

Nordic Nutrition Recommendations 2012

Integrating nutrition and physical activity

Part 2





Nord 2014:004

Nordic Nutrition Recommendations 2012

Part 2
Energy, fat and fatty acids,
carbohydrates, protein, alcohol, fluid
and water balance and physical activity

5th edition

Nordic Nutrition Recommendations 2012 · Part 2

Energy, fat and fatty acids, carbohydrates, protein, alcohol, fluid and water balance and physical activity

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Nordic co-operation

Nordic co-operation is one of the world's most extensive forms of regional collaboration, involving Denmark, Finland, Iceland, Norway, Sweden, and the Faroe Islands, Greenland, and Åland.

Nordic co-operation has firm traditions in politics, the economy, and culture. It plays an important role in European and international collaboration, and aims at creating a strong Nordic community in a strong Europe.

Nordic co-operation seeks to safeguard Nordic and regional interests and principles in the global community. Common Nordic values help the region solidify its position as one of the world's most innovative and competitive.

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Secretary General's Preface

There has been an increasing interest in food and nutritional science in recent years. Food programmes are a staple of most television channels and cookbooks top the bestseller lists. At the same time, it can be a bit of a challenge to find your way through the jungle of advice on what we should eat facing the average consumer.

That is why we need a work like the Nordic Nutrition Recommendations, one of the most well-researched and thoroughly documented works within nutritional science worldwide. They give a scientific basis for formulating dietary guidelines and are an excellent example of what the Nordic countries can achieve when they work together.

The Nordic Council of Ministers funds the extensive scientific effort behind the Nordic Nutrition Recommendations. We do this as a means to inform the public debate on food-related matters. But maybe more importantly, the NNR also serve as the main reference point for the various national nutrition recommendations in the Nordic countries.

The Nordic Nutrition Recommendations are also the foundation for the criteria developed for the Nordic nutritional label the Keyhole, informing the shopping decisions of millions of consumers in the Nordic region on a daily basis.

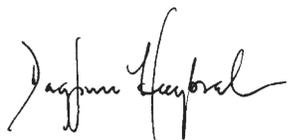
Finally, the NNR form part of the overall Nordic action plan *A better Life through Diet and Physical Activity*. In its aim to ensure the best-possible health for the population at large, this can be seen as an expression of the Nordic model, with its focus on an inclusive and holistic approach to society and the welfare of its citizens.

This is the fifth edition of the Nordic Nutrition Recommendations. As such, this publication is one of many examples of a long and fruitful Nordic co-operation over the last decades.

As a new step, we have decided to publish a free PDF version of the NNR along with a series of e-publications of individual chapters. The NNR will also for the first time ever be published as an e-book and they have thus entered the digital era.

I would like to thank the hundreds of scientists, experts and officials involved in compiling the Nordic Nutrition Recommendations and hope

that the quality of the work itself, as well as the many new forms of publication, will help ensure the widespread use that the NNR deserve.

A handwritten signature in black ink, reading "Dagfinn Høybråten". The signature is fluid and cursive, with the first letter 'D' being particularly large and stylized.

Dagfinn Høybråten
Secretary General, Nordic Council of Ministers

Preface

The 5th edition of the Nordic Nutrition Recommendations, NNR 2012, has been produced by a working group nominated by the Working Group on Food, Diet and Toxicology (NKMT) under the auspices of the Nordic Committee of Senior Officials for Food Issues (ÄK-FJLS Livsmedel). The NNR 2012 working group was established in 2009 and consisted of Inge Tetens and Agnes N. Pedersen of Denmark; Ursula Schwab and Mikael Fogelholm of Finland; Inga Thorsdottir and Ingibjorg Gunnarsdottir of Iceland; Sigmund A. Anderssen and Helle Margrete Möltzer of Norway; and Wulf Becker (Chair), Ulla-Kaisa Koivisto Hursti (Scientific secretary), and Elisabet Wirfält of Sweden.

More than 100 scientific experts have been involved in this revision. Existing scientific evidence has been reviewed for setting dietary reference values (DRVs) that will ensure optimal nutrition and help prevent lifestyle-related diseases such as cardiovascular diseases, osteoporosis, certain types of cancer, type-2 diabetes, and obesity as well as the related risk factors for these diseases. The experts have assessed the associations between dietary patterns, foods, and nutrients and specific health outcomes. The work has mainly focused on revising areas in which new scientific knowledge has emerged.

Systematic reviews (SR) were conducted by the experts, with assistance from librarians, for the nutrients and topics for which new data of specific importance for setting the recommendations has been made available since the 4th edition. Less stringent updates of the reference values were conducted for the other nutrients and topics.

Peer reviewers for each nutrient and topic have also been engaged in the process of reading and commenting on the SRs and the updates conducted by the expert groups. A reference group consisting of senior experts representing various fields of nutrition science both within and outside the Nordic countries has also been engaged in the project. A steering group with representatives from national authorities in each country has been responsible for the overall management of the project.

All chapters were subject to public consultations from October 2012 to September 2013. The responses and actions to the comments by the NNR working group are published separately.

The SRs and the updates form the basis for deriving the DRVs. In the process of deriving the NNR 2012, emphasis has been put on the whole diet and the current dietary practices in the Nordic countries. This evaluation was performed by the NNR 2012 working group and was not part of the SRs conducted by the expert groups. The SRs were used as major and independent components – but not the only components – for the decision-making processes of the working group that was responsible for deriving the NNR 2012.

The SRs are published in the Food & Nutrition Research journal and the other background papers can be found on the Nordic Council of Ministers (NCM) website.

The 5th edition, the Nordic Nutrition Recommendations 2012, is published by the NCM and is also available in electronic form.

The following experts and peer reviewers have been engaged in performing SRs and chapter updates.

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Calcium experts: Christel Lamberg-Allardt, Kirsti Uusi-Rasi and Merja Kärkkäinen, Finland.

Peer reviewers: Christian Mølgaard, Denmark and Karl Michaëlsson, Sweden.

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Peer reviewers: Inger Björck, Sweden, Inge Tetens, Denmark.

Elderly experts: Agnes N Pedersen, Denmark, Tommy Cederholm, Sweden, Alfons Ramel, Iceland.

Peer reviewers: Gunnar Akner, Sweden, Merja Suominen, Finland, Anne Marie Beck, Denmark.

Fat and fatty acids experts: Ursula Schwab and Matti Uusitupa, Finland, Thorhallur Ingi Halldorsson, Iceland, Tine Tholstrup and Lotte Lauritzen, Denmark, Wulf Becker and Ulf Risérus, Sweden.

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Folate experts: Cornelia Witthöft, Sweden, Georg Alfthan, Finland, Agneta Yngve, Norway.

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Peer reviewers: Harri Niinikoski, Finland, Kim Fleischer Michaelsen, Denmark.

Iodine experts: Ingibjörg Gunnarsdottir, Iceland, Lisbeth Dahl, Norway.

Peer reviewers: Helle Margrete Meltzer, Norway, Peter Lauerberg, Denmark.

Iron experts: Magnus Domellöf, Sweden, Ketil Thorstensen, Norway, Inga Thorsdottir, Iceland.

Peer reviewers: Olle Hernell, Sweden, Lena Hulthén, Sweden, Nils Milman Denmark.

Overweight and obesity experts: Mikael Fogelholm and Marjaana Lahti-Koski, Finland, Sigmund A Anderssen, Norway, Ingibjörg Gunnarsdottir, Iceland.

Peer reviewers: Matti Uusitupa, Finland, Mette Svendsen, Norway, Ingrid Larsson, Sweden.

Pregnancy and lactation experts: Inga Thorsdottir and Anna Sigridur Olafsdottir, Iceland, Anne Lise Brantsaeter, Norway, Elisabet Forsum, Sweden, Sjurdur F Olsen, Denmark.

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Peer reviewers: Rikke Andersen, Denmark, Mairead Kiely, Ireland, Karl Michaëlsson, Sweden, Gunnar Sigurdsson, Iceland.

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Peer reviewers: Morten Grønbæk, Denmark and Satu Männistö Finland.

Fluid and water balance expert: Per Ole Iversen, Norway.

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Physical activity experts Lars Bo Andersen, Danmark, Sigmund A Anderssen and Ulrik Wisløff, Norway, Mai-Lis Hellénus, Sweden. Peer reviewers Mikael Fogelholm, Finland, Ulf Ekelund, Norway.

Energy experts: Mikael Fogelholm and Matti Uusitupa, Finland. Peer reviewers: Ulf Holmbäck and Elisabet Forsum, Sweden.

Population groups in dietary transition expert: Per Wändell, Sweden. Peer reviewer: Afsaneh Koochek, Sweden.

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Sustainable food consumption expert: Monika Pearson, Sweden.

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The librarians have been responsible for literature searches in connection with the SRs, other database searches, and article handling.

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Introduction

For several decades, the Nordic countries have collaborated in setting guidelines for dietary composition and recommended intakes of nutrients. Similarities in dietary habits and in the prevalence of diet-related diseases, such as cardiovascular diseases, osteoporosis, obesity and diabetes, has warranted a focus on the overall composition of the diet, i.e. the intake of fat, carbohydrate, and protein as contributors to the total energy intake. In 1968, medical societies in Denmark, Finland, Norway, and Sweden published a joint official statement on “Medical aspects of the diet in the Nordic countries” (Medicinska synpunkter på folkkosten i de nordiska länderna). The statement dealt with the development of dietary habits and the consequences of an unbalanced diet for the development of chronic diseases. Recommendations were given both for the proportion of fat in the diet and the fat quality, i.e. a reduced intake of total fat and saturated fatty acids and an increase in unsaturated fatty acids.

The Nordic Nutrition Recommendations (NNR) are an important basis for the development of food, nutrition, and health policies; for formulation of food-based dietary guidelines; and for diet and health-related activities and programmes. Previous editions mainly focused on setting dietary reference values (DRVs) for the intake of, and balance between, individual nutrients for use in planning diets for various population groups. The current 5th edition puts the whole diet in focus and more emphasis is placed on the role that dietary patterns and food groups play in the prevention of diet-related chronic diseases.

The NNR are intended for the general population and not for groups or individuals with diseases or other conditions that affect their nutrient requirements. The recommendations generally cover temporarily increased requirements, for example, during short-term mild infections or certain medical treatments. The recommended amounts are usually not suited for long-term infections, malabsorption, or various metabolic disturbances or for the treatment of persons with a non-optimal nutritional status. They are meant to be used for prevention purposes and are not specifically meant for treatment of diseases or significant weight reduction. The NNR do, however, cover dietary approaches for sustainable weight maintenance

after significant and intentional weight reduction. For specific groups of individuals with diseases and for other groups with special needs or diets, dietary composition might have to be adjusted accordingly.

After a thorough revision in which experts have reviewed a vast amount of scientific publications, most of the recommendations from the 4th edition (2004) remain unchanged. However, the RIs for vitamin D in children older than 2, adults, and the elderly ≥ 75 years of age and for selenium in adults have been increased. An emphasis has been put on the quality of fat and carbohydrates and their dietary sources. The recommendation for protein has been increased for the elderly ≥ 65 years of age. No recommended intakes have been set for biotin, pantothenic acid, chromium, fluoride, manganese, or molybdenum due to insufficient data, and this represents no change from the 4th edition.

The primary aim of the NNR 2012 is to present the scientific background of the recommendations and their application. A secondary aim is for the NNR 2012 to function as a basis for the national recommendations that are adopted by the individual Nordic countries.

The NNR 2012 are to be used as guidelines for the nutritional composition of a diet that provides a basis for good health. The basis for setting recommendations is defined for each individual nutrient using the available scientific evidence. In many cases, the values for infants and children are derived from adult data using either body weight or energy requirement as a basis for the estimations. As new scientific knowledge emerges with time, the NNR have to be reassessed when appropriate and should, therefore, not be regarded as definitive.

The NNR are based on the current nutritional conditions in the Nordic countries and are to be used as a basis for planning a diet that:

- satisfies the nutritional needs, i.e. covers the physiological requirements for normal metabolic functions and growth, and
- supports overall good health and contributes to a reduced risk of diet-associated diseases.

The NNR are valid for the average intake over a longer period of time of at least a week because the dietary composition varies from meal to meal and from day to day. The recommended intakes refer to the amounts of nutrients ingested, and losses during food preparation, cooking, etc. have to be taken into account when the values are used for planning diets.

The NNR can be used for a variety of purposes:

- as guidelines for dietary planning
- as a tool for assessment of dietary intake
- as a basis for food and nutrition policies
- as a basis for nutrition information and education
- as guiding values when developing food products



Energy

Components of daily energy expenditure

Definitions of energy requirement

The basic principle behind the formulation of energy requirement reference values is energy balance, i.e. the physiological state in which daily energy intake equals energy expenditure and both body weight and energy content (defined by body composition) are constant. For some people, especially those who are over- or underweight, the recommended energy intake might be lower or higher than energy expenditure for a prescribed time period, but long-term energy balance is the ultimate goal even in treatment of undernourishment and obesity. Therefore, the NNR defines the energy requirement in adults as *“the energy intake needed to cover energy expenditure in individuals with body weight, body composition and physical activity compatible with good health. In addition, energy requirement is affected by the energy needed for growth in children, for deposition of tissues during pregnancy and for milk production during lactation”* (1). However, because body energy stores are very large (at least 30 times the daily energy expenditure), there is no need for energy intake and energy expenditure to be equal over short periods of around 1 to 4 days (2).

The daily energy expenditure can be divided into the following components:

- Basal (or resting) energy expenditure (BEE or REE)
- Diet-induced thermogenesis (DIT)
- Energy expenditure caused by physical activity

Energy expenditure is measured in kJ (1000 kJ = 1 MJ) per time unit (usually MJ/d). One kilojoule equals 0.24 kcal (or 1 kcal = 4.184 kJ), a unit that is still often used in the literature.

On average, daily energy expenditure is higher in men than in women but the difference disappears after adjustment for the difference in body

size and body composition between the sexes. Very cold or hot environments, genetic differences, hormonal status (e.g. serum concentrations of thyroid and growth hormones), sympathetic nerve activity, psychological state, pharmacological agents, and several disease states have been shown to increase or decrease energy expenditure, mainly by affecting REE (3, 4).

Basal (resting) energy expenditure

BEE, or basal metabolic rate (BMR), is defined as the energy expenditure of an individual at physical and mental rest in a thermoneutral environment and about 12 hours after the previous meal. REE is measured under less rigorous conditions than BEE and is considered, therefore, to be approximately 5% higher than BEE. The mean energy expenditure is slightly lower during sleep than during waking hours (3). Therefore, sleeping energy expenditure (SEE) is about 10% lower than BEE. Despite small systematic differences, SEE, BEE, and REE are very strongly inter-correlated and they are often used interchangeably.

In individuals with approximately equal physical activity levels, daily energy expenditure is strongly related to body weight and particularly to fat free mass (FFM = body weight - fat mass) (5). Fat mass (FM) also shows a positive correlation with energy expenditure. However, the increase in energy expenditure per unit FM is much smaller than for unit FFM (5). Hence, the inter-individual variations in FFM explain much more of the REE compared to variations in FM. FFM consists of skeletal muscle and organ tissue. When expressed per kg, the metabolic rate in the organs is much higher than in skeletal muscle. In adults, 70%-80% of BEE is derived from organs that comprise only 5% of the total body weight (5). Thus there is an association between total FFM and REE such that when FFM (and hence muscle mass) is low, the slope of BEE against FFM is lower than when FFM (and muscle mass) is high (4). In other words, when the organs make up a higher proportion of the FFM, increases in skeletal muscle mass has less influence on REE.

The inter-individual variation at a given FFM is about 2.1 MJ per day, and this indicates the possible magnitude of the difference in REE between two individuals with similar FFM. Variations in genetic makeup, body composition, hormone concentrations, energy balance, and physical fitness have been found to explain the variation in REE after adjustment for FFM (3, 4, 6, 7).

Diet-induced thermogenesis

DIT, or diet-induced energy expenditure, is defined as the increase above REE in energy expenditure after food intake divided by the energy content of the food ingested (8). The postprandial rise in energy expenditure lasts for several hours, but about 90% of DIT is observed within 4 h of the meal. DIT is assumed to be 10% of the daily energy expenditure in individuals in energy balance who consume a mixed diet with an average composition (9, 10). The DIT of fat is only about 5% of its energy content, but the DIT of protein is approximately 20% of its energy content. The DIT of carbohydrate is around 10% of its energy content, but this figure might be as high as 20% if glucose is directly converted to fat (de novo lipogenesis). However, this process requires an excess of energy from carbohydrates and this situation occurs rarely in healthy individuals consuming diets typical for the Nordic countries (11).

Physical activity

Physical activity (at work or leisure time) is defined as any bodily movement produced by skeletal muscle that results in energy expenditure (12). *Exercise* is a subcategory of physical activity and is a voluntary, deliberate physical activity performed because of anticipated positive effects on physical, psychological, and/or social well-being.

The daily physical activity level (PAL) is defined as total energy expenditure divided by REE (or BEE). This way of quantifying physical activity is based on the assumption that the variation in daily energy expenditure is based on physical activity and body size.

The metabolic equivalent of task (MET = energy expenditure during an activity divided by REE) is a measure of instant physical activity level, and PAL is the daily average of the METs weighted by the time each task was performed (see, for example, Table 8.8.) (13, 14). The inter-individual variation in PAL (roughly 1.4 to 2.0) is much more restricted than for MET, which can range, for example, from 1.2 when sitting to as high as 15 for riding a bicycle at a speed of 30 km/h.

Daily physical activity (and physical activity-induced energy expenditure) can be divided into occupational and leisure activities. The latter can be further divided into exercise and non-exercise activities that have different grades of intensity. Occupational activity can also vary in intensity. Inactivity refers to a state where energy expenditure is close to REE, and

this usually includes sitting or lying down while awake. The associations between physical activity, sedentary lifestyle, and health are described in detail in chapter on physical activity.

Energy balance and health

Body mass index

In obesity, the amount (in kg or as a percentage of body weight) or anatomical distribution (subcutaneous/visceral or abdominal/truncal) of body fat leads to an increased risk for adverse health effects, particularly type 2 diabetes, cardiovascular diseases, musculo-skeletal disorders, and some forms of cancer. Regardless of whether the amount of body fat or the distribution of body fat is used, it is not possible to determine a single point separating normal and healthy body weight from obesity. Moreover, health risks increase with increasing severity of obesity (15-17).

The simplest and probably most common way of assessing the status of obesity is by using body mass index (BMI), that is, body weight (kg) divided by the square of the height (m²). BMI has a U or J shaped association with total mortality and morbidity (15, 17, 18). In general, the BMI compatible with the lowest mortality (and morbidity) in adults is approximately 22-23. According to the WHO definition (15), the normal (or recommended) BMI is between 18.5 and 24.9 (Table 8.1.). The term overweight describes a slightly elevated BMI, and a BMI of 30 or more is considered to be obesity.

Table 8.1. Body mass index; definitions of underweight, overweight, and obesity; and health risks for adults 18-64 years of age

Body mass index	Definition	Morbidity and mortality
<18.5	Underweight	Slightly increased
18.5-24.9	Normal weight	Low
25.0-29.9	Overweight	Slightly increased
30.0-34.9	Grade I obesity	Increased
35.0-39.9	Grade II obesity	Much increased
≥40.0	Grade III obesity	Very much increased

The categories in Table 8.1. are, in principle, applicable in all Nordic countries. However, it should be kept in mind that BMI might represent different

levels of fatness and body fat distribution depending on age, sex, ethnicity, athletic training, and race (26, 27). For instance, the healthy BMI range might be higher for Inuits (19) and lower for individuals of Asian descent (20). Therefore, BMI on the individual level should be used with great caution. Other simple measures, such as waist circumference (see heading *Abdominal obesity*) might help to assess obesity-related health risks.

In a meta-analysis (21), the sensitivity of BMI for detecting high adiposity was 0.50 (95% confidence interval (CI): 0.43–0.57) and its specificity was 0.90 (CI: 0.86–0.94). These data indicate that using BMI leads to both type I errors (true obesity is not detected) and type II errors (obesity is detected even when it is not true) and that type I errors seem to be more common. Okorodudu et al. (21) compared BMI against measures of body fat from body composition analyses and showed that BMI is more prone to underestimate than to overestimate body fatness. In other words, many individuals with a BMI just below a cut-off limit (e.g. 25 or 30) should in reality have been classified as overweight or obese, respectively. Despite a common belief, it is less typical that BMI overestimates fatness (although it certainly does, for example, in people such as bodybuilders).

Obesity in children and adolescents can be defined using BMI, but the cut-off points differ from those presented in Table 8.1. Cole et al. (22) have published international age- and sex-specific BMI cut-off points for overweight (85th percentile) and obesity (95th percentile) for children and adolescents between 2 and 18 years. Many countries also use specific age-adjusted growth charts (weight-to-height for a given age) to assess overweight and obesity.

Studies have found that ageing is associated with decreasing height, weight, and BMI (23, 24) along with a loss of muscle mass and a gain of body fat (30). These changes imply that optimal BMI might be different in older people compared to younger people. Several studies have found the BMI associated with the lowest age-adjusted mortality to be higher in elderly people when compared to recommendations for younger subjects (25–30). Unfortunately the data are inadequate to make any precise recommendations for optimal BMI among the elderly. Studies that relate BMI to functional ability have found both a high and a low BMI to be related to disability (31, 32). However, marked obesity is clearly associated with physical disability and difficulties in performing activities of daily living (33, 34).

The prevalence of adult obesity in the Nordic countries is shown in Table 8.2. These data were obtained from nationally representative surveys, but

the values themselves were self-reported. The prevalence of overweight (BMI between 25 and 29.9) is even higher than the prevalence for obesity. This means that roughly half of the adult population in Nordic countries is either overweight or obese. Because individuals tend to underreport their body weight, the actual prevalence of overweight and obesity is likely to be somewhat higher than shown in the table. Compared to the prevalence of obesity reported in NNR 2004, this condition has become more common in all Nordic countries.

Table 8.2. Prevalence (%) of adult obesity (approximately 25–64 years of age, BMI >30.0) in the Nordic countries as assessed from self-reported body weight

	Women	Men	Reference
Denmark	14.8	15.6	(21)
Finland	19.3	18.2	(22)
Iceland	19.4	19.4	(23)
Norway	22.1	21.0	(24)
Sweden	14	13	(25)

Abdominal obesity

Abdominal fat distribution is an indicator of intra-abdominal fat mass and can also be used as an indicator of obesity (35). Table 8.3. presents cut-off points for waist circumference as suggested by the National Institute of Health (36) and the WHO (15). Intra-abdominal fat mass, or abdominal fat distribution, can be even more strongly associated with metabolic disturbances than the total amount of body fat. The cut-off points are probably higher for elderly subjects (37, 38), but BMI values are interpreted without any age adjustments in all adults older than 18 years.

Table 8.3. Waist circumference (cm) and the risk of metabolic complications in adults (18–64 years)

Risk level	Women	Men
Low	≤79	≤93
Increased	80–87	94–101
High	≥88	≥102

Obesity, weight stability and health

Obesity, and to a smaller extent overweight, is associated with an increased incidence of several diseases (16). This meta-analysis found statistically significant associations between obesity and overweight and the incidence of type 2 diabetes, several types of cancers (breast, endometrial, colorectal, and kidney), cardiovascular diseases, asthma, gallbladder disease, osteoarthritis, and chronic back pain. The strongest association was found between obesity and type 2 diabetes.

According to epidemiological studies, stable weight is related to the lowest total mortality and weight gain is clearly related to increased mortality (39). Many epidemiological studies indicate that weight loss is also associated with increased mortality (e.g. (39-42)). However, these data should be interpreted with caution because of difficulties in separating voluntary and involuntary (due to pre-existing disease) weight reduction. Moreover, epidemiological studies do not separate different techniques or rates of weight reduction or composition of lost body weight (43). Nevertheless, even a modest (5%-10% of body weight) weight reduction in high-risk individuals improves health (15). Weight cycling (weight reduction then increasing to previous weight) might have adverse effects on mortality and morbidity (44, 45), but the data do not provide compelling evidence for this (46). A 25-year study in Gothenburg, Sweden, has found an age-related decrease in body weight from the age of 70 years to 95 years of approximately 0.5-1.0 kg for every 5 years, and this effect is more pronounced in the highest quintiles of body weight (24).

Determinants of obesity and weight control

Weight gain is caused by a positive energy balance. Several retrospective and prospective population-based studies have evaluated factors related to obesity or weight gain. A systematic literature review examining the role of dietary macronutrient composition was carried out as part of the NNR 2012 process (47). The literature search covered the year 2000 to the present, and prospective cohort studies, case-control studies, and interventions were included. The literature search also provided the opportunity to review the role of food consumption and dietary patterns in predicting changes in weight or waist circumference.

The review (47) found probable evidence that high intake of dietary fibre and nuts predicted less weight gain and that high intake of meat predicted more weight gain. Suggestive evidence was found for a protective role against increased weight for whole grains, cereal fibre, high-fat

dairy products, and high scores on an index describing a prudent dietary pattern. Likewise, there was suggestive evidence for both fibre and fruit intake as a protection against increases in waist circumference. Suggestive evidence was found for high intake of refined grains, sweets, and desserts in predicting weight gain, and for refined (white) bread and a high energy density diet in predicting increases in waist circumference. The results of the literature search suggested that the proportion of macronutrients in the diet was not important in predicting changes in weight or waist circumference. In contrast, prospective cohort studies have shown that increased intake of fibre-rich foods and dairy products and a reduction in refined grains, meat, and sugar-rich foods and drinks are associated with a reduction in weight gain.

In a very recent meta-analysis, Te Morenga et al. (48) concluded that the intake of free sugars or sugar-sweetened beverages is a determinant of increasing body weight. These results give additional support for restricting sugar intake as a means to prevent obesity. In another meta-analysis from the same year, Chen et al. (49) did not find evidence that intake of dairy products prevented weight gain. These results are somewhat in contrast to the review by Fogelholm et al. (47). The discrepancies in the results might be related to different selection of studies; Chen et al. (49) scrutinized randomized trials, but Fogelholm et al. (47) examined cohort studies.

A major problem in assessing the grade of evidence was that similar combinations of exposure and outcome variables were quite rare in the literature making comparisons difficult (47). It was decided, therefore, to perform a *post hoc* evidence analysis by first combining the outcome variables (BMI and waist circumference). To increase the number of studies used for evidence grading, foods were grouped by their closeness in terms of nutrient composition. The results of these *post hoc* analyses are shown in Figure 8.1. These analyses suggest that a healthy diet in general (assessed using indices that describe the healthiness of dietary patterns) and fibre-rich foods are clearly associated with less weight gain. Dairy products are only to some extent associated with reduced weight gain. In contrast, meat, refined grains, and sugar-rich foods and drinks are associated with more weight gain.

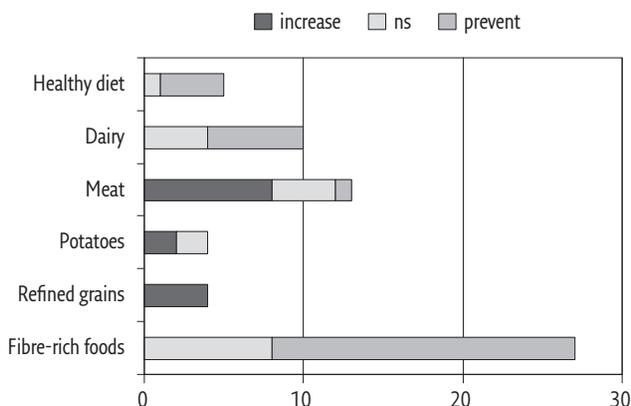


Figure 8.1. Evidence for association between grouped exposure variables against grouped outcome variables (BMI and waist circumference not separated) (47)

Low levels of physical activity are positively associated with obesity and age-related weight gain. Most studies have examined leisure activity, and data on occupational energy expenditure that have been adjusted, for example, to socioeconomic factors in relation to obesity are lacking. High levels of physical activity are also associated with less weight being regained after weight reduction (50). However, most of the above findings are observational and retrospective and the studies are still inconclusive as to whether physical activity can be regarded as a single predictor of weight control. Spontaneous physical activity corresponds to small, involuntary muscle movements, such as fidgeting, and might be related to weight control (51), but the data to support this are limited. Obesity is also associated with education level and socioeconomic status. The general trend is that higher social classes have a lower prevalence of obesity compared with lower social classes (52, 53).

Estimation of energy requirements

There are two main approaches to estimate total energy requirements. The first approach is the doubly labelled water (DLW) technique (54, 55) in which stable isotopes (^2H and ^{18}O) are administered orally. The isotopes are gradually eliminated from the body, ^2H through water and ^{18}O through water and CO_2 . The difference between the elimination rates of ^2H and ^{18}O is related to CO_2 production and, therefore, to energy expenditure. This estimation of total energy expenditure is quite accurate provided that the

experimental and analytical conditions are appropriate. In theory, a large number of DLW measurements could be used as the basis to predict total energy expenditure by deriving equations that describe how total energy expenditure varies as a function of, for example, age, sex, and various anthropometric measures such as weight and body fat. There are several data sets with energy expenditures for a total of several hundred individuals assessed by DLW (56, 57) and pooled analyses (81). However, the populations in these studies were selected and the representativeness of these data cannot be guaranteed.

The second main approach to assess energy expenditure is the factorial method in which total energy expenditure is calculated from the resting (or basal) energy expenditure (REE) and a factor indicating PAL. DLW is more accurate in assessing individuals, but the factorial method provides more opportunity to generalize the results. Therefore, estimates of average energy requirements in NNR 2012 were determined using the factorial method.

Because of technical constraints on REE measurements, determinations of energy requirements are usually based on predicted REE. Table 8.4. shows prediction equations for REE as given by Henry (58). In the previous version of the NNR, the equations of Schofield et al. (59), WHO/FAO/UNU (60), and the Commission of the European Communities (61) were used. However, because a recent validation study has shown that the Schofield equations tend to overestimate REE (62, 63), the new and more accurate equations by Henry (58) have been used in this version of the NNR. The same decision was also made by EFSA in 2013 when formulating reference values for energy (64).

Table 8.4. Equations for calculating the average resting energy expenditure (REE, MJ/d) based on either body weight (W, kg) or a combination of weight and height (H, m) (58)

Age Year	REE	
	MJ/d based on weight	MJ/d based on weight and height
Girls		
<3	$0.246 W - 0.0965$	$0.127 W + 2.94 H - 1.20$
3-10	$0.0842 W + 2.12$	$0.0666 W + 0.878 H + 1.46$
11-18	$0.0465 W + 3.18$	$0.0393 W + 1.04 H + 1.93$
Women		
19-30	$0.0546 W + 2.33$	
31-60	$0.0407 W + 2.90$	$0.0433 W + 2.57 H - 1.180$
61-70	$0.0429 W + 2.39$	$0.0342 W + 2.10 H - 0.0486$
>70	$0.0417 W + 2.41$	$0.0356 W + 1.76 H + 0.0448^a$
Boys		
<3	$0.255 W - 0.141$	$0.118 W + 3.59 H - 1.55$
3-10	$0.0937 W + 2.15$	$0.0632 W + 1.31 H + 1.28$
11-18	$0.0769 W + 2.43$	$0.0651 W + 1.11 H + 1.25$
Men		
19-30	$0.0669 W + 2.28$	
31-60	$0.0592 W + 2.48$	$0.0600 W + 1.31 H + 0.473$
61-70	$0.0543 W + 2.37$	$0.0476 W + 2.26 H - 0.574$
>70	$0.0573 W + 2.01$	$0.0478 W + 2.26 H - 1.070^a$

^a This equation covers all ages above 60 years.

Reference values for energy requirements in children and adolescents

Part of the energy intake of children and adolescents is used for growth, and their energy requirement per kg body weight is, therefore, higher than for adults. During the first four months of life, approximately 27% of the energy intake is used for growth. At the end of the first year of life, this amount decreases to approximately 5%; at age 1-3 years it decreases to approximately 3%; and in older children this value is less than 2% (65).

Reference values for energy requirements of children and adolescents should be based on their REE, their energy expenditure in response to physical activity, and their energy requirements for growth. These values should be consistent with the attainment and maintenance of long-term good health, including recommended levels of physical activity (66).

Age 1-12 months

The estimated energy requirement for infants is based upon the approach of FAO/WHO/UNU (1) where daily energy expenditure is calculated using DLW-derived equations (67) (Table 8.5.).

Table 8.5. Estimated average daily energy requirements (per kg body weight) for children 1–12 months assuming a mixture of breastfeeding and complementary foods (67)

Age months	Average daily energy requirements kJ/kg body weight	
	Boys	Girls
1	486	469
3	411	404
6	339	342
12	337	333

Some studies have shown that breast-fed infants have a lower energy intake than formula-fed infants (68–70), especially infants breast-fed for more than seven months, and that this results in less body weight gain from 6 to 10 months than in infants breast-fed for a shorter period (71, 72). The effect of the infant's food source on energy requirements was found to persist throughout the second year of life in one of the studies used as a basis for the estimated energy requirement (65). This was primarily because of a higher REE in formula-fed than in breast-fed infants (65) although varying digestibility might also play a role (73). However, the differences between feeding groups in terms of energy expenditure never exceeded 20 kJ/kg, and the current NNR gives a single energy requirement that is valid for both breast-fed and formula-fed infants.

Estimated average reference values for children and adolescents

The estimated daily energy requirements according to age for children and adolescents (Table 8.6.) are based on the factorial method. Thus, REE is first estimated using the equations of Henry (58) and daily energy expenditure is then calculated by multiplying REE by an appropriate PAL. Values for body weight related to age in the group aged 0–5 years are based on the mean of the reference values from Denmark (93), Norway (94), Sweden (74), and Finland (75). No values were available for 2–5 year olds in Finland so data for 3–5 year olds in Norway and the other Nordic values were used. Values for growth at school age show increasing weight-to-height ratios and an increased prevalence of overweight (76). This means that using current weight data would base the recommendations on an increasing prevalence of excess body weight. Therefore, values for 6–17 year olds are based on mean values from 1973–1977 (77). The situation is similar for

adults in which the energy requirements are based on a theoretical BMI of 23, not actual BMI data. In the US, the estimated energy requirements for children are based on the median weight and this is slightly higher than the reference weights used in the current NNR (78).

The increase in obesity over the past 30 years in Nordic countries might be leveling off. There are indications showing a decreasing, or at least a stable, prevalence of overweight and obesity among school children in the Nordic countries (79). However, the prevalence of these conditions among young children and adolescents is still high.

Children of the same age vary widely in body weight, particularly in the age groups where only a small fraction of the children have started puberty. The body weight of children of the same age and sex can differ by a factor of two. Therefore, the estimated energy requirement in a certain age group, as illustrated in Table 8.8., must be used with caution. Moreover, the calculated energy requirement for overweight children (>2 SD weight-to-height ratio) is too high when based on body weight because such children have a comparatively high body fat content and the energy requirement is primarily determined by the size of the FFM. Therefore, it is recommended that the energy requirement in overweight children should be based on the weight one SD above normal weight for height or on the weight corresponding to the cut-off value of overweight according to the International Obesity Task Force (15).

PAL values in the NNR2012 are based on a systematic review of the DLW studies that was carried out for the SACN (80) recommendations. The analysis showed no significant differences between the sexes, but did show an increased PAL with age. We have used the first quartile (25th percentile) value as a cut-off for low vs. average activity, and the third quartile (75th percentile) as the cut-off for average vs. high activity (Table 8.6.).

Table 8.6. Estimated daily energy requirements (MJ/d) for children and adolescents (from 2 to 17 years) using the Henry (2005) equations for REE and the physical activity levels from SACN (80)

Age years	Body weight kg	REE	Physical activity level ¹		
			Low	Average	High
Girls					
2	12.5	2.98	4.02	4.14	4.26
3	14.9	3.57	4.82	4.96	5.10
4	16.8	3.53	5.02	5.55	5.97
5	19.2	3.74	5.31	5.87	6.31
6	21.1	3.90	5.53	6.12	6.59
7	23.7	4.12	5.84	6.46	6.96
8	26.1	4.32	6.13	6.78	7.30
9	28.7	4.54	6.44	7.12	7.67
10	31.8	4.80	7.96	8.30	8.88
11	35.5	4.83	8.02	8.36	8.94
12	40.4	5.06	8.40	8.75	9.36
13	45.6	5.30	8.80	9.17	9.81
14	49.9	5.50	9.13	9.52	10.18
15	53.2	5.65	9.39	9.78	10.46
16	54.8	5.73	9.51	9.91	10.60
17	56.0	5.78	9.60	10.01	10.70
Boys					
2	13.2	3.23	4.35	4.48	4.61
3	15.4	3.79	5.11	5.26	5.41
4	17.3	3.77	5.35	5.92	6.37
5	19.4	3.97	5.63	6.23	6.71
6	21.4	4.16	5.90	6.52	7.02
7	24.8	4.47	6.35	7.02	7.56
8	26.5	4.63	6.58	7.27	7.83
9	29.1	4.88	6.92	7.66	8.24
10	32.2	5.17	8.58	8.94	9.56
11	35.3	5.14	8.54	8.90	9.52
12	39.1	5.44	9.03	9.41	10.06

13	43.5	5.78	9.59	9.99	10.68
14	49.2	6.21	10.31	10.75	11.49
15	55.1	6.67	11.07	11.53	12.33
16	60.0	7.04	11.69	12.19	13.03
17	63.6	7.32	12.15	12.67	13.54

¹ Physical activity levels (low, average, high) by age group. 1–3 y: 1.35, 1.39, and 1.43; 4–9 y: 1.42, 1.57, and 1.69; 10–18 y: 1.66, 1.73, and 1.85.

Reference values for energy requirements in adults

The reference values for energy requirements in adults are based on estimates of REE and PAL. Energy requirement is equivalent to the product of REE and PAL. RMR can be calculated from the prediction equations that are presented in Table 8.1., and the PAL values (Table 8.4.) are estimated generalisations (average values) based on studies using DLW. By using more detailed information on daily physical activity (time spent in different activities) and the respective MET values (14), PAL can be approximated for an individual as the daily time-weighted average MET value (Tables 8.7. and 8.8.). For instance, in Table 8.8., an active day is assumed to consist of 8 h rest (mostly sleep), 10 h very light activity (mostly sitting, sometimes standing), and 2 h light activity (e.g. slow walking, cooking, etc.). In addition, the day consists of 1 h moderate activity (e.g. brisk walking) and 1 h vigorous activity (e.g. playing football). To calculate PAL, the MET values of different activity levels are multiplied by the time spent in the corresponding activity divided by 24. Daily energy expenditure is calculated by multiplying PAL by the REE.

Table 8.7. Physical activity level expressed as multiples of the resting energy expenditure according to different levels of occupational and leisure activity (modified from Black et al. (81))

	PAL
Bed-bound or chair-bound (not wheelchair)	1.1–1.2
Seated work with no option of moving around and little or no leisure activity	1.3–1.5
Seated work with some requirement to move around, and with some leisure activity	1.6–1.7
Work including both standing and moving around (e.g. housework, shop assistant) OR seated work with some requirement to move around with regular, almost daily, leisure activity	1.8–1.9
Very strenuous work or daily competitive athletic training	2.0–2.4

Note 1 Moderate leisure physical activity (e.g. brisk walking): 0.025 PAL unit increase for each hour per week.

Note 2 Strenuous leisure physical activity (e.g. running, competitive football): 0.05 PAL unit increase for each hour per week.

Table 8.8. Two examples of how to estimate daily physical activity levels from data on physical activity

Intensity of Activity (MET)	Very inactive day		Active day	
	Time, h	MET × h	Time, h	MET × h
Rest (1.0)	10	10	8	8
Very light (1.5)	12	18	10	15
Light (2.0)	2	4	4	8
Moderate (5.0)	0	0	1	5
Strenuous (10.0)	0	0	1	10
Total	24	32	24	46
PAL		1.33		1.92

Explanation. The time spent in different activities is multiplied by the respective metabolic equivalent value (MET value). To obtain the daily physical activity level (PAL), the sum of daily MET × h is divided by 24. Hence, PAL is the weighted average of daily MET × h. Daily energy expenditure is calculated by multiplying PAL by the resting (or basal) energy expenditure.

An average PAL for adults in Nordic countries is assumed to be around 1.6, which is compatible with sedentary work and some physical activity (56, 57). A totally sedentary lifestyle (PAL 1.4–1.5) is associated with health risks that might be equal to the risk associated with marked obesity (BMI 30–35) or regular smoking. These health risks are offset by approximately 3–4 hours per week moderate physical activity or 2 hours per week of more strenuous leisure-time physical activity (82), which would mean an increase of only 0.1 PAL units. However, it is likely that a PAL of roughly

1.8 would be more optimal for overall health. This level was close to the 75th percentile in the large data sets of Tooze et al. (56) and Mosghfegh et al. (57). This PAL is approximately the same as that observed in moderately active prepubertal children (83). Strenuous athletic training can increase energy requirements to PAL 2.0–2.5 and in extreme cases even up to 4.0 (84, 85). However, it is rare for physical exercise to increase energy requirements by more than 20% compared to energy expenditure during normal daily living. PAL 1.4 is used as the level indicating physical inactivity, and this level is close to the 15th percentile in larger population samples (56, 57).

Table 8.9. shows reference weights based on population data in Denmark, Finland, Iceland, and Sweden. Because of the high prevalence of overweight and obesity, population weights cannot be used directly to estimate reference weights because then the reference energy needs would support the maintenance of overweight and obesity. Therefore, the reference weight needs to be adjusted to a theoretical situation in which all individuals are at normal weight. In the NNR 2012, the reference weight was calculated by using population-based data on height to estimate an age-adjusted weight corresponding to BMI 23. This arbitrary BMI was used to indicate healthy weight. The precise mean point within the WHO normal body weight range (BMI 18.5 to 24.9) would have been BMI 21.7. Because the actual mean BMIs of the populations in all Nordic countries are clearly higher, BMI 23 was chosen as more realistic but still within the normal BMI range. The principle difference in the new recommendations compared to the previous NNR is that in the previous NNR the weight for all overweight and obese individuals was reduced to correspond to BMI 25. The new reference weight is slightly higher for the youngest age group and lower for the oldest age group.

Table 8.9. Reference weights (kg) from Nordic countries calculated as the weight for height corresponding to BMI 23

	Denmark ^a	Finland ^b	Iceland ^c	Sweden ^d	Mean
Men, age in years					
18-30	77.5	73.2	77.2	73.7	75.4
31-60	75.2	72.2	75.5	74.8	74.4
61+	72.0	69.1	74.1	73.0	72.1
Women, age in years					
18-30	65.5	63.1	64.2	65.0	64.4
31-60	64.6	61.7	64.5	63.8	63.7
61+	62.2	58.9	63.3	62.7	61.8

Data sources: ^a (86); ^b (87); ^c (88) ^d (89).

Table 8.10. shows the average estimates of daily energy requirements for men and women with respect to age, different activity levels, and reference weight (Table 8.9.). The values in Table 9.10. are estimations assuming that all individuals have BMI 23. It should be noted that these estimations have a large standard error due to imprecision in both estimation of REE and of PAL. Therefore, the results should be used only for estimations on the group level. In particular, the data for the oldest age group in Tables 8.9. and 8.10. should be used with special caution. Compared to the reference energy requirements in the previous version of the NNR, the new values are lower because the REE equation uses a slightly lower predicted REE and the reference weights have been calculated differently. Due to the age-related weight changes among healthy elderly individuals, 0.5-1.0 kg should be subtracted from the average weights in Table 8.9. for every 5 years above the age of 75.

Table 8.10. Reference energy requirements (MJ/d) in adults based on Nordic reference weights (Table 8.9.) and different activity levels

Age, years	Reference weight, kg ^a	REE, MJ/d ^b	Sedentary PAL ^c 1.4	Average PAL 1.6	Active PAL 1.8
Men					
18-30	75.4	7.3	10.3	11.7	13.2
31-60	74.4	6.9	9.6	11.0	12.4
61-74 ^d	72.1	6.1	8.5	9.7	10.9
Women					
18-30	64.4	5.8	8.2	9.4	10.5
31-60	63.7	5.5	7.7	8.8	9.9
61-74 ^d	61.8	5.0	7.1	8.1	9.1

^a Reference weight corresponds to BMI 23.

^b REE = Resting Energy Expenditure, estimated from the equations of Henry (2005).

^c PAL = Physical Activity Level.

^d The REE for 61-74 year olds was calculated with the equation for 61-70 year olds.

Reference values for energy requirements are based on assumptions regarding weight stability, normal (healthy) weight, and energy balance. However, these assumptions are not always valid. For instance, a negative energy balance is needed for the treatment of obesity. If energy intake is 2.1 MJ/d below the requirement for energy balance, the estimated weight reduction during the first month is approximately 500 g/week. This rate of weight loss is often recommended although a larger negative energy balance (up to 4.2 MJ/d) leading to a weight loss of 1000 g/week still seems to be compatible with a healthy weight reduction (8, 90). The long-term estimation (several months to years) of weight loss due to a fixed reduction in energy intake is much more complicated (91). The reason for this is that energy expenditure decreases with weight loss. Hence, with increasing weight reduction the energy deficit decreases (same intake but less expenditure). Therefore, the 500 g/week weight loss for each 2.1 MJ (500 kcal) reduction in energy intake cannot be used for anything other than predicting initial weight reduction.

The energy requirement for an individual with weight and physical activity different from the values presented in Tables 8.9. and 8.10. can be calculated as follows. First, the RMR is estimated using the appropriate equation in Table 8.4. PAL is then estimated either from Table 8.7. or using the calculation shown in Table 8.8. Finally, the energy requirement

is calculated as $RMR \times PAL$. It should be noted, however, that RMR as well as PAL tend to be imprecise and it is indeed possible to misjudge the daily energy requirement by at least 2 MJ.

Energy requirement during pregnancy

The requirement for energy during pregnancy is based on estimates of weight gain during gestation and the composition of that gain in terms of fat and protein. Hytten and Chamberlain (92) studied reproductive outcomes in healthy women and concluded that a weight gain of 12.5 kg was associated with the best reproductive outcome in mother and infant (92). This value is lower than average values for weight gain in pregnancy in the Nordic countries, and the US Institute of Medicine (IOM) (93) has extended the recommendation of weight gain in pregnancy by taking varying pre-pregnancy weight into consideration.

Pregnant women are in an anabolic dynamic state throughout gestation, and this creates additional needs for energy. Forsum and L f described the partitioning of energy metabolism in the pregnant versus the non-pregnant state (94). According to Butte and King (67), "The energy requirement of a pregnant woman is the level of energy intake from food that will balance her energy expenditure when the woman has a body size and composition and level of physical activity consistent with good health". The energy requirement of pregnant women includes the energy needs associated with the deposition of tissues consistent with optimal pregnancy outcome (67). The energy cost in pregnancy is due to the foetus, placenta, and amniotic fluid as well as the weight gain of the uterus and breasts and increased volumes of blood, extracellular water, and adipose tissue (95).

In 2004, FAO/WHO/UNU published recommendations for energy intake by pregnant women based on a 12.0 kg weight gain during gestation (3). Butte and King (67) modified these figures for women gaining 13.8 kg, a figure in better agreement with values observed for Scandinavian women. The calculations were based on two approaches (1, 67). The first was a factorial approach using estimates of the energy costs due to changes in the resting energy expenditure (REE) and the cost of tissue deposition along with separate estimates for the amount of energy retained and the cost of synthesis associated with this retention. The second approach was based on the increment in total energy expenditure (TEE) and estimates of the amount of energy retained. The two calculations gave similar results for the complete pregnancy (374 and 369 MJ for the first and second alterna-

tive, respectively) but differed with respect to the increase in requirements during the three individual trimesters. The average of the two calculations (430, 1375, and 2245 kJ/24 hours during the first, second, and third trimester, respectively) is considered to represent the additional need for energy during the three pregnancy trimesters, and these are the recommended energy intake values during the three trimesters. A comparison between NNR 2004 and the new recommendations is shown in Table 8.11.

Table 8.11. Additional daily energy requirement during pregnancy: comparison between NNR-2004 and the new recommendations

	NNR 2004	NNR 2012
1st trimester	Value not given	430 kJ (103 kcal)
2nd trimester	350 kcal	1375 kJ (329 kcal)
3rd trimester	500 kcal	2245 kJ (537 kcal)

An additional aspect that should be considered is the potential decrease in energy needs due to a decrease in physical activity during pregnancy. This is a complicated issue where definite answers cannot be provided. Studies have shown that Swedish pregnant women do (96) or do not (97) “save energy” by such a decrease. Thus, as stated by Prentice et al. (98), it cannot be assumed that a high proportion of the energy costs of pregnancy are normally or automatically met by reductions in physical activity.

There is great variation among women regarding the amount of weight gained during pregnancy. Positive associations between this gain and the health of both baby and mother have been observed. However, a very large weight gain is a health risk both for mother and child, especially among women who were overweight or obese prior to pregnancy (e.g. an increased risk for breast cancer in the mother, spontaneous abortion, gestational diabetes, and gestational hypertension) (99, 100). If weight gain during pregnancy is too small, the risk for a low birth weight baby is increased because weight gain in pregnancy is positively correlated to infant size at birth (100). Low birth weight increases the risk for health complications in early life and has been found to be related to increased risks of adult diseases such as coronary heart disease, hypertension, and type 2 diabetes (100–103).

Weight gain during pregnancy among women in the Nordic countries is, on average, 14–16.5 kg (100, 104–107). The average birth size in the

Nordic countries is high (>3500 g), the highest is in Iceland and the Faeroe Islands, and has been increasing for full-term babies in all the Nordic countries in recent years (108). Values on weight gain during pregnancy have been reviewed, and in 2009 the IOM published guidelines with recommended gestational weight gains for women having different BMIs before conception (109). These are the values now recommended by NNR for Nordic women (Table 8.12.). The median value of the recommended weight gain range for women who were normal weight before pregnancy (11.5–16 kg) is the same as the value of 13.8 kg used by Butte and King (67) when calculating energy requirements during pregnancy.

Table 8.12. Weight gain during pregnancy as recommended by the Institute of Medicine (109)

BMI (kg/m ₂) before conception	Recommended weight gain (kg)
<18.5 (underweight)	12.5–18
18.5–24.9 (normal weight)	11.5–16
25.0–29.9 (overweight)	7–11.5
>30.0 (obese)	5–9

In recent years the importance of foetal nutrition has attracted a significant amount of interest. Studies in humans as well as in experimental animals suggest that the supply of energy and nutrients during this very first part of life is related to health later in life. Furthermore, studies have shown that the nutritional situation of the woman before conception is also important and, as indicated above, in the US the recommended weight gain during pregnancy varies according to the pre-pregnancy BMI of the woman. In fact, recent recommendations, also from the US (109), emphasize that “all women should start pregnancy with a healthy weight”, i.e. with a BMI between 18.5 and 24.9. A recent systematic literature review (110) shows that insufficient data are available regarding health outcomes of intended weight loss as a result of dieting prior to conception. It is conceivable that such weight loss might be associated with harmful effects, for example impaired iron and folate status during subsequent pregnancies and a risk for developing eating disorders.

Overweight and obesity is common among Scandinavian women of reproductive age, and this is a serious concern because the pre-pregnancy BMI is a strong predictor of many adverse outcomes of pregnancy (109).

Therefore, it is important that every effort is made to avoid overweight and obesity in women of reproductive age. However, although overweight and obesity are presently the most common nutritional problems in Scandinavian women it should be emphasized that low BMI and insufficient weight gain do occur in some women and are associated with increased health risks for their offspring.

Energy requirement during lactation

The additional energy requirement during lactation is based on estimates of the energy costs for milk production and an estimate of the amount of energy mobilized from the body's energy stores. During pregnancy there is a physiological retention of body fat that, to some extent, can be mobilized postpartum. Thus the energy needs during lactation are dependent on the nutritional status of the mother during pregnancy. According to Butte and King (67), "The energy requirement of a lactating woman is the level of energy intake from food that will balance her energy expenditure when the woman has a body size and composition and a breast milk production which is consistent with good health for herself and her child and that will allow for desirable physical activity".

According to international recommendations (1, 67), energy requirements during lactation for women in developed countries are based on an average milk production of 749 g every 24 hours. Breast milk is considered to contain 2.8 kJ/g and to be produced with an energy efficiency of 80% (95). For partial lactation, the breast milk production is assumed to be 492 g every 24 hours. Table 8.13. shows the energy cost of lactation for women in developed countries during different time periods postpartum (1, 67). These costs should be added to the energy requirement of the non-pregnant and non-lactating woman, and they can be covered by an increased intake of dietary energy or partly covered by mobilized body fat. This contribution of body fat to the energy costs of lactation has been estimated to be, on average, 0.72 MJ every 24 hours during the first six months of lactation. However, the variation between individual women is considerable. A large individual variation is certainly also present with respect to the milk production figures given above. There are no data showing that lactating women decrease their physical activity to "save energy" for milk production. However, because of a risk for weight gain after pregnancy (111), it is recommended that lactating women increase rather than decrease their amount of physical activity.

Table 8.13. Energy cost of milk production (MJ/24 hours) for women in developed countries during exclusive and partial breastfeeding (67)¹

Months post partum ²	0-2	3-5	6-8	9-11	12-23
Exclusive breastfeeding	2.49	2.75	2.81	3.15	-
Partial breastfeeding	2.24	2.40	2.07	1.53	1.57

- ¹ These costs can be covered by an increased intake of energy from food or by mobilized body fat (0.72 MJ/24 hours on average) during the first six months of lactation.
- ² Scandinavian women are recommended to breastfeed exclusively during the first six months postpartum and then breastfeed partially at least until the child is one year old.

The increased prevalence of overweight and obesity among Scandinavian women is also a potential problem during lactation because it has been shown that obese and overweight women tend to have a less successful lactation than normal-weight women (112). Furthermore, there are data from Danish women showing that breastfeeding promotes postpartum weight loss (113). However, this effect is rather weak and it is quite possible to gain weight during lactation if the energy balance is positive, i.e. too much energy from food and/or too little physical activity. A Swedish study showed that dietary advice to overweight and obese lactating women could effectively promote weight loss after pregnancy (114). It is important to stress, however, that breastfeeding is an energy-demanding process and for many lactating women a considerably increased energy intake is recommended.

Energy requirements in the elderly

Daily energy expenditure tends to decline with age (115, 116) mainly due to decreased FFM (117, 118) and decreased physical activity (119, 120). REE is strongly related to FFM, which consists mainly of muscle and organ mass (121). The decrease in REE is not fully explained by the age-related decrease in FFM (122), and Pannemans et al. (115) found that 80% of the variation in REE in elderly subjects was explained by FFM.

Longitudinal (123-125) and cross-sectional (82, 126, 127) studies have found an age-related decrease in REE, but knowledge about daily energy expenditure in the elderly (>75 years) is limited (82). A Swedish study found that the REE among 91-96 year olds was not different from the REE among 70-80 year olds (128), and a US study found a 27% lower REE in very old individuals compared to 60-74 year olds (129). However, a longitudinal follow-up of the 73 year olds at age 78 showed a decrease

in REE as well as TEE but not in active energy expenditure (AEE) (130). The PAL values in the above individuals averaged 1.74 at both ages (73 years and 78 years) indicating a physically active lifestyle for this age group (130). DIT does not seem to be affected by age (127).

A review including 24 studies with measured REE in healthy elderly (mean age 70.6 ± 5.1 years, mean body weight 72.4 ± 6.0 kg, and mean BMI 25.6 ± 1.5) found the mean of the weight-adjusted REE to be about 80 kJ/kg body weight in both males and females, and this value was not significantly different from a group of sick elderly patients (131). The measured PAL obtained from 24 h TEE relative to the REE was 1.66 ± 0.11 among the healthy elderly.

Low energy intake

Lowenstein (132) has suggested a reference value of 1500 kcal/d - corresponding to approximately 6.5 MJ/d - as the minimum daily energy intake necessary for providing an adequate intake of micronutrients from an ordinary diet. In the NNR, *very low energy intake is defined as an energy intake below 6.5 MJ/d*, and an energy intake of 6.5-8 MJ is considered a *low energy intake* with increased risk of an insufficient intake of micronutrients.

A very low energy intake is related to a very low PAL and/or to a low body weight. Low body weight is related to low muscle mass and, therefore, to low energy expenditure. The age-related decrease in energy expenditure might result in a very low energy intake, and such low intakes are also found among people on slimming diets and among subjects with, for example, eating disorders or food intolerances.

Among healthy subjects, very low habitual energy intakes are probably rare - even among sedentary elderly subjects the estimated daily energy requirement is only 7-8 MJ, see Table 8.12. However, with lower body weight among the sedentary elderly, energy intake might become critically low.

Intake of most micronutrients is positively associated with energy intake and, consequently, habitually low energy intake is associated with low nutrient intake. In dietary surveys, the reporting of energy intake is often biased by a widespread underreporting that is independent of age, especially among women and overweight/obese subjects. Thus, it is difficult to explore the consequences of low energy intake on nutritional status based on low-energy *reporters*.

Among elderly subjects, low reported energy intakes were not associated with biochemical signs of nutritional deficiencies (133, 134). This

somewhat surprising result might be explained by underreporting (thus true intakes are higher) or that recommended biochemical levels are already reached at lower intakes than expected. Among elderly Europeans (133), it was not possible to establish a level of reported energy intake that ensured an adequate supply of iron, thiamine, riboflavin, or pyridoxine. At a reported intake of 8 MJ per day, 13% of men and 16% of women still had an inadequate intake of at least one of these four micronutrients.

Energy content of foods

Calculation of energy content

The energy in foods available for metabolism - i.e. the metabolizable energy - is determined by the energy content of the food as assessed in the laboratory by measuring the heat produced when its organic components are fully oxidized. Not all energy in a food item is available to humans, and its energy value must be corrected for losses due to insufficient absorption and, in the case of protein, also for incomplete oxidation and for losses as urea in urine. Accurate calculation of the metabolizable energy content in foods requires knowledge of the foods' macronutrient content as well as of the digestibility of these macronutrients. Because the energy content and the digestibility of each macronutrient vary between foods, it is convenient to use standardised factors based on the energy content and digestibility of macronutrients representing the composition of an average mixed diet.

Due mainly to historical background and tradition, there are different standard factors that differ slightly from each other. In the NNR, the energy content of a mixed diet is calculated based on 17 kJ/g protein and available (glycaemic) carbohydrate and 37 kJ/g fat. Alcohol (ethanol) is considered to yield 29 kJ/g. In kcal, these standard factors are 4 kcal/g protein and carbohydrate, 9 kcal/g fat, and 7 kcal/g alcohol. Note that these numbers include some errors caused by rounding off from kilojoules. To transform values between the two systems of units, the following relationships are used: 1 kcal = 4.2 (or 4.184) kJ and 1 kJ = 0.24 (or 0.239) kcal. These standard factors are not intended for calculating the metabolizable energy content in individual food items because the heat of combustion as well as the digestibility vary slightly between macronutrients from different foods. In a mixed diet, however, these variations balance each other and the standard factors have been shown to be accurate. Specific factors for calculating energy content in individual food items have been presented (135, 136).

As pointed out in chapter 9.1.3, the energy content of foods is not fully available to cover human energy requirements. Large differences exist in the amounts of energy available from different macronutrients because their metabolism *per se* requires different amounts of energy. The post-prandial rise in energy expenditure is highest for proteins (about 20% of the energy content), lower for carbohydrates (about 10%), and lowest for fat (about 5%) (9, 10). In addition, the absorption of macronutrients varies among individuals and is dependent on the specific foods eaten, how they are prepared, and intestinal factors (91).

Carbohydrates and fibre

The values for carbohydrate that are shown in food composition tables are in many cases determined by means of the 'difference method' that defines total carbohydrate as the difference between the total dry matter and the sum of protein, fat, and ash. These values include digestible mono-, di-, and polysaccharides (starch) as well as non-digestible carbohydrates such as lignin and organic acids. The glycaemic or 'available' carbohydrates represent total carbohydrates minus dietary fibre, and are the sum of the total amounts of sugars and starch.

The heat of combustion of glycaemic carbohydrates is slightly lower for monosaccharides than for disaccharides and even higher for polysaccharides (136). However, these differences can be disregarded in most practical situations. When total carbohydrate is analysed 'by difference', available carbohydrate and dietary fibre are considered to contribute with the same amount of metabolizable energy. The energy content will, therefore, be overestimated in diets containing high amounts of dietary fibre if the calculation is based on a carbohydrate content assessed 'by difference'.

In diets containing up to 30 g fibre per day, standard energy factors can be used without significant consequences for the calculated metabolizable energy content of the diet (137). In fact, dietary fibre contributes only a small amount of such energy because its components are, to some extent, fermented in the colon. End products in this process are short-chain fatty acids that can be absorbed and metabolized and thus contribute to the metabolizable energy of the diet. The magnitude of this contribution depends on the type of fibre, but 8 kJ (2 kcal)/g has been suggested as an average value (135, 138). In regulations for specifying the nutritional content of foods, the energy content of fibre is considered to be zero, and dietary fibre is not considered to contribute to the metabolizable energy of diets in the NNR. However, the Codex Alimentarius Commission as well as current

suggestions for revisions of the European Nutrition Labelling Directive, propose that dietary fibre should be given an energy factor of 8 kJ/g.

The digestibility of carbohydrate varies from 90% in fruits to approximately 98% in cereals. The digestibility of flour depends on the fractions included, i.e. the digestibility decreases with a higher content of fibre.

Protein

Protein is not completely oxidized in the body. Therefore, when calculating the metabolizable energy content of protein incomplete digestibility as well as urea losses in the urine must be considered. The digestibility of protein is lowest in legumes (78%) and highest in animal products (97%) (135, 136).

Fat

The heat of combustion for dietary fat is a function of the fatty acid composition of the triglycerides in the diet and the proportion of other lipids in the diet. On average, the digestibility of dietary fat is considered to be 95% in most foods (135, 136).

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10 Fat and fatty acids

Age	6–11 mo.	12–23 mo.	Adults and children from 2 years of age
Cis-MUFA	10–25 E%	10–20 E%	10–20 E%*
Cis-PUFA	5–10 E%	5–10 E%	5–10 E%*
- n-3	≥1 E%	≥1 E%	≥1 E%
SFA	<10 E%	<10 E%	<10 E%
TFA	As low as possible	As low as possible	As low as possible
Total fat	30–45 E%	30–40 E%	25–40 E%

* Cis-monounsaturated (cis-MUFA) and cis-polyunsaturated fat (cis-PUFA) should make up a minimum of 2/3 of the total fat intake.

SFA: saturated fatty acids; TFA: trans-fatty acids.
Fatty acids are expressed as triglycerides.

Introduction

Fat provides the body 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.

Dietary sources and intake

The dietary content of fat and fatty acids in the Nordic countries has changed significantly in recent decades. The total fat content decreased from the 1970s to the 1990s. After being rather stable for several years, the dietary fat content has again increased in recent years in some Nordic countries, e.g. in Finland (1). The content of saturated fatty acids (SFA) has shown a similar trend as total fat, i.e. first it decreased, then levelled

off, and now is increasing in some countries. According to recent surveys, the proportion of SFA is above the recommendations and the ratio of unsaturated to saturated fatty acids is below the recommendations in Nordic countries. The dietary content of trans fatty acids (TFA) has decreased in all Nordic countries since the 1990s primarily through reduced use of partially hydrogenated fats in food production. The dietary content of TFA is currently below 1 E%. The dietary content of cis-polyunsaturated fatty acids (PUFA) increased from the 1960s to the 1980s and has been rather stable ever since. Table 10.1. shows the mean total fat and fatty acid intake in the Nordic countries according to recent surveys.

Table 10.1. The average dietary intake (E%) of total fat and fatty acid sub-categories in the Nordic countries in 2003–2012

	Denmark	Finland	Iceland	Norway	Sweden
	2003–08	2012*	2010–2011	2010–2011	2010–11
Total fat	35	36.1/35.5	36.2	34	34
SFA	14	15.1/15.0	14.5	13	13
TFA	0.6	0.5/0.5	0.8	<1.0**	0.5***
MUFA	12	14.0/13.5	11.6	12	13
PUFA	4.9	6.7/6.7	5.9	6.2	5.6

* Men/Women.

** Household Consumption Survey 2005–2009.

*** Market Baskets 2010.

In Iceland, the intake of total fat decreased from 41 E% to 36 E% between 1990 and 2010/2011. In the same period, the intake of SFA decreased from 19 E% to 14.5 E% and TFA from 2 E% to 0.8 E% (2, 3). In Finland, the intake of SFA was reduced from 19 E% to 14 E% between 1982 and 2002, but it has increased between 2007 and 2012 from 12–13 E% to 15 E% in both women and men (1). In Norway, the dietary content of SFA decreased from 16 E% in 1980 to 14 E% in the 1990s, and it was 13 E% in 2010–11 (4, 5). The dietary content of TFA decreased from 4 E% in the 1970s to 1 E% around the year 2000, and it has been below 1 E% for the past decade. In Denmark, the intake of fat decreased during the period from 1985 to 2001 from 44 E% to 34 E%, mainly due to a decrease in consumption of butter and milk products but also from a decrease in meat consumption (6). However, fat intake in Denmark has increased recently (7). In Sweden, the mean total fat intake has remained stable from 1997

to 2011 (34 E%) and the intake of SFA has slightly decreased from around 14 E% to 13 E% (8, 9). The intake of PUFA has increased from a mean of 4.7 E% to 5.6 E%.

The most important sources of fat are 1) spreads, butter, and oils, 2) milk and milk products, and 3) meat and meat products. Fat-containing dairy products, butter, butter-based spreads, meat products, sweet bakery products, and confectionary are the main sources of SFA. Main sources of TFA are dairy and meat products. Soft margarines, vegetable oils, and fish are the main sources of PUFA, and cis-monounsaturated fatty acids (MUFA) are derived from several food groups.

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. Fatty acids account for about 95% of the triglycerides by weight, and non-esterified fatty acids are uncommon in the diet. 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, on their position in the triglyceride molecule. The unsaturated fatty acids are characterised by the number of double bonds in the molecule: 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 (D) or the methyl end (ω or n-). The human body is capable of synthesising SFA and MUFA - including n-7 and n-9 series MUFA - from acetate, but n-3 and n-6 series PUFA are required from the diet. Linoleic acid (n-6, LA) and α -linolenic acid (n-3, ALA) are metabolised (desaturated and elongated) further in the body by the same enzymes (Figure 10.1.). Naturally occurring unsaturated fatty acids in plants and wild fish are mainly cis-fatty acids.

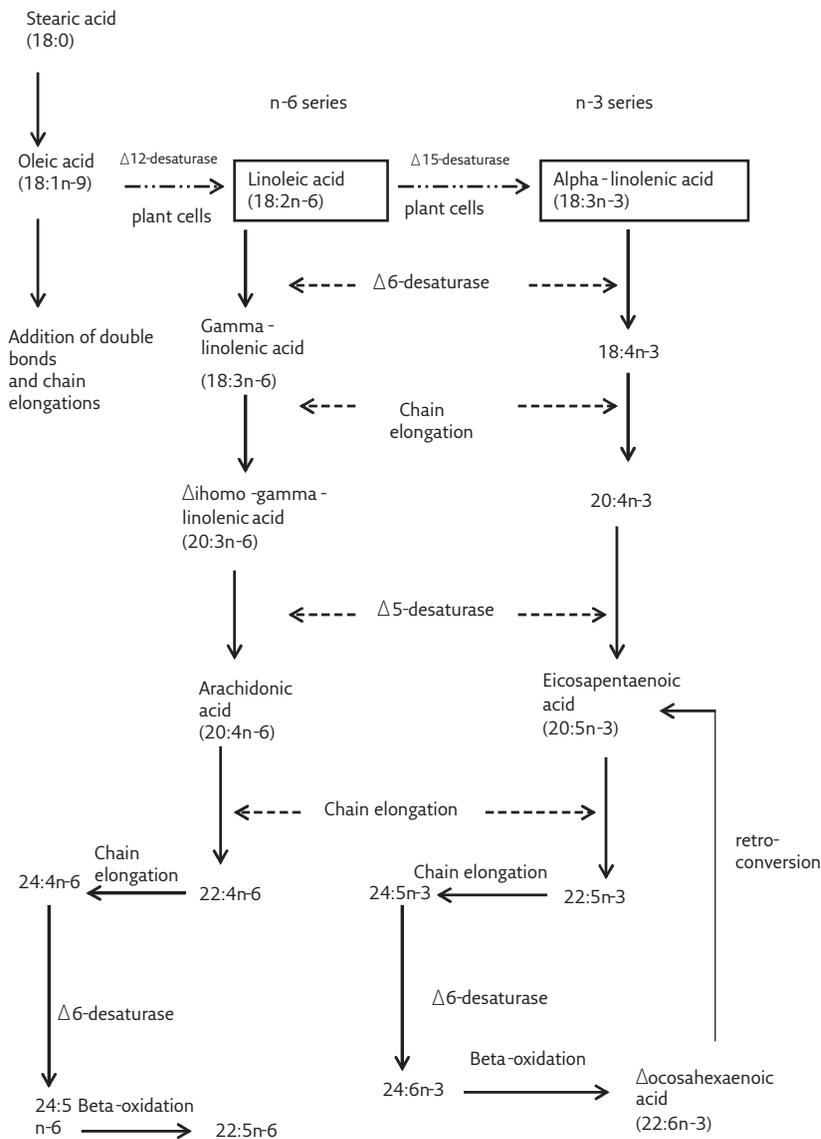


Figure 10.1. Metabolism of polyunsaturated fatty acids

In addition to triglycerides, dietary fats include phospholipids and cholesterol. The most common dietary phospholipid is phosphatidylcholine (lecithin), and cholesterol is found in foods of animal origin. Both phospholipids and cholesterol can be synthesised in the human body. Plants contain small amounts of plant sterols, mainly sitosterol and campesterol

and the corresponding saturated sterols sitostanol and campestanol that are poorly absorbed from the intestine and interfere with the absorption of cholesterol.

TFA are chemically formed by partial hydrogenation and deodorization of vegetable and fish oils (industrial TFA). They are also formed by natural biohydrogenation of fatty acids in the rumen of cattle, sheep, and goats (ruminant TFA) and, therefore, are present in milk and meat. Generally there are more than 10 different trans-18:1 isomers present in ruminant and partially hydrogenated fats (EFSA 2010). The fat of the milk and meat from cattle, sheep, and goats typically contains 3%–6% TFA (as the per cent weight of total fatty acids) of which D11-trans vaccenic acid (18:1t n-7) comprises 30%–50% of the total trans 18:1 isomers (10). There could also be TFA in pork and poultry fat, depending on the feed, but in lower amounts than in ruminant fat. Industrially, partially hydrogenated vegetable oils contain varying amounts of trans isomers with elaidic acid (18:1t n-9) accounting for 20%–30% and trans vaccenic acid accounting for 10%–20% of total trans 18:1 isomers. The TFA profiles of ruminant fat and hydrogenated vegetable oil show considerable overlap for many TFA isomers, but they are present in different proportions (10).

High intake of TFA has been associated with increased risk of coronary heart disease (CHD), sudden death, type 2 diabetes mellitus (T2DM), and increased circulating markers of systemic inflammation (11). The TFA found in partially hydrogenated oils has been associated with increased risk of CHD and appears to be more potent than SFA in the development of CHD (12). FAO recommends a mean population intake of less than 1 E% from both ruminant and industrially derived TFA (13).

One particularly important group of TFA are the conjugated linoleic acids (CLAs) that are formed by bacteria in the rumen and by desaturation of trans MUFA in the organism. The cis-9, trans-11 CLA that is the predominant isomer in milk fat has exhibited anti-carcinogenic properties in experimental animal studies. A chemically produced mixture of CLA isomers reduces fat mass and increases lean body mass in experimental animals. In humans, the effect has been less prominent (14). The trans-10, cis-12 CLA-isomer, which is industrially produced but also present in very low amounts in dairy fat, seems to be responsible for the adipose tissue effects. The same isomer has been found to increase insulin resistance (15) and C-reactive protein levels in humans (16).

Triglycerides are hydrolysed by lipases in the gut to mono-glycerides and fatty acids, which together with bile salts, lysophospholipids, and un-

esterified cholesterol form mixed micelles from which the digested lipids are absorbed in the small intestine. Fats are not soluble in water and are transported in the blood as lipoprotein particles. The core of the lipoprotein particles is formed by triglycerides and esterified cholesterol. The surface of the particles is composed of free cholesterol, phospholipids, and proteins. The lipoproteins are commonly divided into four classes according to density: chylomicrons, VLDL (very low-density lipoprotein), LDL (low-density lipoprotein), and HDL (high-density lipoprotein).

Essential fatty acids

LA (C18:2 n-6) and ALA (C18:3 n-3) are the essential fatty acids (EFA) that must be provided in the diet because the human body lacks the enzymes $\Delta 12$ - and $\Delta 15$ -desaturase that are capable of introducing double-bonds at the n-6 and n-3 positions (Figure 10.1.). These EFA serve important physiological functions. For example, LA, when incorporated into skin ceramides, is essential for maintaining the water-permeability barrier of the skin thereby avoiding excessive trans-epidermal water loss and the accompanying energy loss from water evaporation.

Physiology and metabolism

Both LA and ALA can be elongated and desaturated in the body (Figure 10.1.). LA is metabolised, for example, to γ -linolenic acid (C18:3 n-6), dihomo- γ -linolenic acid (C20:3 n-6, DHGLA), and arachidonic acid (C20:4 n-6, AA). Eicosapentaenoic acid (C20:5 n-3, EPA), docosapentaenoic acid (C22:5 n-3, DPA) and docosahexaenoic acid (C22:6 n-3, DHA) are formed from ALA. There is considerable inter-individual variation in the formation of DHA from ALA related to common polymorphisms in the Δ -5 and Δ -6 desaturase genes FADS1 and FADS2 (17). DHGLA, AA, and EPA are further metabolised by other enzymes (e.g. cyclo-oxygenases and lipoxygenases) into eicosanoids, a group of biologically active substances including prostaglandins, prostacyclins, leukotrienes, and thromboxanes. These highly active substances modulate the regulation of blood pressure, renal function, blood coagulation, inflammatory and immunological reactions, the sensation of pain, and other tissue functions.

The n-6 and n-3 PUFA, particularly the long-chain metabolites, are important structural components of cell membranes. They are essential for various membrane characteristics and functions such as fluidity, permeability, activity of membrane-bound enzymes and receptors, and

signal transduction. DHA is necessary for growth of the brain and other membrane-rich tissues in foetal and early postnatal life and thus plays a significant role in neurological development and visual function.

The n-6 and n-3 PUFA compete for the same enzymes (e.g. desaturases, elongases, and cyclo-oxygenases), and the n-3 series fatty acids have a higher affinity for the enzymes. An imbalance between dietary intakes of LA and ALA might thus influence the further metabolism to more long-chain and unsaturated n-6 and n-3 fatty acids. However, the data from most human studies using radioactive tracers do not show any major impact on ALA conversion in diets with varying n-6:n-3 ratios (18, 19). Some feeding studies show an impact on EPA concentrations in serum phospholipids (20). The interpretation of the results from various studies is further complicated due to differences among studies in absolute intakes and ratios. The total intake of each of the n-6 and n-3 fatty acids is more important than the ratio, as long as basic dietary requirements are covered. This is supported by the FAO report (13) that concludes that the ratio is of limited relevance when dietary intakes are within the recommended reference intakes.

In humans, high intakes of PUFA can potentially result in adverse effects including increased lipid peroxidation, impaired immune function, and increased bleeding tendency (21). Intakes of n-6 fatty acids (LA) up to around 10 E% are considered safe (13, 21). The EFSA concluded that combined long-term supplemental intakes of EPA and DHA up to about 5 g/d did not appear to increase the risk of spontaneous bleeding episodes or bleeding complications or to affect glucose homeostasis, immune function, or lipid peroxidation provided that the oxidative stability of the n-3 long chain PUFA (LCPUFA) was guaranteed (22).

Deficiency

Clinical symptoms of EFA deficiency (skin changes and growth retardation) have been found in healthy, new-born babies fed for 2 to 3 months with a diet low (<1 E%) in LA. EFA deficiency in adults is rare. Reported cases have been associated with chronic diseases or prolonged parenteral or enteral nutrition either without fat or very low in fat. The minimum requirement for LA remains unknown. Combined deficiency of LA and ALA leads to increased formation of the PUFA C20:3 n-9 and an increased C20:3 n-9/C20:4 n-6 ratio. It has not been confirmed, however, that this ratio is a useful indicator of EFA deficiency in humans.

Clinical signs (skin changes) of insufficient supply of ALA have been

reported at intakes of <0.05 E% during enteral nutrition and <0.1 E% during parenteral nutrition, but the specificity of these findings has been challenged. Humans are able to desaturate and elongate ALA to EPA and DPA, but further desaturation to DHA might be limited (Valsta et al. 1996). Conversion is higher in women (up to 21%) than in men (up to 8%) (12, 23, 24). The conversion rate is also affected by the intake of EPA and DHA as well as the intake of LA and of ALA itself (25-27). Two grams per day of DHA has been shown to be superior to the same amount of EPA in erythrocyte membrane incorporation of both EPA and DHA, but an ALA intake of 4 g/d did not increase the proportion of these longer chain n-3 fatty acids in a 6-wk intervention (28). There is also retroconversion of DHA to EPA and DPA. The estimated retroconversion rate varies between 1.4% and 12% depending on, for example, the DHA intake (29, 30).

DHA is found at high concentrations in the synapses of the central nervous system and in the rod outer segment of the photoreceptor cells of the retina where it is essential for the development of normal visual function (31). Studies in preterm infants strongly suggest that DHA is essential for normal development of visual function and perhaps for optimal psychomotor development (32, 33). These findings support the concept that it is necessary to consume n-3 fatty acids at least in amounts sufficient to replace physiological losses.

Several studies indicate that the enzymes that are responsible for the metabolism of EFA cannot synthesize enough long-chain PUFA from their parent fatty acids to meet the needs at birth, at least not in infants born before term, although the capacity for PUFA synthesis in preterm infants might be higher than in term infants (34). Therefore, AA and DHA, which are present in human milk, should be considered conditionally essential for a limited time after birth. It is recommended that a small proportion of AA and DHA, resembling the amounts in human milk, should be included in infant formula intended for preterm infants. There is as yet no consensus as to whether these fatty acids are also conditionally essential for infants born at term, although it has been recommended that formula intended for term infants should also be supplemented with AA and DHA (35). Supplementation of infant formula with long-chain PUFA has been associated with lower blood pressure during later childhood (36). Such supplementation is in accordance with the European directive on infant formula and follow-on formula intended for term infants, although the directive does not give a specific recommendation for supplementation with AA and DHA (37).

Intake of long chain n-3 fatty acids during pregnancy improves the n-3 status of the foetus and new-born child (38) and might be beneficial for the mental development of the child as assessed by their IQ (39). Plasma n-3 long chain fatty acid concentrations that are optimal for both mothers and infants have to be defined before general recommendations for intake are made (40). The n-3 content of breast milk is affected by the mother's intake (41), and this in turn can affect the development of visual acuity in the breastfed infant (42).

Cholesterol

Cholesterol is formed in various types of cells in the human body, and it is used for the production of bile acids and steroid hormones and for cell membrane structures. Cholesterol synthesis is highly regulated, and its uptake by cells reduces endogenous synthesis. About one gram of cholesterol is synthesised in human adults every day, and this is 3 to 4 times the amount absorbed from the average adult Nordic diet.

The most important dietary sources of cholesterol are meat and of-fal, eggs, and dairy products. The fractional absorption of cholesterol is reduced when the intake increases. On average, 40% to 50% of dietary cholesterol is absorbed, but absorption varies between individuals and can range from 20% to 80%. According to a meta-analysis of 17 randomized controlled trials (RCTs) published from 1974 to 1999 (24), 100 mg of dietary cholesterol increased serum total cholesterol by 0.056 mmol/L and HDL-cholesterol by 0.008mmol/L and slightly increased the total-cholesterol to HDL-cholesterol ratio by 0.020 units. In individuals with the apoprotein E4 allele, dietary cholesterol has a more pronounced effect on serum cholesterol concentration whereas in those without the apoprotein E4 allele dietary cholesterol has a weaker effect on serum cholesterol concentration (43).

Several expert groups, mainly in the US, have recommended that cholesterol intake in adults should be kept below 300 mg/d and those at high risk of cardiovascular diseases, e.g. those with T2DM, should not exceed 200 mg/d (44, 45). The average cholesterol intake in the Nordic countries is 250–350 mg/d. It is anticipated that the dietary guidelines promoting increased consumption of vegetable foods and limiting excessive intake of fatty dairy and meat products will lead to a reduction in cholesterol intake (46). Therefore, the current NNR does not set an upper intake level for cholesterol. Apparently, the endogenous capacity to synthesise cholesterol

is sufficient to meet the needs even of preterm infants. Therefore, there is no recommendation that infant formula should contain cholesterol even though cholesterol is a natural constituent of human milk.

Dietary fat, fatty acids, and health

Dietary fat and fatty acid composition has been linked to the risk of cardiovascular diseases (CVD), certain types of cancer, obesity, and gallstones. The relatively high amount of fat, especially SFA, in the diet in the Nordic countries during the 1960s and 1970s has contributed to the high prevalence of CVD in these countries (47). Serum/plasma LDL-cholesterol concentration has been identified as an important and causal risk factor for atherosclerosis, and a high serum/plasma HDL-cholesterol concentration and a low LDL-cholesterol to HDL-cholesterol ratio are associated with a reduced risk for atherosclerosis. A higher risk profile already seen in childhood is associated with an increased risk of atherosclerosis and CHD (48, 49).

For the update of NNR 2012, a systematic review (SR) was carried out with the aim of assessing the effect of, and to grade the evidence in regard to, the amount and type of dietary fat and biomarkers of the quality of dietary fat on risk factors and risk of non-communicable diseases, i.e. CVD, T2DM, and cancer, and body weight in healthy subjects or subjects at risk for these diseases (50). The SR included papers published from 2000 up to February 2012.

Serum lipid profile

Serum LDL-cholesterol concentration has been causally related to atherosclerosis based on modern genetic studies, and the ratio of LDL-cholesterol to HDL-cholesterol, as well as the non-HDL-cholesterol concentration, are good markers for CVD risk (51-53).

When SFA and TFA are replaced by cis-MUFA and PUFA, the LDL-cholesterol concentration in serum is reduced while the HDL-cholesterol concentration usually remains unchanged, i.e. the total-cholesterol to HDL-cholesterol ratio improves (54-62).

The NNR SR by Schwab and co-workers (50) included 45 RCTs investigating the effect of different fatty acids on serum lipids. The evidence that the serum/plasma concentrations of total cholesterol and LDL-cholesterol are reduced when SFA is replaced by cis-MUFA or PUFA was evaluated as *convincing* (50). There was no adverse effect on serum/plasma HDL-cholesterol concentration. Substituting cis-MUFA for SFA or carbohydrates

might even have a favourable effect on HDL-cholesterol concentration and the total-cholesterol to HDL-cholesterol ratio (57, 63). However, there is no direct evidence that increasing the HDL-cholesterol concentration itself lowers the risk of CHD (64). If total fat intake is markedly reduced in addition to replacing SFA or carbohydrates with cis-MUFA, the HDL-cholesterol concentration decreases and the concentration of triglycerides increases (57). Increased physical activity might counterbalance the effects of reduced fat intake on HDL-cholesterol (65, 66). In the SR by Schwab and co-workers (50), the evidence for replacing SFA by cis-MUFA or PUFA in regard to concentrations of serum/plasma HDL-cholesterol was evaluated as *limited - no conclusion*, and the effect on concentration of serum/plasma total triglycerides was *unlikely*. The evidence for replacing carbohydrates with cis-MUFA or PUFA was evaluated as *limited - no conclusion* for serum/plasma concentrations of total cholesterol, HDL-cholesterol, and total triglycerides. For LDL-cholesterol concentration, the evidence was evaluated as *unlikely*.

RCTs investigating dietary interventions aiming to improve serum or plasma lipid profiles in free-living individuals who have decreased the amount or improved the quality of dietary fat have shown a mean reduction in serum total cholesterol of 8.5% in 3 months and 5.5% in 12 months, but the dietary goals were seldom achieved (67). In metabolic ward studies, the compliance was better and the serum cholesterol concentrations were reduced by 10% to 15% (68).

The effects on LDL-cholesterol of specific SFA differ to some extent. Myristic acid (C14:0), palmitic acid (C16:0), and lauric acid (C12:0) increase both LDL- and HDL-cholesterol concentrations (C14:0>C16:0>C12:0), but stearic acid (C18:0) has a neutral effect comparable to that of oleic acid (n-9 C18:1). TFA from partially hydrogenated vegetable oils or fish oils increase LDL-cholesterol concentrations almost as much as the C12-C16 SFA but reduce HDL-cholesterol concentrations (34, 35, 36). Replacing 1 E% of TFA with 1 E% SFA, cis-MUFA, or PUFA decreases the total-cholesterol to HDL-cholesterol ratio by 0.31, 0.34, and 0.67 units, respectively (69). The source of partially hydrogenated fat might also have an effect because TFA from partially hydrogenated fish oils have been shown to affect LDL- and HDL-cholesterol concentrations more than partially hydrogenated soybean oil (70).

Long chain n-3 PUFA (EPA and DHA) can increase serum LDL-cholesterol concentrations (71, 72). In subjects with T2DM the increase of serum LDL-cholesterol concentration is 11% on average (73). The effects

of fish oil supplements and DHA might differ such that DHA has a stronger effect than fish oil when compared with cis-MUFA (50). The evidence for the effect on LDL-cholesterol concentration was evaluated to be *suggestive* for DHA and *inconclusive* for fish oil. In contrast to the effect on serum/plasma cholesterol concentrations, long chain n-3 PUFA decrease the concentration of serum triglycerides (73). In the SR by Schwab et al. (50), the evidence of the effect on the concentration of total triglycerides in serum/plasma was evaluated as *probable* for fish oil, i.e. EPA + DHA, but *inconclusive* for DHA alone. No adverse effects from EPA or DHA have been reported when consumed in the form of fish (74).

The SR by Schwab and co-workers (50) concluded that the evidence for the hypotriglyceridemic effect of fish oil supplementation compared with cis-MUFA was *probable*, whereas DHA supplementation did not seem to have a hypotriglyceridemic effect. In comparisons of fish oil with other types of PUFA, the evidence for the effect on serum/plasma total cholesterol concentration was evaluated as *unlikely*, and the evidence for its effect on concentrations of LDL- and HDL-cholesterol and total triglycerides was evaluated as *limited - no conclusion*.

Glucose tolerance and insulin sensitivity

The NNR SR by Schwab and co-workers (50) included 11 RCTs investigating the effect of dietary fat on insulin sensitivity and 20 studies on fasting serum/plasma insulin concentration.

In comparisons of the effects of cis-MUFA or PUFA and SFA on insulin sensitivity measured either by insulin sensitivity index (SI) or homeostasis model insulin resistance (HOMA-IR), insulin sensitivity was improved in both cis-MUFA-enriched and PUFA-enriched diets (56, 60). There are also studies showing no difference for cis-MUFA or SFA (58, 59, 75). However, obesity might alter the effect of the quality of fat on insulin sensitivity. When cis-MUFA has been compared with both carbohydrates and SFA (76-79), cis-MUFA resulted in improved HOMA-IR and SI. The amount of fat in the diet might also modify the effect of the quality of fat. In the KANWU study, it was shown that replacing SFA with MUFA improved insulin sensitivity in healthy subjects whose fat intake was below the median intake, i.e. 37 E% (56). There is also evidence that carbohydrates might result in better SI than SFA (80). The SR by Schwab and co-workers (50) concluded that the evidence for a favourable effect of cis-MUFA on insulin sensitivity or fasting serum/plasma insulin concentration in comparison with carbohydrates and SFA was *probable*.

In a meta-analysis of RCTs, n-3 fatty acids did not affect insulin sensitivity (81). The effects of fish oil as fish oil supplements and ALA do not differ in terms of SI (82). Fish oil supplements did not have an effect on SI, first phase insulin secretion, disposition index (K_G) when added either to the diet high in cis-MUFA or SFA (56, 83). Regarding insulin sensitivity, 12 out of 15 studies included in a meta-analysis found no effect of the quality of dietary fat, but the quality of the studies was questionable (84).

When unsaturated fatty acids have been compared with SFA, no differences in fasting plasma or serum glucose concentrations have been found (56, 58, 60, 75). Regarding plasma or serum insulin concentrations, only one of these studies showed a favourable effect from MUFA (56). In a comparison between MUFA and carbohydrates, MUFA resulted in better fasting plasma/serum glucose concentration in one study (76) but in other studies no difference has been found (77, 78, 80). On the contrary, in all of these studies fasting plasma or serum insulin concentrations were improved while on a MUFA-rich diet compared to a SFA-rich diet. Most of these studies, however, were very short term, and in some of these studies the diets were high in fat (about 40 E%) (58, 78).

The SR by Schwab and co-workers (50) concluded that the evidence for an effect on blood glucose by replacing SFA with cis-MUFA or PUFA was *unlikely*. The evidence for replacing SFA with cis-MUFA or carbohydrates was evaluated as *limited - no conclusion*.

Blood pressure

Dietary fat composition might influence blood pressure, and it is possible that a very low intake of n-3 PUFA increases blood pressure (85). However, at intakes exceeding the minimum requirement blood pressure is not affected by PUFA intake.

In comparisons of cis-MUFA and SFA, diets rich in cis-MUFA have resulted in lower blood pressure in some studies (86-88). Recently, a prospective randomized intervention found lower blood pressure among those with lower intake of SFA from infancy (89). The amount of fat might play a role in the effect of changing the quality of dietary fat on blood pressure. In one study with a high intake of fat (about 40 E%), no difference between MUFA and SFA was found (58) and in another study the beneficial effect of MUFA was more pronounced in those subjects with a fat intake below 37 E% (87).

Fish oil supplement has been observed to give varying results. In randomized trials, it decreased blood pressure in young overweight adults (90),

in infants (91), and in adolescents (92). A recent observation found lower blood pressure among elderly people taking fish oil supplements (93), but such intake was found to increase blood pressure in pregnant women and decrease the size at birth of their babies (94, 95).

The SR of Schwab and co-workers (50) concluded that the evidence for an effect of any modification of the quality of dietary fat on blood pressure was *limited – no conclusion*.

Body weight

Intervention studies have shown that reduced-fat diets consumed *ad libitum* contribute to weight reduction (96), although the effect is limited and RCTs with durations from six months to over eight years have shown average weight losses of 1.4-1.6 kg. Results from prospective cohort studies do not indicate any association between fat intake and body weight (97) in contrast to the somewhat different results from the RCTs. The evidence from RCTs for a positive association between the amount of dietary fat and body weight was found to be *probable* in the SR by Schwab and co-workers (50). There is no evidence that the quality of fat has any effect on body weight (50).

CVD

The NNR SR included 29 publications on the association between dietary fat and fatty acids and cardiovascular outcomes (50). For *total fat intake*, the results from the included studies showed no difference with respect to the risk of any of the CVD outcomes (50). Mean intakes of total fat in the prospective studies varied from 35 E% to about 45 E%. In summary, a direct association between total fat intake and CVD outcomes is *unlikely*. For SFA, MUFA, and PUFA, a meta-analysis comprising 28 cohort studies and 16 RCTs found no association between the intake of SFA and the risk of CHD independent of the intake of unsaturated fat or a healthy dietary pattern (98).

In a meta-analysis of 48 RCTs, substituting unsaturated fatty acids for SFA reduced CVD events by 14% (99). Meta-analyses of intervention studies showed that reducing SFA intake by reducing and/or modifying dietary fat reduced the risk of cardiovascular events by 14%. The reduction in CVD events was seen in studies of fat modification of at least two years' duration in which the risk reduction was 22%. Significant risk reductions were seen in men, but not in women (99), and the effect might be more pronounced in younger subjects (100).

A meta-analysis of eight RCTs compared the effect on CHD of interventions with increased intake of PUFA as a replacement for SFA. PUFA intake in the intervention groups was 15 E% compared to 5 E% in the control groups. The results of those studies showed a significant overall risk reduction of 19%, which corresponds to a 10% reduced CHD risk for each 5 E% increase of PUFA intake. Studies of longer duration showed greater benefits (101). A pooled analysis of data from 11 US and European prospective cohort studies showed a 20% decreased risk of CHD in both men and women when 5% of the energy was changed from SFA to PUFA (102). Furthermore, pooled evidence from different types of studies showed a ≥ 2 -3% reduced risk of CHD when 1 E% SFA was replaced with PUFA (103).

The SR by Schwab and co-workers (50) concluded that there is *convincing* evidence that partial replacement of SFA with PUFA decreases the risk of CVD, especially in men.

Substituting MUFA for SFA does not affect the CHD risk in epidemiological studies (102). In long-term prospective cohort studies, MUFA have been found to have a favourable effect on the risk of CHD, although unfavourable effects have also been reported (88).

The SR by Schwab and co-workers (50) concluded that the evidence for the favourable effect of cis-MUFA on CHD was *unlikely* for a direct association. It is of note, however, that the intake of MUFA correlates highly with the intake of SFA except in countries where olive oil is used in abundance (104).

When SFA is replaced with carbohydrates without regard to the quality of the carbohydrates, no beneficial effect on CHD risk has been found. In the pooled analysis of 11 US and European cohort studies, the effect of replacing SFA with carbohydrates on ischaemic heart disease was unfavourable (102). In populations with very low intake of both total fat and SFA (<15 E% and <5 E%, respectively), such as in rural China, CHD is rare (105). This discrepancy is most likely explained by the quality of carbohydrates in the diet and lower BMI as well as the level of physical activity (106). However, differences in life expectancy compared to the Nordic countries might also explain this discrepancy. In a Danish prospective cohort study, replacement of SFA with carbohydrates with high glycaemic index increased the risk of myocardial infarction whereas a replacement with carbohydrates with low glycaemic index did not affect the risk (107).

Dietary intake of n-3 fatty acids of animal origin such as fish and, in some studies, of ALA of plant origin has reduced mortality in patients with

CHD (108-111) as has the use of fish oil supplements (112). However, whole fish might be more beneficial than fish oil supplements (111, 113). The effect of n-3 fatty acids might be mediated by reduced risk of cardiac arrhythmias (113). An intake of 200-250 mg/d of EPA + DHA has been shown to be effective with no further benefit with an increasing dose (114, 115). A recent Scandinavian study reported an increased overall risk of CVD at a very low intake (<0.06 g/d) of n-3 long chain PUFA (85). In the SR by Schwab and co-workers (50), the evidence of long chain n-3 PUFA on CVD risk was evaluated as *suggestive*. However, it is important to pay attention to the source of long chain n-3 PUFA - either fish or fish oil supplement - when assessing the evidence.

In a Finnish study, the proportion of ALA in serum lipids showed a similar inverse association with the risk of CHD death as the proportions of EPA and DHA (46). ALA intake has also been shown to be associated with decreased risk of CHD (98, 116, 117). The evidence for ALA intake on CVD risk was evaluated as *suggestive* in the SR by Schwab and co-workers (50). However, the proportion of total PUFA, n-6 PUFA, and LA in plasma lipids also showed a favourable effect, and the evidence for the inverse association of these variables with CVD mortality was evaluated as *suggestive*.

A high intake of TFA has been associated with increased risk of CVD in some prospective studies (118-120) as well as in a meta-analysis of cohort studies (98). In a study using principal component analysis of the plasma phospholipid fatty acid composition, TFA were related to a greater risk of CVD risk and progression of atherosclerosis in women with ischaemic heart disease (121).

Stroke

Long chain n-3 PUFA in the form of fish intake reduces the risk of stroke mortality (122), and a very low long chain n-3 PUFA intake (<0.06 g/d) increases the risk of stroke (85). The meta-analysis by Hooper and co-workers (99) included stroke as a secondary outcome. Eleven trials reported on stroke events and showed no significant overall effect of reduced and/or modified fat intake, although this was largely driven by results from the WHI trial (123) that mainly focused on reduction in total fat intake.

T2DM

Total fat intake and SFA intake were associated with a higher risk of T2DM in a prospective cohort study, but these associations were not independent of BMI (124). An increase in PUFA intake from 3 E% to around 6 E% in

exchange for SFA or carbohydrates might be associated with a 20% reduction in the risk of T2DM (125-127). According to Schwab and co-workers (50), there is *probable* evidence that LA intake has a favourable effect on the risk of T2DM. The evidence for a favourable effect of the proportion of LA in plasma phospholipids and cholesteryl esters was *suggestive* (50). Non-significant associations have been reported as well (128).

An increase in the intake of long chain n-3 PUFA from <100 mg/d to >360 mg/d has been associated with a 20%-40% increased risk of T2DM (124, 126, 128, 129). However, in a recent meta-analysis, including studies examining the effect of both fish intake and fish oil supplementation, the effect of long chain n-3 PUFA on the risk of T2DM was not significant (130). As mentioned in the section on CVD, it is important to pay attention to the source of long chain n-3 PUFA - either fish or fish oil supplement - when assessing the evidence.

The proportion of SFA in plasma phospholipids and cholesteryl esters might be associated with increased risk of T2DM, and the evidence for this was evaluated as *suggestive* in the SR by Schwab and co-workers (50). Odd chain SFA (C15:0 and 17:0) might have an inverse association with the risk of T2DM, and the evidence for this was evaluated as *suggestive* (50). Odd chain fatty acids have been considered as a biomarker of dairy fat intake, but both C15:0 and C17:0 also exist in fish, even in higher amounts than in dairy fat (131-133). In the EPIC study, there was a strong positive correlation ($r = 0.8$) between total fish intake and C17:0 in plasma phospholipids (133).

Reduction of total fat and SFA intake in conjunction with modest weight reduction, increased intake of dietary fibre, and increased physical activity reduces the risk of diabetes in subjects with glucose intolerance (134-138). In the Finnish Diabetes Prevention Study (DPS), diabetes incidence was associated with a diet high in fat and low in carbohydrate. Thus, a diet high in fat might even be detrimental in people with impaired glucose metabolism, i.e. in 25-40% of the population (135).

Cancer

In experimental animal studies, fat intake promotes the development of breast cancer and colon cancer. Experimental studies also suggest that n-3 fatty acids counteract cancer cell proliferation, and n-6 fatty acids tend to have the opposite effect (139, 140). However, epidemiological studies examining the link between dietary fats and cancer risk have reached mixed results. Such discrepancies can partly be explained by the complex

composition of dietary fat and the presence of other bioactive compounds in the diet (141) or by diverse confounding factors such as drug use (142), unstable food habits over time (143), or genetic variations in key metabolising enzymes (140).

Breast cancer is the major cancer type in women, and the studies included in the SR by Schwab and co-workers (50) did not find any association between the risk of breast cancer and the intake of total fat in postmenopausal women. The evidence regarding both the intake and tissue markers of the quality of dietary fat was also evaluated as *inconclusive* in the SR by Schwab and co-workers (50). No association with the intake of total fat or quality of fat with other types of cancer, i.e. endometrial, colorectal, pancreatic, oesophageal, gastric, renal cell, bladder, lung, or skin cancer, was found in the SR by Schwab and co-workers (50). However, for ovarian cancer the evidence for a positive association with the intake of SFA is *suggestive*. There is also *suggestive* evidence for an inverse association with the intake of ALA and risk of prostate cancer.

Although the criteria for including studies were slightly different for the SR by Schwab and co-workers (50) and the World Cancer Research Fund/American Institute of Cancer Research (WCRF/AICR) report (139), the overall conclusions are generally similar. However, the WCRF/AICR report concluded that the evidence is *limited-suggestive* for a link between total fat and an increased risk of postmenopausal breast cancer and lung cancer. There was also *limited-suggestive* evidence for an increased risk of foods containing fat of animal origin and colorectal cancer. According to the WCRF/AICR report (139) there is an indirect link between energy-dense diets and cancer, and the evidence indicates a *probable* or *convincing* link between body fatness and most cancer types. Interestingly, an SR of food pattern studies (144) concluded that so called “prudent” dietary patterns are associated with reduced breast cancer risk while “Westernized” dietary patterns are associated with increased risk (See chapter on Foods and food patterns: Guidelines for a healthy diet). The quality of dietary fat is potentially closer to the recommendations in “prudent” dietary patterns compared to “Westernized” dietary patterns.

Pregnancy and lactation

DHA is necessary for growth of the brain and other membrane-rich tissues in foetal and early postnatal life. There is considerable inter-individual variation in the formation of DHA from ALA due to common polymorphisms in the Δ -5 and Δ -6 desaturase genes FADS1 and FADS2 (17).

Evidence from RCTs in which EPA + DHA was administered to pregnant women (from 150–200 mg DHA/d up to 1200 mg DHA/d) showed reduced risk of preterm birth (145, 146).

A consensus statement published in 2007 (147) advised that pregnant and lactating women's intake of DHA should be at least 200 mg/d. This is in line with the FAO (13), which set the average nutrient requirement (ANR) to 200 mg/d.

A Cochrane Review of six RCTs did not find convincing evidence for supplementation of n-3 LCPUFA with doses varying from 200 mg/d of DHA to between 1.5 and 2.7 g/d of EPA + DHA or 10 mL/d of cod liver oil to breastfeeding mothers for improving their children's neurodevelopment and visual acuity. However, supplementation had a positive effect on head circumference in two studies (10 mL/d of cod liver oil or 200 mg/d DHA) and decreased risk of allergic sensitisation in one study with a dose of 2.7 g/d of EPA + DHA (148). The results on allergic diseases and cognition differ somewhat between the effects of fish oil and other PUFA supplementation (149, 150). An intake of DHA slightly greater than 1 g/d or a total intake of 2.7 g/d of n-3 LCPUFA (both EPA and DHA) is considered safe (147).

Recommendations

Adults and children from 2 years of age

SFA: Intake of SFA should be limited to less than 10 E% calculated as triglycerides. Even lower levels might be desirable in persons with hypercholesterolaemia. Detailed recommendations on intakes of specific SFA are not given. The cholesterol-raising fatty acids are generally found in the same foods that are the main sources of total SFA.

TFA: The intake of TFA both from fat-containing dairy products and partially hydrogenated, industrially produced fats should be as low as possible. A reduction in SFA intake will generally also reduce the intake of TFA and dietary cholesterol.

Cis-MUFA should contribute 10–20 E%.

Intake of cis-PUFA (the sum of n-6 and n-3 fatty acids) should contribute 5–10 E%, including at least 1 E% from n-3 fatty acids. An intake of EPA + DHA up to 200–250 mg per day has been associated with decreased risk of CVD.

Intake of cis-MUFA and cis-PUFA should make up at least two thirds of the total fatty acids.

Because minimum requirements of cis-PUFA for adults are not known, the estimates are based on threshold intake data from children. The recommendation for EFA, i.e. LA and ALA, is 3 E%, of which at least 0.5 E% should be ALA.

For pregnant and lactating women, the contribution of cis-PUFA should be at least 5 E%, including 1 E% from n-3 fatty acids of which 200 mg/d should be DHA.

The upper intake range for total PUFA intake is 10 E%. Increased intakes are not recommended due to potential adverse effects.

The evidence for defining an optimal ratio between n-6 and n-3 PUFA is insufficient and in NNR 2012 no recommendation for the ratio of n-6 to n-3 has been set.

The total fat recommendation is 25–40 E%, which is mainly based on the recommended ranges for fatty acid categories. Very low-fat diets tend to reduce HDL-cholesterol concentration and increase triglyceride concentration in serum/plasma and might impair glucose tolerance in susceptible individuals (151). Furthermore, it is difficult to ensure sufficient intake of fat-soluble vitamins and EFA if total fat intake is below 20 E%.

A limitation of the total fat intake will facilitate achieving the recommended intakes of certain micronutrients and dietary fibre. The amount of dietary fat is also associated with body weight. Experiences from the STRIP and Young Finns studies show that a moderate fat intake (about 30 E%) with a reduction in SFA intake results in a better CVD risk profile and fewer atherosclerotic changes in early life (152–155).

For dietary planning purposes, a suitable target is the middle value of the range, i.e. about 32–33 E%, given that careful attention is paid to the quality of both fat and carbohydrates and the amount of dietary fibre.

Children 6–23 months

Due to the rapid growth rate during infancy, fat accounts for about 50% of the total energy intake in human milk and infant formula. Because exclusive breastfeeding is recommended during the first 6 months of life, and because the fat content of infant formula and follow-on formula is regulated (40–55 E% in infant formula and 35–55 E% in follow-on formula) (37), no further recommendations are given for the first 6 months of life. After 6 months of age, this high energy density is reduced with increasing amounts of complementary foods. Thus the fat intake can decline rapidly to around 30 E% at the end of infancy depending on the composition of the complementary food and the extent of partial breastfeeding. It is also common that after the first

year the proportion of fat increases gradually until 3 years of age to levels common among adults. If the proportion of fat and, therefore, the energy density of the diet becomes too low in the first year or in early childhood this might result in insufficient energy intake because children of this age have limited capability for ingesting more voluminous servings.

According to the EFSA, total fat intake below 25 E% has been associated with low vitamin intakes in some young children (10). The German-Austrian-Swiss recommendation (156) of total fat intake for infants 4 to 11 months of age is 35–45 E%. The US Institute of Medicine has set an adequate intake (AI) for 7–12 month olds to 40 E% (157). Similarly, the EFSA has set an AI at 40 E% for children aged 7–12 months based on AI and consensus reports (10, 158). In the NNR 2012, the intake of total fat for infants between 6 and 11 months of age is recommended to be kept between 30 E% and 45 E%, and this is the same as in the NNR 2004.

Some studies in children indicate that a fat content around or below 30 E% of the total energy is already applicable after the age of 1 year because fat intakes at these levels did not adversely influence children's growth and neurological development in the Finnish STRIP study (159). The German-Austrian-Swiss 2008 recommendation of total fat intake for children aged 1 to 3 years is 30 E% to 40 E% (156) and similarly the US Institute of Medicine set an acceptable macronutrient distribution range (AMDR) for the proportion of fat to 30–40 E% for children 1 to 3 years of age (157). The EFSA set the recommended intake for the same age group (1–3 years old) to 35–40 E% (10).

In NNR 2012, the intake of total fat for children from 12 to 23 months of age is recommended to be kept between 30 E% and 40 E%. From the age of 2 years, the recommendation of total fat intake is the same as for older children and adults in NNR 2012.

The quality of dietary fat is also important in infancy and childhood. From the age of 12 months, the intake of SFA should be less than 10 E%. The intake of TFA both from dairy fat and partially hydrogenated, industrially produced fats should be kept as low as possible. Partial breastfeeding is recommended from 6 months and throughout the child's first year, and can be continued for as long as it suits the mother and the child. Half or more of the energy from human milk is fat. Typical fatty acid composition (wt%) in mature breastmilk is 40–45% SFA, 40–45% MUFA and 13–16% PUFA (160–164) There is, however, no evidence for a higher recommendation of saturated fat intake for 6–11 month old children than the recommendation for older children, i.e. lower than 10 E%.

For the intake of cis-PUFA in childhood, no new convincing evidence has emerged for changing the recommendations from NNR 2004. The EFSA's AI for LA is 4 E% and AI for ALA is 0.5 E%, but the EFSA also adds an AI for DHA of 100 mg/d (10). This is primarily based on cohort studies and a few randomized trials with DHA supplementation to the foetus or young infants that indicate a positive effect on visual acuity, cognitive function and attention, maturity of sleep patterns, spontaneous motor activity, and immunity.

In the NNR 2012, it is recommended that the total intake of cis-PUFA for children 6–23 months of age should constitute 5–10 E% and that this should include at least 1 E% from n-3 fatty acids, including DHA, as in NNR 2004. However, the optimum ratio of n-6 to n-3 fatty acids is not known.

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Carbohydrates

	Dietary fibre	Added sugars, E%	Carbohydrates Total, E%
6–11 mo	–	< 10 E%	45–60*
1–17 years	2–3 g/MJ	< 10 E%	45–60*
≥ 18 years	>3 g/MJ	< 10 E%	45–60*
- women	≥ 25 g/d		
- men	≥ 35 g/d		

* acceptable range.

Introduction

Carbohydrates provide energy in the diet mainly as starch and sugars and to a lesser extent as dietary fibre and sugar alcohols. Generally used energy conversion factors are 17 kJ per gram of available (glycaemic) carbohydrates, 8 kJ per gram of total fibre, and 10 kJ per gram of sugar alcohols (polyols). Conversion factors partly depend on the methods used to derive carbohydrate values, e.g. “by difference” or by analysis of individual constituents (see the chapter on energy).

Dietary sources and intake

Cereals and potatoes are the major sources of carbohydrates in the Nordic diet. Fruit, fruit juices, berries, and milk provide sugars (mono- and disaccharides). Sweets, soft drinks, fruit syrups, sweetened bakery products, and sweetened dairy products are main sources of refined, added sugars.

Wholegrain cereals, fruits, berries, and vegetables provide the main proportion of the dietary fibre intake. In the Nordic diets, total carbohydrates contribute on average 43–52 E%, including 10–16 E% added sugars, and provide 25–27 g of fibre per 10 MJ.

Chemical classification

The chemical classification of carbohydrates is usually based on molecular size and monomeric composition. The three principal carbohydrate groups are sugars (1 or 2 monomers), oligosaccharides (3–9 monomers), and polysaccharides (10 or more monomers) (1, 2). The most important food carbohydrates are glucose, fructose, and galactose (monosaccharides); sucrose, lactose, and trehalose (disaccharides); oligosaccharides; and polysaccharides. There are two main classes of polysaccharides, starch and non-starch polysaccharides (NSP). Starch is a homopolymer of glucose and comes in two main forms, amylose (basically unbranched) and amylopectin (highly branched). NSP include a host of different polymers and are highly variable in terms of molecular size and structure as well as in monomeric composition. The main classes of NSP are cellulose, hemicelluloses, pectins, and hydrocolloids. Due to their structural variability, different NSP can have very different physical-chemical properties and these are of key importance for their physiological effects. Cellulose is insoluble in water, and pectins and hydrocolloids, e.g. guar gum and mucilages, can form highly viscous solutions in water.

Nutritional classification

Nutritionally, carbohydrates can be divided into two broad categories. The first include those that are digested and absorbed in the human small intestine providing carbohydrates to body cells, and the second include those passing on to the large intestine forming substrates for the colonic microflora (3, 4). The concept of “glycaemic carbohydrate”, meaning “providing carbohydrate for metabolism”, was introduced by the FAO/WHO (1, 2). The non-digestible (unavailable) carbohydrates (NDC) are commonly referred to as “dietary fibre”.

Glycaemic carbohydrates

The main glycaemic carbohydrates are:

- Glucose and fructose (monosaccharides)
- Sucrose and lactose (disaccharides)
- Malto-oligosaccharides
- Starch (polysaccharide)

The term “sugars” covers monosaccharides and disaccharides. In the literature, various terms are used to differentiate between sugars naturally occurring in foods, i.e. “intrinsic” sugars, and sugars and sugar preparations added to foods, i.e. “added” or “extrinsic” sugars (5, 6). In the NNR, the term “added sugars” refers to refined sugars such as sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup), and other isolated sugar preparations used as such or added during food preparation and manufacturing.

Fructose and glucose are mainly found in fruits, berries, juices, and some vegetables. Free galactose is rare in foods except in fermented and lactose-hydrolysed milk products. Fruits, berries, and juices also provide some intrinsic sucrose. Sucrose is found in varying amounts in manufactured foods, e.g. soft drinks and sweets, and is used as a sweetener and cooking ingredient in the household. More or less completely hydrolysed starches or high-fructose syrups, in which about half the glucose is isomerised to fructose, have been increasingly used to replace sucrose in confectionary and carbonated drinks. Lactose occurs exclusively in milk and milk products. Malto-oligosaccharides originate mainly from partially hydrolysed starch. Bread and other cereal products, potatoes, and tubers are major sources of starch (1, 2).

Sugar alcohols (polyols) such as sorbitol, xylitol, mannitol, and lactitol, are usually not included in the term “sugars”. However, they are absorbed to some extent by the body and are included in “carbohydrates” according to the European legislation for nutritional labelling (7).

Organic acids such as lactic acid, citric acid, and malic acid, which occur in fermented foods, fruits, and berries, respectively, can contribute to carbohydrates if measured “by difference.”¹

1 (100–(sum of protein, total fat, ash, and water)).

Table 11.1. Classification of main food carbohydrates and related substances, all included in carbohydrate as assessed “by difference”. Adapted from EFSA (8) and NNR 2004

Class (DP *)	Sub-group	Components	Monomers	Digestibility**	Analytical methods			
					Sugars	Starch	Dietary fibre AOAC ¹	
Sugars (1 or 2)	Mono-saccharides	Glucose		+	+			
		Galactose		+	+			
		Fructose		+	+			
	Disaccharides	Sucrose	Glu, Fru		+	+		
		Lactose	Glu, Gal		+(-)***	+		
		Trehalose	Glu		+	+		
Oligo-saccharides (3–9)	Malto-oligo-saccharides	Malto-dextrins	Glu	+	+	+		
		Other oligo-saccharides	a-Galactosides (GOS)	Gal, Glu	-	+/-		
	Fructo-oligo-saccharides (FOS)	Fructo-oligo-saccharides (FOS)	Fru, Glu		-	+/-		
		Poly-dextrose	Glu		-			
		Resistant dextrins	Glu		-			
	Polyols	Maltitol, sorbitol, xylitol, lactitol			+(-)****			
	Poly-saccharides (>9)	Starch	Amylose, Amylo-pectin	Glu	+(-)		+	+(-) ³
Modified starch			Glu	-		+/- ²	+	

Analytical methods							
Class (DP*)	Sub-group	Components	Monomers	Digestibility**	Sugars	Starch	Dietary fibre AOAC ¹
		Resistant starch	Glu	-		+/- ²	+
		Inulin	Fru	-			+ (-) ³
	Non-starch polysaccharides	Cellulose	Glu	-			+
		Hemi-celluloses		-			
		Pectins	Uronic acids	-			
		Other hydrocolloids, e.g. gums, mucilages, β -glucans	Variable	-			
Related substances		Lignin		-			+
		Tannins/polyphenols		+ (-) ⁴			+ (-) ³
		Phytate		-			+ (-) ³
		Organic acids		+			-

* DP = Degree of polymerisation.

** Denotes digestibility in the small intestine: + digestible, +(-) mainly digestible, +/- partly digestible, - non-digestible.

*** Lactose is poorly digested by individuals with low intestinal lactase activity.

**** Polyols are partly and variably absorbed.

¹ AOAC: Association of Official Analytical Chemists.

² +/- Determined in some but not all methods.

³ Partly determined.

⁴ Includes soluble, partly absorbable, and insoluble (non-absorbable) forms.

Fru = Fructose, Glu = Glucose, Gal = Galactose.

Dietary fibre

The main types of dietary fibre are:

- Non-starch polysaccharides - cellulose, hemicelluloses, pectins, hydrocolloids, etc.
- Resistant oligosaccharides - fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS), and other resistant oligosaccharides

- Resistant starch
 - Physically enclosed starch
 - Some types of raw starch granules
 - Retrograded amylose
 - Chemically modified starches
- Lignin (and other usually minor components associated with the dietary fibre polysaccharides)

The term dietary fibre was originally defined as “that portion of food which is derived from cellular walls of plants which are digested very poorly by human beings” (9). The recognition that polysaccharides added to foods, notably hydrocolloids, could have effects similar to those originating from plant cell walls led to a redefinition of dietary fibre to include “polysaccharides and lignin that are not digested in the human small intestine” (10).

Internationally, definitions of dietary fibre vary somewhat. The EFSA (8) defined dietary fibre as “non-digestible carbohydrates plus lignin including non-starch polysaccharides (NSP) – cellulose, hemicelluloses, pectins, hydrocolloids (i.e. gums mucilages, β -glucans), resistant oligosaccharides – fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS), other resistant oligosaccharides, resistant starch – consisting of physically enclosed starch, some types of raw starch granules, retrograded amylose, chemically and/or physically modified starches, and lignin associated with the dietary fibre polysaccharides”. This definition is the basis for the EC legislation on labelling (Commission directive 2008/100/EC), which also requires that beneficial physiological effects have to be demonstrated before natural or synthetic fibre can be added to foods. This is in accordance with the definition by Codex Alimentarius (11), although whether non-digestible carbohydrates with 3–9 monomeric residues should be included or not is so far left to the national authorities. The US Institute of Medicine (IoM) (12) uses the term “total fiber”, which is the sum of “dietary fiber” consisting of non-digestible carbohydrates and lignin that are intrinsic and intact in plants and “functional fiber” consisting of isolated, non-digestible carbohydrates that have beneficial physiological effects in humans.

With any definition of dietary fibre, NSP from plant cell walls, such as cellulose, hemicelluloses, and pectins, are the dominant components. Hydrocolloids can be either naturally occurring cell wall or storage components or added to foods as ingredients to obtain specific technological and/or nutritional benefits. Resistant oligosaccharides and resistant starch have partly similar physiological and nutritional effects as non-starch polysaccharides.

Cellulose is insoluble in water and occurs together with hemicelluloses in cereals. The lignified outer layers in wholegrain products are the predominant source of cellulose, and this type of fibre is most resistant to fermentation by the colonic microflora. Oats and barley contain high levels of β -glucan, a soluble viscous polysaccharide. Pectins – the main type of dietary fibre in fruits and vegetables – have similar properties.

The absorption of polyols in the small intestine depends on their structure and on the amount consumed, and when consumed in excess amount may cause gastrointestinal discomfort such as diarrhoea.

Considerable amounts of lactose can reach the colon in infants due to the high content of lactose in breast milk, and this can cause gastrointestinal discomfort and diarrhoea. This is also the case in children and adults with low intestinal lactase activity. A limited capacity to absorb fructose seems to be rather common, especially if this sugar is consumed alone without glucose, and this can be another cause of diarrhoea (13).

Analytical methods

Dietary fibre is usually analysed using enzymatic gravimetric or enzymatic chemical methods that include NSP, analytically resistant starch, and lignin. Methods measuring NSP alone give lower estimates in foods containing resistant starch and/or lignin, e.g. wholegrain flour and cereals processed in a way that generates resistant starch. Resistant oligosaccharides are not included in any of the current dietary fibre methods and, therefore, have to be measured separately and added to the total fibre estimate. Dietary fibre methods that include resistant starch measure the fraction resistant to the enzymes used in the assay. This “analytically resistant starch” includes mainly retrograded amylose, and the analytical methods need to be fine-tuned to correspond better to the physiologically resistant starch (3, 14).

In both epidemiological studies and mechanistic intervention studies, the term “dietary fibre” is usually used for non-digestible plant material as measured by official analytical methods approved by the Association of Official Analytical Chemists (AOAC). This means that dietary fibre includes NSP as the main component along with lignin and analytically resistant starch. Other NDC such as resistant oligosaccharides and inulin are not included and usually make up only a small part of the NDC in Nordic diets.

The dietary fibre recommendation in NNR 2012 refers to dietary fibre naturally occurring in plant foods as measured by AOAC methods for total dietary fibre.

With the methods currently used, some overlap might occur and some components might be missed (see Table 11.1.).

Physiology and metabolism

Glycaemic carbohydrates

The glycaemic carbohydrates provide carbohydrate to the cells of the body mainly in the form of glucose. In practice, only monosaccharides can be absorbed in the small intestine. The enzymatic degradation of starch begins by the action of salivary amylase and is continued in the small intestine by pancreatic amylase. The degradation products - mainly maltose and oligosaccharides - are further hydrolysed to glucose by a set of enzymes (disaccharidases) that are bound to the brush border membrane of the enterocytes. The same enzymes hydrolyse the dietary disaccharides. Glucose and galactose are absorbed efficiently by a secondary active carrier coupled with sodium (glucose transporter, GLUT 2), whereas fructose is absorbed by facilitated diffusion that does not involve sodium co-transport (GLUT 5). Absorption of monosaccharides is generally regarded as the rate-limiting step, but down-regulation of lactase (hypolactasia) occurs in most humans from 1 to 2 years of age to the teenage years resulting in a limited lactose absorption capacity. The same is true for sucrose in the rare case of sucrase deficiency (15-24) and for fructose in the hereditary congenital disorder fructose intolerance (25).

Absorbed sugars are transported to the liver and then to the systemic circulation, and cellular uptake is mediated by GLUT 1-4 that are variously expressed in different tissues. Insulin is a key hormone for the uptake and metabolism of carbohydrates, and the plasma insulin concentration increases immediately after ingestion of glycaemic carbohydrates. One important effect of increased insulin is an increase in the translocation of glucose transporters (GLUT 4) to cell membranes. This increases peripheral uptake and counteracts an excessive rise in blood glucose. Glucose is a preferred fuel for most body cells, and can be stored as glycogen in the liver and in the muscles. The storage capacity is limited to around 500 g, of which 300-400 g can be stored in the muscles. Liver glycogen is used to maintain normal blood glucose levels between meals, and muscle glycogen is used primarily as a source of energy within the muscles. Unlike glucose, fructose enters mainly liver cells without the need for insulin. The metabolism of fructose favours lipogenesis more than glucose. Galactose, arising from hydrolysis of lactose, is also transformed to glucose mainly in

the liver, and this transformation is inhibited by alcohol (ethanol).

The glycaemic carbohydrates reach the peripheral circulation mainly as glucose, and insulin is secreted in response to the elevated blood glucose concentration after a meal. Vagal signals, gastrointestinal hormones (incretins), and certain non-carbohydrate food components - especially amino acids - contribute to the stimulation of insulin secretion. The blood glucose level is determined by the following three main factors: 1) The rate of intestinal carbohydrate uptake, 2) the net liver uptake or elimination, and 3) the peripheral glucose uptake, which is dependent on both insulin production by the pancreas and the level of peripheral insulin sensitivity or resistance. Even with a constant dietary glycaemic carbohydrate load, there is a wide variety of blood glucose responses between individuals. These responses form a continuum from low responses to impaired glucose tolerance to type-2 diabetes.

Glycaemic index (GI)

The concept of glycaemic index (GI) was introduced by Jenkins and co-workers in 1981 (26) as a way to rank foods in a standardised way with regard to their effects on blood glucose levels after a meal. The FAO/WHO Expert Consultation on Carbohydrates in Human Nutrition (1, 2) defined GI as the incremental area under the blood glucose response curve after a 50 g carbohydrate portion of a test food expressed as a percentage of the response to the same amount of carbohydrate from a standard food taken by the same subject. Glucose and white bread have been used as standards. GI values obtained with the white bread standard are about 40% higher than those obtained with the glucose standard. An ISO standard that uses glucose as the standard for the determination of GI in foods was approved in 2010 (27). Application of this standard will contribute to more reliable and comparable GI values. It has been recommended that GI should only be used to rank food items with at least 10–20 g of glycaemic carbohydrates in the portion of the food analysed (28).

The factors that determine the GI of a carbohydrate are generally unrelated to the molecular size of the carbohydrate. For instance, both fructose and sucrose have a lower GI than white bread (29). Starchy foods, on the other hand, can have a low, intermediate, or high GI depending on the composition (amylose/amylopectin ratio), amount of resistant starch, and physical/chemical state. The swelling and dissolution of starch with wet heat treatment, known as gelatinisation, is particularly important in making starch more readily accessible to digestive enzymes (29).

Physical barriers such as intact cereal grains, the cellular structures in leguminous seeds, parboiled rice, and whole fruits, and the protein network in pasta products are food-related factors lowering the GI (29). Organic acids (acetic acid, propionic acid, and lactic acid) decrease the glycaemic response to foods or meals mainly due to inhibition of gastric emptying (30). Viscous, soluble types of dietary fibre can also delay gastric emptying in addition to their inhibitory effect on diffusion and transport in the small intestine (31). Other factors such as physical activity influence glucose metabolism and have the potential to influence insulin sensitivity and, therefore, the glycaemic response to any meal (See chapter on physical activity, (31) and (32)).

The concept of glycaemic load (GL) was introduced in 1997 by Harvard epidemiologists to quantify the glycaemic effect of a portion of food (33). GL is defined as the amount of glycaemic carbohydrate in a food multiplied by the GI of the food divided by 100. The glycaemic response to a meal can also be influenced by the protein and fat content as well as by the size of the meal and the amount of drink taken with the food. Several groups, however, have demonstrated that the glycaemic response to a meal can be predicted from properly determined GI of the constituent foods (34, 35). Lack of consistency in other studies (36) using published GI values might be due to these values not being applicable to the foods in question.

In conclusion, standardized analytical methods exist to measure GI. However, when studying the physiological effects of glycaemic carbohydrates many other factors have to be taken into consideration. This limits the use of GI in the prediction of the physiological effects of carbohydrates in meals and habitual diets.

Dietary fibre

Dietary fibre constituents pass through the upper gastro-intestinal tract and enter the colon substantially unmodified. In the colon, they are subject to anaerobic fermentation by the colonic microflora. The extent of fermentation is dependent on both substrate and host factors such as the molecular structure and physical form of the substrate, the bacterial flora, and the transit time. Less fermentable types of fibre, such as the lignified outer layers of cereal grains, generally have the most prominent faecal bulking effects due to their ability to bind water in the distal colon. Fermentable fibre also contributes to the faecal bulk through increased microbial mass. The main fermentation products are short-chain fatty acids (SCFA) such

as acetate, propionate, and butyrate and gases, the most notable of which are hydrogen and methane.

Dietary carbohydrates and health

Two reviews were conducted for the update of NNR 2012, one systematic review (SR) on dietary sugars (37) covering studies in adults and an overview on dietary fibre and glycaemic index (38). These reviews included original papers and expert reports published between 2000 and December 2011.

Total and glycaemic carbohydrates

Plasma lipids, glucose, and insulin

The influence of carbohydrates on plasma lipid, glucose, and insulin levels depends on several factors such as food source, physical form, and the amount and type of macronutrient replaced. Generally, a transient increase in fasting triglycerides and decreased HDL-cholesterol levels is seen when total dietary carbohydrate intakes are increased from 30–40 E% up to 60–70 E% (8). However, long-term effects depend on dietary carbohydrate sources. In an earlier controlled eight-month intervention study with healthy subjects following a fat-modified diet rich in fruits and vegetables, complex carbohydrates, and dietary fibre conforming to the NNR, an initial increase in triglyceride levels was seen that diminished with time (39).

A specific triglyceride-elevating effect of fructose has been demonstrated in animal experiments. In earlier, mainly short-term human studies, a high intake of refined sugars (> 20 E% sucrose or > 5 E% fructose) resulted in elevated triglyceride levels (8, 37). Results from more recent randomised controlled trials (RCTs) with high intakes (up to 17 E%) of refined sugars are partly conflicting, and evidence from epidemiological studies is also limited (8, 37). Results from two prospective studies reviewed by Sonestedt et al. (37) showed a positive association between intake of sugar-sweetened beverages and dyslipidaemia, i.e. elevated triglycerides and low HDL-cholesterol, and one study also showed a positive association with elevated LDL-cholesterol. Results from two prospective cohort studies and two RCTs on blood glucose and insulin levels/response did not show any consistent associations with intake of sugars or sugar-sweetened beverages (37). The EFSA evaluated five RCTs, three of which were published before 2000 (8). Two of those three studies showed increased insulin concentrations at high

intakes of sucrose (18–30 E%) compared to low intakes (3–10 E%), but no difference was seen in the third study. An effect on glucose concentration was observed in one study with no difference seen in another.

In summary, there is *insufficient* evidence to draw conclusions on effects on plasma lipids with respect to fructose or sucrose (of which half is fructose) in the general population. The evidence for effects on glucose and insulin response is *limited*. There is *limited-suggestive* evidence that high intake of sugar-sweetened beverages might be associated with dyslipidaemia indicating that the specific food source of sugar might influence metabolic response.

Blood pressure

The NNR SR on sugars included three prospective cohort studies and one RCT on the association between intake of sugars and blood pressure (37). A significant positive association with sugar-sweetened beverages was found in one of the prospective studies (40), but no significant association was found in the other two. The authors concluded that no consistent evidence for an association between dietary sugar intake and blood pressure was found. However, intake of sugar-sweetened beverages ≥ 1 serving per day (1 bottle, glass, or can) was significantly associated with incident hypertension after 16 to 38 years follow-up in three US cohorts (41).

It can be concluded that there is *suggestive* evidence that frequent consumption of sugar-sweetened beverages has an unfavourable effect on blood pressure. This is in line with studies examining whole diets low in sugar and fat and high in natural fibre-rich foods (e.g. the DASH studies) (42).

Type-2 diabetes

Results from trials aiming at preventing type-2 diabetes have shown that fat-modified, high-fibre diets together with 20–30 min daily physical activity reduced type-2 diabetes onset in high-risk, glucose-intolerant individuals (43–45).

The NNR SR included nine prospective cohort studies (8 of good quality and 1 of low quality) that examined the association between intake of sugars and incidence of type-2 diabetes (37). The results showed no consistent association between intake of total sugars, sucrose, or fructose and type-2 diabetes. Four of the six studies that investigated the association with sugar-sweetened beverages reported a significantly increased relative risk of type-2 diabetes with increasing intake. In one study, a significant positive association was found in the model that did not adjust for BMI.

The NNR SR concluded that there is *probable* evidence that high consumption of sugar-sweetened beverages increases the risk of type-2 diabetes (37), and this supports a limitation on the intake of added sugars. The apparent inconsistency in the studies examining total or individual sugars might be due to confounding because sugars in the form of whole fruits and vegetables have different effects compared to refined, added sugars, especially as sugar-sweetened beverages.

Body weight

The role of carbohydrate intake as a primary determinant for body weight maintenance has been less studied. Most studies using iso-energetic or ad-libitum designs have focused on changes in fat and protein content with changes in carbohydrate intake as a consequence of dietary modifications. Results from an SR and meta-analysis of 38 prospective studies and 30 RCTs showed that reduced intake of sugars was associated with a modest decrease in weight among adults (0.80 kg, 95% CI: 0.39-1.21; $P < 0.001$), and an increased sugar intake was associated with a weight increase of similar magnitude (0.75 kg; 95% CI: 0.30-1.19; $P = 0.001$) (46). Results for children showed generally no association, although high intakes of sugar-sweetened beverages were associated with an increased risk of obesity. The authors conclude that the observed effect on body weight “seems to be mediated via changes in energy intakes, since isoenergetic exchange of sugars with other carbohydrates was not associated with weight change” (46). Previous reviews and meta-analyses have found a positive association (47, 48) or no association (49) between intake of sugar-sweetened beverages and body weight.

Regarding the association between total carbohydrates and body weight, most studies have focused more on the effects of variations in total fat and less on the details of the type and dietary sources of carbohydrates replacing fat. An SR and meta-analysis of 33 RCTs on adults, without intentional weight loss as endpoint, found that a reduced total fat intake was associated with less weight gain of 1.4-1.6 kg, and nine of these studies showed a lower BMI of -0.51 kg/m^2 (50). Total fat intake was 28-43 E% at baseline and was typically 5-15 E% lower in the intervention arms. The reduction in fat intake was generally achieved through a proportional increase in total carbohydrates. Results from the NNR SR covering prospective cohort studies published from 2000 to 2011 suggest that the proportion of macronutrients in the diet has a limited role in prevention of obesity, and that plenty of fibre-rich foods and dairy products - and fewer

refined grains, meats, and sugar-rich foods and beverages – were associated with less weight gain (51). The results indicate that gross macronutrient composition per se might have only a limited impact on long-term weight change or maintenance. The observed effects on body weight changes might, therefore, be partly mediated by food-related factors that affect long-term energy intake.

Meta-analyses of intervention trials aiming at weight-reduction among overweight and obese individuals have shown that low-carbohydrate, high-protein diets resulted in similar or lower body weight compared to fat-reduced diets up to 6 months (52, 53). Long-term effects of such dietary changes, however, were less clear.

Fructose has been suggested to play a specific role in weight gain and insulin resistance syndrome (54). Unlike glucose, fructose is preferentially metabolised to lipids in the liver. On the other hand, fructose has a low glycaemic index. Fructose induces metabolic alterations typical for insulin resistance syndrome (metabolic syndrome) in animal models, but data in humans are less clear. A meta-analysis of human intervention trials did not find any evidence that fructose causes weight gain when substituted for other carbohydrates in diets with similar energy content. High intakes of free fructose providing excess energy intake were associated with modestly increased body weight (55). There is no basis for specific recommendations regarding fructose beyond the general limitation of added refined sugars.

Pregnancy outcomes

Results from a Norwegian pregnancy cohort have shown that consumption of sugar- sweetened beverages were associated with adverse pregnancy outcomes including preeclampsia and preterm delivery (56, 57). It should be noted that consumption of artificially sweetened beverages was also associated with preterm delivery both in the Norwegian study and a parallel study in a Danish pregnancy cohort (57, 58).

Dental caries

Caries develop as tooth tissues demineralise upon pH decrease due to fermentation of carbohydrates by tooth-colonising bacteria. Thus, dental caries is an infectious disease but sucrose and other easily fermentable mono- and disaccharides play a key role (59, 60). Foods rich in starch can also contribute to dental caries, especially when the starch molecule is easily available for degradation by amylase. The presence of sucrose intensifies the cariogenic potential of starch, but acid production from lactose

is normally low (61). Bacterial fermentation of sugar, mainly to lactic acid, causes pH decreases well below 5.5, which is considered critical for the development of caries in the enamel (the tooth crown). In tooth roots, the critical pH for demineralisation is approximately 6.5, a pH reached already when bread without added sugar is consumed. In addition to lactic acid, sucrose induces production of insoluble extracellular glucose polymers, i.e. glucans and mutans, leading to voluminous biofilms that favour colonisation of cariogenic streptococci on the surfaces of the teeth surfaces.

The prevalence of dental caries has declined in the Nordic countries in recent decades up to around the year 2000, but no corresponding reduction in total sugar intake was observed in this time period. Prophylactic use of fluoride and improved oral hygiene are important factors that modify the effect of sugar intake (62). However, in Norway and Sweden an increase in the prevalence of dental caries has been observed in some age groups in recent years (63, 64). Substantial socioeconomic and geographical differences still persist in caries prevalence, especially among children (65, 66).

Previous SRs have found a weak correlation, or no correlation, between sugar intake and caries development (67, 68). Intake data, however, were not provided in these reviews, which limits the interpretation of the results. The review by Burt and Pai (67) included all main forms of sugars, including added mono- and disaccharides and starch hydrolysates, while the review by Anderson et al. (68) mainly focused on sucrose. Generally, sugars other than sucrose contribute significantly to both the intake of total sugars and total refined sugars.

Results from a Finnish longitudinal study indicate that intake of sucrose and sucrose-containing foods is associated with caries development during childhood, including those children with fluoride prophylaxis (69–71). The main sources of sucrose were sweetened milk products, sugared drinks and juices, sweets, and chocolate (69, 71). Results from a Swedish study showed that frequent intake of sugar-containing foods among children aged 2–3 years was associated with caries prevalence (72) and that intake at 2 years of age predicted caries at 3 years of age (73).

As stated above, factors such as fluoride prophylaxis, oral hygiene, meal pattern, and meal composition interact (62). Frequency of sugar intake was found to be moderately related to caries development in the review by Anderson et al. (68), and there was generally a close correlation between intake frequency and intake amounts. Limiting the frequency of intake of refined sugars, and especially limiting sugar-rich foods as snacks, might contribute to reduced caries risk. A general level of safe refined sugar intake

cannot be provided because net caries development upon sugar challenge is modified by various other life-style factors (exposure to fluoride, meal frequency, and diet composition), heredity, illness, medication, malnutrition, and the flow and composition of the saliva.

Nutrient density

Nutrient density is the amount of essential nutrients found in foods per unit of energy content. An adequate nutrient density is essential for providing recommended intakes of nutrients, especially in individuals with a low energy intake. Added refined sugars mainly provide energy and no or only a few nutrients and thus tend to decrease the nutrient density. A review of 15 cross-sectional studies mainly from Europe and North America comprising children and adults concluded that there are insufficient and conflicting data with respect to the relation between intake of sugars and the density of selected micronutrients (74). This can be partly attributed to different definitions of the terms “sugars” or “added sugars” and on contributions of micronutrients from food fortification. Dietary fibre was not included in the analysis. However, studies among both children and elderly nursing home residents in the Nordic countries (75–78) have shown that a high intake of refined sugars (>10–15 E%) might adversely affect the intake of essential nutrients and dietary fibre. High intakes of sugar-rich foods might also be associated with poor dietary habits, e.g. low fruit and vegetable intake (78). A limitation of the intake of refined sugars is of special importance for children and adults with low energy intake. In the Finnish STRIP project, increasing sucrose intake was associated with a generally poorer quality of the diet in childhood (79). Thus higher sucrose intake (>10 E%) is associated with lower intake of many micronutrients and dietary fibre and a higher intake of saturated fatty acids.

GI, GL

Type-2 diabetes

Based on an evaluation of international expert reports (2, 28, 80, 81) and Nordic studies published from 2000 to December 2011, Øverby et al. (38) concluded that there is *suggestive*, but inconsistent, evidence that GI is associated with an increased risk for type-2 diabetes, especially in overweight and obese subjects. A meta-analysis of 13 prospective studies conducted in the US (8 studies), Europe (2 studies), Australia (2 studies), and China (1 study) published between 1997 and 2010 found a significantly increased

risk of type-2 diabetes when comparing the highest to lowest GI categories (Dong et al. 2011). Relative risk (RR) was 1.16 (95% CI: 1.06–1.26) with evidence of heterogeneity ($p = 0.02$). In a Finnish study on male smokers included in the review by Øverby et al. (38), but not in the meta-analysis by Dong et al. (82), no significant association with GI was found (83).

Cancer

SRs and meta-analyses of prospective cohort studies do not support an independent association between diets with high GI or GL and colorectal cancer (84) or breast cancer (85). A meta-analysis of four prospective cohort studies and one hospital-based case-control study on endometrial cancer published between 2003 and 2007 showed an increased risk when comparing the highest to lowest GL categories (86). RR was 1.20 (95% CI: 1.06–1.37), with a higher RR among obese women (RR = 1.54; 95% CI: 1.18–2.03). No significant associations were observed for GI.

In summary, an association between GI or GL and colorectal or breast cancer is unlikely. There is *limited-suggestive* evidence for an association between GL and endometrial cancer.

Blood lipids, glucose, insulin

Results from controlled, mainly short-term, intervention studies in humans have led to conflicting results with respect to effects on metabolic risk factors (38). Evidence from epidemiological studies is also conflicting. There are a number of methodological problems involved, including reliability of GI measurement, diet composition, and other food characteristics and constituents.

Pregnancy outcomes

Pregnancy is a physiological condition in which the GI might be of particular relevance because glucose is the primary fuel for foetal growth (87). High glycaemic load has been shown to be associated with the risk of gestational diabetes mellitus (88) as well as excessive gestational weight gain and post-partum weight retention (89).

Conclusions

The review by Øverby et al. (38) concluded “that 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 dietary fibre, whole grains, legumes and fruits and vegetables. Issues regarding methodology, validity and practicality of the GI remain to be clarified”.

Dietary fibre

Dietary fibre has several physiological effects including faecal bulking and colonic fermentation and it affects blood glucose response, blood lipid levels, and blood pressure. These effects differ between various fibre constituents and food sources.

Laxation

Insoluble fibre, especially lignified types of fibre such as those in wheat bran, has the most prominent effects on faecal bulk. The increase in faecal weight ranges from 1.3 g for each gram of ingested pectin to 5.7 g for each gram of ingested wheat bran fibre (90). Oligosaccharides and resistant starch can also provide some faecal bulk (91).

Plasma lipids

Viscous types of soluble fibre lower plasma levels of total cholesterol and LDL-cholesterol. Although fasting triglyceride levels are generally not affected, different kinds of fibre – especially soluble and viscous types – can reduce post-prandial hyperlipidaemia (92). These effects are related to diminished cholesterol and/or bile acid absorption (93) and hypothetically also to products of colonic fermentation. The effects on lipid metabolism that have been demonstrated by resistant starch and resistant oligosaccharides in experimental animals have so far not been reproduced in man.

Blood pressure

Dietary fibre, mainly in the form of viscous fibre, can modulate blood pressure. In a meta-analysis by Streppel et al. (94) including 24 RCTs, fibre supplementation with an average dose of 11.5 g/d for a mean duration of 9 weeks was associated with a 1.13 mm Hg decrease in systolic blood pressure (95% CI: -2.49 to 0.23) and a 1.26 mm Hg decrease in diastolic blood pressure (95% CI: -2.04 to -0.48). Reductions in blood pressure

tended to be larger in individuals older than 40 years of age and in hypertensives. Similar results were found in a meta-analysis by Whelton et al. (95) that included 25 RCTs. These findings are in line with those of whole-diet trials such as the DASH studies (42). The potential mechanisms for the blood pressure-lowering effect are less documented, but they might involve effects on insulin response, vascular endothelial function, and mineral absorption (94). Foods rich in fibre such as fruit and vegetables also contain potassium and magnesium, which might contribute to reductions in blood pressure.

Blood glucose attenuation

Intake of viscous, soluble fibre has been shown to contribute to lower post-prandial blood glucose and insulin response (8, 96, 97). The mechanism of action might in part be reduced absorption of food carbohydrates (98, 99). Of the expert reports included in the review by Øverby et al. (38), the EFSA (8) concluded that fibre intakes > 2.6 g/MJ were associated with reduced risk of impaired glucose control.

Colonic fermentation

Dietary fibre components are subject to anaerobic fermentation by the colonic microflora. The main fermentation products are SCFA such as acetate, propionate, and butyrate and gases, most notably hydrogen and methane. The decrease in pH of the colonic content has been shown to be protective against colon cancer through the reduced formation of bile salt metabolites that have been implicated in carcinogenesis. Furthermore, butyrate is recognised as a main source of energy for colonocytes and this has effects on cell differentiation and apoptosis that might be protective (100, 101). Acetate and propionate are absorbed and have possible systemic effects on carbohydrate and lipid metabolism. Propionate has been shown to inhibit liver cholesterol synthesis in experimental animals, but the importance of such a mechanism in humans remains to be established (92). The proportions of different SCFA differ with the fermentation substrate. Resistant starch and oat fibre have been shown to produce large amounts of butyrate (for a review, see (91)).

Certain oligosaccharides, such as FOS (i.e. inulin and shorter molecules) have been shown to increase the numbers of bifidobacteria in the colon, and this seems to be a general effect of increased amounts of non-digestible carbohydrates such as other oligosaccharides (GOS and resistant malto-oligosaccharides) and resistant starch (91, 102).

Cancer

The reduced transit time and increased faecal weight with dilution of the intestinal contents and improved laxation were factors behind the early hypothesis that an appropriate intake of dietary fibre would reduce the risk of both colorectal cancer and diverticular disease. There is an inverse relationship between faecal weight (influenced by non-starch polysaccharide intake) and risk of colon cancer (103).

Results from epidemiological studies show clear evidence of a protective effect of dietary fibre on colorectal cancer (38). Aune et al. (84) assessed the association between fibre intake and colorectal cancer using data from 25 prospective cohort studies. The summary RR risk of developing colorectal cancer expressed per 10 g/d of total dietary fibre (16 studies) was 0.90 (95% CI: 0.86–0.94). Corresponding analyses for different food sources showed significantly reduced risk for cereal fibre (RR = 0.90; 95% CI 0.83–0.97, 8 studies). No significant associations were seen in separate analyses of fibre from fruits, vegetables, or legumes. Based on these results, the WCRF upgraded the evidence that foods containing dietary fibre protect against colorectal cancer from *probable* to *convincing* (104). Similar estimates were observed in the large European prospective cohort study EPIC (105) where an increase of 10 g/d of total dietary fibre was inversely associated with colorectal cancer (hazard ratio = 0.87, 95% CI: 0.79–0.96). Fibre from cereals and fibre from fruits and vegetables were inversely associated with colon cancer, but for rectal cancer such an inverse association was only seen for fibre from cereals.

An SR and meta-analysis of 16 prospective cohort studies covering breast cancer showed a significant moderate risk reduction (106). The summary RR for a 10 g increase in fibre intake was 0.95 (95% CI: 0.91–0.98). Effects were mainly seen in studies with a large range of fibre intakes.

In summary, there is *convincing* evidence for a protective effect of dietary fibre against colorectal cancer and *limited-suggestive* evidence for a protective effect against breast cancer.

Cardiovascular disease (CVD)

Based on the review of recent international expert reports, SRs, and Nordic studies, there is clear evidence that a high intake of dietary fibre is protective against CVD (38). This is supported by results from the large European cohort study EPIC on the association between fibre intake and ischaemic heart disease mortality in which 306,331 subjects free from CVD were followed for a mean of 11.5 years (107). The RR for an increase in fibre intake

of 10 g/d was 0.85 (95% CI: 0.73–0.99, $P = 0.031$). The associations for various food sources of dietary fibre were in the same direction, but not statistically significant.

An SR including eight cohort studies found an inverse association between total intake of dietary fibre and risk of both haemorrhagic and ischaemic stroke as well as some evidence of heterogeneity between studies (RR per 7 g/d = 0.93; 95% CI: 0.88–0.98; $I^2 = 59\%$). Viscous, soluble fibre intake of 4 g/d was not significantly associated with reduction in stroke risk and there was evidence of low heterogeneity between studies (108).

In conclusion, there is *probable* evidence for a protective effect of dietary fibre from various foods against CVD.

Body weight

Adults

Several physiological effects of foods rich in dietary fibre, including diminished energy density, slower gastric emptying, short-term increase in satiety, and decreased rate of nutrient absorption, might be important for body weight regulation. Of the expert reports included in the review by Øverby et al. (38), the EFSA (8) concluded that dietary fibre is associated with lower body weight, and the SR included in Dietary Guidelines for Americans 2010 (80) showed “moderate evidence that intake of wholegrain and grain fibre is associated with lower body weight”.

Results from mainly short-term intervention studies on adults have shown that increased intake of various fibre types resulted in moderate weight loss (109, 110). The SR by Wanders et al. (110) studied 59 RCTs in which the effects of different fibre types on body weight were assessed. Effect sizes were calculated as an average that was weighted by the number of subjects who completed the study. Study durations varied from 3 weeks to 13 weeks with large variations in fibre intakes in the intervention arm from 3.0 g/d to 48 g/d. The average change in body weight was a reduction of 0.72 kg (1.3%), corresponding to a reduction of 0.4% every 4 weeks.

Results from prospective cohort studies on adults published since 2000 generally show that increased dietary fibre intake is associated with lower body weight (51) and waist circumference (111). The SR by Fogelholm et al. (51) included five prospective cohort studies, and the evidence linking high fibre intake to prevention of weight gain was judged to be *probable*. Reported effect sizes, however, were variable. In a European cohort study including 89,432 subjects free from cancer, CVD, and diabetes at baseline, an increased intake of total fibre of 10 g/d was associated with a -39 g/y

(95% CI: -71, -7 g/y) weight change and a -0.08 cm/y (95% CI: -0.11, -0.05 cm/y) waist circumference change over an average of 6.5 years (111).

In summary, there is *probable* evidence that dietary fibre intake is associated with lower weight gain in adults.

Children

Of the expert reports included in the review by Øverby et al. (38), the EFSA (8) used normal laxation as a criterion to set an adequate intake of dietary fibre of 2 g/MJ for children > 1 year of age. The SR included in Dietary Guidelines for Americans 2010 (80) concluded that the evidence for an association between dietary fibre and adiposity in children is insufficient.

A few studies, mainly on British vegan children, indicated slower growth in some of the children (112, 113), but it is not clear if this can be attributed to the fibre content of their diet (114). Studies among children consuming a mixed diet do not indicate that a high fibre intake would compromise growth (114). A high fibre intake is often linked to higher intake of fruits, vegetables, and cereals and might be an indicator of more favourable dietary habits (115, 116). Results from the Finnish intervention study STRIP (Special Turku Coronary Risk Factor Intervention Project) show that children who were allocated a diet in line with the NNR from 7-8 months of age grew and developed normally (116-118). Intake of fibre was positively related to nutrient intake (116). Mean intake of fibre was 9.2 g/d at 13 months of age and 11.8 g/d at 5 years of age corresponding to 2.3 g/MJ and 2.0 g/MJ, respectively (119). There were no differences in weight or growth in relation to fibre or fat intake. At age 7, fibre intake varied from 12.3 g/d to 15.5 g/d corresponding to 1.9-2.4 g/MJ (120). Results from studies among German children who had been followed from 6 months to 18 years of age showed that the fibre intakes, expressed as g/MJ, were highest at 1 year (3 g/MJ) and then decreased somewhat to 2.5 g/MJ in pre-school and school-age children (77). In a subsequent study, no clear association between increased fibre intake from 2 to 7 years of age and per cent body fat or BMI was found. Mean fibre intake was 2.5 g/MJ. Increased fibre intake was associated with lower percentage of body fat among children who at 2 years of age consumed fewer than six meals per day (121).

Pregnancy outcomes

Intake of dietary fibre and dietary patterns characterised by vegetable foods with high content of dietary fibre (>22 g/d) have been shown to be asso-

ciated with decreased risk of gestational diabetes (88) and preeclampsia (122, 123).

Type-2 diabetes

The NNR review by Øverby et al. (38) concluded that there is moderate evidence that high intake of dietary fibre is associated with a lower risk of type-2 diabetes. This was mainly based on the EFSA opinion (8) and results from a Finnish prospective cohort study (124). Intake of wholegrain and cereal fibre has been associated with reduced risk of type-2 diabetes (125). A meta-analysis by Schulze et al. (126) comprising nine prospective cohort studies and 393,385 subjects, which was evaluated in the EFSA report, showed a reduced risk for type-2 diabetes with higher cereal fibre intake (RR for extreme categories = 0.67, 95% CI: 0.62–0.72).

In summary there is *probable* evidence that dietary fibre intake is inversely associated with type-2 diabetes. This might partly be mediated by wholegrain intake.

Requirement and recommended intake

Glycaemic carbohydrates

Only cells in the central nervous system, red blood cells, and some other cells dependent on anaerobic glycolysis have an absolute requirement for glucose. In the body, glucose can be synthesised from proteins and glycerol, and it has been assumed that there is no need for dietary carbohydrates as long as adequate amounts of fat and of protein for de novo synthesis of glucose are consumed. With prolonged glucose deficit, brain cells can partially adapt by utilising fat-derived metabolites such as β -hydroxybutyric acid and acetoacetic acid. A very low carbohydrate diet (below 50 g/d), however, results in chronically increased production and increased plasma levels of these acids resulting in a condition known as ketosis. An intake of 50–100 g/d of glycaemic carbohydrates generally prevents ketosis.

An intake of 50–100 g/d of glycaemic carbohydrates per day is sufficient to avoid ketosis among children and adults, and an intake of 130 g/d for both children older than 1 year and adults has been estimated to cover the glucose needs of the brain (5). This intake corresponds to about 20 E% and 25 E% in adult males and females using a reference intake of 11 MJ and 9 MJ/d, respectively. For children up to 11–13 years of age, this intake corresponds to 25–45 E%. These levels are used in NNR 2012 as average requirements (AR) for glycaemic carbohydrates.

Added sugars

A restriction in the intake of added, refined sugars is important to ensure adequate intakes of micronutrients and dietary fibre (nutrient density) as well as supporting a healthy dietary pattern. This is especially important for children and persons with a low energy intake. 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. Frequent consumption of sugar-containing foods should be avoided to reduce the risk of dental caries. The recommendation from NNR 2004 is maintained.

Added sugars (sucrose, fructose, and starch hydrolysates) should be kept below 10 E%.

Dietary fibre

In NNR 2004, the recommendation of dietary fibre intake was mainly based on the amounts required for bowel regularity and for maintaining a faecal bulk that was associated with a diminished risk of colon cancer (90, 103). Since then, a number of studies have been published supporting the beneficial effects of dietary fibre and/or dietary fibre-rich foods such as wholegrain cereals, fruit, and vegetables on a number of diseases. An adequate intake of dietary fibre reduces the risk of constipation. There is *convincing* evidence for a protective effect of dietary fibre against colorectal cancer, *probable* evidence for a protective effect against CVD, and *limited-suggestive* evidence for a protective effect against breast cancer and type-2 diabetes. Moreover, fibre-rich foods help in maintaining a healthy body weight. The evidence supporting the recommendation from NNR 2004 has been strengthened by these recent studies. There is also evidence that intake of appropriate amounts of dietary fibre from a variety of foods is important for children.

Adults: Intake of dietary fibre should be at least 25–35 g/d, i.e. approximately 3 g/MJ. Wholegrain cereals, whole fruit, vegetables, pulses, and nuts should be the major sources.

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.

Note: These recommendations are based on AOAC methods for total dietary fibre.

Total carbohydrate

The health effects of dietary carbohydrates are related to the type of carbohydrate and the food source. Dietary patterns associated with reduced risk of chronic diseases are characterised by abundant intake of fibre-rich foods consisting mainly of slowly digestible carbohydrates such as wholegrain cereals, whole fruit, berries, vegetables, and pulses (127). These foods should be the major sources of dietary carbohydrates. Typical ranges of total carbohydrate intakes in studies on dietary patterns associated with reduced risk of chronic diseases among adults are 45–60 E%. This is considered a reasonable range of total carbohydrate intake in NNR 2012, and this range is also applicable for children from about 6 months of age.

For planning purposes, the focus should be on achieving the recommended amounts of dietary fibre and added sugars. The ranges for total carbohydrates can be used as complementary goals using the middle value (52–53 E%) as an appropriate target.

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12

Protein

	Adults 18-64 y	≥65 y	Children 2-17 y	12-23 mo.	6-11 mo.
E%	10-20	15-20	10-20	10-15	7-15

The range of 10-20 E% for adults corresponds to about 0.8-1.5 g protein/kg body weight/d, provided a physical activity level (PAL) of 1.6 for an intake of about 10 E%, and a PAL of 1.4 for an intake of about 20 E%, respectively.

The range of 15-20 E% for the elderly corresponds to about 1.1-1.3 protein/kg body weight/d, provided a PAL of 1.6 for an intake of about 15 E%, and a PAL of 1.4 for an intake of about 20 E%, respectively.

Introduction

Proteins are a constituent of all organic material in the cells of animals and plants and are built from 20 unique amino acids. Within the body, proteins provide enzymatic activity, antibody activity, and muscle work; are involved in repair processes and the transport of various substances; and are the building blocks for several cellular structural elements. Dietary protein has two roles in nutrition; it has a specific role as source of nitrogen and amino acids and a non-specific role as an energy source. In individuals in energy balance and with a moderate physical activity level, the protein requirement is defined as the lowest intake of protein to maintain nitrogen balance (N-balance). In the NNR, the energy content from protein in a mixed diet is calculated as 17 kJ/g.

Dietary sources and intake

Dietary proteins are found in almost all foods of animal and plant origin. Meat, fish, milk, and eggs have large quantities of high-quality protein. Pulses, nuts, and seeds also have high protein content. This makes them important sources of proteins in vegetarian diets, especially for vegans who also exclude milk and eggs from their diet.

The average protein intake among adults is high in the Nordic countries, ranging from 15 E% in Denmark to 18 E% in Norway and Iceland according to national dietary surveys.

Physiology and metabolism

During digestion and absorption, dietary proteins are broken down into their constituent amino acids. Within the body, amino acids absorbed into the blood are incorporated into tissue protein and other nitrogen-containing compounds such as neurotransmitters, creatinine, and drug elimination ligands. Thus, the protein requirement is actually a requirement for amino acids and nitrogen.

Body proteins are continually being broken down and synthesised. The protein turnover (which is about 300 g/d in adults) is many times higher than the amount of proteins consumed from the diet. This indicates an extensive reutilisation of amino acids in protein metabolism. Nitrogen from the amino acids leaves the body via the urine in the form of urea, uric acid, creatinine, etc. Small quantities of nitrogen are also lost from faeces, sweat, and other secretions and from the skin, hair, and nails. The body needs amino acids to compensate for these losses, and amino acids are also needed for protein synthesis during anabolism, e.g. during growth, pregnancy, and lactation.

It is usually assumed that almost all of the dietary nitrogen is incorporated as protein. Dietary nitrogen \times 6.25 is accepted as a reasonable approximation of the amount of protein in the diet because the average protein contains 16% nitrogen. However, because the nitrogen content of various amino acids ranges from 7.7% to 32.2%, the nitrogen content of protein in individual foods depends on the amino acid composition. Thus the conversion factor can vary from 5.83 in wheat to 6.38 in milk (1). N-balance is the difference between nitrogen intake and nitrogen output. A negative N-balance (i.e. losses greater than intake) is seen during fasting and starvation. A positive N-balance is seen during active growth. On a

long-term basis, healthy adult subjects should be in nitrogen equilibrium, i.e. intake and losses should be equal.

Amino acids from dietary protein are classified as either essential (indispensable) amino acids that cannot be synthesised in the human body and thus must be provided in the diet, or nonessential (dispensable) amino acids that are synthesised within the body from other amino acids (transamination) provided that there is an adequate nitrogen supply. The essential amino acids in humans are isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, valine, and histidine. Histidine is considered essential although it does not fulfil the criterion of reducing protein deposition and inducing negative nitrogen balance when removed from the diet (2, 3).

In humans, the nonessential amino acids are alanine, arginine, asparagine, aspartic acid, cysteine, glutamine, glutamic acid, glycine, proline, serine, and tyrosine (4). Conditionally essential amino acids, including arginine, cysteine, glutamine, glycine, proline and tyrosine, are amino acids whose synthesis requires the availability of another amino acid either as the carbon donor or as the donor of an accessory group, e.g. the sulphur group of cysteine (5). Under normal conditions, conditionally essential amino acids are synthesised in sufficient amounts but during certain conditions, such as prematurity (6) and illness (7), synthesis might not support all of the body's metabolic needs.

Protein constitutes 15–20% of the human body, which corresponds to approximately 12 kg in a person with a body weight of 70 kg.

Dietary proteins differ in their nutritional quality due to differences in amino acid composition, the total amount of each amino acid, and the digestibility of the protein. In 1991, the FAO/WHO (8) introduced the Protein Digestibility-Corrected Amino Acid Score (PDCAAS) based on an age-related amino acid reference pattern that is representative of human requirements combined with estimates of the digestibility of the protein. For mixed diets or whole foods, PDCAAS values >1 are not used, but extrapolated to 1. The WHO (9) defined good-quality protein as proteins with a PDCAAS value of 1.0.

Most recently, the FAO (10) has introduced the Digestible Indispensable Amino Acid Score (DIAAS). The main difference between DIAAS and PDCAAS is that in DIAAS the true ileal amino acid digestibility for the indispensable amino acids is used rather than a single faecal crude protein digestibility value.

Dietary proteins of animal origin (meat, fish, milk, and eggs) or a com-

bination of plant protein from, for example, legumes and grains, will give a good distribution of essential amino acids. Most proteins are also reasonably well digested although those found in grains have slightly lower digestibilities.

The quality of dietary proteins is usually high in the typical Nordic mixed diet. In practice, the differences in quality between proteins might be less important in diets containing a variety of protein sources (11).

Requirement

The WHO/FAO/UNU (9) define the protein requirement of an individual as *“the lowest level of dietary protein intake that will balance the losses of nitrogen from the body, and thus maintain the body protein mass, in persons at energy balance with modest levels of physical activity, plus, in children or in pregnant or lactating women, the needs associated with the deposition of tissues or the secretion of milk at rates consistent with good health.”*

Despite limitations in the method that are mainly related to accuracy of the measurements and interpretation of the results, N-balance remains the method of choice for determining the protein requirement in adults in the absence of validated or accepted alternatives and in the absence of a reliable biological marker of protein status.

For the NNR 2012 update, two systematic reviews (SRs) were conducted to assess the scientific evidence behind the dietary requirement for protein. One SR included healthy adults (12), and the other included healthy elderly populations with a mean age of ≥ 65 years (13).

The two SRs included one meta-analysis (14) and four additional balance studies (15–18). The meta-analysis by Rand et al (14) included 19 N-balance studies, and they found no statistically significant differences in estimated requirements between the locations of the study sites, the adult age of the participants, gender, or source of dietary protein, although there was an indication that women might have a lower requirement. The authors emphasized that the data did not provide sufficient power to detect possible differences. The median estimated average requirement (EAR) of good-quality protein was 105 mg nitrogen, which corresponds to 0.66 g protein/kg body weight (BW)/d. The estimated recommended dietary allowance (RDA) was set to 0.83 g good-quality protein/kg BW/d, corresponding to the 97.5th percentile. Only one study in elderly persons was included (19). The median nitrogen requirement was 130.5 mg/kg BW/d in the elderly group versus 103.9 mg/kg BW/d in the younger group. This

corresponded to a difference of 0.17 g protein/kg BW/d or a 26% higher requirement in the elderly group. Rand et al (14) concluded that healthy elderly persons might have higher requirements, but there was not enough evidence to make different recommendations for this group.

The objective of the high-quality N-balance study by Campbell et al (16) was to study the effect of age on the EAR for protein. They found no difference in the EAR between the young and old participants, and the calculated adequate protein allowance of 0.85 g good-quality protein/kg BW/d for all participants combined was not statistically different from the RDA estimated in the meta-analysis by Rand et al (14). An earlier study by Campbell et al (15) also found 0.8 g protein/kg BW to be sufficient to obtain N-balance in 10 elderly participants. Steady state was reached at week 2 indicating that the protein intake was adequate for the participants. However, after two weeks the urinary nitrogen excretion decreased and was associated with a loss in mid-thigh muscle area. The authors suggested that the protein intake might have been marginally inadequate and resulted in long-term accommodation in skeletal muscle (15).

In a controlled metabolic study by Morse et al (17), 11 healthy elderly women were provided eucaloric diets with three different protein intake levels. These included a low-protein diet of 0.5 g/kg BW; a medium-protein diet of 0.75 g/kg BW; and a high-protein diet of 1.0 g/kg BW during three periods of 18 days with a minimum of one week habitual diet between the periods. N-balance was determined on week 2 and 3 of each diet. The mean dietary allowance was estimated to be 0.90 g protein/kg BW at week 2 and 0.76 g/kg BW at week 3, but the urinary nitrogen excretion decreased between week 2 and 3 indicating that a steady state was not yet reached.

A short-term study was also included in the SR because it compared a high protein (HP) intake in the test meal versus usual protein (UP) intake (18). Young men and women (UP 1.04 g/kg BW and HP 2.08 g/kg BW, respectively) versus old men and women (UP 0.89 g/kg BW and HP 1.79 g/kg BW, respectively) were tested for 10 days on each diet in a crossover design. There was no age-related difference in N-balance. However, in the elderly participants with a habitual low glomerular filtration rate (GFR) the HP diet corresponding to about 24 E% did not result in the expected increase in GFR after intake of the HP diet.

From these studies, the evidence was assessed as *probable* regarding a median daily EAR of nitrogen of 105 mg/kg BW. This corresponds to 0.66 g good-quality protein/kg BW/d regardless of sex or age.

It should be noted that two of the studies among the elderly (15, 17) found a decrease in the urinary nitrogen excretion. In the study by Campbell et al (15), this was associated with loss of muscle mass, but this was not the case in the study by Morse et al (17).

The use of N-balance as a basis for establishing dietary protein recommendations is a matter of debate. This methodology provides an indirect determination of protein turnover, and no information about whole-body nitrogen or protein turnover, or various protein metabolic pathways, can be obtained. Furthermore, obtaining complete urine collections and strict measurements of energy intake/balance are challenging in field studies. If the energy balance changes during the study, this will influence the results, thus eucaloric diets are needed. Also, low protein intake might induce protein sparing and thus lead to underestimation of needs.

Over the last several years more direct methods of measuring turnover of various body proteins have been developed, including stable isotope tracer methods. This has enabled a mechanistic approach to the effects of various dietary proteins. In 1992, Tarnopolsky et al (20) used stable isotopes. They also studied sedentary individuals at three different protein intakes for 13 days at each intake and found the average requirement (AR) to be slightly less than 0.7 g protein/kg BW. This led to a recommendation of 0.89 g protein/kg BW. The main limitation is the lack of prolonged studies using this methodology as evidenced by the fact that most articles using stable isotope methods from 2000 onward only describe acute effects of protein or amino acid intake (12) and these are mainly focused only on muscle protein metabolism. However, the WHO (9) used stable isotope studies to increase the estimated requirements for essential amino acids based on the biologically sound criterion that the point of intake where oxidation of the essential amino acids investigated begins to increase reflects the point at which intake is above requirements. Similar logical reasoning cannot be applied to whole-body protein turnover beyond what can already be deduced from N-balance studies. Rates of whole-body protein synthesis and degradation are usually reported to increase in parallel with protein intakes above the amount required for N-balance, but the relation between whole-body protein turnover rate and health or body functions needs to be established. Similarly, studies of muscle protein turnover have not yet added to an understanding of muscle function because no studies are available demonstrating a correlation between, for example, muscle strength or endurance and the dynamics of muscle protein turnover. Thus, in the future it will be important to use more advanced methodologies in strictly

controlled long-term studies to establish mechanistic links between health outcomes and protein intake from various sources.

Severe protein deficiency results in oedema, muscle weakness, and changes to the hair and skin. Protein deficiency is often linked to energy deficiency and protein-energy malnutrition, as well as deficiency of other nutrients based on a general nutrition deficiency. Sarcopenia has recently been defined as the loss of muscle mass and function leading to adverse clinical outcomes. The diagnosis is based on the combined finding of reduced muscle mass/lean tissue and reduced power or strength (21). At present, there are no specific blood tests for protein deficiency. Plasma albumin and other plasma proteins decrease in very severe malnutrition, but this is difficult to distinguish from dilution due to hunger oedema.

Protein intake and health

For the NNR 2012 update, three SRs were conducted on the health effects of protein intake. One study was in infants and children ≤ 18 months of age (22), one was in healthy adults (12), and one was in healthy elderly populations with a mean age of ≥ 65 years (13). The literature searches covered the years 2000 to 2011, and the SRs assessed the health effects of varying protein intakes to evaluate the evidence for an optimal protein intake.

Mortality

The two SRs carried out in adults and elderly (12, 13) only included two studies that addressed protein intake *per se* in relation to all-cause mortality. In the PREVEND study (23), the protein intake was calculated from two 24-hour urinary urea excretions and was expressed as protein intake in grams per kilogram of “ideal” BW, i.e., after correcting BW to a BMI corresponding to 22. Thus, the level of protein intake could not be assessed because the correction probably overestimated intakes and because no correction was made for possible loss of urine in the collections. After 7 years, they found that quintiles of protein intake were inversely associated to all-cause mortality and non-cardiovascular mortality. Among British elderly, Bates et al (24) found a decreased risk of all-cause mortality associated with total protein intake after 14 years, but their study was flawed by underreported energy intake. Thus, the evidence for a relation between total protein intake and all-cause mortality was assessed as *inconclusive* (12, 13).

The SR (12) also assessed three longitudinal cohort studies that included about 200,000 men and women. The studies used a low-carbohydrate/

high-protein (LC/HP) diet score based on the protein E%, and one of the studies also used a LC/HP- high fat score (25). All three studies found an increased risk of all-cause mortality, thus the evidence was assessed as *suggestive* regarding an increased risk of all-cause mortality in relation to an LC/HP diet with total protein intakes of at least 20–23 E% (12). In addition to the SR, a Swedish population-based cohort study from 2012 included 77,319 men and women aged 30–60 years at baseline who were followed for a mean of 10 years (26). The study used a LC/HP score and found no statistically significant association to all-cause, cancer, or cardiovascular disease mortality after accounting for saturated fat intake. It should be noted that 97.2% of the participants had a reported protein intake in line with the recommended range of 10–20 E%.

For cardiovascular mortality, the evidence was assessed as *suggestive* for an inverse relation to vegetable protein intake based on three studies in which the protein intake was expressed in E% and on one study with an LC/HP diet score based on vegetable protein (12). Additionally, in a Swedish cohort study in elderly men comparing dietary patterns, an LC/HP diet was associated with increased risk, but a Mediterranean-like diet was associated with decreased risk of cardiovascular mortality (27). However, very few participants were in the highest protein score corresponding to 18 E% (n = 7) compared to the lowest protein score of 12 E% (n = 12).

In the study by Fung et al (25) that included 44,548 women and 85,168 men in the Nurses' Health Study and Health Professionals' Follow-up Study, respectively, they found that an animal-based LC/HP score was associated with a higher risk of cancer mortality.

Generally, the use of an LC/HP score makes it uncertain whether the effects result from reduced carbohydrate or increased protein and/or fat, and thus the effect of protein *per se* cannot be assessed from LC/HP diets.

Cancer

The overall association between cancer and protein intake was assessed as *inconclusive* (12). Most studies on the relation between protein intake and cancer are food based (28) and, therefore, cannot isolate the effect of the protein intake *per se* from other nutrients or ingredients in the foods. For instance, The World Cancer Research Fund (28) found that the consumption of red and processed meat was associated with several cancers, especially colorectal cancer.

Cardiovascular diseases and serum lipids

For cardiovascular diseases, the association between protein intake and coronary heart disease and stroke was statistically non-significant in six cohort studies, and the evidence was regarded as *inconclusive* (12). However, apart from the SR in a cohort study from 2012 of 43,396 Swedish women, Ligiou et al (29) found that an LC/HP diet was associated with an increased risk of cardiovascular disease after 15.7 years.

The evidence for an association between protein intake and blood pressure was assessed as *inconclusive* for total and animal protein, but an inverse association with vegetable protein intake was assessed as *suggestive* (12) and this is in agreement with the WHO/FAO/UNU report (9). The SR included one feeding study among African-Americans and non-Hispanic white participants, the OmniHeart study (30), that was based on a carbohydrate diet similar to the Dietary Approaches to Stop Hypertension (DASH) diet but with 15 E% protein versus 25 E% protein and with the 10 E% protein replaced with carbohydrates. The subgroup analysis in the Caucasian group found no significant relation between protein intake and blood pressure (30). Both the SUN cohort study (31) and the Chicago Western Electric Study (32) found an inverse relation with vegetable protein (expressed as E%) and the risk of hypertension, and blood pressure change, respectively, and the most recent meta-analysis with soya intake in controlled trials (33) supported the conclusion that the evidence is *suggestive* for an inverse association between hypertension and intake of vegetable protein (12).

In the elderly, the Rotterdam prospective cohort study looked at the association between risk of hypertension and intake of energy-adjusted tertiles of total, animal, and vegetable protein among persons ≥ 55 years of age without hypertension at baseline. The lowest tertile of total protein intake was 70 ± 15 g/d (14 E%) and the highest was 97 ± 19 g/d (19 E%). They found no statistically significant associations except in persons ≥ 70 years of age where animal protein intake was related to an increased risk of hypertension after 6 years of follow-up (13). A recent comprehensive SR regarding protein and blood pressure included the above mentioned studies and studies before 2000, but also cross-sectional studies and studies among risk groups (overweight/obese, hypertensive, and diabetic patients) and in non-Nordic settings, and they found a small beneficial effect of dietary protein, especially for vegetable protein (34).

Two high-quality meta-analyses of randomized controlled studies (35, 36) found a statistically significant inverse effect for a mean daily intake

of 25–30 g soya protein, corresponding to 1 or 2 servings per day, on LDL-cholesterol concentration. Studies with participants with the highest baseline LDL-cholesterol concentration had greater reductions than studies with the lowest values, thus the effect might be smaller in normo-cholesterolemic persons. The evidence was assessed as *probable to convincing* in regard to the effect of soya protein on LDL-cholesterol concentration (12), but the intake level in those studies was much higher than in the present Nordic diet so the relevance of these results for the average Nordic diet is questionable.

Bone health

The role of dietary protein on bone health has been controversial. Urinary calcium loss increases in high-protein intakes, but at the same time protein increases calcium absorption and bioavailability and these seemingly contradictory effects make it uncertain as to what the net effect of high protein diets is on calcium metabolism and bone health (37). Any negative effect of protein might be opposed by an increase in the protein-sensitive anabolic mediator insulin-like growth factor, IGF-1.

In the SR by Pedersen et al (12), the evidence for beneficial or adverse effects of higher protein intake in relation to bone health was assessed as *inconclusive*. Also, the EFSA (38) found the available evidence regarding protein and bone health to be insufficient. The assessment of protein intake and risk of bone loss was based on three small and low-quality cohort studies carried out mainly in women (12), and a single good-quality meta-analysis (39) that found a “small benefit of protein on bone health”. Based on Darling et al (39) and three cohort studies that included risk of fractures, the association between bone health and protein intake was assessed as *inconclusive* (12). However, there seems to be an interaction with the intake level of calcium. Under conditions of low calcium intake an increased risk of fractures was found to be related to high animal-protein intake, but under conditions of high calcium intake (>800 mg) a decreased risk of fractures was related to high animal-protein intake. This finding is supported by an older Norwegian study of 39,787 middle-aged men and women that showed an elevated risk of hip fracture in women with a high intake of animal (non-dairy) protein under conditions of low calcium intake (40). The evidence for an association between vegetable protein intake and fracture risk was *inconclusive* (12), and this finding was supported by an older study of 32,050 postmenopausal women that showed a decreased risk of hip fracture related to a high animal protein intake but not to vegetable

protein intake (41). Fenton et al (42) published an SR and meta-analysis on the association between dietary acid load (including protein intake) and bone health and did not find support for the hypothesis that an “acidic” diet causes osteoporosis or that an “alkaline” diet prevents osteoporosis.

In the elderly, Pedersen & Cederholm (13) assessed the evidence as *suggestive* in regard to a positive association between protein intake and bone mineral density based on one intervention study and three prospective cohort studies. The evidence was assessed as *inconclusive* regarding the relation of protein intake to bone loss and risk of fractures. Interestingly, in the included randomized controlled study with calcium and vitamin D supplementation by Dawson-Hughes & Harris (43) the highest tertile of protein intake (20 E%, or 1.2 g/kg BW) was associated with less bone loss compared to the lowest tertile (14 E%, or 1.1 g/kg BW) but only in the intervention group. The habitual mean intake in the placebo group was 871 mg calcium and about 7 µg vitamin D per day, which is close to the recommended values in the NNR 2004 (44). Thus, the possible effect of protein intake on bone health might depend on an intake of calcium and vitamin D above this level.

Energy intake and body weight control

Higher satiety after protein intake than after carbohydrate and fat intake has been reported in test-meal and short-term studies (45). An SR covering the period of 1966–2003 on the effect on weight loss of LC/HP diets in the treatment of obesity showed that weight loss was associated with decreased energy intake and not with the macronutrient composition of the diet (46) but a recent SR including studies from 2002 to 2007 found LC/HP diets to be effective in reducing body weight at 6 months and up to one year (47). However, long-term results have so far been disappointing (48), and perhaps this is a reflection of difficulties with adherence to the diets. It should be emphasized that LC/HP diets cannot be used to assess the effect from protein *per se*.

An SR of dietary macronutrients and food consumption as determinants of long-term weight change in adults was conducted for the update of NNR 2012 (49). The authors found that the evidence for an association between the dietary macronutrient composition in prevention of weight gain after prior weight loss was *inconclusive*. The results suggested that the proportion of macronutrients in the diet was not important in predicting changes in weight or waist circumference.

The majority of studies addressing protein and energy intake, appetite

regulation, and body weight have been performed in overweight/obese persons, and very few studies have assessed the prevention of overweight/obesity in normal-weight populations. The NNR intend to prevent an undesired increase in body weight and overweight, thus the SR addressing protein intake in relation to energy intake and body weight control in healthy adults (12) excluded studies on overweight/obese participants or participants on weight-loss diets. Based on one prospective cohort study and two intervention studies, Pedersen et al (12) assessed the evidence for an association between protein intake and energy intake as *inconclusive*. The evidence for an association between protein intake and body weight change was also assessed as *inconclusive* (12). This assessment was based on a cohort study of 89,432 men and women that found weight gain to be significantly positively associated with total and animal protein intake, on two small cohort studies that found no significant associations, and on two small controlled trials, one low-quality and one good-quality study (50), that found high protein intake to be related to weight loss.

Based mainly on intervention studies with overweight/obese participants, the EFSA (38) assessed the protein intake data as insufficient to establish reference values in relation to body weight control.

Muscle mass, strength, and function

Adequate muscle mass and function is crucial for body function and survival and for the prevention of sarcopenia, i.e. the age-related loss of muscle mass and function, is highly relevant. Advanced sarcopenia is associated with increased risk of physical frailty and, therefore, is associated with increased likelihood of falls and impairment in the ability to perform activities of daily living (51).

The SR by Pedersen et al (12) included only one randomized controlled trial with body composition as outcome (52). This study included 15 physically active men who were prescribed either a high-protein diet (1.9 g/kg BW per day (22 E%)) or a normal diet (1.3 g/kg BW per day (15 E%)) for 6 months. No association between protein intake and change in fat mass or fat-free mass was found.

Based on one intervention study and two prospective cohort studies in the SR of the elderly (13), the evidence was assessed as *suggestive* with regard to a positive relation between muscle mass and a total protein intake in the range of 13 E% to 20 E%. In the included Health ABC Study (53), which was the first longitudinal study to examine the role of dietary protein on changes in body composition using state-of-the-art body-composition

measurements, the mean protein intake was 0.9 g/kg BW and the mean 3-year loss of lean body mass was 0.68 ± 1.9 kg. Participants in the highest quintile of protein intake (≈ 19 E%) lost less lean mass compared to those in the lowest quintile (≈ 11 E%). It is notable that there was no statistically significant association between total protein intake and 3-year loss of muscle mass adjusted for physical activity in the 49.5% of the participants who were weight stable, and this raises the awareness of a sufficient energy intake among the elderly. In a strictly controlled metabolic study with a focus on N-balance (15) and on resistance training (54), a protein intake of 0.8 g/kg BW (≈ 10 E%) for 14 weeks resulted in a loss of mid-thigh muscle area in the sedentary control group during a period of body weight stability.

Frailty is a geriatric term (characterized by slowness, weakness, fatigue, low physical activity, and unintentional weight loss) indicating that older persons are at increased risk of developing adverse health outcomes such as the onset of disability, morbidity, institutionalization, or mortality (55). An important and fundamental component of frailty is sarcopenia (56). The SR by Pedersen & Cederholm (13) included only one study that addressed protein intake and the relation to frailty, and this study found that reduced protein intake was associated with an increased risk of frailty after three years (57).

Muscle function, expressed as activities of daily living (ADL) or physical performance, is the clinically relevant outcome of muscle mass and muscle strength. Very few studies have addressed the association of protein intake to physical performance, and most of the ones that have been performed have been among disabled or frail elderly (58) and in combination with exercise (59–61). Tieland et al (58, 59) found improvements in physical performance after protein intervention, but the older studies (60, 61) found no effect from protein supplements on physical performance in the frail elderly.

Type-2 Diabetes

Based on four prospective cohort studies with long-term LC/HP diets, including one study with an LC/HP and high fat diet, the SR by Pedersen et al (12) assessed the evidence as *suggestive* regarding the relation of total and animal protein intake to increased risk of type-2 diabetes. In two of the included studies, this association was most clearly associated with intake of animal protein, but this could be a reflection of the fact that animal protein was the main protein source. Again it should be emphasized that LC/HP diets cannot be used to assess the effect from protein *per se*.

Renal function and kidney stones

The evidence for associations between protein intake and kidney function and kidney stones was regarded as *inconclusive* in the SR by Pedersen et al (12). Also, the EFSA (38) assessed the available evidence as insufficient to derive an upper level of protein intake based on kidney function.

Protein and physical exercise

Whether there is an increased protein requirement as a result of heavy physical exercise is still a matter of debate. Aerobic exercise leads to increased protein oxidation in the muscles in absolute terms. However, the relative contribution of protein to energy turnover is remarkably reduced in relation to that of fat and carbohydrate. Because the body gives priority to covering its energy needs – even when protein turnover is increased – it is important when analysing data to ensure that energy needs are being met before concluding that there are increased protein requirements during physical exercise. A critical analysis of the background data in many studies that give support for increased protein needs indicates that energy needs were not being met.

An increased demand for protein during physical exercise might be due to increased muscle mass as a result of training, increased breakdown of muscle tissue and protein turnover during strenuous physical activity, and increased gluconeogenesis from muscle protein if energy needs are not met leading to muscle protein catabolism and negative N-balance (62, 63). Studies using both the N-balance technique and stable isotope technique have suggested that the daily protein requirement might be as high as 1.4–1.8 g/kg BW in athletes with heavy training (20, 64–67). Long-term studies using stable isotope techniques (68–70) indicate, however, that there seems to exist a compensatory reduction in leucine oxidation during the recovery phase after aerobic exercise indicating a homeostatic response to conserve body protein. For strengthening exercise, the acute anabolic response to exercise and amino acid nutrition, measured by stable isotope technology, was found to reflect the 24 hour response in regularly active young persons (71). Resistance training, however, attenuated the response to an acute bout of strength training but elevated the resting muscle protein turnover (72, 73). Thus, protein utilisation might improve and become more efficient as a result of prolonged training (74, 75).

The SR by Pedersen et al (12) assessed the evidence regarding the effect

of physical training on protein requirements as inconclusive based on three intervention studies with protein intakes at or above the RDA for healthy adults. The evidence was assessed as *suggestive* for the effect of training on whole-body protein retention. The literature search on the interaction between physical activity and protein intake resulted for the most part in studies of short duration, studies in athletes, or studies of specific protein or amino acid supplements, and these studies were not eligible for the review. The conclusion from the SR (12) is in agreement with the most recent position statement on 'Nutrition and Athletic Performance' from the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine (76) that states that protein requirement for N-balance, even in athletes, generally can be met through diet alone without use of protein or amino acid supplements. Thus, the same should be valid for healthy adults who are physically active. The position statement does, however, recommend a slightly higher daily protein intake of 1.2 to 1.7 g/kg BW in endurance and strength-trained athletes.

Even though strenuous physical training could potentially double the daily protein requirements (as suggested by earlier studies), the daily energy need in such populations would also be very high. Therefore, there are no data to support the use of protein supplements in athletes consuming a variable, mixed diet (62, 63), and thus there are no data to support such use in healthy adults performing regular physical activity.

Exercise among the elderly, especially resistance training, is a strong anabolic muscle stimulus with proven effects (77), but only a few studies have addressed the issue of dietary protein intake for the optimal effect of physical exercise in older adults. The SR by Pedersen & Cederholm (13) included only two intervention studies (54, 78). Due to the small number of included studies, the evidence was assessed to be *inconclusive*. Studies of short duration or studies of specific protein or amino acid supplements were not eligible for the review. However, it has been suggested from short-term human studies in the elderly that provision of essential amino acids (79, 80), especially the branch-chained essential amino acid leucine (81, 82), might have specific protein anabolic effects, but more long-term studies on leucine supplementation are needed.

Although supplementation does not seem to be needed to ensure adequate protein intake, the timing of protein supplementation in relation to exercise might be important for the anabolic effect of the supplementation in both young (67) and older (83) subjects. These findings might be explained by interacting effects between acute exercise and nutrition

on net muscle-protein balance in the post-exercise period (84). However, long-term dietary protein-based studies are still lacking.

There has also been discussion as to whether there is a threshold for post-exercise protein intake to initiate an augmented protein synthesis response. A minimum amount of approximately 25–30 g of high-quality protein is necessary to maximally stimulate muscle protein synthesis (67). Accordingly, a short-term study by Symons et al (85) demonstrated that the protein synthesis effect was the same irrespective of an intake of 30 grams or 90 grams of protein during one meal.

Despite the above findings, it is still premature to make recommendations regarding timing and distribution of protein intake in the elderly based on the present scientific evidence.

Recommended intake

Adults

Based on Rand et al's meta-analysis (14), the WHO/FAO/UNU (9) recommends 0.83 g good-quality protein/kg BW/d based on an estimated average requirement (EAR) of 0.66/kg BW per day. The values are based on the 97.5th percentile to allow for individual variability, and these values are about 10% higher than the previous values proposed by the FAO/WHO/UNU report from 1985 (86).

The 2002 US recommendations from Institute of Medicine, IoM, (87) for protein were also based on the meta-analysis of N-balance studies by Rand et al (14) and cite an EAR of 0.66 g/kg BW per day and an RDA of 0.8 g good-quality protein/kg BW per day. These recommendations are for healthy adults based on a coefficient of variation of 12% and with no significant differences according to adult age or sex. In addition, IoM 2002 (87) recommends an Acceptable Macronutrient Distribution Range of 10–35 E% from protein. The EFSA (38) also based their Population Reference Intake of 0.83 g good-quality protein/kg BW per day on the meta-analysis by Rand et al (14).

For the update in NNR 2012, two SRs were conducted to assess the evidence behind the dietary requirement of protein. One SR included healthy adults (12) and one included healthy elderly populations (13), and they both assessed the evidence as *probable* for a median EAR of nitrogen of 105 mg/kg BW per day. This corresponds to a daily intake of 0.66 g good-quality protein/kg BW and a subsequent RDA of 0.83 g good-quality protein/kg BW per day regardless of sex or age.

The SRs also assessed the possible health effects of varying protein intake in order to evaluate the evidence for an optimal protein intake. For most outcomes, the evidence of a relation to protein intake was assessed as *inconclusive* (e.g. all-cause mortality, cancer mortality and cancer diseases, cardiovascular disease, bone health, body weight control, body composition, and renal function). However, an inverse relation of intake of vegetable protein to cardiovascular mortality and blood pressure was assessed as *suggestive* (12). Thus, despite some studies finding a decreased risk of outcome associated with vegetable protein intake, there is at present insufficient evidence for a recommendation of an increased intake of protein from vegetable food sources.

Many of the protein intake data from the observational studies were flawed by misreporting, and poor adherence to the diets was an issue in some of the intervention studies. Thus, the SR by Pedersen et al (12) failed to identify high-quality studies that could alter the classical criterion for protein recommendations, i.e. studies based on N-balance. Also the EFSA Panel (38), WHO (9), and IoM (87) considered several health outcomes associated with protein intake and concluded that currently available data were insufficient to establish reference values or recommendations.

At reference energy intakes (see the chapter on Energy), a protein intake of 0.8 g/kg BW/d corresponds to approximately 10 E% from protein provided a moderate physical activity level (PAL) of 1.6. Thus, 10 E% protein might represent the *lower intake range* for healthy adults with a PAL of 1.6.

Based on the available evidence, and according to the Nordic dietary habits, a protein intake corresponding to 10–20 E% is recommended. Thus, the recommended range is the same as in NNR 2004.

The range of 10–20 E% corresponds to about 0.8–1.5 g protein/kg BW/d provided a PAL of 1.6 for an intake of about 10 E% and a PAL of 1.4, for an intake of about 20 E%, respectively.

For food planning purposes with energy intake in the range of 8–12 MJ, an appropriate target is 15 E% and this corresponds to about 1.1 g protein/kg BW/d. This intake of protein should also adequately meet the requirements for essential amino acids. With decreasing energy intake below 8 MJ (e.g., decreased physical activity or during intentional weight loss), the protein E% should increase accordingly and still correspond about 1.1 g protein/kg BW/d.

In relation to physical activity, the protein requirement can generally be met through a diet that meets the energy need. Thus, a protein intake

corresponding to 10–20 E% is recommended for adults, and an intake corresponding to 15–20 E% is recommended for the elderly.

Elderly

Chronic diseases are more frequent in the elderly, and such conditions might lead to periodic temporary losses of body protein through catabolic exacerbations of the disease, temporary periods of bed rest, or loss of appetite. The losses must be replaced from the diet and thus represent an added need for dietary protein (88). In addition, older individuals exhibit a gradual loss of muscle mass and strength with age (sarcopenia). This is estimated to be a daily loss of 0.5 mg nitrogen per kg BW (89) that occurs naturally and is not simply due to decreased physical activity (90).

Based on N-balance studies, Pedersen & Cederholm (13) assessed the evidence as *probable* regarding a median EAR of nitrogen of 105 mg/kg BW per day which corresponds to 0.66 g high-quality protein/kg BW per day and the subsequent RDA of 0.83 g high-quality protein/kg BW per day to represent the *minimum dietary protein intake* for virtually all healthy elderly persons. Despite being in N-balance, two studies (15, 17) also found a decreased urinary N-excretion, which in the study by Campbell et al (15) also was related to a loss of muscle mass, indicating that a higher protein intake might be necessary to maintain muscle mass among the elderly. Thus, N-balance *per se* might not reflect a preservation of muscle mass.

The SR (13) also assessed the health effects of varying protein intakes in order to evaluate the evidence for an optimal protein intake. For maintenance of bone mass, muscle mass, and strength, as well as for a relationship with morbidity and mortality, the assessment of the evidence ranged from *suggestive* to *inconclusive*. Results from prospective cohort studies in particular suggested that a safe intake of up to at least 1.2–1.5 g protein/kg BW/d or approximately 15–20 E%, represents an *optimal intake level*.

Short-term studies, including studies with protein hydrolysates or amino acids and studies that have looked at the effect of timing or meal distribution, point to specific anabolic effects but larger long-term studies are still needed as background for future recommendations.

Based on data from N-balance studies in relation to maintenance of muscle mass (supported by prospective cohort studies and by suggestive health effects) it is recommended to increase the protein intake for those ≥ 65 years of age. In relation to the age-related decrease in energy intake, a diet with a protein content in the range of 10–14 E% might not sufficiently cover the need for protein in absolute amounts.

A protein intake corresponding to 15-20 E% is recommended, and with decreasing energy intake the protein E% should be increased accordingly because the protein needs do not change in a corresponding manner.

The range of 15-20 E% corresponds to about 1.1-1.3 g protein/kg BW/d provided a PAL of 1.6 for 15 E%, and a PAL of 1.4 for 20 E%, respectively, as estimated from body weights and energy expenditure by Gaillard et al. (91).

For food planning purposes, the recommendation is 18 E%, which corresponds to about 1.2 g protein/kg BW/d. This is an increase of 20% compared to the NNR 2004 recommendation.

Infants and children

Recommended protein intakes for infants and children are based on the factorial method. The calculation is based on estimates of the need for maintenance and growth, the efficiency of conversion from dietary protein to body protein, and intra individual variation in growth. There is, however, considerable discussion about the appropriate values to use for these calculations during the first year of life, and this has led to large differences in the recommendations for protein intake during the first year of life, especially the first 6 months.

A highprotein intake results in a high renal solute load. However, it is only during the first months of life that the kidneys cannot handle a high solute load (92). For NNR 2012 (as well as for the previous NNR 2004), no adequate intake for protein is given for the first 6 months. During this period, infants are either breastfed or receive infant formula. The protein content of breast milk is considered adequate for term infants and the protein content of infant formula is regulated by EC legislation. The protein content of infant formula and especially follow-on formula has decreased over the years. According to the current regulation/directive (REGULATION (EC) No 1243/2008 and Directive 2006/141/EC), the protein content of infant formula should be between 0.45 and 0.7 g/100 kJ and the protein content of follow-on formula should be between 0.45 and 0.8 g/100 kJ.

Based on N-balance studies, the WHO/FAO/UNU (9) calculated a maintenance requirement of 0.66 g protein/kg BW between 6 months and 18 years of age. Adding a requirement for growth results in an estimated average requirement that falls very rapidly during the first two years of life (1.12 g/kg BW at 6 months and 0.79 g/kg BW at two years) and then falls more slowly reaching 0.75 g/kg BW at 10 years and 0.69 and 0.66 g/kg BW for boys and girls, respectively, at 18 years. The “safe level of intake”

is calculated by adding 1.96 standard deviations (SD) to the average requirement. Compared to previous calculations (86), the recommended safe level has decreased for all ages but especially for the first two years. The EFSA (38) has accepted the WHO/FAO /UNU 2007 (9) figures, and these form the basis of the recommended levels for children aged 6 months to 18 years in NNR 2012 (Table 12.1.).

In relation to body weight, the WHO/FAO/UNU (9) gives the reference values of 0.9 g/kg BW per day from 3 to 18 years of age for boys and from 3 to 15 years of age for girls. This value decreases slightly for girls to 0.8 g/kg BW per day between 15 and 18 years of age. The protein energy percentage necessary to cover the adequate protein intake can be calculated by combining these reference values with the reference values for energy intake for age and sex. The average requirement calculated as E% is about 5.3 E% at 6 months, followed by a decline to 4.3 E% at 2 years. Thereafter there is a gradual increase to about 7 E% and 9 E% at 17 years of age for boys and girls, respectively.

Table 12.1. Safe level of protein intake (average requirement + 1.96 SD) in weaned infants and children

Age	Protein g/kg BW	E%	g/100 kJ
6–11 months	1.1	7–15	0.4–0.9
12–23 months	1.0	10–15	0.6–0.9
2–17 years	0.9	10–20	

Expressed as E%, the protein intake increases considerably during the first 1 to 2 years of life when the infant gradually changes from breast milk with a protein content of about 5 E% to the family diet that typically provides around 15 E% from protein. The average protein intake among children varies between 13 E% and 16 E% from about 1 year of age in most European countries, including the Nordic countries (38).

With regard to later risk of non-communicable diseases such as cardiovascular disease, both the quantity and quality of protein intake in infancy and childhood is of interest. In the Nordic setting, the quantity is more important because the protein sources are usually of animal origin and quality is not a concern. The upper level of a healthy protein intake in infancy and childhood, however, has yet to be firmly established.

One of the SRs carried out for the update of NNR 2012 (22) aimed

to review the scientific data published between 2000 and 2011 on the short- and long-term health effects of different levels of protein intake in infancy and childhood. Special focus was on growth, serum lipids, glucose and insulin, blood pressure, body weight, body composition, and bone mineral density. The authors concluded that the evidence was *convincing* that higher protein intake in infancy and early childhood contributes to increased risk for obesity later in life. Which age period is most sensitive to high protein intake was not clear, but with regard to the available data the authors state that the first two years of life seems probable and that a protein intake between 15 E% and 20 E% in early childhood increases the risk of being overweight later in life. A high-quality European multi-centre, double-blind, randomized controlled trial included in the SR tested the effect on growth of a low vs. a high protein intake during the first two years of life (93). The protein content in the formulas and follow-on formulas used in the trial represented approximately the lowest and highest acceptable levels in the range given in the European Union (EU) directives from 1991 (when the directives for follow-on formula, at 0.5-1.0 g/100 kJ, were slightly higher than the present) and corresponded at 12 months to 14.0 E% in the low-protein formula group and 16.7 E% in the high-protein formula group. The higher intake in the latter group was associated with increased risk for overweight at 24 months. In several Nordic countries, mean protein intake is close to 15 E% during the first years of life indicating that a large proportion of young children have a higher protein intake that might contribute to increased risk of later obesity (22).

With regard to other outcomes, the evidence was not as strong (22). The protein source appears to be important and there was *suggestive* evidence that intake of animal protein, especially from dairy products, has a stronger association with growth, and particularly with weight gain, than vegetable protein. The evidence was also *suggestive* that higher intake of animal protein was associated with earlier onset of puberty and that total protein intake was positively associated with bone mineral content. Other associations with early protein intake and different health outcomes were assessed to be *inconclusive*. Part of the lack of evidence could be due to different effects depending on BMI, phenotypes, or gender, and the effects of these factors warrant further studies.

The recommendations for protein intake in children in the NNR 2012 are the same as in the NNR 2004, i.e. 7-15 E% from 6 to 11 months of age, 10-15 E% for 12 to 23 months of age, and 10-20 E% for 2 to 17 years of age.

Pregnant and lactating women

During pregnancy, the average protein requirement is increased to provide additional protein for deposition in maternal (blood, uterus, and breasts), foetal, and placental tissues. Additional protein is also needed to maintain the increased mass of the pregnant body. According to the WHO/FAO/UNU report (9), the additional safe intake of protein for a healthy woman gaining 13.8 kg body weight during her complete pregnancy is 0.7, 9.6, and 31.2 g/d during the first, second, and third trimester, respectively. This represents less than 12 E% protein for a reference woman of reproductive age assuming a PAL of 1.6. The dietary protein content in the Nordic countries is generally higher than 12 E%, and the protein quality is generally high. Consequently, most pregnant women are able to cover their protein needs by consuming their normal diet in a quantity that allows a weight gain within the recommended limits. However, it is recommended that the increased intake of protein during pregnancy – due to increased energy intake – should consist of normal food rather than of high-protein supplements. The basis for this is that studies (94, 95) have indicated that supplements with a high protein content during pregnancy might result in adverse pregnancy outcomes.

The average protein requirement is also increased during lactation when the breast milk produced by a woman provides all of the protein needed by her infant. The WHO/FAO/UNU (9) recommends that the safe level of additional protein for a lactating woman in full lactation is 18–20 g per day. This figure is applicable during the first six months of lactation. During partial lactation, i.e. 6–12 months post partum, the recommended amount is 12.5 g/d. This represents less than 12 E% protein and, therefore, a lactating as well as a pregnant woman can, in most cases, cover her protein requirements with her normal diet if her energy requirements are covered.

Based on this, the recommended E% protein during pregnancy and lactation is the same as for non-pregnant women.

Reasoning behind the upper intake range

Based on the risk of mortality and morbidity, the SR by Pedersen et al (12) also assessed the evidence for potential adverse effect of a high protein intake.

There was no indication of adverse effects of protein intake in relation to bone health provided a sufficient calcium intake, and an included meta-

analysis did not find support for the hypothesis that “acid” from the diet causes osteoporosis (42).

The evidence was assessed as *suggestive* regarding an increased risk of all-cause mortality and type 2 diabetes in relation to long-term LC/HP diets with a total protein intake of at least 20–23 E% (12). However, the use of an LC/HP score makes it uncertain whether the effects result from reduced carbohydrate or increased protein intake. A biologically plausible explanation might be that diets rich in plant foods like vegetables, fruits, nuts, and whole-grain cereals are associated with a lower risk of chronic diseases (96) while protein intake in the form of red and processed meat increases the risk of cancers, especially colorectal cancer (28). Notably, the data evaluated the health consequences of long-term habitual dietary intakes and should not be interpreted as indicating that short-term use of LC/HP diets is detrimental to health.

One study with elderly subjects (34) found that animal protein intake was related to increased risk of hypertension among persons ≥ 70 years of age. The lowest tertile of total protein intake was 14 E% and the highest was 19 E%.

With regard to renal function, the SR by Pedersen et al (12) called for reflection. An increase in GFR is a physiological adaptation to increased protein intake (97). Walrand et al (18) found that a high protein intake did not increase GFR in the elderly participants in their study from a baseline GFR that was lower than that of the young participants. This was probably due to the reduced kidney function in the elderly because patients individuals with mild to moderate chronic kidney disease also do not show the usual protein-induced increase in GFR (98). Caution is also required due to the observation of a decline in GFR among women with mild kidney insufficiency (99), and because older adults might have severely reduced GFR without knowing it.

With regard to microalbuminuria, one experimental study found an increase in urinary albumin after seven days on a high protein intake of 2.4 g/kg BW per day, but a similar increase in protein intake in another short-term experimental study of healthy young men did not find an increase in 24 hour urinary albumin excretion (12). Further studies are needed to settle whether this discrepancy is due to the different durations of the studies or due to different methods of analysis of albumin in the urine. A review by Friedman (100) cites an earlier 3-week study showing a reduction in proteinuria with reduced protein intake (from 75 g per day to 43 g per day). Caution is required until this matter is settled.

The upper range for protein intake in adults of 20 E% is unchanged from the NNR 2004. This recommendation takes into account the potential harmful effects of a long-term dietary protein intake above 20–23 E% seen in studies with protein *per se* and with LC/HP and/or high-fat diets, the caveat from renal function studies, and a consideration of the recommendations for fat and carbohydrates.

Although possible negative consequences of a high-protein intake have not been clearly demonstrated in infants and children, a decrease in the upper levels for the ages of 6 to 23 months is deemed prudent. The following upper ranges for protein intake are suggested, assuming sufficient intake of other nutrients: 0–6 months, 10 E%; 6–11 months, 15 E%; 12–23 months, 17 E%; and 2 years and older, 20 E%.

Upper intake levels

No upper intake level could be established based on the present evidence.

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13

Alcohol

	Adults	Children and adolescents
Women	< 10 g/d (< 5 E%)	Abstain
Men	< 20 g/d (< 5 E%)	

Introduction

Alcohol (ethanol) is generally consumed as beer (about 2.5–6 vol% alcohol), wine (about 12 vol%), or spirits (about 40 vol%). The energy liberated upon oxidation of alcohol in the body corresponds to 29 kJ per gram. At high alcohol consumption, however, the energy efficiency appears to be lower with relatively higher heat dissipation than with the other energy-yielding nutrients (1). Alcohol is efficiently absorbed through passive diffusion, mainly in the small intestine and is distributed throughout the total water compartment of the body. Most of the absorbed alcohol is oxidized in the body but a small amount (5%–10%) is lost through expired air and in the urine.

In the Nordic countries, mean alcohol consumption accounts for about 2% to 6% of the total energy intake in adults, but the intake is very unevenly distributed.

Nutritional aspects

Replacing part of the food intake with alcoholic beverages can impair the quality of the diet. In particular, the consumption of dairy products, fruits, and vegetables appears to decrease when the intake of alcohol is increased. Some exceptions to this pattern, however, are noted. For example, a Danish study showed a strong positive association between fruit and vegetable consumption and wine intake (2). A high level of alcohol consumption can also result in impaired absorption of nutrients and increased nutrient loss

in the urine. From a nutritional point of view, therefore, it is reasonable to recommend moderation in alcohol intake. Nutritional status among high alcohol consumers is always affected (3), and deficiencies in ascorbic acid, thiamine, magnesium, phosphorus, vitamin D, and protein are frequent (4, 5).

Alcohol and health

Alcohol is a toxic substance that affects all organs of the body. Both acute and chronic alcohol-induced damage contributes significantly to morbidity and mortality. From a public health perspective, it is important to bear in mind that overall consumption is a main determinant of the alcohol-related harm rates in the population (6). The negative health effects of alcohol are primarily determined by the total amount of alcohol to which the body is exposed. This means that alcohol damage might develop in individuals who have not been visibly drunk. It is likely that daily consumption of 70 g alcohol will result in alcohol-related damage (7).

A review of the health aspects of alcohol consumption was carried out for the revision of the Nordic Nutrition Recommendations (NNR) with a focus on those areas in which new scientific knowledge has emerged since the 4th edition and that has special relevance for the Nordic setting (8). The literature search covered articles published between January 2000 and November 2010, with a complementary search up to February 2012. The majority of the research covered the following topics: Cardiovascular disease and related metabolic risk factors, total mortality, cancer, weight change/outcome, and pregnancy or birth outcomes.

Cardiovascular disease

Alcohol has been associated with coronary heart disease (CHD), atrial fibrillation (AF), ischemic stroke, haemorrhagic stroke and congestive heart failure (CHF).

Coronary heart disease

A meta-analysis and review comprising data from 84 prospective cohort studies with a total of 3,159,720 study participants assessed the relative risk (RR) of various cardiovascular outcomes. CHD among drinkers and non-drinkers (9). The pooled, adjusted RRs for alcohol drinkers relative to non-drinkers were 0.71 (95% CI: 0.66–0.77) for incident CHD (29 studies) and 0.75 (95% CI: 0.68–0.81) for CHD mortality (31 studies). These results

persisted after excluding former drinkers from the category of abstainers. In analyses exploring dose-response, alcohol consumptions of 2.5-14.9 g/d, 15-29.9 g/d, or 30-60 g/d were all associated with similar and statistically significant reductions in the RR of CHD relative to non-drinkers. The highest consumption category (> 60 g/d) was associated with an RR of 0.76 (95% CI: 0.52-1.09). There is evidence, therefore, of a maximal upper range of intake for the cardioprotective effect of alcohol but no indication of a higher risk among the heaviest drinkers. In contrast, an earlier meta-analysis found that the association between alcohol and CHD risk was J-shaped implying a minimum RR of 0.80 at 20 g/d, a significant protective effect up to 72 g/d, and a significantly increased risk at intakes above 89 g/d (10).

The impact of drinking pattern has been addressed in fewer studies, but the majority of these find a non-beneficial or even harmful effect of a drinking pattern that involves drinking large amounts of alcohol per occasion (binge drinking).

The finding that CHD risk is lower in light to moderate drinkers compared with non-drinkers is very consistent across study populations with different distributions of confounders and potential effect modifiers (9). Due to the heterogeneity of the exposure in studies investigating the independent effects of drinking patterns, it is premature to make a firm conclusion of the exact measure of drinking pattern that most accurately captures the non-beneficial effect. However, most evidence suggests that a drinking pattern in line with the NNR 2004 recommendations (< 10 g/d for women and < 20 g/d for men) is not detrimental. In conclusion, the current evidence is in accordance with NNR 2004.

Atrial fibrillation

In a meta-analysis and review comprising data from five case control and nine prospective cohort studies, of which six were hospital based, and included a total of 138,020 participants, high alcohol intake was associated with increased risk of AF (11). In pooled analyses, the RR for the highest versus the lowest alcohol category was 1.51 (95% CI: 1.31-1.74), but the definition of 'high' intake differed from study to study (11). In dose-response analyses, each 10 g/d increment was associated with an increased risk of AF (RR = 1.08, 95% CI: 1.05-1.10). Results of the meta-analysis indicate that the risk of AF is probably increased by heavy drinking, while the effect of light to moderate intake is more uncertain due to a lack of high-quality studies. In conclusion, the current evidence is in accordance with the recommendations in NNR 2004.

Stroke

Two meta-analyses and reviews assessed the association between alcohol intake and stroke (9, 12). These papers analysed the results of 16 studies on haemorrhagic stroke and 20 studies on ischemic stroke that included a total of 737,038 study participants. The results of the analyses show that high alcohol intake is consistently associated with an increased risk of both haemorrhagic and ischemic stroke. With moderate intakes of up to 3 drinks per day, the results are inconsistent; moderate consumption seems to be protective against ischemic stroke, but neutral or slightly detrimental for haemorrhagic stroke. In conclusion, the current evidence is in accordance with the recommendations in NNR 2004.

Congestive heart failure

A meta-analysis and review (13) included six prospective cohort studies with a total of 164,479 study participants. Compared with never drinkers, the pooled RRs for CHF were 1.16 (95% CI: 0.90-1.51) for former drinkers and 0.90 (95% CI: 0.83-0.98), 0.80 (95% CI: 0.73-0.88), 0.78 (95% CI: 0.65-0.95), and 0.77 (95% CI: 0.63-0.95) for current drinkers of 0.1-0.9, 1-7, 8-14, and >14 drinks/week, respectively (13). There was no heterogeneity in the findings between the six individual studies. Light to moderate drinking was not associated with increased CHF risk, and at best was associated with a lower risk of CHF. In conclusion, the current evidence on this subject is in accordance with the recommendations in NNR 2004.

All-cause mortality

A meta-analysis and review was carried out on 34 prospective cohort studies published up to 2005 reporting on mortality from Australia, China, Japan, Europe, and the US that included 1,015,835 study participants (14). A J-shaped relationship between alcohol and all-cause mortality was found in adjusted analyses of both men and women. Consumption of alcohol of up to 2-4 drinks per day in men and 1-2 drinks per day in women was inversely associated with total mortality, with a reduction of 18% in women (99% CI: 13%-22%) and 17% in men (99% CI: 15%-19%). Higher intakes of alcohol were associated with increased mortality. Risk reductions were somewhat lower in analyses adjusting for age, socioeconomic status, and dietary markers and were apparent at up to 3 drinks per day for men and up to 2 drinks per day for women. The calculated reversion point (the dose of alcohol at which the protection against mortality was

not statistically significant at the 99% confidence level) was 30 g per day in the adjusted model. Because the relative incidences of alcohol-related diseases and outcomes differ by age, the J-shaped association between alcohol and all-cause mortality also differs by age. The nadir (representing the alcohol intake at the lowest risk of mortality) is achieved at a lower intake at younger ages. In a British study, the lowest mortality risk among women 16 to 34 years old and men 16 to 24 years old was observed among the non-drinkers (15). Hence, a beneficial effect of alcohol is not observed among the young, and instead alcohol is directly associated with mortality in this age group.

Results from studies regarding the role of drinking pattern consistently imply an increased mortality risk associated with drinking large amounts of alcohol per session, or binge drinking (16). Furthermore, there is good evidence that the protective effect of alcohol on cardiovascular disease only occurs if the pattern of drinking is not a binging pattern (16). Hence, the J-shaped association between alcohol intake and all-cause mortality depends upon the drinking pattern.

The association between alcohol and all-cause mortality is J-shaped; the nadir of the 'J' reflects a relatively lower risk of CHD among light to moderate drinkers compared with abstainers and the ascending leg of the J is reflective of an increased risk of alcohol-related diseases such as liver cirrhosis, pancreatitis, upper gastrointestinal cancers, cardiomyopathy, polyneuropathy, and deaths from accidents and violence among excessive alcohol users. Because the association between alcohol and all-cause mortality represents the sum of the numerous diseases and outcomes that are related to alcohol, the shape and nadir of the risk curve depends upon the distribution of other variables such as age, relative incidences of diseases, the prevalence of drunk-driving, etc. Thus, the association between alcohol and all-cause mortality does not have the same causal interpretation as associations between alcohol and singular endpoints.

In conclusion, light to moderate drinking is not associated with increased mortality risk and is at best associated with a lower risk among middle-aged and older adults who do not engage in episodes of heavy drinking. Total abstinence is associated with the lowest risk of mortality in young adults, and binge drinking should be avoided in all age groups.

Cardio-metabolic risk markers

Serum lipids

A comprehensive high-quality meta-analysis of intervention studies was published in 2011 (17) and found that alcohol significantly increased levels of high-density lipoprotein and adiponectin and significantly decreased levels of fibrinogen. These favourable changes in cardiovascular biomarkers provide indirect physiological support for a protective effect of moderate alcohol use against CHD.

Hypertension

There is convincing evidence that high alcohol intake is associated with increased blood pressure (18) and risk of hypertension (19). During recent years, there has been some discussion as to whether light to moderate alcohol intake is associated with lower blood pressure and lower risk of hypertension, especially among women (19).

Insulin and glucose concentrations

Reviews and meta-analyses are sparse in this area, but individual studies have found that an alcohol intake of 1–2 drinks per day is associated with reduced fasting insulin concentration and improved insulin sensitivity (20–24). Furthermore, fasting glucose levels were similar in non-drinkers and moderate alcohol drinkers in a prospective cohort study (25).

Cancer

The evidence that intake of alcohol is related to several types of human cancers has been strengthened since the mid-1990s. Alcohol (ethanol) is classified as a human carcinogen by the International Agency for Cancer Research (26). The 2007 World Cancer Research Fund report included an extensive systematic review of the available evidence on the association between alcohol intake and the development of cancer (27). Evidence was graded as “convincing” for an increased risk of cancer of the mouth, pharynx, larynx, and oesophagus and for colorectal cancer among men and breast cancer among women. There was “probable” evidence for an association between alcohol intake and the risk of liver cancer and colorectal cancer among women. Several subsequent meta-analyses and reviews have been published. For the cancers with sufficient evidence in the WCRF report (27), new studies have supported the evidence of a relation between alcohol intake and cancer risk (28–38). This is especially the case for can-

cers of the upper aerodigestive tract and colorectal cancer and for breast cancer among women.

The WCRF review concluded that a substantial effect on risk was unlikely with regard to renal cell cancer (27). A subsequent meta-analysis by Song et al. (39) included 20 case-control studies, 3 cohort studies. A pooled analysis of the cohort studies found an inverse association with the greatest reduction at the moderate level of intake, while an alcohol intake >15 g per day does not confer additional benefits for prevention of renal cell cancer.

A meta-analysis indicated an association between heavy alcohol intake (≥ 3 drinks/d) and increased risk of pancreatic cancer (33). There was no association between moderate drinking and pancreatic cancer risk. However, because smoking is a strong risk factor for pancreatic cancer, residual confounding is a potential problem in these studies. This could also be the case in the studies between alcohol intake and lung cancer, where a suggestive increased risk has been shown (40). No strong association was shown for alcohol intake and the risk of ovarian, endometrial, or non-Hodgkin lymphoma (41-43). A suggested possible protective effect of alcohol intake on lymphoma risk might differ by lymphoma type.

There is some evidence suggesting that alcohol increases the risk of liver cancer through alcohol-associated fibrosis and hepatitis (27, 44). Liver cirrhosis was found to be present among 80% of patients with liver cancer (45).

A review on alcohol consumption and prostate cancer (46) concluded that a daily consumption up to about 3 drinks per day does not appear to influence prostate cancer risk, but heavy consumption of 7 or more drinks per day might be associated with an increased risk. However, the data on high exposure in this review were limited to only one prospective study and four case-control studies.

In a meta-analysis on alcohol intake and bladder cancer, the overall estimate showed no association (47). Sub-analysis did show a relation between beer and wine intake and a reduced risk of bladder cancer in a dose-dependent manner, and this should be explored further in future studies.

The overall conclusion is that the evidence for associations between alcohol intake and cancer does not show any "safe limit" of intake. This is especially true for breast cancer where even very moderate intake has been shown to increase the risk (48). The effect is from ethanol irrespective the type of drink (27). The current evidence on the relationship between alcohol and cancer risk is in accordance with the recommendations in NNR 2004.

Weight maintenance

Results from a review including 31 publications with 13 prospective cohort studies and 4 clinical trials did not show any consistent associations between alcohol intake and weight gain (49). Some studies, however, found that higher levels of consumption (> 2–3 drinks/d) were associated with weight gain. The type of beverage seems to be of importance with a lower weight gain observed for wine compared to beer and spirits. Only four prospective studies reported on the relation between alcohol intake and waist circumference or waist to hip ratio. The findings were inconsistent with studies finding positive, negative, or no associations. The effect of alcohol on weight gain and waist circumference is not clear from the current evidence, and no final conclusion could be drawn.

Prenatal alcohol exposure

Alcohol can affect the developing foetus in a dose-dependent manner. Alcohol is teratogenic and can lead to Foetal Alcohol Syndrome (FAS), which is characterized by cranioccephal abnormalities, physical and mental retardation, and cardiac and joint abnormalities (50). These effects are mainly seen with an alcohol intake above 24–48 g/d.

A systematic review of prenatal alcohol exposure (51) found that low to moderate levels of alcohol consumption had no consistently significant effects on miscarriage, stillbirth, intrauterine growth restriction, prematurity, birth weight, small for gestational age at birth, or birth defects. However, weaknesses in the evidence preclude the conclusion that drinking at moderate levels during pregnancy is safe.

Alcohol intake during lactation

Although no effects of alcohol consumption on the infant during breastfeeding have been established, some studies (52) - but not all (53) - have suggested that there is impaired development in infants whose mothers consume alcohol when lactating. Reduced milk production (54), reduced milk intake (55), and sleep disturbances in the child (56) have been described. These effects are transient and compensated for by the child within 24 hours if the mother does not continue to drink during that time. No medical consequences have been seen in the child if a lactating mother occasionally drinks small amounts of alcohol (57). Mothers in Sweden are advised that there are no positive effects of alcohol intake while breastfeeding, but also that occasional intake of small amounts (not exceeding 1–2 small glasses of wine 1–2 times per week) is not harmful to the child.

Recommendation

Alcohol consumption is associated with both negative and positive health effects and tends to have a negative effect on diet quality. The evidence shows that regular, moderate alcohol consumption might confer cardio-protective effects among middle-aged and older individuals, but alcohol consumption among young adults is detrimental. For most cancers, there is convincing evidence that alcohol consumption increases the risk and it not possible to set any “safe limit” of intake. This is especially true for breast cancer, where even very moderate intake has been shown to increase the risk. Light to moderate regular alcohol consumption is not associated with increased mortality risk among middle-aged and older adults. Among young adults, however, alcohol consumption is associated with increased mortality.

Based on the overall evidence, it is recommended to limit alcohol intake. Based on estimates of the maximal mortality risk reduction associated with moderate alcohol consumption (15, 16), the intake should not exceed 10 g (approximately 1 unit*) per day for women and 20 g (approximately 2 units*) per day for men. The consumption of alcohol should not exceed 5% of the energy intake in adults.

Pregnant women, children, and adolescents are recommended to abstain from alcohol. Lactating women are recommended to limit alcohol intake.

* 1 unit is defined as 12 g alcohol (41) corresponding to the alcohol content in one bottle of beer (330 mL), one glass of wine (120 mL), or one glass of spirits (40 mL). The definition of a unit varies in different countries from approximately 8 g to 12 g (17).

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Fluid and water balance

	Adults and children > 14 y	Children 2-13 y
Guiding value*	1-1.5 L/d	1 L/d

* From water and fluids. In addition to water from foods.

Introduction

Safe water for drinking and sanitation is critical to maintaining good health. This pivotal role of water is described in several human rights provisions, including the Convention of the Rights of the Child (Article 24) and the International Convention on Economic, Social, and Cultural Rights (elaborated on in General Comment 15), and is highlighted in Voluntary Guideline 8c on the Right to Adequate Food as adopted by the Food Agricultural Organisation (FAO). According to the World Health Organisation (WHO), 2.5 million children suffer annually from diarrhoea and malnutrition due to unsafe water, and improvement in water standard in large parts of the world could have a profound impact on the incidence of many infectious diseases that currently affect millions of people of all ages.

Dietary intake

The usual volume of ingested water and other fluids amounts to 1,000-2,000 mL per day in the Nordic countries. Foods contain on average 1,000 to 1,500 mL water per day. This brings the total amount of available water to 2,000-3,500 mL per day, which is about 10% of total water content of the body.

Physiology and metabolism

Water is the main component of the human body and is vital for organ functions and for thermoregulation. Water content as a fraction of body weight is usually lower in women than in men. It also varies with age and is about 75% in new-borns and about 50% in the elderly. Approximately two thirds of the total body water is confined to the intracellular compartment and the remaining third is located extracellularly. In the extracellular compartment, about 75% of the water is in the interstitium and 25% is a component of blood plasma (1).

The regulation of fluid balance is closely linked to the regulation of electrolyte balance. In the kidneys, the excretion of water and electrolytes is regulated by hormones, in particular the antidiuretic hormone and aldosterone. When there is excess water in the body, diluted urine is excreted. If the concentration of electrolytes in body fluids becomes too high, the thirst centre in the brain is stimulated and this leads to a feeling of thirst and reduced excretion of water by the kidneys.

Foods provide an average of 1,000 mL to 1,500 mL of water per day. The water content in food items varies considerably and ranges from about 5% in nuts to 90% in fruits and vegetables. Intake of drinking water and beverages also provide varying amounts of water, and the oxidation of fat, carbohydrates, and protein yields an additional 300 mL to 350 mL of water per day. Loss occurs by through urinary output, the water in the stools, and evaporation from the respiratory tract and the skin. The daily urinary output exceeds 600 mL in healthy adults and is normally between 1,000 and 2,500 mL. The water content of stools is generally 100 mL to 200 mL per day, but this amount can increase considerably when a person suffers from diarrhoea. The daily insensible losses by evaporation are on average 300 mL to 500 ml per square meter of body surface in a temperate climate. Losses by sweating are generally small, but they can increase to several litres per day in a warm and humid environment or with heavy exercise in temperate conditions.

During total parenteral nutrition, the daily requirement for total water is generally considered to be 30 mL per kg body weight and this corresponds to 2,250 mL for a 75 kg healthy person living in temperate conditions and performing moderate physical activity.

Requirement and recommended intake

The vast majority of healthy people meet their daily hydration needs by letting thirst be their guide. It is virtually impossible to give exact recommendations on daily water intake for healthy subjects because the requirement for fluids shows considerable inter-individual variations, and it is confounded by physical activity patterns and the ambient climate. Moreover, the evidence is insufficient to establish water intake recommendations as a means to reduce the risk of chronic diseases such as cancer and cardiovascular and metabolic disorders (2, 3).

The U.S. Institute of Medicine has set general recommendations for adequate intake (AI) of approximately 2.7 litres and 3.7 litres of total water from all beverages and foods daily for women and men, respectively, but has not set an upper level for total water intake (3). Moreover, in the U.S. the AI for total water was set to 1.3 litres per day for children 1-3 years old, 1.7 litres per day for children 4-8 years old, 2.4 and 2.1 litres per day for 9-13-year-old boys and girls, respectively, and 3.3 and 2.3 litres per day for 14-18-year-old boys and girls, respectively (3).

The European Food Safety Authority (EFSA) set the AI of total water to 2.0 and 2.5 litres per day for adult women and men, respectively (4). The AI for total water per day was set to 0.8-1.0, 1.1-1.2, 1.3, and 1.6 litres per day for children aged 0.5-1, 1-2, 2-3, and 4-8 years, respectively. Furthermore, the daily AI for 9-13-year-olds was set to 2.1 litres for boys and 1.9 litres for girls. The recommended AI for children aged 14 years and older is similar to that of adults.

The EFSA also recommends an additional 0.3 litres of water per day for pregnant women (4). Lactating women increase their fluid intake in relation to the volume of breast milk that they produce. A volume of 750 mL per day of breast milk during the first six months increases the requirement for fluid by about 600-700 mL per day. This is generally compensated for by a self-regulatory increase in fluid intake of about 12-16% (6). The EFSA recommends that lactating women have the same daily AI of total water as non-lactating women plus an extra 0.7 litres (4). For elderly people whose capacity to concentrate the urine is limited and who often have impaired feelings of thirst, a broader safety margin might be needed. The EFSA, however, does not recommend a specific AI for total water intake among the elderly (4).

In NNR 2004 guiding values for daily intake of water and fluids, in addition to water derived from foods, were set to 1 litre for adults and children

and 1.5 litre for elderly. In NNR 2012 the guiding value for daily intake of drinking fluids for adults and children performing moderate physical activity and living under moderate temperate conditions is 1–1.5 litres of water in addition to the water derived from foods. Lactating women increase their fluid intake in relation to the volume of breast milk. A volume of 750 ml per day of breast milk during the first six months increases the requirement for fluid by about 600–700 ml per day. This is generally compensated for by a self-regulatory increase in fluid intake.

Lower and upper limits of intake

Mild dehydration – defined as a 1% to 2% loss of body weight due to fluid losses – can result in headache, fatigue, loss of appetite, and vertigo. Dehydration in excess of 3% to 5% of body weight can decrease endurance and strength and contribute to heat exhaustion (5, 6). Dehydration of 15% to 25% of body weight lost as water is fatal (7).

Acute water toxicity has been reported (8) due to rapid consumption of large quantities of fluids that greatly exceed the kidney's maximal excretion rate of 0.7–1.0 L/hour (4). Excessive ingestion of water can increase the risk of water intoxication and hyponatraemia during pregnancy (9). However, it is not possible to define a maximum daily amount of water that can be tolerated by a population group without taking into account individual and environmental factors (4).

Hydration status in relation to coffee and alcohol

Coffee is reported to increase 24-hour urine excretion in subjects with no habitual intake (10), while hydration status seemed unaffected in habitual coffee drinkers (11). Because the main diuretic compound in coffee and tea is caffeine, it appears that caffeine tolerance develops after habitual consumption but there is reportedly no basis for restricting caffeine consumption to avoid either dehydration or overhydration (12).

Alcohol (ethanol) has a diuretic effect by inhibiting the secretion of antidiuretic hormone, but moderate amounts of alcohol such as beer and wine appear to have little or no effect on hydration status (13).

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9 Physical activity

Recommended minimum physical activity in addition to normal daily activities.

	<i>Minutes per week</i>	<i>Intensity</i>
Adults	150 or	Moderate
	75	Vigorous
	<i>Minutes per day</i>	
Children and adolescents	60	Moderate to vigorous
All	Reduce sedentary behaviour	

Introduction

There is a lack of data for making direct comparisons of past and present levels of energy expenditure and physical activity among different populations, and differences in definitions of physical activity across studies usually preclude meta-analyses of the existing data. However, both the average weight and the percentage of women and men in the Nordic countries who are overweight/obese have increased in recent decades (1-4) even though energy intake in the adult population has remained relatively stable from the mid-1970s until 1997 (5;6). However, many nutritional studies are affected by under-reporting of energy-dense foods that are high in fat and sugar. Nevertheless, this suggests that the level of physical activity among Nordic populations has been decreasing. Furthermore, Church et al estimated that the average daily occupational-related energy expenditure has decreased by more than 100 kcal (420 kJ) over the last 50 years, and they suggested that this could account for a significant proportion of the average weight gain over the same period (7). This trend is likely due to structural changes in society that might have resulted in a decrease in overall physical activity in daily life. As a result, large segments of the population can be characterized as physically inactive. Indeed, objective

measurements of physical activity in both Sweden and Norway show that adults and older people spend the vast majority of their time being sedentary and that adherence to physical activity recommendations is low (8). However, trend data from high-income countries indicate that leisure-time physical activity has increased among adults while occupational physical activity has decreased (9).

The understanding of how physical activity and insufficient physical activity is associated with health outcomes has increased considerably over the past decades. Epidemiologic research, clinical interventions, and mechanistic studies have contributed to the evidence that physical activity is essential to preventing disease, improving health, and improving quality of life. The reference list in this chapter includes several key references but does not intend to cover the entire body of literature regarding the effects of physical activity.

Physical activity in the prevention of various diseases

The effect of insufficient physical activity on the global burden of major communicable diseases has been quantified (13). According to conservative estimates, insufficient physical activity causes 9% of premature mortality and more than 5 million deaths a year worldwide. The risk factor of being inactive is, therefore, similar to established risk factors such as smoking and obesity (13).

Cardiovascular disease, metabolic syndrome, and type 2 diabetes

Several studies have shown an inverse relationship between physical activity (14–19) or physical fitness (20–23) and coronary heart disease (CHD) in both genders and in different age groups. People who are sedentary run twice as great a risk of developing CHD as those who are physically active (24). This is probably an underestimation due to the dilution of relative risk (25). A study from Norway (26;27) found that women and men below the median peak oxygen uptake ($<35.1 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and $<44.2 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, respectively) were five and eight times more likely to have a cluster of cardiovascular risk factors compared to those in the highest quartile of peak oxygen uptake ($\geq 40.8 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and $\geq 50.5 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in women and men, respectively). Each $5 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ lower peak oxygen uptake corresponded to ~56% higher odds of cardiovascular risk factor clustering.

A study by Stensvold and colleagues (28) showed that individuals with

Box 9.1. Definitions

Physical activity is a comprehensive concept that encompasses many terms related to movement of the body. It is defined as any bodily movement achieved by contraction of skeletal muscles that increases energy expenditure (EE) above resting levels (10).

Physical inactivity is insufficient physical activity and is defined as a failure to meet the current recommendations.

Sedentary behaviour refers to any waking activity characterized by an energy expenditure ≤ 1.5 metabolic equivalents and a sitting or reclining posture (11). In general, this means that any time a person is sitting or lying down they are engaging in sedentary behaviour. Common sedentary behaviours include TV viewing, video game playing, computer use (collectively termed “screen time”), driving automobiles, and reading.

Exercise is any planned, structured, and repetitive bodily movement carried out to improve or maintain one or more components of physical fitness.

Physical fitness is a set of attributes related to the ability to perform physical activity and is something that people “have” or “strive to achieve” (12). The term includes cardiorespiratory fitness, strength, coordination, flexibility, etc.

Cardiorespiratory fitness relates to the ability of the circulatory and respiratory systems to supply and utilize oxygen during sustained physical activity (12).

MET (metabolic equivalent) is a unit used to estimate the metabolic cost (oxygen consumption) of physical activity. One MET equals the resting metabolic rate and corresponds to approximately $3.5 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

Light intensity activity is defined as activity corresponding to an EE between 1.5 to 3 METs such as standing or walking slowly ($<3.5 \text{ km/h}$).

Moderate intensity physical activity is defined as activity that requires three to six METs.

Vigorous intensity physical activity is activity requiring more than 6 METs (8;12).

Muscle-strengthening/resistance training is exercise designed to increase strength and power.

Endurance training is repetitive, dynamic use of large muscles (e.g. swimming, walking, or bicycling).

the metabolic syndrome (a clustering of risk factors for cardiovascular disease) had an increased risk of premature mortality from cardiovascular causes (hazard ratio 1.78, 95% CI 1.39–2.29) compared with the risk in healthy counterparts. Additionally, those with the metabolic syndrome who reported being highly active had about a 50% reduced risk of cardiovascular mortality compared to inactive individuals with metabolic

syndrome. The study also showed that, compared to insufficient physical activity, even low levels of physical activity were associated with reduced cardiovascular mortality.

There is sufficient evidence to clearly establish a dose-response association between physical activity and fitness and CHD morbidity and mortality (29;30). Paffenbarger et al demonstrated that those who had an extra energy expenditure on activities of at least moderate intensity corresponding to approximately 500-1000 kcal per week had a 22% lower mortality compared to a group who were sedentary (31). Leon et al. showed that people who were regularly physically active for 30 minutes a day during their leisure time, corresponding to an energy expenditure of 150 kcal (630 kJ), had a 36% lower risk of dying from CHD after adjustment for other important CHD risk factors (17). One study found that a weekly energy expenditure of 2000 kcal might represent a threshold, at least for the risk of heart attack in men (32). Interestingly, Lee et al. (33) showed that apparently healthy elderly men who exercised one to two times per week (so-called “weekend warriors”), had a ~60% lower risk of all-cause mortality compared with sedentary, apparently healthy men. In addition, a dose-dependent association has been indicated, suggesting an additional benefit among those who attain an even higher activity level (29). A Norwegian study (34) found that a single weekly bout of exercise of high intensity reduced the risk of cardiovascular death, both in men (~40%) and women (~50%), compared with those who reported no activity. In contrast to studies of male college graduates, in which mortality from ischaemic heart disease was gradually reduced with increasing energy expenditure from 500 to 3500 kcal per week (35), no additional benefits were found to be associated with as many as four high-intensity sessions per week compared with a single weekly bout (34).

Some studies have suggested that physical activity and cardiovascular fitness have independent effects on overall mortality (36;37), but these associations appear to be complex. In one recent study, Lee et al (38) found that the preventive effect of following the guidelines for physical activity was completely attenuated when adjusting for fitness. This meant that the protective effect was confounded by high or low levels of fitness. In contrast, Hein and colleagues found that men who were inactive and highly fit had similar mortality rates from ischaemic heart disease as men who were inactive and unfit, while men who were active and unfit were protected compared to those who were inactive and unfit (20). Although further studies are needed to examine the combined effects of activity

and fitness on morbidity and mortality and whether fitness modifies the association between activity and mortality, the scientific evidence to date is consistent in suggesting that being physically active provides protection against all-cause mortality and cardiovascular disease regardless of fitness level.

Physical activity/physical fitness and metabolic risk factors

Regular physical activity and high levels of physical fitness are favourably associated with plasma lipid levels (triglycerides, HDL-, and LDL-cholesterol) (39) (40) blood pressure (41), insulin sensitivity (42), haemostasis/fibrinolysis (39;43), and endothelial function (44). Increased physical activity has the potential to influence all of these factors in a favourable manner at the same time. The “effect size” and the amount of physical activity needed to improve these factors are not fully understood, but some data in this regard are available for plasma lipids, blood pressure, and insulin sensitivity.

The average expected changes in lipids and lipoproteins following exercise are an increase in HDL-cholesterol of 4.6%, a reduction in LDL-cholesterol of 3.7%, and a reduction in triglycerides of 5% (45). There is also evidence of a beneficial effect on LDL sub-classes (40). The baseline levels of these metabolic risk markers strongly influence the effect of physical activity, and greater beneficial effects are seen in those with poor lipoprotein profiles. The improvements are probably more related to the amount of activity and not to the intensity of the activity or to improvement in cardiorespiratory fitness (40).

A meta-analysis of randomised controlled trials has shown that the effect of exercise on systolic/diastolic blood pressure reduction is on average 3/2 mm Hg in normotensive and 8/6 mmHg in hypertensive individuals (41). Engaging in moderate intensity physical activity 3 to 5 times per week with a duration of 30–60 minutes appears to be effective in reducing blood pressure. There is strong scientific evidence that regular physical activity has a beneficial effect on insulin sensitivity (42;46). Prospective studies have shown that regular physical activity brings about a linear decrease in the age-adjusted risk of developing type 2 diabetes (47–49). Importantly, the protective effect is also independent of general and central adiposity (50). The decrease in risk is on the order of 6% for each 500 kcal expended in physical activity during weekly leisure time (49). It appears that those who are at greatest risk of developing type 2 diabetes benefit the most from regular physical activity (48).

Overweight and obesity

Physical activity has profound effects on body composition and metabolism. It increases EE and helps to maintain and increase muscle mass, and this might result in an increased basal metabolism and an increased capacity for mobilising and burning fat both while using the muscles and while resting (51;52). Thus, regular physical activity is likely to be of importance in long-term regulation of body weight. However, there is limited evidence of a prospective association between physical activity and later body weight, and the association might be bi-directional. Regular physical activity is important for obese people because health benefits can be achieved through improved physical fitness regardless of whether or not weight loss occurs (53). The mortality and morbidity related to being overweight are substantially reduced in people who, despite being overweight, are physically fit (30;54;55). However, in a systematic review by Fogelholm it was concluded that having a high body mass index (BMI), even with high levels of physical activity, was a greater risk factor for the incidence of type 2 diabetes and the prevalence of cardiovascular and diabetes risk factors than having a normal BMI with low levels of physical activity (54). Only in short-term studies (16 weeks or shorter duration) is it possible to find evidence of a linear dose-response relationship between the amount of physical activity and the amount of weight loss when diet is controlled for. The amount of weight loss is consistent with the excess energy expended (56). In practice, a weight loss of around 3 kg, with large individual variations, might be expected following increased physical activity in obese persons (57). Even though there is a lack of conclusive data, it seems that the amount of activity needed to avoid weight gain is about 60 minutes of moderate-intensity activity per day or a shorter duration if the activity is of vigorous intensity (58;59).

Cancer

Physical activity is an essential modifiable lifestyle risk factor that has the potential to reduce the risk of some major forms of cancer (13;60). The risk reduction for active individuals is 10-70% for colon cancer, but this is dependent on intensity and duration (61). With respect to breast cancer, regular physical activity corresponding to an intensity of 6 METs and with a duration of four hours per week might reduce the risk by 30-50% (62;63). Physical activity might also prevent the development of endometrial cancer (62-64). The evidence is weaker for lung and prostate cancers and is generally either null or insufficient for all remaining cancers (63;64).

There are several possible biological mechanisms through which physi-

cal activity might prevent cancer. They include, among others, the effect of physical activity on body composition and energy metabolism, insulin resistance, sex steroid hormones, inflammation, and immune function. In a review by Fridenreich et al, it was estimated that between 9% and 19% of cancer cases in Europe can be attributed to lack of sufficient physical activity (64). They also found that public health recommendations for physical activity and cancer prevention generally suggest 30–60 min of moderate- or vigorous-intensity activity performed at least 5 days per week. Recently, several observational studies, as well as some randomised clinical trials, have found that physical activity might improve survival in breast and colon cancer patients. However, the effects of physical activity on site-specific cancer survival have not yet been fully established.

Musculo-skeletal disorders

Reversible risk factors for falls include weak lower limb muscle strength, poor balance, and a poor level of overall physical fitness, all of which can be improved by regular physical activity (65–68). Muscle strength and muscle endurance diminish with increasing age and decreasing activity level (69), and physical activity can counteract and reverse this trend to a substantial degree and keep older people independent in daily life longer (66;70).

Loss of calcium can lead to osteoporosis. This risk increases with age, particularly in post-menopausal women. Physical activity contributes to increased bone density and can counteract osteoporosis, and physical activity immediately before and during puberty seems to yield greater maximum bone density in adult life (71–74). For adults and the elderly, physical activity retards bone loss (75). To be beneficial for bone mass and structure, exercise should preferably be weight-bearing (76) and repeated weight-bearing and loading, such as walking and running, is more beneficial than activities such as swimming and cycling. Even better for bone health are activities with high impacts (e.g. tennis, squash, and aerobics) or high volume loading (weight training). However, there is a lack of information about the dose-response relationship between activity/exercise and osteoporosis (76).

Exercises that strengthen and stabilize the muscles of the back reduce the incidence of back problems. This is particularly true in people with a history of back problems, but these exercises are also effective to a certain degree among those who have not previously experienced such problems (77). Regular physical activity might have a preventive effect on lower back pain, but the type of the activity that has the most benefit has yet to be determined (76).

Mental health and quality of life

A positive association has been found between physical activity habits and both self-esteem and psychological well-being in children and young and middle-aged adults (12). There is also evidence that regular physical activity reduces symptoms of anxiety and poor sleep. Furthermore, observational studies have shown that those who are physically inactive are at greater risk of developing depression than those who are physically active (78;79). However, there is not enough data to determine clear-cut dose-response relationships between physical activity and depression and anxiety (80). There is evidence supporting the hypothesis that physical activity can prevent the development of vascular dementia (81) compared to a sedentary lifestyle. Further research is needed to study the volume and mode of physical activity that is most psychologically beneficial and to explore the mechanisms through which physical activity improves mental health. For more details see http://www.health.gov/paguidelines/Report/pdf/G8_mentalhealth.pdf

Sedentary behaviour

Knowledge regarding the importance of reducing the amount of time spent sitting and engaging in daily physical activities has grown significantly in recent years. Several cross-sectional and prospective studies have demonstrated a relationship between sedentary behaviours, especially during leisure time, and obesity (81;82). Recently, prospective studies have also demonstrated a dose-response relationship between TV viewing and cardiovascular mortality as well as total mortality (83). Although residual confounding by unmeasured or poorly measured confounders (e.g. unconscious or poorly reported diet intake while viewing TV) cannot be excluded, these studies suggest that the association might be independent of physical activity level and exercise habits (82). Even in individuals fulfilling the recommendations for physical activity, sitting for prolonged periods might compromise metabolic health (81).

The underlying mechanisms are yet not fully known, but substantially decreased lipoprotein lipase activity as well as an instantaneously insulin-resistant state during sitting might contribute to adverse health effects (81). Energy expenditure differs substantially when comparing sitting still with standing, walking, or light intensity indoor activity (84), and a study from Australia showed that the frequency of breaks during prolonged sitting is associated with a favourable metabolic profile (85). Reducing sedentary time should be considered as an additional strategy in combination with the promotion physical activity

as a means of improving public health. Recommendations regarding reduced sedentary time are now being incorporated along with recommendations on physical activity in various countries, for example, the UK (gov.uk/government/publications/UK-physical-activity-guidelines).

Recommendations on physical activity

There is strong evidence that vigorous intensity physical activity that is sufficient to improve cardiorespiratory fitness has a major impact on different health outcomes at all ages (12). In fact, previous recommendations on physical activity were equal to the quantity and quality of exercise sufficient to develop and maintain cardiorespiratory fitness. However, as previously described in this chapter, clinical and epidemiological studies have established that activity of a moderate intensity, even without associated improvements in cardiorespiratory fitness, also has favourable effects on several risk factors for CHD and type 2 diabetes (12;86). Therefore, it is important to emphasize that substantial health gains can be achieved through moderate intensity physical activity. Nevertheless, evidence from large population-based studies in healthy individuals (34, 87) demonstrates that physical activity with high intensity gives more robust risk reduction compared to that achieved by physical activity at low and moderate intensities. These observations are in line with the cardiovascular adaptations observed after high-intensity endurance training compared to those observed after low- to moderate-intensity activities in small-scale randomized studies (88). Interestingly, Stanaway et al followed 1,705 men aged 70 years or older for a mean of 59.3 months and observed that men who normally preferred to walk faster than 3 km/h were 23% less likely to die compared with those walking at a slower speed during the follow-up period (89).

Examples of energy requirements corresponding to 3-6 METs (moderate intensity activity) and > 6 METs (vigorous intensity activity) are given in Table 9.1. Cardiorespiratory fitness decreases as people age and also as a consequence of insufficient physical activity. Activity of a certain MET value, therefore, requires a greater percentage of a person's cardiorespiratory fitness (Table 9.1.) as he or she ages. Note that activity of a certain energy cost might be perceived differently by different groups. For instance, climbing stairs might be perceived as a light intensity activity for a 30-year-old but hard for a 70-year-old.

Table 9.1. Energy requirements for performing various activities in different age groups shown as METs and as percentages of cardio-respiratory fitness (\approx maximal oxygen uptake)

Activities	Energy cost in METs	Energy requirements as percentages of cardiorespiratory fitness (\approx maximal oxygen uptake) and corresponding rating of perceived exertion (Borg scale, raised and in bold) according to age group in years.*			
		Young 20–39	Middle-aged 40–59	Old 60–79	Very old 80+
Watching TV/reading	1.3	10 <10	13 <10	15 <10	18 <10
Light household chores	2.5	20 <10	25 10–11	29 10–11	35 10–11
Driving a car	1.5	12 <10	15 <10	18 <10	21 <10
Moderate physical activity					
Climbing stairs	5.5	42 10–11	55 12–13	64 14–16	77 15–17
Walking (4.8 km/h)	3.5	27 10–11	35 10–11	41 10–11	49 12–13
Walking (6.4 km/h)	5.0	39 10–11	50 12–13	59 14–15	70 14–16
Snow clearing (snow blower)	3.0	23 <10	30 10–11	35 10–11	42 10–11
Snow clearing (manual)	6.0	47 12–13	60 14–16	70 14–16	84 15–17
Lawn mowing (manual)	4.5	35 10–11	45 12–13	53 12–13	63 14–16
Vigorous					
Jogging 8.0 km/h	7.0	55 12–13	80 14–16	93 17–19	>100 20

* Activity of a certain energy cost might be perceived differently by people both as a function of age and of insufficient physical activity. For instance, climbing stairs might be perceived as light activity for a 30-year-old but hard for a 70-year-old. Rating of perceived exertion (Borg scale)(63): Very light <10; Light 10–11; Somewhat hard 12–13; Hard 14–16; Very hard; 17–19; Very, very hard 20.

The total amount of physical activity (the combination of intensity, duration, and frequency) is related to a number of health variables in a dose-response relationship. The preventive effect (the health gain) increases with increasing activity level, but the relationship is curve-linear (Figure 9.1.). Those who are physically inactive might achieve the greatest health gains by increasing their physical activity, and this applies even in old age (12;16;90). The health gain seems to be dependent on the amount of physical activity, but the intensity of the aerobic physical activity might compensate for duration or frequency and provide further health benefits than moderate intensity alone as described above. Another aspect is whether several short bouts of activity are as effective in influencing health outcome as one longer session of the same total duration (91). Although aerobic physical activity is the type primarily recommended, data also show

that resistance training also have a protective effect on the incidence of CHD (92) and also all-cause mortality and cancer in men (93). It is recommended that regular resistance training involving the major muscle groups of the upper and lower body two or three times a week is sufficient to have an impact on health (94).

The question of how much physical activity is needed to improve health depends on initial health status and the group of interest: the young, the elderly, overweight individuals, etc. It is important, however, to keep in mind that physical activity might have different dose-response relationships with different health outcomes and that these effects might also be dependent on the type of activity.

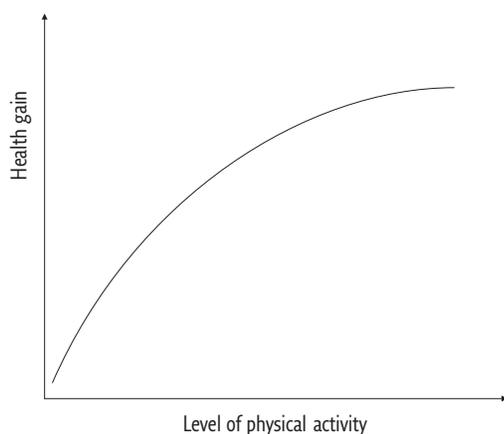


Figure 9.1. Dose-response curve for physical activity and health (95). Different health outcomes probably have different dose-response relationships

Children and adolescents

Regular physical activity is necessary for normal growth and the development of cardiorespiratory endurance, muscle strength, flexibility, motor skills, and agility (96–100). In addition, physical activity during the formative years strengthens the bones and connective tissues and yields greater maximum bone density in adult life (96;101;102). Physical activity that provides high impact loading on bones is important for bone development, particularly during early puberty (103). There is also evidence of an association between cardiorespiratory fitness and physical activity and cardiovascular disease risk factors in children and adolescents (27;61;104). Furthermore, risk factors such as fatness, insulin glucose ratio, and lipids

tend to cluster in children and adolescents with low cardiorespiratory fitness and low levels of physical activity (27;61;104).

Regular physical activity is associated with wellbeing and seems to promote self-esteem in children and adolescents. Furthermore, children and adolescents who are involved in physical activity seem to experience fewer mental health problems (105–108). There is no indication that increased physical activity in school represents any risk of impairing children's cognitive skills as a result of less time for theoretical school subjects (109). However, a higher fitness level in young adults is associated with better cognitive function and higher future educational level (110).

There is convincing evidence regarding the health effects of regular physical activity in children and adolescents (111). Recent literature reviews have prompted the WHO and the U.S. health authorities to refine their recommendations of physical activity guidelines for children (112–115). The following is recommended for children and adolescents:

1. *Children and adolescents should accumulate at least 60 minutes of moderate to vigorous-intensity physical activity daily.*
2. *Physical activity of amounts greater than 60 minutes daily will provide additional health benefits.*
3. *Vigorous-intensity activities should be incorporated, including those that strengthen muscle and bone, at least 3 times per week.*
4. *Reduce sedentary behaviour*

Activities should be as diverse as possible in order to provide optimal opportunities for developing all aspects of physical fitness including cardiorespiratory fitness, muscle strength, flexibility, speed, mobility, reaction time, and coordination. Varied physical activity provides opportunities to develop both fine-motor and gross-motor skills. Active children get the exercise they need while playing in the neighbourhood, at day-care, and on the school playground and by participating in children's sports.

In NNR 2012, recommendations for children and adolescents are identical to those of the WHO (112). The WHO also recommends that inactive children and youth undergo a progressive increase in activity to eventually achieve the recommendations mentioned above. Also, the WHO states that the recommended levels of physical activity for children and adolescents should be above and beyond the physical activity accumulated in the course of normal daily non-recreational activity.

Adults

The evidence in the literature suggests that adults who are insufficiently physically active gain considerable health benefits from participating in moderate to vigorous intensity physical activity for about 30 min per day. The optimal health effects are likely to be achieved from the combination of two modalities including at least 75 minutes of vigorous intensity physical activity per week and daily moderate intensity physical activity (see Figure 9.2.). Based on those mentioned above and other international guidelines (112) (113) (116), the recommendations on physical activity for adults are the following:

- 1. Adults should engage in at least 150 minutes of moderate-intensity physical activity throughout the week or engage in at least 75 minutes of vigorous-intensity physical activity throughout the week or engage in an equivalent combination of moderate- and vigorous-intensity activity preferably spread out over most days during the week.*
- 2. Physical activity should be performed in bouts of at least 10 minutes duration.*
- 3. For additional health benefits, adults should increase their moderate-intensity physical activity to 300 minutes per week or engage in 150 minutes of vigorous-intensity physical activity per week or engage in an equivalent combination of moderate- and vigorous-intensity activity.*
- 4. Muscle-strengthening activities should be performed involving major muscle groups on 2 or more days a week.*
- 5. Reduce sedentary behaviour.*

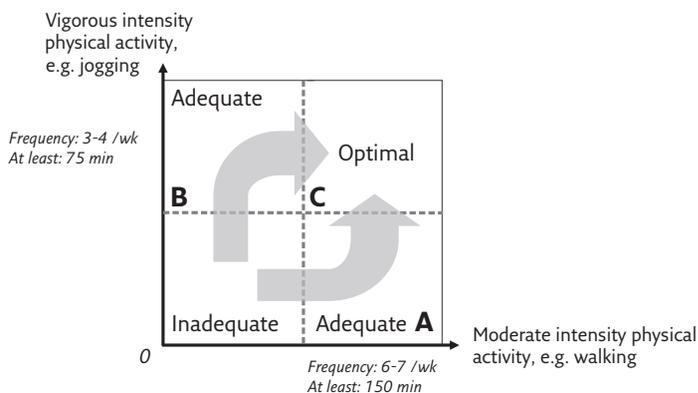


Figure 9.2. Three modalities of physical activity that are sufficient to provide health benefits: **A)** Physical activity of moderate intensity – e.g. walking, household chores, and playing – 6 or 7 times per week and for a minimum of 150 min per week **B)** Physical of vigorous intensity – e.g. jogging, swimming, tennis, resistance training, circuit training, and cross-country skiing – 3 or 4 times a week and for a total of 75 min per week **C)** The *optimal* activity dose might be the combination of A) and B) (both moderate intensity physical activity and moderate to vigorous intensity exercise)

Elderly

Regular physical activity in elderly people is associated with improved strength and functional ability (117), is inversely related to mortality (118), and has been found to be strongly associated with maintaining mobility during a 4-year follow up study (119).

Endurance training in the elderly has been found to improve oxygen consumption (VO_2 max) by approximately 23% in a meta-analysis (120). Hard endurance training results in improved VO_2 max, increased muscle mass, unchanged body weight, and unchanged daily energy expenditure because of a compensatory decline in physical activity during the remainder of the day (121;122).

Resistance training increases basal energy expenditure, muscle mass, muscle strength (90;123), and daily energy expenditure in the elderly (124) and might counteract the age-related accumulation of fat (125). Even engaging in high-resistance training less than 3 times per week still provides beneficial outcomes in the elderly (126). Low-intensity and moderate-intensity physical activity might be beneficial for the institutionalised elderly (127), and positive effects of resistance training have been seen even in 85- to 97-year-old subjects (128). In general, healthy elderly people are advised to follow the recommendations for the adult population. This applies especially to the advice to become more physically active in daily life.

The following recommendations apply:

1. *The elderly should engage in at least 150 minutes of moderate-intensity physical activity throughout the week or at least 75 minutes of vigorous-intensity physical activity throughout the week or engage in an equivalent combination of moderate- and vigorous-intensity activity preferably spread out over most days during the week.*
2. *Physical activity should be performed in bouts of at least 10 minutes duration.*
3. *For additional health benefits, the elderly should increase their moderate intensity physical activity to 300 minutes per week or engage in 150 minutes of vigorous-intensity physical activity per week or engage in an equivalent combination of moderate- and vigorous-intensity activity.*
4. *Adults of this age group with poor mobility should perform balance exercises to enhance balance and prevent falls on 3 or more days per week.*
5. *Muscle-strengthening activities should be performed involving major muscle groups on 2 or more days per week.*
6. *Reduce sedentary behaviour.*

When adults of this age group are unable to participate in the recommended amounts of physical activity due to health conditions, they should be as physically active as their abilities and conditions allow. The intensity can be increased by climbing stairs or hills of increasing steepness, preferably on uneven terrain (which is an advantage for improving balance). Other forms of aerobic exercise that can be engaged in as an alternative to walking include swimming and other water activities, dancing, cycling, rowing, and the use of equipment such as exercise bicycles, rowing ergometers, etc.

Because resistance training is particularly valuable in maintaining muscle strength, a varied, progressive programme of weight training is recommended for older people. Strengthening exercises should be tailored to the needs of the individual with regard to types of exercises, number of sets, number of repetitions, and frequency of training sessions. Strengthening exercises should optimally be combined with aerobic, balance, and mobility training.

Pregnancy and lactation

Pregnancy is associated with extensive physiological and anatomical changes. Despite this, regular physical activity or exercise has minimal risk and has confirmed benefits for most women (129). Women who are moderately physically active during pregnancy experience easier pregnan-

cies and deliveries, have better self-esteem, gain less weight, have more normal deliveries, and have fewer perinatal complications than women who have not engaged in physical activity during their pregnancy (130–132). Except for complicated pregnancies and a few circumstances in which exercise is contraindicated (see (129) for details), the following recommendations apply:

1. *Women who have previously not been physically active should engage in moderate intensity physical activity during pregnancy with a gradual progression of up to at least 150 minutes per week*
2. *Women who were regular exercisers before pregnancy should continue to engage in physical activity at an appropriate level.*
3. *Training the muscles of the pelvic floor is particularly important during pregnancy and after giving birth.*
4. *Activities with a high risk of falling (such as horseback riding and downhill skiing) and contact sports (such as handball, basketball, and ice hockey) increase the risk of trauma and should be avoided. Scuba diving should be avoided throughout the pregnancy.*
5. *There are no restrictions regarding the kind of activities that can be carried out during lactation.*
6. *Lactating women should be encouraged to be physically active as much as possible. This is especially important for overweight and obese lactating women and for women having gained more weight than recommended during pregnancy.*

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