77: Add . after “Mullee et al 2012)	Done Done
National Food Agency, Sweden	Detailed comments 9: Since no recommendations are given –maybe make this clear in the beginning of the chapter- with a box or something else. (This could be done with all the micronutrients that lack recommendation in a uniform way) Comments to row 53-74. Efsa has published an opinion on substantiation of health claims related to chromium and contribution maintenance of normal blood glucose concentrations. http://www.efsa.europa.eu/en/efsajournal/doc/1732.pdf This has resulted in an authorised health claim published in the Regulation of authorised health claims, with the wording as follows: Chromium contributes to the maintenance of normal blood glucose levels http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2012:136:0001:0040:EN:PDF Since this is now allowed on food products in Europe, NNR5 should check the references which are the basis for this claim, compare them to your references, and perhaps change the text in NNR5. 93 SCF is introduced without explanation- state what the abbreviation stands for. 104 EFSA is introduced into the text without explaining what the abbreviation means 109- states that the 2004 edition did not give any recommendation- but what about the current edition- make a clear statement.	Box is added Acknowledged. However, this does not provide evidence for setting a DRV. References are covered in the chapter. A clear statement is provided in lines 113- .

NNR 2012: Public consultation – Chapter 39 (Manganese)

Organisation/ Company	Manganese General and detailed comments	WG observations and comments
Selskab for Ernæringsforsk- ning, Denmark	Line 23-30: Move to “Chapter Intake of vitamin and minerals in the Nordic countries” Line 60-61 and 67-68 are identical Line 20,29,38,41,46, 56, 57,73,76: Replace “/day” with “/d”	OK done

Organisation/ Company	Manganese General and detailed comments	WG observations and comments
National Food Agency, SwedenFA	<p>Detailed comments</p> <p>9 Box or text that there is no RI or UL set for this nutrient</p> <p>23 Please, use data from new Swedish market basket survey published as report 7/2012, page 4: The daily estimated per capita exposure was 4.0 mg manganese. http://www.slv.se/upload/dokument/rapporter/kemiska/2012_livsmedelsvetket_7_market_basket_2010.pdf 86 We suggest removing the sentence that</p> <p>Manganese is regarded the least toxic trace elements. In Sweden, excess manganese content has been discussed in relation to foods for infants and young children. A review of the literature indicates that manganese intakes not too high above general intakes children may be associated with negative health effects. Although study quality does not allow definite conclusions or for setting an UL, these potential associations should be mentioned. Extract from the coming report: "Many epidemiologic studies including children have been carried out in order to investigate potential associations between intake of manganese from drinking water and/or levels of manganese in blood/hair on the one hand and results of a variety of tests regarding behaviour, hyperactivity, learning ability and intelligence on the other (98-100). In conclusion, these studies indicate that relatively low intakes of manganese by children may affect the nervous system in a negative way, which has been demonstrated predominantly in disturbances of the behaviour (91) and/or in intellectual impairments (99)". 91. Ljung K, Vahter M. Time to re-evaluate the guideline value for manganese in drinking water? Environ Health Perspect. 2007 Nov;115(11):1533-8. 98. Menezes-Filho JA, Bouchard M, Sarcinelli Pde N, Moreira JC. Manganese exposure and the neuropsychological effect on children and adolescents: a review. Rev Panam Salud Publica. 2009 Dec;26(6):541-8. 99. Bouchard MF, Sauve S, Barbeau B, Legrand M, Brodeur ME, Bouffard T, et al. Intellectual impairment in school-age children exposed to manganese from drinking water. Environ Health Perspect. 2011 Jan;119(1):138-43. 100. Henn BC, Schnaas L, Ettinger AS, Schwartz J, Lamadrid-Figueroa H, Hernandez-Avila M, et al. Associations of early childhood manganese and lead coexposure with neurodevelopment. Environ Health Perspect. 2012 Jan;120(1):126-31.</p>	<p>Added</p> <p>Results from Market basket survey have been included.</p> <p>The possible association between Mn exposure and neuropsychological effects has been included in the chapter.</p>

Organisation/Company	Molybdenum General and detailed comments	WG observations and actions
Selskab for Ernæringsforskning, Denmark	General comments Line 54,59, : Replace "/day" with "/d"	Corrected
National Food Agency, Sweden	Detailed comments 9 Box or text that there is no RI or UL set for this nutrient 30 Use data from new Swedish market basket survey published as report 7/2012 Livsmedelsverket if possible. Daily estimated per capita consumption of Mo was 157 µg. http://www.slv.se/upload/dokument/rapporter/kemiska/2012_livsmedelsverket_7_market_basket_2010.pdf	Done

NNR 2012: Public consultation – Chapter 41 (Fluoride)

Fluoride

General and detailed comment	WG observations and actions
<p>University of East Anglia, UK</p> <p>General comments</p> <ol style="list-style-type: none"> Line 14 says that the essentiality of fluoride is debated but on lines 46-47 it is stated that it is not considered essential. Lines 22-23 give a greater sense of certainty than is warranted. There seems little point in calculating the intake of fluoride given a certain concentration in the drinking water when the levels in water are so variable. Instead it would be interesting to provide a range of intakes using low fluoride and high fluoride water found in Nordic countries for the estimate. Is fluoride added to water in any of the countries (as is carried out regionally in the UK)? The report is similar to NNR4 2004. Lines 38 onwards could perhaps be updated so that the 'today' of 2004 is not the 'today' of 2012/13! <p>Detailed comments</p> <ol style="list-style-type: none"> Line 13. Perhaps replace 'surface' with enamel? Line 21. Ranges require two figures. These are reported intakes, not ranges. Are they means or medians? Which countries? Line 25. All age groups will acquire fluoride in the enamel from toothpaste - it does not need to be 	<p>Comments considered. Revised in final version</p>

<p>swallowed. The distinction should be made clear between fluoride entering the enamel (as prevention against caries is the primary endpoint for setting requirements) and systemic fluoride obtained from ingested food, water and swallowed toothpaste.</p> <p>4. Line 33. Change 'adsorbed' to absorbed.</p>	
<p>Vitenskapskomiteen for mattrygghet (VKM), Norway</p> <p>General comments</p> <p>Line 54-65: EFSA established ULs for different age groups in a rather comprehensive opinion from 2005. In the NNR 2012 draft chapter for fluoride no UL is set. It should be explained why no UL is set in NNR 2012, and if NNR disagrees with the conclusions in the EFSA opinion, an explanation should be given.</p> <p>Detailed comments</p> <p>Line 63-65 The referral to the EFSA opinion from 2005 should be more precise and complete. In the NNR 2012 draft for fluoride it is not clear why "The EFSA NDA (6) panel considered that an intake of less than 0.1 mg F/kg BW/day in 63 children up to 8 years old corresponds to no significant occurrence of "moderate" forms 64 of fluorosis in permanent teeth" is the only part of the EFSA opinion that is referred to.</p>	<p>Accepted.</p> <p>More detail on ULs added in the final version.</p>
<p>Minivent AB, Sweden</p> <p>I am missing information about the goitrogenic effect of F in food and tooth paste. It is well known among biochemists that F may replace I in T1, T2, T3 and T4 thus these thyroid hormones do not fit the thyroid receptors in the cells as the F atom is so much smaller than the I atom.</p>	<p>It is acknowledged that high fluoride intake/exposure has been associated with effects on thyroid metabolism, However, at exposures necessary for caries prevention provided from tooth paste or drinking water has not been shown to affect thyroid function.</p> <p>Some references added.</p>