

Nordic Nutrition Recommendations 2012

Integrating nutrition and physical activity

Part 4





Nord 2014:006

Nordic Nutrition Recommendations 2012

Part 4
Food, food patterns and health –
Guidelines for a healthy diet,
breastfeeding, sustainable food
consumption and dietary antioxidants

5th edition

Nordic Nutrition Recommendations 2012 · Part 4

Food, food patterns and health – Guidelines for a healthy diet, breastfeeding, sustainable food consumption and dietary antioxidants

ISBN 978-92-893-2681-0

<http://dx.doi.org/10.6027/Nord2014-002>

Nord 2014:006

ISSN 0903-7004

© Nordic Council of Ministers 2014

Layout and ebook production: [Narayana Press](#)

Cover photo: ImageSelect/Jette Koefoed

Typeface: Fresco Pro

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.

Nordic Council of Ministers

Ved Stranden 18

DK-1061 Copenhagen K

Phone (+45) 3396 0200

www.norden.org

Contents

Secretary General's Preface 7

Preface 9

Introduction 15

5 Food, food patterns and health outcomes – Guidelines for a healthy diet 19

4 Breastfeeding 53

6 Sustainable food consumption – Environmental issues 71

14 Dietary antioxidants 89

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.

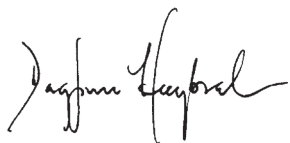
Finally, the NNR form part of the overall Nordic action plan *A better Life through Diet and Physical Activity*. In its aim to ensure the best-possible health for the population at large, this can be seen as an expression of the Nordic model, with its focus on an inclusive and holistic approach to society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

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.

Systematic reviews

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

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

Carbohydrates – including sugars and fibre experts: Emily Sonestedt, Sweden, Nina C Överby, Norway, Bryndis E Birgisdottir, Iceland, David Laaksonen, Finland.

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.

Peer reviewers: Jan I Pedersen, Norway, Ingibjörg Hardardottir, Iceland, Antti Aro, Finland, Jorn Dyerberg, Denmark, Göran Berglund, Sweden.

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

Peer reviewers: Margaretha Jägerstad and Jörn Schneede, Sweden.

Food based dietary guidelines experts: Lene Frost Andersen, Norway, Asa Gudrun Kristjansdottir, Iceland, Ellen Trolle, Denmark, Eva Roos and, Eeva Voutilainen, Finland, Agneta Åkesson, Sweden, Elisabet Wirfält, Sweden.

Peer reviewers: Inge Tetens, Denmark, Liisa Valsta, Finland, Anna Winkvist, Sweden.

Infants and children experts: Agneta Hörnell, Sweden, Hanna Lagström, Finland, Britt Lande, Norway, Inga Thorsdottir, Iceland.

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, Sjurður F Olsen, Denmark.

Peer reviewers: Bryndis E Birgisdottir, Iceland, Majjaliisa Erkkola, Finland, Ulla Hoppu, Finland.

Protein experts: Agnes N Pedersen, Denmark, Jens Kondrup, Denmark, Elisabet Börsheim, Norway.

Peer reviewers: Leif Hambraeus and Ingvar Bosaeus, Sweden.

Vitamin D experts: Christel Lamberg-Allardt, Finland, Magritt Brustad, Norway, Haakon E Meyer, Norway, Laufey Steingrimsdottir, Iceland.

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

Overviews

Alcohol experts: Anne Tjønneland and Janne Schurmann Tolstrup, Denmark.

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

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

Vitamin B₆, Vitamin B₁₂: Chapters revised by the NNR5 working group.

Thiamin, Riboflavin, Niacin, Biotin, Pantothenic acid: Hilary Powers, United Kingdom. Evaluation of need for revision. Revised by the NNR5 working group.

Vitamin K expert: Arja T Erkkilä, Finland. Peer reviewer: Sarah L. Booth, USA.

Dietary Antioxidants expert: Samar Basu, France. Peer reviewer: Lars Ove Dragsted, Denmark.

Vitamin A: Håkan Melhus, Sweden. Evaluation of need for revision. Chapter revised by the NNR5 working group.

Vitamin E expert: Ritva Järvinen, Finland. Peer reviewer: Vieno Piironen, Finland.

Vitamin C expert: Mikael Fogelholm, Finland. Peer reviewer: Harri Hemilä, Finland.

Phosphorus expert: Christel Lamberg-Allardt, Finland. Peer reviewer: Susan Fairweather-Tait, United Kingdom.

Magnesium, Zink, Manganese experts: Ingibjörg Gunnarsdottir, Iceland, Helle Margrete Meltzer, Norway. Peer reviewer Lena Davidsson State of Kuwait.

Chromium, Molybdenum experts: Ingibjörg Gunnarsdottir, Iceland, Helle Margrete Meltzer, Norway.

Copper expert: Susanne Gjedsted Bügel, Denmark Peer reviewer: Lena Davidsson, State of Kuwait.

Sodium as salt and Potassium expert: Antti Jula, Finland. Peer reviewer: Lone Banke Rasmussen, Denmark.

Selenium experts: Antti Aro, Finland, Jan Olav Aaseth and Helle Margrete Meltzer Norway. Peer reviewer: Susanne Gjedsted Bügel, Denmark.

Fluoride expert: Jan Ekstrand, Sweden. Peer reviewer Pia Gabre, Sweden.

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.

Use of NNR experts: Inge Tetens, Denmark, Agneta Andersson, Sweden.

Sustainable food consumption expert: Monika Pearson, Sweden.

Librarians

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

Jannes Engqvist, Sweden

Birgitta Järvinen, Finland
Sveinn Ólafsson, Iceland
Hege Sletsjøe, Norway

Steering group

Else Molander, chair, Denmark
Suvi Virtanen, Finland
Holmfrídur Thorgeirsdóttir, Iceland
Anne Kathrine O. Aarum, Norway
Irene Mattisson, Sweden

Reference group

Lars Johansson, Norway
Mairead Kiely, Ireland
Dan Kromhout, The Netherlands
Marja Mutanen, Finland
Hannu Mykkänen, Finland
Berndt Lindahl, Sweden
Susan Fairweather-Tait, United Kingdom
Lars Ovesen, Denmark
Dag Thelle, Norway

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

5 Food, food patterns and health outcomes – Guidelines for a healthy diet

Guidelines for a healthy diet

The current scientific evidence indicates that a micronutrient- and fibre dense dietary pattern should be adopted in order to promote the future health and wellbeing in Nordic populations.

The dietary pattern should include natural fibre-rich foods such as vegetables (e.g. dark-green leaves, fresh peas and beans, cabbage, onions, root vegetables, and fruiting vegetables), pulses, fruits, berries, nuts, seeds, and whole grains as well as fish and seafood, vegetable oils, vegetable oil-based fat spreads, and low-fat dairy products.

Such dietary patterns, especially if low in energy density and combined with physically active lifestyles, will reduce the risk of weight gain in the population. In contrast, dietary patterns characterized by high intakes of processed meat, red meat, and food products made from refined grains and sifted flour as well as those high in sugar, salt, and saturated and *trans*-fatty acids are associated with adverse health effects and chronic disease.

Introduction

Nutrition research has traditionally strived to identify the specific mechanisms, imbalances, and health impacts of single nutrients, but the 5th edition of the Nordic Nutrition Recommendations (NNR 2012) puts the whole diet in focus. Similar to previous editions, the 5th edition sets dietary reference values (DRVs) for individual nutrients, which are intended as a tool when planning diets for various population groups, assessing dietary intakes in the population, and formulating public health nutrition programs and policies. Most food items, however, contain many nutrients that interact with each other. Therefore, the concept of *food-based dietary guidelines* (FBDGs) was introduced by the FAO. FBDGs are defined as advice expressed at the food level that represents a ‘translation’ of energy and nutrient intake recommendations into foods and is aimed at the general population or specific population groups (1).

Non-communicable diseases are not simply caused by single nutrient imbalances, but are diseases with multifaceted aetiologies (2, 3). The search

for preventive measures against chronic disease, therefore, needs to take a broad approach. Over the past 15 to 20 years, a large number of observational studies and experimental trials have recognized the complexity of the diet and thus have focused on the impact of whole diets and of patterns of food consumption when examining diet-disease associations. Such an approach has resulted in a significant amount of new and original data.

The dietary habits in the Nordic countries have several common features, and food consumption trends tend to be similar. Some characteristics of these diets are an ample supply of milk and dairy products, moderate to high consumption of meat, and moderate consumption of vegetables and fruit. Consumption of fish is moderate to high overall, but lower in Denmark. Potatoes and cereal products are also consumed in moderate to high amounts. Cultural and culinary traditions differ, however, in terms of meal patterns, food choices, and traditional dishes and each Nordic country has developed and formulated national FBDGs.

Reports with a focus on the impact of food consumption on health that are relevant for Nordic countries include the extensive and systematic reviews (SRs) of the World Cancer Research Foundation/American Institute of Cancer Research WCRE/AICR (4, 5), the Norwegian comprehensive review of dietary guidelines for health (6), Danish reports on the consumption of fruits and vegetables, whole grains, and milk (7–9), a report on meat consumption from the Nordic council of Ministers (10), and the new Danish Dietary Guidelines (11). In addition, several systematic reviews (SRs) were undertaken to provide information on the health impact of food groups and food patterns in preparation for the 5th edition of the NNR (12–15).

Food sources of nutrients and other bioactive substances

Most foods contain a broad range of nutrients, with some exceptions such as refined sugar and household salt, and the distribution of nutrients differs across foods and food groups. Foods also contain a multitude of bioactive constituents other than nutrients that can affect the bioavailability, uptake, and metabolic response of nutrients. Diets are planned with the aim of promoting and maintaining optimal body function. A variety of common foods should be used in order to ensure that essential nutrients are provided as well as other food components for which human requirements have been less well defined. The descriptions of major food groups and their nutrient contributions given below are largely based on information provided in the Norwegian report of dietary guidelines for health (6).

Vegetables, fruits, and berries usually contain plenty of dietary fibre; vitamins such as ascorbic acid (vitamin C), carotenoids (pre-vitamin A), folate, tocopherol (vitamin E), and vitamin K; and minerals such as potassium and magnesium. Beans and peas are good sources of protein, minerals (iron, zinc, magnesium, and potassium), B-vitamins (except B₁₂), fibre, and starch. Nuts and seeds contain significant amounts of monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) as well as protein, magnesium, zinc, copper, potassium, vitamin E, vitamin B₆, niacin, and several antioxidants. Although the energy density of many plant foods is low, others such as nuts and seeds, olives, root vegetables, legumes, and cereals are comparatively energy dense. The nutrient concentration per weight unit might be comparatively low when the water content of vegetables, fruits, or berries is high.

Potatoes are comparatively rich in carbohydrates (starch), several minerals (such as potassium and magnesium), and vitamins such as vitamin C. Potatoes have traditionally been important sources of vitamin C and protein, but today fruits and vegetables are the most important sources of vitamin C and animal products provide most of our protein.

Whole grain is defined as intact grain (or cereal), and in processed whole grains the fractions of endosperm, bran, and germ are present in the same proportions as in the intact grain. Cereals are good sources of carbohydrates, such as the starch concentrated in the endosperm, and, therefore, are major sources of dietary energy. Whole grains also provide fibre, resistant starch, minerals (iron, zinc, phosphorous, and magnesium), vitamins (vitamin E, thiamine, riboflavin, niacin, and vitamin B₆), and phytochemicals (see below). Phytic acid in cereals can reduce the absorption of both iron and zinc. Prolonged fermentation of bread (e.g. sourdough) and germination of seeds can reduce this negative effect of phytic acid, and vitamin C (ascorbic acid) enhances the absorption of iron from plant foods.

Cereals are processed and manufactured into a variety of products including many different types of flour, breads, and pasta and in mixed and complex products such as breakfast cereals, baked goods and bread. Because micronutrients and other bioactive compounds are mostly found in the germ and bran fractions, refined cereal products (made from sifted flour) generally have lower nutrient content and also often contain higher amounts of added sugar, fat, and salt (see below).

All plant foods (including vegetables, beans and peas, root vegetables, fruits, berries, nuts and seeds, and whole grains) naturally contain a wide variety of phytochemicals such as polyphenols, salicylates, phytosterols, saponines, glucosinolates, monoterpenes, phytoestrogens, sulphides, terpenes, and lectins. Most of these have important functions in the plant cells and can also influence biological functions in the human body via a wide variety of mechanisms. Many are antioxidants with the potential to reduce oxidative stress, and others can influence signalling systems, cell cycles, repair systems, and inflammation reactions. The currently estimated number of bioactive phytochemicals is around 100,000 (6) and a single plant-based meal might provide around 25,000 different phytochemicals – albeit with comparatively small amounts of each. The observed health effects associated with vegetable, fruit, berry, and whole grain consumption can likely be explained by the combined action of many different phytochemicals and other nutrients.

Vegetable oils, margarine, vegetable oil-based fat spreads, and butter are used in cooking and with bread and by the food industry to produce foods such as mayonnaise, dressings, baked goods, and soups. Vegetable oils are manufactured by pressing oil from seeds or plants such as rapeseeds, sunflower seeds, flaxseeds, soya beans, olives, maize kernels, palm fruit, and coconuts. Margarine and fat spreads are mixtures of different vegetable oils and fats, and butter is made from the fat of cow's milk. Vegetable oils, vegetable oil-based fat spreads, and butter contain fat, and thus dietary energy, and fat-soluble vitamins such as vitamins A, D, E, and K. Vegetable oils and vegetable oil-based fat spreads also contain essential fatty acids. Vitamins A and D are usually added (regulated by legislation) to vegetable oil-based fat spreads. Vegetable oils contain 100% fat, but margarines and spreads contain varying amounts of fat. The fatty acids composition can vary considerably depending on the fat source used in manufacturing. Soybean, maize, and sunflower seed oils are rich in PUFA, and rapeseed oil and especially olive oil are rich in MUFA. Rapeseed and soybean oils have comparatively high content of omega-3 fatty acids. Vegetable oils and fats from marine sources, e.g. fish oils, contain more unsaturated fatty acids than fat from land-living animals, e.g. lard and tallow. However, palm and coconut oils have high contents of saturated fatty acids (SFA). Fish oils are generally rich in very long omega-3 PUFA. Butter and fat from ruminants (e.g. tallow) tend to have high contents of SFA and contain cholesterol. Butter and ruminant fat naturally contain 3% to 5% trans-fatty acids (TFA).

In the Nordic countries, the TFA content of margarines and vegetable oil-based fat spreads has decreased considerably during the last decades (to less than 1%) due to changes in raw materials and processing methods.

Fish and seafood contain 20%–35% protein. Lean fish such as cod, haddock, saithe, plaice, and pike contain less than 2 g of fat per 100 g, medium-fat fish such as winter-mackerel, halibut, catfish, and tuna contain 2–8 g of fat per 100 g, and fatty fish such as herring, summer-mackerel, trout, salmon, and eel contain more than 8 g of fat per 100 g. Medium-fat and fatty fish are the major dietary sources of the marine omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Fish also contain MUFA and SFA including odd-chain fatty acids (e.g. C15:0 and C17:0) (17, 18). Fatty fish are a major source of dietary vitamin D, and some lean fresh-water fish (e.g. pike-perch) also contain high amounts of vitamin D (19, 20). Fatty fish, and especially cod liver, contain high amounts of vitamin A (retinol). Fish and seafood are also good sources of vitamin B₁₂, iodine, and selenium. However, the nutrient content might vary between wild fish and farmed fish depending on the feed.

Fish and seafood can contain environmental toxins. In general, fish captured in the open sea have lower concentrations of pollutants than fish from the Baltic Sea or Norwegian fjords. Some marine fish (e.g. large tuna and halibut) and freshwater fish from certain areas might contain elevated levels of methyl mercury. Lean fish generally contain low levels of persistent organic pollutants (POPs). As a consequence the national food agencies of the Nordic countries have issued specific advice on fish consumption for specific population groups (i.e. children and women of fertile age).

Milk from ruminants is both a food in itself and a raw material for different dairy products such as cheese, butter, fermented milk, yoghurt, and cream. Milk and milk products are good sources of protein, fat, vitamin A, riboflavin, vitamin B₁₂, calcium, and iodine. Fat-soluble vitamins are often added to skim and low-fat milk. Two thirds of the fat in whole milk consists of SFA, and the major unsaturated fatty acid is oleic acid (C18:1). Milk also contains short-chain fatty acids and the odd-chain fatty acids C15:0 and C17:0 (21). The fat content varies from 0.1 g to around 4 g per 100 g, the protein content is about 3.0–3.5 g per 100 g, and the carbohydrate content (lactose) is about 4–5 g per 100 g. Whole milk and low-fat milk contain about the same amounts of calcium (120 mg per 100 g) and have the same proportions of fatty acids. Cheese has a high content of cal-

cium (750–940 mg per 100 g). Although milk products are generally good mineral sources, they usually contain very little iron (exceptions are whey products). Currently, several plant-based “milks” (e.g. those based on soy or rice) enriched with calcium, vitamin B₁₂, and vitamin D are available.

Eggs are high in protein, fat, riboflavin, vitamin A, and vitamin D relative to their energy content. The egg yolk contributes together with dairy products, meat, and fish to the dietary intake of cholesterol.

Meat from beef, pork, mutton, and game (e.g. reindeer and moose) is generally defined as “red” meat, and meat from chicken and turkey is defined as “white” meat. The term “processed meat” is defined by the WCRF/AICR as meats (usually red meats) preserved by smoking, curing, or salting or by the addition of preservatives (e.g. nitrites). Examples of such processed meats are ham, bacon, salami, different kinds of sausages, and smoked meat. Meat that is boiled, fried, dried, fermented, or frozen is usually not categorized as processed (4).

Meat and meat products contain 20%–35% protein and are usually good sources of vitamin B₆, vitamin B₁₂, iron, zinc, and selenium. The content of energy, fat, fatty acids, and salt can vary considerably between different types of meat. Fat content can vary from less than 1% to more than 40%. Also, the types of fatty acids vary between different animals depending both on species and feed; the typical proportion of SFA is 30% in chicken, 35%–40% in pork, and 40%–55% in beef and mutton. The level of TFA is less than 1% in chicken and pork, but 3%–5% in the meat of ruminants such as beef and mutton. The salt content is low in raw, unprocessed meat, but can be much higher in processed meat. Game meat usually has a lower fat content.

Alcohol (ethanol) is a toxic substance that is rapidly absorbed and distributed in the body and can influence all organs. Since the weight of alcohol is lighter than water (i.e., 1 litre of water equals 1000 grams, but 1 litre alcohol weighs 789 grams), the alcohol content of beverages is expressed in volume per cent (vol%). Alcoholic beverages have varied alcohol contents that range from 2–10vol% for beer to 10–15 vol% for wine to 30–60 vol% for liquor. In addition, alcoholic beverages can contain a wide variety of phytochemicals.

Non-alcoholic beverages

Coffee and tea contain stimulants such as caffeine but no macronutrients and, therefore, no energy. Pure juice made from fruits and berries is comparatively high in natural sugars (fructose) and contains most of the nutrients found in raw fruits and berries. Such juices, however, lack dietary fibre.

Breast milk

Breast milk provides infants with all nutrients, except vitamin D, in a combination that is efficiently absorbed. Breast milk also contains immune-related factors and hormonal factors that are important for infant health and growth. If breastfeeding is not possible or is not chosen, commercial infant formula prepared according to Codex standards is recommended (22). (For influences on health please see the chapter on breastfeeding).

Dietary supplements

Dietary supplements providing vitamins, minerals, protein, fatty acids, or non-essential nutritional factors derived from food can be purchased in all Nordic countries. These often include nutrients at doses similar to the recommended intake (RI), or even higher doses. A high intake of one nutrient, however, might disturb the bioavailability of other nutrients or be associated with other complications. Modern preparations of fish oil and fish liver oil are cleansed of high doses of vitamin A and pollutants. In Norway and Iceland, fish oil is classified as a food, not a supplement, and is a recommended source of the marine omega-3-fatty acids EPA and DHA and of vitamin D. It is not uncommon in the Nordic countries to advise the use of specific supplements at specific periods in life such as during pregnancy or for the frail elderly.

Characteristics of dietary patterns

Westernized dietary patterns (DP) are typically dense in energy and are characterized by high intakes of fat and SFA and processed and red meats. There is also a greater use of food products manufactured from refined cereals (sifted flour) and with added refined sugars, fat, and salt such as soft drinks, candy bars, desserts, sweet bakery goods, some highly sugared breakfast cereals and milk-products, deep-fried potatoes, savoury snacks, etc. In such products, the salt, fat, and sugar content is often disproportionate to the natural content of essential vitamins and minerals and to other

bioactive substances important for health. Especially substances found in plant foods that are naturally rich in fibre tend to be low in Westernized DP.

In contrast, the traditional diet of the Mediterranean region typically includes plant foods in abundance, fresh fruit, olive oil as the principal source of fat, pulses, cheese, yoghurt, fish, poultry, and wine consumed in low to moderate amounts. Such diets also include only small amounts of red meat. Data-driven food pattern studies have identified “prudent dietary patterns” (23, 24) that typically include plenty of plant foods and have characteristics similar to the Mediterranean-like diets. Biomarker studies have demonstrated that Westernized DP are associated with lower concentrations of micronutrients than the prudent patterns (25, 26).

The traditional diets of Nordic countries have lately been advocated as healthy alternatives to the Mediterranean-like diets (27, 28). Foods common across Nordic countries include whole-grain rye, oats, and barley, berries, fruits such as apples, pears, and plums, root vegetables, cabbages, onions, peas, beans, fish (e.g. herring), boiled potatoes, and dairy products and the use of rapeseed oil (29, 30). Although traditional Mediterranean and healthy Nordic diets exist in many varieties, both include large amounts of unrefined plant foods and are dense in micronutrients.

Most individuals today depend on food products supplied by the food industry, which over time has evolved into a complex global food production system. Food products are largely safe, tasty, nutritious, diverse, convenient, inexpensive, and readily accessible (31), but the identification of so-called unhealthy commodities (e.g. sugar-sweetened beverages) as major culprits in the worldwide spread of non-communicable diseases is of increasing concern (32). The imbalance of essential micronutrients in these foods is also a concern along with the potentially adverse health effects of other substances found in these foods. For instance, the health effects of TFA in processed foods have been recognized and documented over the last 10 to 15 years. In response, the food industry in the Nordic countries has changed raw materials and processing methods, and this has resulted in very low concentrations of TFA (close to zero) in most food products (33–38). Substances that still could be a concern are those added during the manufacturing process (e.g. nitrites in processed meat) or those formed during prolonged treatment at very high temperatures (e.g. deep-frying) such as heterocyclic amines, acryl amide, and advanced glycation/lipidoxidation end products.

Interestingly, studies within the EPIC (European Prospective Investigation into Cancer and Nutrition) cohorts report that the use of moderately

processed and non-processed foods is lower in Northern and Central European study centres compared to Mediterranean EPIC centres (39, 40). In these studies, the mean food intakes (from 24 hour recall data) were computed according to their degree of food processing (highly, moderately, or non-processed foods) using a specifically designed classification system (39). These studies also examined a biomarker of food processing (40).

The health impact of specific food groups

Because of the complexity of the diet, a search for the health effect of single nutrients might be misleading (41–43), and, therefore, an increasing number of studies are examining the link between food consumption (rather than nutrient intakes) and health outcomes. This section summarizes conclusions from comprehensive literature reviews regarding associations between food group intakes and the risk of major chronic diseases – including cardiovascular disease (CVD), type-2 diabetes, and cancer – and weight gain.

Vegetables, fruits, berries, and nuts

Prospective studies consistently conclude that high vegetable, fruit, and berry intakes are associated with reduced risk of CVD and lower levels of risk markers of CVD (6, 7). The comprehensive review by Mente et al (44), concluded that higher intake of vegetables and nuts was associated with strong evidence for protection against coronary heart disease (CHD) and myocardial infarction (MI) (44). Although the scientific evidence regarding different cancer types is less clear, the WCRF/AICR concludes that vegetables, fruits, and berries probably protect against most cancers in the gastric system and against lung cancer (4). Very few studies have examined the specific health influence of potato consumption (15). For instance, the evidence linking potato consumption to weight change is limited (13).

Whole grain

Prospective cohort studies indicate significant inverse associations between whole grain intakes and total risk of CVD, CHD, and stroke (6, 8). Prospective cohort studies also indicate protective associations between whole grain intakes and the risk of weight gain or obesity (13), and several larger cohort studies show convincing, protective associations between intake of whole-grain products and type-2 diabetes (6, 8).

The WCRF/AICR report of 2007 and the update report of 2011 (4, 5) both conclude that there is convincing evidence for a protective effect

of dietary fibre from plant foods on colorectal cancer risk. So far there is insufficient evidence for a direct link between whole grains and cancer.

Fish

Many reports indicate that there is convincing evidence for health benefits of replacing dietary SFA with unsaturated fat and PUFA from fish, nuts, seeds, vegetable oils, and vegetable oil-based fat spreads (6, 11). Several prospective cohort studies examining the direct health impact of fish consumption have concluded that fish reduce the risk of cardiovascular mortality, especially of MI and stroke (6, 11). The evidence seems clearer for secondary rather than for primary prevention (8, 45). A multicentre European randomized trial of young overweight adults found that fatty fish consumption was associated with reduced blood pressure (46) as well as improved insulin sensitivity (47). There is also possible evidence that fish consumption is related to reduced risks for type-2 diabetes, impaired cognitive function, and age-related macular degeneration (6).

A recent SR and meta-analysis of 21 cohort studies concluded that dietary marine n-3 PUFA was associated with reduced breast cancer risk (48). The WCRF/AICR concludes that there is limited-suggestive evidence that fish and foods containing vitamin D protect against colorectal cancer (4).

Milk

There is no convincing evidence that consumption of milk or dairy products is related to increased risk of CVD (6, 9, 15). Some reports indicate that milk consumption is related to a reduced risk of metabolic syndrome, type-2 diabetes, hypertension, and stroke (6, 9). However, a meta-analysis of long-term randomised controlled trials (RCTs) indicates that there is no beneficial effect on body weight and body fat loss by increasing dairy consumption without concomitant energy restriction (49).

The WCRF/AICR report (4) and the update report (5) concluded that milk consumption and high calcium intake probably reduce the risk of colorectal cancer. However, the 2007 report concluded that the consumption of diets high in calcium probably increases the risk for prostate cancer, while the evidence is weaker (limited-suggestive) for an association between milk and dairy products and increased risk of prostate cancer. No conclusion can be made on the link between milk and breast cancer. There is limited-suggestive evidence linking butter consumption to increased lung cancer risk.

Although nutrients important for bone health come from many foods,

there is probable evidence for an increased risk of osteoporosis with insufficient intakes of calcium and vitamin D, high alcohol intakes, low levels of physical activity, and low BMI (6, 11, 50).

Meat

Population studies consistently report that high consumption of processed meat is associated with an increased risk of type II diabetes and CHD (51–53). Similar but weaker associations were observed in a meta-analysis of red meat consumption (53). Replacing processed and red meat with vegetarian alternatives such as pulses or with fish and poultry was associated with a reduced risk.

The WCRF/AICR report of 2007 and the update report of 2011 (4, 5) both concluded that there is convincing evidence that high consumption of processed meat and red meat increases the risk of colorectal cancer. There is limited-suggestive evidence that foods containing animal fats are associated with increased colorectal cancer risk (4). There is also limited-suggestive evidence that processed and red meats are linked to other cancers (e.g. lung cancer). As a consequence, the WCRF has recommended that the consumption of processed meats should be reduced considerably, or avoided altogether, and that the consumption of red meat should be limited to an average intake of 500 g/week. A recent Nordic study examined the consequences on micro- and macronutrient intakes associated with reduction in processed and red meat according to the WCRF/AICR guidelines (10). That study concluded that the average consumption of red meat in the Nordic countries is currently at the comparatively low level recommended by the WCRF. However, in order to fulfil the WCRF recommendation for processed meat, a considerable reduction would be needed. The conclusion from the Nordic study was that a reduction of meat consumption would not have a detrimental impact on essential nutrient intakes in Nordic populations (10).

Alcohol

Light to moderate alcohol consumption has been associated with reduced risk of CVD and all-cause mortality in middle-aged and older subjects, whereas alcohol consumption among young adults is detrimental. High alcohol consumption is associated with increased risk of hypertension and stroke (11). High alcohol consumption is also convincingly associated with increased risks of several cancers such as those of the mouth, pharynx, larynx, and oesophagus (4). In women, there is convincing evidence for

an association between high alcohol consumption and increased breast cancer risk and probable evidence for increased colorectal cancer risk. In men, alcohol consumption is convincingly linked to increased colorectal cancer risk (4).

Sugar-sweetened beverages

A recent SR and meta-analysis of 30 RCT and 38 prospective cohort studies concluded that intake of free sugars and sugar-sweetened beverages (SSB) is a determinant of body weight in individuals with self-selected diets and that this effect is likely mediated via energy intake (54). Another SR and meta-analysis of 24 controlled intervention studies concluded that low to moderate doses (i.e. ≤ 100 g per day) of iso-caloric fructose in exchange for carbohydrates had no effect on cholesterol levels, but at high doses (> 100 g per day) blood levels of total and LDL cholesterol were significantly increased (55). In addition, a meta-analysis of eight prospective cohort studies concluded that high consumption of SSB was associated with increased risk of type 2 diabetes (56).

Energy density of food

There is limited-suggestive evidence linking total fat intake per se to increased risk of postmenopausal breast cancer and lung cancer (4). However, high fat intake as well as refined carbohydrates and sugars contribute to higher energy density. The WCRF/AICR report points out that because obesity and excessive body fat are major risk enhancers of many cancer types (including breast cancer), the low energy density of plant foods will likely have an indirect protective effect (4).

Three systematic reviews for the 5th edition of the NNR

Three SRs using the guidelines specifically prepared for the revision of the NNR (NNR5 working group (57)) were conducted and provided information on the health impacts of certain food choices relevant for the Nordic situation and health outcomes (12, 13, 15).

Five food groups

The SR of Nordic foods examined papers published from 2000 to 2010 to evaluate the scientific basis of dietary guidelines in relation to five food groups: potatoes, berries, whole grains, milk and dairy products, and red and processed meat (15). Out of the eligible abstracts, a total of 86 pub-

lished papers were extracted and quality graded and 64 papers were of sufficient quality for evidence grading. There was insufficient evidence to draw any conclusions regarding the health impact of potatoes and berries. Very few studies had examined the health impact of potatoes, especially boiled potatoes. Most of the identified studies of berries were conducted in North America and did not examine the types of berries consumed in Nordic countries. Also, there were still too few studies to draw a conclusion regarding red meat and processed meat intake and CVD risk. The end-point diversity in the reviewed studies contributed to the conclusion of insufficient evidence.

The review concluded that there was probable evidence (with a moderate evidence grade) for whole grains to be associated with protection against type-2 diabetes and against CVD, but only limited evidence for whole grains to protect against colorectal cancer. There was limited-suggestive evidence for total dairy consumption to be associated with decreased risk of type-2 diabetes. In contrast, there was suggestive evidence (with a low evidence grade) for total dairy consumption to be associated with increased risk of prostate cancer.

The papers identified regarding the association between red and processed meat and colorectal cancer were all reviewed in the WCRF/AICR report (4) and the update report in 2011(5). In line with these very detailed SRs, the NNR SR concluded that a high consumption of red and processed meat is a convincing cause of colorectal cancer. Because meat consumption is an important contributor to iron intake in Nordic populations, there is a concern that certain population groups might be at higher risk of iron deficiency if meat is not replaced by plant foods with sufficient mineral content (e.g. pulses/legumes). The NNR SR search identified only one relevant RCT. This study indicated an improvement in iron status among 12 to 20-month-old toddlers with increased intake of red meat (58), but more studies are needed to draw any conclusions.

Sugar

The second SR for the 5th edition of the NNR examined the effect of sugar intakes (SSB, sucrose, and fructose) on metabolic risk factors and related diseases (12). This review selected studies of adults from 2,743 potential abstracts. Out of 17 extracted studies of sufficient quality, 15 were prospective cohort studies and two were randomised controlled crossover trials. It was concluded from prospective cohort studies published in the years 2000 to 2011 that SSB probably increase the risk of type-2 diabetes.

However, too few studies were available to draw conclusions on other types of sugars or foods and on the links with related metabolic risk factors, CVD, or all-cause mortality. With respect to the incidence of type 2 diabetes, four of six prospective cohort studies found a significant positive association with SSB intake. Larger cohort studies with longer follow-up more often reported positive associations, and BMI seemed to mediate part of the increased risk.

Macronutrients, food, and weight maintenance

The third NNR SR examined prospective cohort, case-control, and intervention studies on the role of dietary macronutrient composition in predicting change in body weight or waist circumference in adults (13). This review also included comprehensive, albeit non-systematic, data on the associations between food consumption, dietary patterns, and weight change. The literature search covered studies published between the years 2000 and 2012. Out of 1,517 abstracts, 50 papers were extracted and quality graded. All data on food consumption and weight gain were taken from the 21 prospective cohort studies that were identified in this search. No conclusion could be made regarding the preventive role of the dietary proportion of macronutrients on weight regain after prior weight loss. Currently there is not enough evidence linking potato consumption to weight change (13). However, probable evidence was found for high intake of dietary fibre and nuts to predict less weight gain, and for high meat intake to predict more weight gain. Limited-suggestive evidence was found for whole grains, cereal fibre, high-fat dairy products, and prudent dietary patterns to protect against weight increase. Evidence was also limited-suggestive for dietary fibre and fruit intake to protect against larger increases in waist circumference. Similarly, plenty of fibre-rich foods and dairy products, and less refined grains, meat, and sugar-rich foods and beverages, were associated with less weight gain in prospective cohort studies. In contrast, there was limited-suggestive evidence for high intakes of refined grains, sweets, and desserts to predict weight gain, and for refined (white) bread and high energy density foods to predict larger increases in waist circumference.

Conclusions

These three NNR SRs concluded that red and processed meat is a convincing cause of colorectal cancer, but there is too little evidence to make a conclusion regarding red and processed meat and CVD (15). High intakes

of SSB probably increase the risk of type-2 diabetes (12), and whole grains probably protect against type-2 diabetes and CVD (15). There is limited-suggestive evidence that total dairy protects against type-2 diabetes but increases the risk of prostate cancer (both with low evidence grade) (15). No effect was seen for total dairy consumption and CHD risk, and there was no evidence for dairy consumption to increase the risk of breast cancer (15). There is probable-suggestive evidence that “prudent dietary patterns” rich in plants foods, fibre-rich foods such as whole grains, nuts, and dairy products protect against weight gain and a larger waist circumference, and that diets with large amounts of meat, refined grains, sweets, SSB, and desserts predict more weight gain and larger waist circumference (13).

Scientific evidence from Whole Diet Trials – “The power of food”

Nordic studies

In the late 1980s, a Danish trial was launched to examine the direct influence of the whole diet on risk markers of cardiovascular health (59). Young adults (18 men and 12 women) consumed a diet for eight months that was planned according to the NNR (3rd edition, 1989). The results indicated that changing diets from an average Danish diet to one in accordance with the NNR was associated with favourable changes in a range of CVD risk markers including continuous decreases in blood lipids and blood pressure, unintended decreases in body weight and fat mass, and favourable changes in the haemostatic system (59–61).

In later years, a range of experimental whole-diet trials, including multicentre collaborative projects (e.g. the SYSDIET and DiOGenes studies) have been launched. These are intervention trials to examine the health impact of dietary components commonly consumed in Nordic countries (e.g. the NORDIET and SYSDIET studies) (62) or of combinations of dietary components with specific biological activities (63). The DiOGenes study is a Pan-European study targeting issues related to the macronutrient composition of the diet in relation to obesity that takes genetic predisposition into account (64).

The SYSDIET study, which was one of three projects in the Nordic Centre of Excellence Programme on Food Nutrition and Health, was launched in 2007 (65, 66). This randomized controlled dietary trial examined the impact on insulin sensitivity, lipid profile, blood pressure, and inflammatory markers in adults over a period of 18 or 24 weeks (66). The experimental

diet was based on the NNR 2004 and included whole-grain products, berries, fruits, vegetables, rapeseed oil, vegetable oil-based margarines (i.e. > 2/3 of fat in the diet was unsaturated fatty acids), three fish meals per week, and low-fat dairy products. Key dietary components were provided to the participants, and an average Nordic diet was served as a control diet. Favourable and significant changes between the groups were found in blood lipid profiles and in markers of low-grade inflammation. With the iso-caloric diets, body weight remained stable and no changes were observed in insulin sensitivity or blood pressure.

Thus a range of intervention studies suggest that changing from an average diet to one planned according to the NNR and/or using healthy foods commonly found in Nordic countries is clearly associated with health benefits (59–66). Similarly, several prospective epidemiological studies have concluded that healthy Nordic dietary patterns are associated with important health benefits (29, 30, 67).

Two international trials

In preparation for the 5th edition of the NNR, an exploratory literature search was conducted to identify review articles of studies that had examined food or dietary patterns in relation to chronic disease that were published from 2000 to January 2011 (14). This search identified several review articles that described and discussed two large secondary prevention trials, both of which have received much international attention. An important design aspect of these two trials was that foods were provided to the study participants ensuring internal validity of the exposure and treatment (68).

The Lyon Heart Study – conducted in France in the late 1980s – was a randomized dietary trial in survivors of a first MI (69). Participants, men and women younger than 70 years of age, were carefully instructed to adopt a Mediterranean-like diet including more bread, more root and green vegetables, more fish, less meat (i.e. replace red meat like beef, lamb, and pork with poultry), and no days without fruit. Butter and margarine were replaced by margarine based on rapeseed oil, which was supplied by the study. The margarine content of SFA and MUFA was similar to that of olive oil (i.e. SFA made up 15% and MUFA made up 48% of the total fatty acids), and the content of linoleic acid (LA) was 2-fold (16.4%) higher and α -linolenic acid (ALA) was 8-fold (4.8%) higher. After 2 years, the experimental group had significantly less heart disease (in terms of both the number of MIs and deaths) compared to the control group, and after 4 years

the experimental group showed a 50%–70% reduction in heart disease. Thus the data confirmed the preventive effect of the Mediterranean-like diet on heart disease (69).

These observations were recently supported by a large ($n = 7447$) intervention study that examined the effects of different Mediterranean diets on cardiovascular disease (70). The participants were randomized into three groups: a Mediterranean diet supplemented with extra-virgin olive oil (i.e. enriched with MUFA and polyphenols), a Mediterranean diet supplemented with mixed nuts (i.e. enriched with different PUFAs and polyphenols), or a low-fat control diet. Total fat was close to 40% in the Mediterranean diets, whereas the control diet had both poorer quality (especially in terms of type of fatty acids) and a slightly lower proportion of total fat (37%). This resulted in similar total mortality in all three groups, but the incidence of cardiovascular events was significantly reduced (by about 28%–30%) with the Mediterranean diets compared to the control diet.

The Dietary Approaches to Stop Hypertension (DASH) feeding studies were a series of controlled trials carried out in the 1990s among US men and women aged 22 years or older. DASH was designed to test the effect of a whole-diet modification on blood pressure (BP) (43, 71–73). The DASH diet emphasized fruits, vegetables, and low-fat dairy products and included whole grains, poultry, fish, and nuts. The diet was also reduced in dietary fats (especially SFA), red meat, sweets, and SSB (74). The most effective diet was the reduced-salt DASH diet (74). This diet was not only rich in minerals such as potassium, magnesium, and calcium and in dietary fibre, but also reduced in both SFA and sodium. The result was a clear reduction in systolic BP both in normotensives and in hypertensives. Because each dietary factor only has a modest effect, the best interpretation of the impressive reduction is that multiple dietary factors influence BP and that the influence of a combination of factors can be substantial (72).

Since the time of the DASH studies, researchers in the US have designed and conducted several similar trials (75, 76). The conclusion from these studies is that well-balanced diets that meet the recommended intakes of minerals, vitamins, macronutrients, and micronutrients (as recommended by the US Food and Nutrition Board of the National Research Council, and US national health organizations (77, 78)) can improve risk factors of CVD (79). In addition, two recent SRs on prospective cohort studies have concluded that a diet following the DASH principles is associated with reduced incidence of type 2 diabetes (80) and CVD (81).

Systematic reviews of Food and Dietary Pattern studies

Epidemiological studies that construct food patterns or DPs have been designed to examine the impact of the whole diet on health. These studies include a combination of many food items, nutrients, and other food factors and have the potential to capture dietary factors that would be hard to detect in studies focusing on single components. The number of epidemiological studies that use DPs as the exposure variable to estimate disease risks has increased rapidly in the last 15 to 20 years. DPs are typically constructed either using data-driven statistical (*a posteriori*) or index (*a priori*) methodologies.

Data-driven statistical (*a posteriori*) methodologies

Factor analysis, principal component analysis, and cluster analysis are examples of data-driven methodologies that were originally developed in the social sciences to handle large datasets. When using these statistical methods with dietary data in epidemiological studies, researchers will obtain DPs that reflect the diets reported by the study participants. These patterns are typically a mix of many different foods and can contain foods both with and without health benefits.

Researchers conducting DP studies tend to label emerging patterns similarly, e.g. “prudent” and “Western” patterns. It should be noted, however, that even if the chosen labels are similar across studies, the food habits that exist in the examined populations, the methodologies used to assess diets, and the specific methodologies used to construct the patterns will all influence the emerging food patterns. Thus even if DPs largely appear similar across populations, details within the patterns might still vary considerably.

Index (*a priori*) methodologies

Dietary indices are typically constructed using the general dietary recommendations as a base. High scores of a dietary index will, therefore, reflect how well the examined individuals adhere to the recommendations. Because the basis of the *a posteriori* and *a priori* methodologies differ, the emerging patterns might share some characteristics but also show dissimilar features.

The Mediterranean diet index has been used in many studies. However, the index typically needs to be adjusted to every new population examined due to population-specific differences in food habits. Also, it should be noted that the scores obtained for the separate components of the index

commonly use the population medians as cut-offs to indicate adherence or non-adherence to the diet. Several food consumption differences exist between Northern European and Mediterranean countries, and these could influence the ability and efficiency of the Mediterranean diet index to rank individuals. For instance, because the population median, and the index cut-off, of vegetables and fruits is considerably lower in Northern European countries compared to the Mediterranean populations, it can be argued that this index is not able to assess adherence to a Mediterranean diet in Northern European populations. At best, a “Mediterranean-like diet” is reflected.

Three systematic reviews

The exploratory literature search of food patterns studies (14) identified three SRs that used independent reviewers and strict inclusion and quality assessment criteria in line with those issued for the revision of the NNR (NNR5 working group (57)).

Breast cancer. One SR and meta-analysis by Brennan et al. (82) examined DP studies using either factor analysis or principal component analysis in relation to the risk of breast cancer. This SR identified 16 articles published between 2001 and 2009 that defined and labelled DPs as “prudent/healthy”, “Western/unhealthy”, and/or “drinker” (i.e., DPs characterised by high/frequent consumption of alcoholic beverages). This review concluded that the prudent DP was associated with a significantly decreased risk of breast cancer. There was more heterogeneity among case-control studies with non-significant risk estimates, but less heterogeneity among prospective cohort studies with significant protective associations with the prudent DP. The drinker DP was significantly associated with increased risk of breast cancer, and no evidence of heterogeneity across studies was seen. The Western DP was associated with increased breast cancer risk in case-control studies, but no significant associations were seen in cohort studies. However, one cohort study using a diet-history methodology to assess diet (as opposed to a less extensive food-frequency questionnaire) showed significantly increased risk with the Western DP and decreased risk with the prudent DP (82).

CHD. The SR and meta-analyses of cohort studies and RCT on the associations between diet and CHD and MI by Mente et al. (44) evaluated studies according to four of the Bradford Hill criteria for causality: strength,

consistency, temporality, and coherence (44). This SR indicated strong evidence for a causal link (i.e., support from all four criteria) between DPs and CHD. Protective associations were observed with Mediterranean-like and high-quality DPs, but it should be noted that “high-quality” was not defined in the paper. In studies of high methodological quality, the evidence was strong both for protection of prudent DPs and for adverse effects of Western DPs. Beneficial effects of the Mediterranean-like diet were observed in RCTs (44). In addition, this SR found that some separate dietary factors were associated with strong evidence for protection, including higher intakes of vegetables, nuts, and MUFA. The evidence for harmful effects of higher intakes of TFA and of foods with high glycaemic index or glycaemic load was also strong.

Mediterranean-like diet patterns. A third SR and meta-analysis of prospective cohort studies by Sofi et al. (83) evaluated the effects of adherence to the Mediterranean-like diet (as assessed with population-specific index scores) on several health outcomes. Previous observations of convincing protective effects of the Mediterranean-like diet on all-cause mortality, mortality from and incidence of CVD, the risk of neoplastic diseases, and the occurrence of neurodegenerative diseases were confirmed, and no significant heterogeneity was observed across studies (83).

These SRs of prospective epidemiological studies that used independent reviewers and strict inclusion and quality assessment criteria indicate consistently that DPs high in vegetables, fruits, nuts, legumes, fish, vegetable oil, and low-fat dairy products (such as the prudent DP or the Mediterranean-like diets) are associated with decreased risk of chronic diseases such as breast cancer, CHD, and MI and of all-cause mortality. In contrast, Westernized DPs characterized by high fat (especially SFA), processed meats, refined grains, sifted flour, and sugar-rich products – and low in plants foods, whole grains, fish, and vegetable oils – are linked to increased risks of these diseases.

Implications of documented diet-disease risks

Based on the scientific evidence documented in the 5th edition of the NNR regarding associations between food and food patterns and risk of chronic disease, and the current situation in the Nordic countries, an overall micronutrient-dense DP can be identified with the potential to promote future health and wellbeing in Nordic populations. A combination of food

selection changes should be implemented to fulfil all aspects of dietary improvement.

Decrease energy density, increase micronutrient density, and improve carbohydrate quality

Diets dominated by naturally fibre-rich plant food (e.g. vegetables, pulses, fruits and berries, nuts and seeds, and whole grains) will generally be lower in energy and higher in micronutrients compared to diets dominated by animal food. The energy density is generally higher in food products high in fat and sugar (e.g. desserts, sweets, candy bars, cakes and biscuits, savoury snacks, some breakfast cereals, ice-cream, and some dairy products). Whole grains and whole-grain flour are rich in dietary fibre and have lower energy density compared to refined grains and sifted flour.

Limit sugar-sweetened beverages.

A limited consumption of SSB will contribute to an increased micronutrient density and a reduced intake of added sugars.

Improve dietary fat quality by balancing the fatty acid proportions

Fatty fish, nuts and seeds, avocados, olives, vegetable oils and vegetable oil-based fat spreads high in unsaturated fat should largely replace butter, high-fat meat, and meat products. A switch from high-fat to low-fat dairy will also improve the dietary fat quality while sustaining micronutrient density.

Replace processed and red meat with vegetarian alternatives such as pulses or with fish and poultry

A limited consumption of processed meat and red meat, and a switch from high-fat to low-fat red meat, will contribute both to an improvement of dietary fat quality and to lower energy density of the diet.

Limit the use of salt in food production and food preparation

Manufactured food products provide a large proportion of the total salt intake. A reduced salt intake can be achieved by choosing low-salt varieties and limiting the salt added during food preparation.

Table 5.1. Dietary changes that potentially promote energy balance and health in Nordic populations

Increase	Exchange	Limit
Vegetables Pulses	Refined cereals → Wholegrain cereals	Processed meat Red meat
Fruits and berries	Butter Butter based spreads → Vegetable oils Vegetable oil based fat spreads	Beverages and foods with added sugar
Fish and seafood	High-fat dairy → Low-fat dairy	Salt
Nuts and seeds		Alcohol

Vegetarian diets

Modern vegetarian diets come in many varieties (See Tables 5.2. and 5.4.). A vegan diet consists only of plant foods, and avoids all food products of animal origin. A vegetarian diet might include foods from living animals such as dairy and eggs. Other diets that exclude meat and meat products, but include fish and/or poultry are not strictly speaking vegetarian diets.

Individuals adopt a vegetarian diet for several different reasons including health, nutritional, ethical, religious, philosophical, environmental and economic concerns (84–86). However, there is a concern that modern vegetarian diets are not always selected with care. If so, and if certain food groups are consistently excluded from the diet, dietary imbalances can result that can lead to health problems.

A well planned vegetarian diet includes a variety of plant foods such as vegetables, fruits and berries, pulses, nuts and seeds, and whole grain cereals and generally provides high amounts of dietary fibre, folate, potassium, magnesium and antioxidants such as vitamin C, vitamin E, and β -carotene. Well planned vegetarian diets also include other health enhancing bioactive substances including phytochemicals such as phytoestrogens. A well-balanced lacto-ovo-vegetarian or lacto-vegetarian diet provides sufficient amounts of energy and essential nutrients for adults, including pregnant and lactating women, as well as children. The brief review *below* is mainly based on a report by the Swedish National Food Agency (87).

Table 5.2. Vegetarian diets can include, or exclude certain animal foods

	Plant foods	Dairy	Egg	Fish	Poultry
Lacto-vegetarian	•	•			
Ovo-vegetarian	•		•		
Pescitarian	•			•	
Pollotarian	•		•		•
Lacto-Ovo-vegetarian	•	•	•		
Lacto-Ovo-Pesci-vegetarian	•	•	•	•	

Health effects

Research studies have most often examined the health effects of the common lacto-ovo-vegetarian diet that is based on plant foods and includes both dairy products and eggs (87). In general, research indicates that vegetarian diets are associated with lower risk of chronic diseases such as CVD, type 2-diabetes, and obesity (88–92). In addition, vegetarians often have lower blood-lipid levels and lower blood pressure (90, 93), and are likely to live longer (89). Besides the dietary influence, it is probable that the overall lifestyle also contributes to a better health status among vegetarians because they often tend to be more health conscious and physically active, and less likely to smoke than non-vegetarians (89).

Nutritional considerations

If certain foods are consistently excluded from the diet, some nutrient intakes might be systematically lower compared to a mixed, conventional diet (Table 5.3.). This could result in deficient or inadequate intakes of essential micro-nutrients and other food components that are important for health. The content of some nutrients or bioactive constituents might, on the other hand, be higher or closer to current recommendations in vegetarian diets.

Protein

Pulses, whole grain cereals, nuts and seeds are good and Important sources of vegetable protein in all vegetarian diets. Other plant foods also provide protein, although in lower amounts. All essential amino acids are found

in plant foods, but the proportions are not as optimal as in animal foods. Protein is not a problem among vegetarian adults consuming varied diets, as long as energy needs are met and a variety of foods providing vegetable proteins are consumed. However, because the digestibility of vegetable protein is slightly lower compared to animal protein, individuals adopting vegan diets might require somewhat higher protein intakes.

Also, lectins found in many varieties of pulses could cause unfavourable health effects such as nausea, vomiting, bowel pain and diarrhoea if beans and peas are improperly cooked, or are consumed uncooked. Dried beans and peas should be soaked in water overnight, and boiled until soft (94).

Fat and fatty acids

Although, the content of SFA is usually lower in vegetarian diets compared to regular Westernized mixed diets, lacto-ovo-vegetarian diets can contain high amounts of SFA and cholesterol if high fat dairy products are used (89).

There are two essential fatty acids; α -linolenic (ALA) and linoleic acid (LA). Vegetarian diets often provide only limited amounts of long-chain omega-3 fatty acids. Therefore, a sufficient amount of the essential short chain omega-3 fatty acid ALA from plant foods needs to be included in the diet. The most important dietary source of ALA is rapeseed oil, and vegetable oil-based fat spreads made from rapeseed oil. Flax seed oil and camelina oil have a high content of ALA. Other good sources are also soybean oil, hempseed oil and walnuts. Eggs might contain some long-chain omega-3, depending on the feed content. The other essential omega-6 fatty acid LA is the most common fatty acid in nuts and seeds, and is usually included in sufficient amounts in most vegetarian diets.

Dietary fibre

A vegetarian diet based on naturally fibre-rich foods such as whole grain cereals, vegetables, pulses, nuts, fruits and berries, generally has a high and adequate content of dietary fibre. In contrast, with mixed diets based on animal foods an insufficient intake of dietary fibre is often a concern.

Vitamin D

Because vitamin D is produced in the skin when exposed to UV-light from the sun, the dietary supply is especially important at Nordic latitudes during the winter season. The vitamin is mainly found in fish, eggs (the yolk), and dairy products. It can also be found in some mushrooms. Vegan diets

need to be complemented with supplemental vitamin D all year round, whereas individuals following a vegetarian diet might need supplemental vitamin D in the winter season. Vegetable oil-based spreads are often enriched with vitamin D, and in Finland, Norway and Sweden fat-free and low-fat milk is enriched with the vitamin. This is not the case in Denmark and Iceland, although, some low-fat milk available is enriched with vitamin D. Some other food products might also contain added vitamin D since the introduction of voluntary fortification as per EU legislation in 2006.

Vitamin B₁₂

Vitamin B₁₂ is only found in animal foods. Thus; vegan diets need to be complemented with supplemental vitamin B₁₂. Studies have, however, shown that low vitamin B₁₂ status is not uncommon even among vegetarians with less strict diets (95). Therefore, all individuals consuming vegetarian diets should consider the use of vitamin B₁₂ supplements (see the chapter on vitamin B₁₂).

Riboflavin

Animal foods, especially dairy products are the major riboflavin sources in Nordic diets. If dairy products are excluded from vegetarian diets plenty of pulses, dark green leaves and wholegrain cereals should be included to provide sufficient riboflavin intake. Also, many plant milks (e.g. soy bean or rice based) are fortified with calcium and riboflavin.

Vitamin B₆

Meat, potatoes, fish and dairy products are major sources of vitamin B₆ in mixed conventional diets. In vegan diets dark green leaves, pulses, whole grain cereals, almonds, sesame seeds, wheat germs and yeast are important sources (see the chapter on vitamin B₆).

Calcium

Dairy products are major sources of calcium, but in vegan diets whole grain cereals, pulses, nuts, seeds and dark-green leaves are major contributors. Because the oxalic acid found in some dark green leaves can inhibit calcium absorption, a good variety of plants foods should be included in the diet.

Iron

The iron content of plants foods such as pulses, whole grain cereals, nuts, seeds and dark green leaves is comparatively high. Iron absorption is gen-

erally good when eating varied vegetarian diets, but less is absorbed compared to the iron ingested from meat (i.e., heme iron). Because vitamin C enhances the absorption of iron ingested from plants foods, a sufficient intake of vitamin C might ensure sufficient iron absorption (89).

Zinc

Wholegrain cereals, pulses, nuts and seeds are good sources of zinc, but as with iron absorption, zinc absorption from animal foods is better than from plant foods. Phytic acid in cereals can reduce both iron and zinc absorption. However, prolonged fermentation of bread (*e.g.* sour-dough) and germination of beans and seeds can potentially reduce the negative effect of phytic acid on absorption.

Selenium

The major sources of selenium in the Nordic countries are fish, meat, eggs and milk. Although, lentils, peas, chick peas and mushrooms are important plant food sources of selenium, excluding one or several animal product categories from the diet might lead to low selenium intake. In addition, the soil content of selenium is generally low in Nordic countries, and plant foods grown on such soils have low selenium contents. The selenium content of imported plant foods vary due to varying selenium levels in soil around the world. In Finland, selenium is added to fertilizers, and cereals grown there are, therefore, a good source of the mineral. It is also added to animal feed, which means that dairy products and eggs generally are good sources. However, this might not be the case for all organically produced products.

Iodine

Iodized salt, sea salt, fish, shell-fish, eggs and dairy products are important sources of iodine in the Nordic countries. Algae are often suggested as a vegetarian source of iodine, especially in vegan diets. However, because the content of iodine in algae vary considerably (some varieties might even contain toxic amounts), the iodine content of algae should be known before the food is consumed.

Infants and children

A vegetarian diet including dairy has generally been associated with adequate growth, and a well-balanced lacto-ovo-vegetarian diet is nutritionally adequate for the growing child (96). There are few studies on children adopting vegan diets, although the results of the available studies indicate a somewhat slower growth among some of the children (96, 97). Vitamin B₁₂-deficiency has been observed in breastfed infants born to mothers consuming vegan diets, and in children from families following such a DP. This emphasises the need for adequate supplementation in both mothers and children (98, 99). Thus, vegan, lacto-vegetarian and lacto-ovo-vegetarian diets should be able to satisfy the nutrient needs of infants, children, and adolescents and promote normal growth if they are appropriately planned (89), but vegan diets always need to be supplemented with vitamin B₁₂ and vitamin D.

Table 5.3. Nutritional recommendations in vegetarian diets

Type of diet	Critical nutrients	Recommendation
Vegan	Vitamin B ₁₂ Vitamin D	Supplements are needed
	Protein	Careful combination of pulses, whole grain, nuts and seeds
	Essential fatty acids; ALA	Search for sources of ALA
	Riboflavin Calcium, iron, zinc, selenium and iodine	Search for appropriate sources
Lacto-ovo vegetarian	Vitamin B ₁₂ Vitamin D Fat quality; ALA Iron, zinc, and selenium	Search for appropriate sources

Table 5.4. Modern diets can be defined in many different ways¹

General principles	
Vegan – veganism	“...a way of living that seeks to exclude, as far as possible and practicable, all forms of exploitation of, and cruelty to, animals for food, clothing and any other purpose”. ² Diets exclude all animal products and animal-derived substances and are based on fruits, legumes, grains and vegetables.
Macrobiotic ³	These diets typically consist of 50%-60% organically grown whole grains; 20%-25% locally, organically grown fruits and vegetables; and 5%-10% soups with vegetables, seaweed, grains, beans, and miso (fermented soy); The diets allow the occasional consumption of fresh white fish, nuts, seeds, pickles, Asian condiments, and non-stimulating and non-aromatic teas. These diets exclude all other animal products. The diets discourage consumption of coffee, sugar, stimulant and aromatic herbs and processed foods, some vegetables (potatoes, tomatoes, eggplants, peppers, asparagus, spinach, beets, zucchini, and avocados) and fruits not grown locally.
Fruitarian ⁴	Diet includes only dried or raw fruits, berries, nuts, seeds (e.g. cereals) and fruit vegetables (e.g. tomatoes, cucumbers, olives and avocados).
Live-Raw foodism ⁴	Diet consists of uncooked, unprocessed, often organic or wild foods and might include raw fruits, vegetables, nuts, seeds, sprouted whole grains, eggs, fish (e.g. sashimi), meat (e.g. carpaccio), and non-pasteurized/non-homogenized dairy products (e.g. raw milk, or cheese and yogurt made from raw milk).

¹ Depending on the specific restriction the impact on health and well-being can vary.

² The Vegan Society (<http://www.vegansociety.com>).

³ Source: the American Cancer Society (<http://www.cancer.org/>).

⁴ Information from Wikipedia.

References

1. FAO/WHO. Preparation and use of food based dietary guidelines. Geneva 1998.
2. Gerber M. The comprehensive approach to diet: a critical review. *J Nutr.* 2001;131(11 Suppl):3051S-5S.
3. Thomson CA, Thompson PA. Dietary patterns, risk and prognosis of breast cancer. *Future Oncol.* 2009;5(8):1257–69.
4. Food, Nutrition, Physical Activity and the prevention of Cancer: a Global Perspective. Washington, DC: World Cancer Research Fund / American Institute for Cancer Research 2007.
5. Food, Nutrition, Physical Activity and the prevention of Cancer: a Global Perspective. Second Expert report (Continuous updates). Washington, DC: World Cancer Research Fund /American Institute for Cancer Research 2011.
6. Nasjonalt råd for ernæring. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer. Metodologi og vitenskapelig kunnskapsgrunnlag. Oslo, Norway: Helsedirektoratet, Avdelningen for nasjonalt folkehelsearbeid 2011.
7. Hallund J, Dragsted L, Halkjær J, Madsen C, Ovesen L, Højgaard Rasmussen H, et al. Frugt, grøntsager og sundhed Opdatering af vidensgrundlaget for mængdeanbefalingen 2002–2006. Søborg: Fødevareinstituttet DTU, Avdelningen for Ernæring 2007.
8. Mejbom H, Biltoft-Jensen A, Trolle E, Tetens I. FULDKORN Definition og vidensgrundlag for anbefaling af fuldkornsindtag i Danmark. Søborg: DTU Fødevareinstituttet, Avdelningen for Ernæring 2008.
9. Beck A, Hoppe C, Hess Ygil K, Lyhne Andersen N, Pedersen A. Vidensgrundlag for rådgivning om indtag af mælk, mælkeprodukter og ost i Danmark, 2010. Søborg: DTU Fødevareinstituttet, Avdelningen for Ernæring 2010.

10. Tetens I, Hoppe C, Frost Andersen L, Anni Helldán, Warensjö Lemming E, Trolle E, et al. Nutritional evaluation of lowering consumption of meat and meat products in the Nordic context. Copenhagen: Nordic Council of Ministers 2013.
11. Tetens I, Andersen L, Astrup A, Holmboe Gondolf U, Hermansen K, Jakobsen M, et al. Evidensgrundlaget for danske råd om kost og fysisk aktivitet.: Report for DTU Fødevarerinstitutionen, Afdelingen for Ernæring (Søborg) 2013.
12. Sonestedt E, Overby N, Laaksonen D, Birgisdóttir B. Does high sugar consumption exacerbate cardiometabolic risk factors and increase the risk of type 2 diabetes and cardiovascular disease? *Food Nutr Res.* 2012;56.
13. Fogelholm M, Anderssen S, Gunnarsdóttir I, Lahti-Koski M. Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review. *Food Nutr Res.* 2012;56.
14. Wirfalt E, Drake I, Wallstrom P. What do review papers conclude about food and dietary patterns? *Food Nutr Res.* 2013;57.
15. Akesson A, Andersen LF, Kristjansdóttir AG, Roos E, Trolle E, Voutilainen E, et al. Health effects associated with foods characteristic of the Nordic diet: a systematic literature review. *Food Nutr Res.* 2013;57.
17. Copeman L, Parrish C. Lipids Classes, Fatty Acids, and Sterols in Seafood from Gilbert Bay, Southern Labrador. *J Agric Food Chem.* 2004;52:4872–81.
18. Özogul Y, Özogul F, Cicek E, Polat A, Kuley E. Fat content and fatty acid compositions of 34 maries water fish species from the Mediterranean Sea. *International Journal of Food Science and Nutrition.* 2009;60(6):464–75.
19. Fineli [database on the Internet]. Available from: www.fineli.fi
20. Sök näringsinnehåll i livsmedel [database on the Internet]. Available from: www7.slv.se/Naringssok/
21. Saadatian-Elahi M, Slimani N, Chajès V, Jenab M, Goudable J, Biessy C, et al. Plasma phospholipid fatty acid profiles and their association with food intakes: results from a cross-sectional study within the European Prospective Investigation into Cancer and Nutrition. *Am J Clin Nutr.* 2009;89(1):331–46.
22. Hörnell A, Lagström H, Lande B, Thorsdóttir I. Breastfeeding, introduction of other foods and effects on health: A systematic literature review for the 5th Nordic Nutrition Recommendations. *Food and Nutrition Research.* 2013;57:208–23.
23. Slattery ML, Boucher KM, Caan BJ, Potter JD, Ma KN. Eating patterns and risk of colon cancer. *American Journal of Epidemiology.* 1998;148(1):4–16.
24. Hu F. Dietary pattern analysis: a new direction in nutritional epidemiology. *Current Opinion in Lipidology.* 2002;13(1):3–9.
25. Kant A. Dietary patterns: biomarkers and chronic disease risk. *Appl Physiol Nutr Metab.* 2010;35(2):199–206.
26. Román-Viñas B, Ribas Barba L, Ngo J, Martínez-González MA, Wijnhoven TM, Serra-Majem L. Validity of dietary patterns to assess nutrient intake adequacy. *Br J Nutr.* 2009;101(Suppl 2):S12–20.
27. Bere E, Brug J. Towards health-promoting and environmentally friendly regional diets – a Nordic example. *Public Health Nutrition.* 2009;12(1):91–6.
28. Mithril C, Dragsted L, Meyer C, Blauert E, Holt M, Astrup A. Guidelines for the New Nordic Diet. *Public Health Nutrition.* 2012 Jan 17:1–7.
29. Olsen A, Egeberg R, Halkjær J, Christensen J, Overvad K, Tjønneland A. Healthy aspects of the Nordic diet are related to lower total mortality. *Journal of Nutrition.* 2011;141(4):639–44.
30. Kanerva N, Kaartinen N, Schwab U, Lahti-Koski M, Männistö S. Adherence to the Baltic Sea diet consumed in the Nordic countries is associated with lower abdominal obesity. *British Journal Nutrition.* 2012;1–9.
31. Floros J, Newsome R, Fisher W, Barbosa-Canovas G, Chen H, Dunne C, et al. Feeding the World Today and Tomorrow: The Importance of Food Science and Technology. *Comprehensive Reviews in Food Science and Food Safety.* 2010;9(5):572–99.

32. Moodie R, Stuckler D, Monteiro C, Sheron N, Neal B, Thamarangsi T, et al. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. *Lancet*. 2013;381:670–79.
33. Korver O, Katan MB. The Elimination of Trans Fats from Spreads: How Science Helped to Turn an Industry Around. *Nutrition Reviews*. 2006;64(6):275–9.
34. Pietinen P, Paturi M, Reinivuo H, Tapanainen H, Valsta LM. FINDIET 2007 Survey: energy and nutrient intakes. *Public Health Nutr*. 2010 Jun;13(6A):920–4.
35. Darnerud PO, Törnkvist A. Market Basket 2010 – chemical analysis, exposure estimation and health-related assessment of nutrients and toxic compounds in Swedish food baskets Uppsala: Livsmedelsverket 2012. Report No.: 7 – 2012.
36. Utviklingen i Norsk Kosthold. Matforsyningsstatistikk og Forbruksundersøkelser. Helsedirektoratet 2011.
37. Ærendahl Miikkelsen A, Bysted A, Langkilde S. Transfedtsyrer i udvalgte fødevarer: Danmarks Tekniske Universitet, Fødevareinstituttet 2011. Report No.: projekt 2009–20–64–00151.
38. Wallin L, Wretling A, Mattisson I. Transfettsyror i kakor/kex och chips –märkning och halter (In Swedish) Uppsala 2009. Report No.: 18.
39. Slimani N, Deharveng G, Southgate D, Biessy C, Chajès V, van Bakel M, et al. Contribution of highly industrially processed foods to the nutrient intakes and patterns of middle-aged populations in the European Prospective Investigation into Cancer and Nutrition study. *Eur J Clin Nutr*. 2009;63(Suppl4):S206–25.
40. Chajès V, Biessy C, Byrnes G, Deharveng G, Saadatian-Elahi M, Jenab M, et al. Ecological-level associations between highly processed food intakes and plasma phospholipid elaidic acid concentrations: results from a cross-sectional study within the European prospective investigation into cancer and nutrition (EPIC). *Nutrition and Cancer*. 2011;63(8):1235–50.
41. Slattey M. Analysis of dietary patterns in epidemiological research. *Appl Physiol Nutr Metab*. 2010;35(2):207–10.
42. Jacobs Jr DR, Tapsell LC. Food, Not Nutrients, Is the Fundamental Unit in Nutrition. *Nutrition Reviews*. 2007;65(10):439–50.
43. Appel LJ. Dietary patterns and longevity: expanding the blue zones. *Circulation*. 2008;118(3):214–5.
44. Mente A, de Koning L, Shannon H, Anand S. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. *Arch Intern Med*. 2009 Apr 13;169(7):659–69.
45. Turunen A, Verkasalo P, Kiviranta H, Pukkala E, Jula A, Männistö S, et al. Mortality in a cohort with high fish consumption. *Int J Epidemiol*. 2008;37(5):1008–17.
46. Ramel A, Martinez J, Kiely M, Bandarra N, Thorsdottir I. Moderate consumption of fatty fish reduces diastolic blood pressure in overweight and obese European young adults during energy restriction. *Nutrition*. 2010;26(2):168–74.
47. Ramel A, Martínéz A, Kiely M, Morais G, Bandarra N, Thorsdottir I. Beneficial effects of long-chain n-3 fatty acids included in an energy-restricted diet on insulin resistance in overweight and obese European young adults. *Diabetologia*. 2008;51(7):1261–8.
48. Zheng J, Hu X, Zhao Y, Yang J, Li D. Intake of fish and marine n-3 polyunsaturated fatty acids and risk of breast cancer: meta-analysis of data from 21 independent prospective cohort studies. *British Medical Journal*. 2013;346:f3706.
49. Chen M, Pan A, Malik V, Hu F. Effects of dairy intake on body weight and fat: a meta-analysis of randomized controlled trials. *Am J Clin Nutr*. 2012;96(4):735–47.
50. Osteoporos – prevention, diagnostik och behandling. Stockholm, Sverige 2003.
51. Männistö S, Kontto J, Katja-Tuomola M, Albanes D, Virtamo J. High processed meat consumption is a risk factor of type 2 diabetes in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention study. *Br J Nutr*. 2010;103(12):1817–22.

52. Fretts A, Howard B, McKnight B, Duncan G, Beresford S, Mete M. Associations of processed meat and unprocessed red meat intake with incident diabetes: the Strong Heart Family Study. *Am J Clin Nutr*. 2012;95(3):752–8.
53. Micha R, Michas G, Mozaffarian D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes--an updated review of the evidence. *Curr Atheroscler Rep*. 2012;14(6):515–24.
54. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomized controlled trials and cohort studies. *British Medical Journal*. 2012;346:e7492.
55. Zhang Y, An T, Zhang R, Zhou Q, Huang Y, Zhang J. Very high fructose intake increases serum LDL-cholesterol and total cholesterol: A meta-analysis of controlled feeding trials. *Journal of Nutrition*. 2013;143(9):1391–8.
56. Malik V, Popkin B, Bray G, Depres J, Willett W, Hu F. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care*. 2010;33:2477–83.
57. NNR5 working group. A guide for conducting Systematic Literature Reviews for the 5th edition of the Nordic Nutrition Recommendations. Revised ed. Copenhagen: Nordic Council of Ministers 2011.
58. Szymlek-Gay E, Ferguson E, Heath A-L, Gray A, Gibson R. Food-based strategies improve iron status in toddlers: a randomized controlled trial. *American Journal of Clinical Nutrition*. 2009;90(6):1541–51.
59. Sandström B, Marckmann P, Bindeslev N. An eight-month controlled study of a low-fat high-fibre diet: effects on blood lipids and blood pressure in healthy young subjects. *Eur J Clin Nutr*. 1992;46(2):95–10.
60. Raben A, Jensen N, Marckmann P, Sandström B, Astrup A. Spontaneous weight loss during 11 weeks' ad libitum intake of a low fat/high fiber diet in young, normal weight subjects. *Int J Obes Relat Metab Disord*. 1995;19(12):916–23.
61. Marckmann P, Sandström B, Jespersen J. Favorable long-term effect of a low-fat/high-fiber diet on human blood coagulation and fibrinolysis. *Arterioscler Thromb*. 1993;13(4):505–11.
62. Adamsson V, Reumark A, Fredriksson I, Hammarström E, Vessby B, Johansson G, et al. Effects of a healthy Nordic diet on cardiovascular risk factors in hypercholesterolaemic subjects: a randomized controlled trial (NORDIET). *J Intern Med*. 2011;269(2):150–9.
63. Tovar J, Nilsson A, Johansson M, Ekesbo R, Aberg A, Johansson U, et al. A diet based on multiple functional concepts improves cardiometabolic risk parameters in healthy subjects. *Nutr Metab (Lond)*. 2012;2(9):29.
64. Gögebakan O, Kohl A, Osterhoff M, van Baak M, Jebb S, Papadaki A, et al. Effects of weight loss and long-term weight maintenance with diets varying in protein and glycemic index on cardiovascular risk factors: the diet, obesity, and genes (DiOGenes) study: a randomized, controlled trial. *Circulation*. 2011;124(25):2829–38.
65. de Mello V, Schwab U, Kolehmainen M, Koenig W, Siloaho M, Poutanen K, et al. A diet high in fatty fish, bilberries and wholegrain products improves markers of endothelial function and inflammation in individuals with impaired glucose metabolism in a randomised controlled trial: the Sysdimet study. *Diabetologia*. 2011;54(11):2755–67.
66. Uusitupa M, Hermansen K, Savolainen MJ, Schwab U, Kolehmainen M, Brader L, et al. Effects of an isocaloric healthy Nordic diet on insulin sensitivity, lipid profile and inflammation markers in metabolic syndrome -- a randomized study (SYSDIET). *J Intern Med*. 2013 Jul;274(1):52–66.
67. Drake I, Gullberg B, Sonestedt E, Wallström P, Persson M, Hlebowicz J, et al. Scoring models of a diet quality index and the predictive capability of mortality in a population-based cohort of Swedish men and women. *Public Health Nutrition*. 2012;29:1–11.
68. Tucker K. Dietary patterns, approaches, and multicultural perspective. *Appl Physiol Nutr Metab*. 2010;35(2):211–8.
69. de Lorgeril M, Salen P. The Mediterranean-style diet for the prevention of cardiovascular diseases. *Public Health Nutr*. 2006;9(1A):118–23.

70. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013 Apr 4;368(14):1279–90.
71. Appel L, Moore T, Obarzanek E, Vollmer W, Svetkey L, Sacks F, et al. A Clinical trial of the Effects of Dietary Patterns on Blood Pressure. *N Engl J Med*. 1997;336(16):1117–24.
72. Appel LJ. Lifestyle modification as a means to prevent and treat high blood pressure. *J Am Soc Nephrol*. 2003;14(7 Suppl 2):S99–S102.
73. Appel LJ. The effects of protein intake on blood pressure and cardiovascular disease. *Curr Opin Lipidol*. 2003;14(1):55–9.
74. Harsha D, Lin P, Obarzanek E, Karanja N, Moore T, Caballero B. Dietary Approaches to Stop Hypertension: a summary of study results. DASH Collaborative Research Group. *J American Dietetic Association*. 1999;99(8 Suppl):S35–9.
75. Craddock S, Elmer P, Obarzanek E, Vollmer W, Svetkey L, Swain M. The DASH diet and blood pressure. *Curr Atheroscler Rep*. 2003;5(6):484–91.
76. McCarron D, Oparil S, Chait A, Haynes R, Kris-Etherton P, Stern J, et al. Nutritional management of cardiovascular risk factors. A randomized clinical trial *Arch Intern Med*. 1997;157(2):169–77.
77. Krauss RM, Deckelbaum RJ, Ernst N, Fisher E, Howard BV, Knopp RH, et al. Dietary Guidelines for healthy American adults: A statement for health professionals from the Nutrition Committee of the American Heart Association. *Circulation*. 1996;94:1795–800.
78. Program NCE. Report of the Expert panel on Population Strategies for Blood Cholesterol Reduction: executive summary. *Arch Intern Med*. 1991;151:1071–84.
79. McCarron DA, Reusser ME. The power of food to improve multiple cardiovascular risk factors. *Curr Atheroscler Rep*. 2000;2(6):482–6.
80. Shirani F, Salehi-Abargouei A, Azadbakht L. Effects of Dietary Approaches to Stop Hypertension (DASH) diet on some risk for developing type 2 diabetes: a systematic review and meta-analysis on controlled clinical trials. *Nutrition*. 2013 Jul-Aug;29(7–8):939–47.
81. Salehi-Abargouei A, Maghsoudi Z, Shirani F, Azadbakht L. Effects of Dietary Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular diseases-Incidence: A systematic review and meta-analysis on observational prospective studies. *Nutrition*. 2013;29(4):611–8.
82. Brennan SF, Cantwell MM, Cardwell CR, Velentzis LS, Woodside JV. Dietary patterns and breast cancer risk: a systematic review and meta-analysis. *Am J Clin Nutr*. 2010;91(5):1294–302.
83. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated systematic review and meta-analysis. *Am J Clin Nutr*. 2010;92(5):1189–96.
84. Fox N, Ward K. Health, ethics and environment: a qualitative study of vegetarian motivations. *Appetite*. 2008 Mar-May;50(2–3):422–9.
85. Pribis P, Pencak RC, Grajales T. Beliefs and attitudes toward vegetarian lifestyle across generations. *Nutrients*. 2010 May;2(5):523–31.
86. Ruby MB. Vegetarianism. A blossoming field of study. *Appetite*. 2012 Feb;58(1):141–50.
87. Johansson U. Vegetarisk kost. Råd om mat för barn 0–5 år – vetenskapligt underlag med risk- eller nyttovärderingar och kunskapsöversikter Rapport 21 – 2011. Uppsala, Sweden: Livsmedelsverket/ National Food Agency; 2011. p. 259–74.
88. Key TJ, Appleby PN, Rosell MS. Health effects of vegetarian and vegan diets. *Proc Nutr Soc*. 2006 Feb;65(1):35–41.
89. Craig WJ, Mangels AR. Position of the American Dietetic Association: vegetarian diets. *J Am Diet Assoc*. 2009 Jul;109(7):1266–82.
90. Fraser GE. Vegetarian diets: what do we know of their effects on common chronic diseases? *Am J Clin Nutr*. 2009 May;89(5):1607S–12S.
91. Barnard ND, Cohen J, Jenkins DJ, Turner-McGrievy G, Gloede L, Green A, et al. A low-fat vegan diet and a conventional diabetes diet in the treatment of type 2 diabetes: a randomized, controlled, 74-wk clinical trial. *Am J Clin Nutr*. 2009 May;89(5):1588S–96S.

92. Tonstad S, Butler T, Yan R, Fraser GE. Type of vegetarian diet, body weight, and prevalence of type 2 diabetes. *Diabetes Care*. 2009 May;32(5):791–6.
93. Ferdowsian HR, Barnard ND. Effects of plant-based diets on plasma lipids. *Am J Cardiol*. 2009 Oct 1;104(7):947–56.
94. Andersson C. Lektiner i baljväxter. Råd om mat för barn 0–5 år – vetenskapligt underlag med risk- eller nyttovärderingar och kunskapsöversikter Rapport 21 – 2011. Uppsala, Sweden: Livsmedelsverket/ National Food Agency; 2011. p. 93–112.
95. Pawlak R, Parrott SJ, Raj S, Cullum-Dugan D, Lucas D. How prevalent is vitamin B(12) deficiency among vegetarians? *Nutr Rev*. 2013 Feb;71(2):110–7.
96. Van Winckel M, Vande Velde S, De Bruyne R, Van Biervliet S. Clinical practice: vegetarian infant and child nutrition. *Eur J Pediatr*. 2011 Dec;170(12):1489–94.
97. Sanders TA, Reddy S. Vegetarian diets and children. *Am J Clin Nutr*. 1994 May;59(5 Suppl):1176S–81S.
98. Jacobs C, Dwyer JT. Vegetarian children: appropriate and inappropriate diets. *Am J Clin Nutr*. 1988 Sep;48(3 Suppl):811–8.
99. Roed C, Skovby F, Lund AM. [Severe vitamin B12 deficiency in infants breastfed by vegans]. *Ugeskr Laeger*. 2009 Oct 19;171(43):3099–101.

4 Breastfeeding

The benefits of breastfeeding are well documented. The WHO recommends that infants are breastfed exclusively for about 6 months and given breast milk as part of the diet throughout the first 2 years and that this is continued as long as it suits the mother and child (1). Exclusively breastfed means that the infant is given no food or liquid other than breast milk but can receive additional vitamins, minerals, and/or medications. Vitamin D supplements are recommended for all infants in the Nordic countries from the first weeks of age. Vegan and vegetarian mothers who are breastfeeding should ensure an adequate intake of vitamin B₁₂ in order to avoid risk of deficiency in the infant.

Breast milk gives the new-born essential nutrients in an efficiently absorbed combination (2, 3) and has positive health effects on the infant as well as later in life. In addition to the macronutrients, vitamins, and minerals in breast milk, breast milk also contains immune-related factors and hormonal factors that are important for infant health and growth (4, 5). The contents of human breast milk differ significantly from those of other animal milks such as cow's milk (6, 7). If breastfeeding is not possible or is not chosen, commercial infant formula prepared according to Codex standards is recommended.

The impact of breastfeeding and the level of evidence often vary for different health-related outcomes. Research in this area has been active not least because of the high interest in the programming effect that diet can have on future health. It was, therefore, highly relevant to systematically evaluate the scientific evidence for the NNR 2004 (8) to be able to update the guidelines for the NNR 2012. A systematic literature review (SR) was performed on the scientific data valid in a Nordic setting on the short- and long-term health effects of both exclusive breastfeeding, any breastfeeding and breastfeeding in combination with the introduction of other foods (9). The SR only covered immediate and later health effects in the child. Health effects in the mother and other potential effects, such as on the bonding between mother and child, were not reviewed. Studies where the mother

or child was sick at start or at increased risk for disease were excluded from the SR. Studies involving preterm infants were also excluded, but it is likely that the health effects of breastfeeding are more pronounced in vulnerable infants such as these. In addition, too few studies existed to enable a review of the importance of the product vs. the mode of delivery, e.g. differentiating between breastfeeding and breast milk given in a bottle. The number of studies investigating the health effects of partial breastfeeding for 12 months or longer was also too small for any definitive conclusions to be drawn from the literature. The SR graded the evidence for relevant outcomes as convincing, probable, suggestive, or limited/inconclusive.

There is convincing evidence that breastfeeding protects against the development of overweight and obesity and prevents infections in infancy and early childhood (9). The strength of the evidence was lower for other health outcomes. There is probable evidence that breastfeeding has a role in diminishing cholesterol and blood pressure in adulthood; has beneficial effects on children's IQ and developmental scores; has a protective effect against inflammatory bowel disease (IBD) and celiac disease; and, when comparing any breastfeeding to none, has a protective effect against type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM) (9). Evidence that breastfeeding protects against cancer is scarce. There is, however, limited but suggestive evidence for a reduced risk of childhood leukaemia, with the protective effect coming after breastfeeding for longer than 6 months, and possibly of other childhood cancers (9). The evidence is insufficient and no conclusions can be drawn regarding breastfeeding and the risk of atopic diseases, asthma, wheezing, and eczema (9). More information is needed especially with regard to exclusive breastfeeding and allergies because this is the effect for which the results vary the most between studies. There is, however, currently no evidence for an association between decreased risk of allergies and later or earlier introduction of supplementary foods. Longitudinal studies in cohorts of new-born infants could help clarify the relationships described above. A summary of the grading of the evidence for relevant outcomes in the SR is shown in Table 4.1. and discussed further in the following text.

Table 4.1. Grading of evidence for health effects associated with breastfeeding in industrialized countries (9)

Outcome	Evidence grading
Acute otitis media	Convincing evidence (grade 1) that breastfeeding protects against acute otitis media.
Gastrointestinal infection	Convincing evidence (grade 1) that breastfeeding protects against gastrointestinal infections.
Lower respiratory infection	Convincing evidence (grade 1) that breastfeeding protects against respiratory tract infections.
Overweight/obesity	Convincing evidence (grade 1) that longer duration of exclusive breastfeeding or any breastfeeding is associated with a protective effect against overweight and obesity in childhood and adolescence. Suggestive evidence (grade 3) that breastfeeding protects against overweight and obesity in adulthood.
General growth	Probable evidence (grade 2) that exclusive breastfeeding for longer than 4 months is associated with slower weight gain during the second half of the first year.
Blood pressure	Probable evidence (grade 2) that breastfeeding has a small but significant reductive effect on blood pressure.
Serum cholesterol	Probable evidence (grade 2) that breastfeeding provides a small reduction in blood cholesterol levels later in life or adulthood.
Type 1 diabetes mellitus (T1DM)	Probable evidence (grade 2) that any breastfeeding has a protective effect against T1DM. The evidence for a stronger protective effect for longer duration of breastfeeding is limited but suggestive (grade 3).
Type 2 diabetes mellitus (T2DM)	Probable evidence (grade 2) that any breastfeeding has a protective effect against T2DM. The evidence for a stronger protective effect for longer duration of breastfeeding is limited but suggestive (grade 3).
IQ, neurological development, visual acuity	Probable evidence (grade 2) that prolonged breastfeeding is beneficial for IQ and developmental scores in children.
Celiac disease	Probable evidence (grade 2) that breastfeeding has a protective effect against celiac disease if gluten is introduced in small amounts while still breastfeeding. It is unclear whether the protection only delays the onset of celiac disease or if it provides permanent protection. The evidence is insufficient (grade 4) to conclude which age is best for the introduction of gluten.
Inflammatory bowel disease (IBD)	Probable evidence (grade 2) that breastfeeding provides protection against IBD.
Cancer	Limited but suggestive evidence (grade 3) for a protective effect of breastfeeding for 6 months against leukaemia and possibly other childhood cancers.
Atopic disease	Limited and inconclusive evidence (grade 4) and no conclusions can be drawn for any preventive effects of breastfeeding on the risk for atopic diseases in children.
Asthma	Limited and inconclusive evidence (grade 4) and no conclusions can be drawn for any preventive effects of breastfeeding on the risk for asthma in children.

Health benefits of breastfeeding

Energy yielding nutrients in human milk have multiple functions, such as providing amino acids in adequate amounts and proportions to support growth and maintenance of muscle tissue in breastfed infants and to facilitate optimal development of important physiological functions in new-borns. Increasing numbers of studies have also indicated long-term beneficial effects of breastfeeding on health.

The positive effects seen for breastfeeding depend to a considerable degree on the breast milk itself and its unique composition. They may also depend on the avoidance of certain other foods given to the infant, on the physical closeness during the act of breastfeeding, or on other associated factors. Interpreting the results of different studies is made difficult because the definition of breastfeeding varies and the methodology used to assess breastfeeding is often unclear. However, numerous nutrients and biologically active substances are found in breast milk, including vitamins, minerals, fatty acids, and immune factors, and many of these have proven positive effects on health. Data from prospective longitudinal infant cohorts in Nordic populations are urgently required because such data are likely to provide the best evidence for any benefits associated with the duration of both exclusive and any breastfeeding. In general, more high quality research, such as randomized controlled trials, is needed in this area.

Breastfeeding and infections

There is convincing evidence that breastfeeding has a protective effect against overall infections, acute otitis media, gastrointestinal infections, and respiratory tract infections (9). Breast milk contains many protective factors that might exert long-term health benefits, and the immunological protection against infections appears to last for some years after cessation of breastfeeding. The magnitude of the effect varies depending on the specific outcome and the exclusiveness of breastfeeding. A protective dose/duration-response effect on gastrointestinal or respiratory tract infections was found in the SRs of Duijits et al and Kramer et al (10, 11) as well as in the prospective studies by Fisk et al and Ladomenou et al (12, 13). In other reports, reduced risks of about 20% for otitis media, 50% for gastroenteritis, and about 30% for lower respiratory tract infections have also been cited (1, 3).

Breastfeeding and growth, overweight, and obesity

There is convincing evidence for a protective effect of breastfeeding, exclusive or any, against overweight and obesity in childhood and adolescence. The evidence is probable that exclusive breastfeeding for longer than 4 months is associated with slower weight gain during the second half of the first year compared with shorter duration. No negative health effects are reported for this slower weight gain, and instead the slower growth in infancy appears to help reduce the risk of overweight or obesity later in life (9). Only a few studies have been performed on the association between breastfeeding and overweight or obesity in adulthood, thus the evidence was evaluated to be only suggestive for a protective effect of breastfeeding (9).

The slower weight gain during the second half of the first year seen in children who are exclusively breastfed for longer than 4 months might partly explain the beneficial long-term effects on body weight development. The discrepancy in growth rates between those breastfed and those not breastfed becomes prominent during the latter half of the first year of life (14, 15). Several physiological mechanisms have been suggested to link early nutrition with later obesity. One hypothesis is the programming of high serum leptin concentrations relative to fat mass through formula feeding and faster growth in infancy (16). Another hypothesis is that obesity is an inflammatory condition and that the interactions between brain growth and development and long chain polyunsaturated fatty acids, pro-inflammatory cytokines, neurotransmitters, and bone morphogenic proteins might explain the relationship between breastfeeding and obesity (17). Better self-regulation of energy intake among breastfed infants (18) might also play a part, and this has been linked to a lower metabolic rate and lower weight gain in breastfed infants compared with formula-fed infants (19, 20).

An often overlooked explanation for the protective effect of breast milk against the development of overweight is the issue of the amount and type of other foods used to replace breast milk. Replacement with other foods might result in high protein intake, and this is convincingly associated with growth rate and higher risk of overweight and obesity as shown by Escribano et al (21) and other investigators and is thoroughly discussed in a SR on protein intake in childhood (22) and in the protein chapter in NNR 2012. Infant formula has previously been much higher in protein than breast milk, but the levels have been decreasing and are now closer to that of breast milk. This change will probably decrease the difference

in protein intake between infants fed breast milk and those fed infant formula (REGULATION EC No 1243/2008 and Directive 2006/141/EC).

Selective reporting or publication bias in this area cannot be totally excluded, and well-performed prospective studies with longer duration of breastfeeding and follow-up data are still needed. Kramer et al (23) found that smaller body size was strongly associated with increased risks of premature weaning and of discontinuing exclusive breastfeeding, especially when the infant was between 2 and 6 months old. In other words, if a child is small the parents are more likely to start giving other foods in addition to or instead of breast milk. This is problematic because it makes it more difficult to study and interpret studies on the associations between infant feeding and growth. In an Icelandic randomized controlled trial, all infants were exclusively breastfed for 4 months and then randomized to either continued exclusive breastfeeding until they reached 6 months of age or continued breastfeeding combined with introduction to complementary foods at 4 months (24). Similar growth rates and body compositions were seen at 6 months in the two groups of infants.

International reports have concluded that breastfeeding might reduce the occurrence of overweight and obesity by about 20% in the general population (1, 3, 25), and the WHO has developed new growth charts that are more relevant than the charts formerly used (26). The new charts are based on breastfed infants and give a better picture of normal growth. This might take the pressure off breastfeeding women to give their babies formula or other foods too early and might also decrease the risk of overfeeding (27).

In a recent update of a Cochrane review, Kramer and Kakuma (11) did not find exclusive breastfeeding for 6 months to have long-term effects on reducing the risk of obesity when compared to exclusive breastfeeding for 3–4 months. However, that review compared infants whose breastfeeding probably differed too little to see a difference, i.e., exclusively breastfed for at least 6 months followed by mixed breastfeeding compared to infants exclusively breastfed for 3–4 months of age followed by mixed breastfeeding until or beyond 6 months. The infant groups compared in the Nordic SR, which serves as the main reference for NNR 2012, usually differed more in the duration of exclusive breastfeeding (9).

Breastfeeding and risk factors for cardiovascular disease

The influence of breastfeeding on the risk of cardiovascular disease is unclear, but there is probable evidence of small but significant effects on

the reduction of blood pressure levels and serum cholesterol levels later in adulthood (9). Breastfeeding has also been associated with lower blood pressure levels in childhood and adolescence (28), but more evidence is needed to support such an association. Even though the physiology behind these effects is still unclear, possible explanations might include a higher intake of n-3 fatty acids by breastfed infants that results in increased elasticity of the blood vessels. Another reason could be lower salt intake by breastfed infants. The lower cholesterol in adulthood found among those who were breastfed in infancy might be the result of the metabolic effects of constituents in breast milk such as cholesterol and n-3 fatty acids.

The macronutrient content of breast milk is relatively stable, but the fat composition varies depending on the mother's diet. For example, the levels of long-chain polyunsaturated fatty acids are highly dependent on the mother's intake of seafood, linoleic and linolenic acids, and dietary supplements (29–31). It is also possible that a high intake of omega-6-fatty acids decreases the synthesis of omega-3-fatty acids (29, 31), and the content in breast milk.

Breastfeeding and diabetes mellitus

There is probable evidence that any breastfeeding is protective against type 1 and type 2 diabetes mellitus. The evidence of a larger protective effect based on the duration of breastfeeding is limited, though suggestive, for type 1 diabetes mellitus (9). This protective effect of breast milk might depend on the different proteins in breast milk compared with infant formula, but other routes of association between diabetes and short duration of breastfeeding or the introduction of food and drinks have been suggested. In a study on a high risk population, Virtanen et al found that the overall or exclusive duration of breastfeeding was not associated with the risk of beta cell autoimmunity (which could be a sign for later development of type 1 diabetes mellitus), but it must be noted that all participants in that study were breastfed to some extent (32). A report from the EFSA in 2009 suggested that introduction of gluten-containing foods between 4 and 6 months while still breastfeeding might decrease the risk for type 1 diabetes mellitus (33). A joint statement by COT/SACN in 2011 found the evidence insufficient for a specific age to introduce complementary food except that such introduction should not occur before the age of at least 3 completed months (34). Both SACN (25) and the WHO (1) state that infants who are not breastfed are at greater risk of type 2 diabetes mellitus.

Breastfeeding and IQ, neurological development and visual acuity

There is probable evidence that breastfeeding has beneficial effects on IQ and developmental scores of children and that the benefits increase with increasing duration of breastfeeding (9). The favourable effect of breastfeeding on the healthy neurological development of the infant might be caused by the high content of docosahexaenic acid (also known as DHA) in breast milk because this fatty acid is present in high amounts in nerve cell membranes. The interpretation of studies on the association between breastfeeding and neurological development is complicated because the outcome is not only influenced by whether the child is breastfed or not or by what children are fed instead of breast milk and the exposure that this gives, but also by the facilitating effects breastfeeding can have on mother-infant bonding. Not all studies have found a beneficial effect of breastfeeding on neurological development, but no study has found detrimental effects or that formula feeding is advantageous in comparison. Several strong cohort studies have shown positive effects of breastfeeding, and the few studies showing no or non-significant effects can be explained by their study design. Thus the evidence is probable that breastfeeding is beneficial for IQ and developmental scores in children and that increased benefits are associated with increased duration.

Oken and co-workers (35) studied developmental milestones at 18 months of age, and children who were breastfed for 2–3, 4–6, or >6 months all showed higher scores for motor developmental milestones and total developmental milestones compared to those breastfed <1 month. Children breastfed >6 months also showed higher scores for social and cognitive developmental milestones in comparison to children breastfed <1 month. A stepwise increase in IQ was found with longer duration of breastfeeding, and the highest IQ points and developmental scores were found with breastfeeding that lasted longer than 6 months (36, 37).

Positive results from the PROBIT study in Belarus that compared control areas to intervention areas in which breastfeeding was promoted also provide quite strong support for positive associations between breastfeeding and neurological development (38). The non-results in another paper from the same group (39) can probably be explained by the fact that this latter paper compared children who were exclusively breastfed for 3 or 6 months from both the intervention and control areas. This likely resulted in smaller differences than when the authors compared the two areas with their large differences in overall breastfeeding patterns. In other studies, Zhou and co-workers found a positive association that was attenuated and no lon-

ger significant after adjustment for socioeconomic characteristics (40), but Oddy and co-workers concluded that although the effect sizes were small breastfeeding for 4 months or longer was associated with improved neurological outcomes in children aged 1 to 3 years after adjustment for multiple confounders (41).

Breastfeeding and celiac disease

There is probable evidence for protection against celiac disease if gluten is introduced in small amounts while still breastfeeding (9). The evidence is insufficient, however, to conclude which age is best for the introduction of gluten (9).

In a systematic review, Akobeng and co-workers found a negative association between breastfeeding and celiac disease. The authors found a 50% lower risk if the child was still breastfed when gluten was introduced, but they stated that it was not clear whether breastfeeding only delays the onset of celiac disease or if it provides permanent protection (42).

Both EFSA (33) and ESPGHAN (43) support introducing gluten-containing foods while still breastfeeding, but not later than 6 months of age or too early (<4 months). In a joint statement, COT/SACN (34) agrees with the introduction of gluten while breastfeeding, but it does not consider the evidence sufficient to support a precise statement about age of introduction of gluten except that introduction should not occur before 3 completed months.

Breastfeeding and inflammatory bowel disease (IBD)

The evidence is probable that breastfeeding provides protection against IBD, but it is insufficient to give exact estimates of the risk reduction (9). Well-performed prospective studies with reliable, well-defined breastfeeding data are needed to enable such estimates. Klement and co-workers (44) included 17 studies in an SR and found breastfeeding to have protective effects against ulcerative colitis, giving about a 45% reduction in risk, and an even greater effect against Crohn's disease with close to a 55% reduction in risk.

Breastfeeding and cancer

There is limited but suggestive evidence that breastfeeding decreases the risk for leukaemia and possibly other childhood cancers (9). The effect on childhood leukaemia seems to be greater with longer breastfeeding duration (>6 months), but the amount of data is too small to rigorously

assess this effect (9). In a systematic review, Ip et al concluded that there is an association between a history of breastfeeding of at least 6 months duration and a reduction in the risk of leukaemia (3). Existing research is insufficient to assess any associations between breastfeeding and cancers in adulthood.

Breastfeeding and atopy and asthma

The evidence regarding infant feeding and development of atopic diseases and asthma is conflicting. Previous advice on allergy prevention has included delayed introduction of food items to the infant's diet. Immunomodulatory qualities of breast milk and avoidance of allergens, or a combination of these and other factors, were thought to prevent conditions such as asthma especially if a family history of atopy was present (45). This recommendation has changed due to lack of evidence.

At present the Swedish Paediatric Society (46) concludes that breastfeeding gives some protection against infection-induced, asthma-type airway symptoms but states that breastfeeding has not been proven to decrease the risk of atopy and allergies, and also that there are no advantage in avoiding allergens, neither during pregnancy or in infancy. The American Association of Pediatrics (47) states, however, that there is evidence that breastfeeding for at least 4 months, compared with feeding formula made with intact cow's milk protein (which can be found in some countries) prevents or delays the occurrence of atopic dermatitis and cow's milk allergy in early childhood.

Certain foods are more allergenic than others (i.e. milk, eggs, fish, nuts, and shellfish), and over the past decade it has been discussed if total elimination or if early introduction of these foods in the diet protects against atopic disease and asthma. Findings of prospective studies on high-risk populations suggest that early age at introduction of new foods is associated with decreased risk of atopic asthma and other allergic diseases (48–50). It is not clear whether this is related to the infant's age per se or to increased chances of introduction occurring while breastfeeding is on-going which have been shown to be protective when it comes to gluten introduction and celiac disease. There are studies in progress trying to elucidate if introduction of small amounts of complementary foods while still breastfeeding is beneficial.

The SR performed for the 5th NNR (9) found the existing scientific evidence limited and contradictory. The studies on the association between breastfeeding and asthma found contradictory results, and the evidence

linking breastfeeding or introduction of solid foods to asthma and wheezing was inconclusive. This makes the evidence limited and no firm conclusions can be drawn (9). Two SRs and meta-analysis studying the effect of exclusive breastfeeding for longer than 3 months on the risk for atopic disease got contradictory results. One study found a protective effect (3). The other study found no significant effect for longer duration of exclusive breastfeeding regardless of heredity but did find that any breastfeeding was protective when compared to no breastfeeding (51). Another SR looked at early introduction of solid food (<4 months of age) and concluded that early solid feeding might increase the risk for eczema but that little data supported an association between early solid feeding and other allergic conditions (52). The prospective studies included in the 5th NNR SR did not change the grading of the evidence.

Longitudinal studies in cohorts of new-born infants could help clarify the relationship between exclusiveness and/or duration of breastfeeding, as well as the introduction of solid foods, and atopic diseases. It is important to include data about whether the infants are introduced to new food when still breastfed. It has also been shown that genes might have modifying effects on the associations between breastfeeding and outcomes such as asthma, and this suggests that genetic aspects should be included in future studies (53). Very little is known about active prevention of allergies and asthma by adding specific food components to the diets of pregnant or lactating women or to the diets of infants. Any positive effect of giving different dietary supplements (n-3 fatty acids, pre- and probiotics, or vitamins) remains to be shown.

Breastfeeding and vitamin D and iron status

It has been questioned if breastfeeding provides sufficient amounts of vitamin D and iron to the breastfed infant. Sun exposure is insufficient to prevent rickets in the Nordic countries, and breast milk does not contain sufficient vitamin D for prevention even if the mother takes vitamin D supplements (54). If not given a vitamin D supplement, there is a rapid decrease in the level of 25-OH vitamin D stored in the infant's body during the first weeks of life to the level usually seen in rickets (55). It has long been known that all infants and young children living at northern latitudes need vitamin D supplements. These should be in the form of drops or as cod liver oil, and 10 µg/d is recommended for new-borns from the first weeks of age as discussed in the chapter on vitamin D in NNR 2012.

A recent randomized trial on exclusive breastfeeding for 4 vs. 6 months has reported on the iron status of the infants at 6 months of age. Ferritin levels were lower in the group exclusively breastfed for 6 months compared with the group exclusively breastfed for 4 months and given other food together with breastfeeding until 6 months, but there were no indications or evidence that the difference was of biological or clinical importance (56). An earlier study found iron status to be negatively affected by exclusive breastfeeding for 9 months (57). Another study, however, found that there is no need to give iron supplementation to infants who are breastfed longer than 6 months (58). It seems apparent that the total diet after the age of 6 months, and the choice and amount of breast milk, cow's milk, or formula, is important (59, 60). A recent study, however, suggests that delayed umbilical cord clamping might be an important and easy way to improve iron status in later infancy (61) without risking the overconsumption of iron that can occur with iron supplementation.

Prevalence of breastfeeding in the Nordic countries

Compared to the rest of the world, all of the Nordic countries have relatively high breastfeeding rates. After birth virtually all mothers breastfeed their infants, and between 58% and 80% of the infants are still breastfed at 6 months (Table 4.2.). In spite of the high breastfeeding rates, a relatively low proportion of infants in the Nordic countries are breastfed as recommended, i.e. exclusively breastfed for around the first 6 months of life and partly breastfed until 12 months of age (9). The rate of exclusive breastfeeding is high the first months, but this decreases quickly to only 23% to 63% of infants being exclusively breastfed at 4 months. The majority of infants in the Nordic countries are introduced to other foods before 6 months of age.

Table 4.2. Reported breastfeeding rates (% exclusive and any breastfeeding) among children born in the Nordic countries

	1 week		1 month		2 months		3 months		4 months		5 months		6 months		9 months		12 months	
	Excl	Any	Excl	Any	Excl	Any	Excl	Any	Excl	Any	Excl	Any	Excl	Any	Any	Any	Any	Any
Denmark ¹	95	80							60				12					
Finland ²			46	87	39	80	34	77	23	68	9	66	0	58	39		34	
Iceland ³	86	98	87	94	80	91	67	86	63	84	35	79	8	74	45		27	
Norway ⁴			82	95	73	91	63	88	46	85	25	82	9	80	63		46	
Sweden ⁵	83	97			67	87			51	76			11	63	34		16	

¹ Children born in 2008 and 2009 in 14 municipalities in Denmark. (62).

² Children born in 2010 Finland. Health and Welfare report 2012 (63).

³ Children born in 2005–2006 in Iceland, Nationwide randomized cohort (60) and children born in 2004–2008, Directorate General of Health, Iceland (64).

⁴ Children born in 2006 in Norway. National dietary survey. (65, 66).

⁵ Children born in 2010 in Sweden. National statistics 2012 (67).

The definition of exclusive breastfeeding that is used in the studies is important to take into consideration when looking at breastfeeding statistics and comparing countries and comparing rates within a country over time.

Breastfeeding rates have been increasing in all of the Nordic countries since the mid-1970s. The prevalence of exclusive breastfeeding at 4 months of age presented in the fourth edition of the NNR (8) was 50% in Denmark compared to the current rate of 60% (62, 68); 15% in Finland compared to the current rate of 23% (63, 69); 46% in Iceland compared to the current rate of 63% (15, 60, 64); and 44% in Norway compared to the rate of 46% in 2006 (65, 66, 70). However, in Finland exclusive breastfeeding at 4 months increased from 10% in 1995 to 15% in 2000 and further to 34% in 2005, but decreased in 2010 to 23%. The rate of any breastfeeding in Finland has, though, consistently increased from 1995 to 2010 (63, 69). In Sweden, a decline in breastfeeding rates, both exclusive and any, has been seen since 2004. The reason behind the decline in exclusive breastfeeding rates in Sweden is, at least in part, due to a change to the more strict definition of exclusive breastfeeding given by the WHO. Rates of any breastfeeding have also declined in Sweden for reasons unknown at this time, but the frequency of breastfeeding in Sweden is still high compared to the rest of the world (67, 71). Considering the

decrease in breastfeeding prevalence seen in Sweden, and the decreasing prevalence of exclusive breastfeeding with infant age seen in the Nordic countries, it is deemed very important to further protect, promote, and support breastfeeding in all of the Nordic countries.

Recommendation for breastfeeding

Exclusive breastfeeding is recommended until the infant is about 6 months old. This is in accordance with the latest recommendation from the World Health Assembly and the WHO and is not changed from the NNR 2004 (8, 72–75). Exclusive breastfeeding means that the child only receives breast milk but, if necessary, can be supplemented with vitamins, minerals, and medications. The WHO recommendation applies to all countries and populations regardless of economic status or developmental level. In the Nordic countries, breastfed infants only need to be supplemented with vitamin D in the form of drops or as cod liver oil. If for some reason breastfeeding is not possible during the first 6 months of life, the infant should be given expressed breast milk, the mother's own or from others, or commercial infant formula formulated according to relevant regulations to serve as the only food for infants. Parents should when required be given guidance on how to prepare and feed formula to their babies. Some infants will need complementary feeding before 6 months of age, but experts agree that solid food should not be introduced before the age of 4 months.

Exclusive breastfeeding for about 6 months is recommended by most official bodies, including the AAP in 2008 and 2012 (47, 76), EFSA in 2009 (33), ESPGHAN in 2008 and 2009 (43, 77), SACN in 2011 (25), and the WHO (73, 74). At the same time, EFSA (33) and ESPGHAN (43) do not find any disadvantages with starting to give complementary foods in addition to breastfeeding in the age range of 4 to 6 months in Europe. But there is also no evidence that giving complementary foods between 4–6 months provides any health benefits beyond those of exclusive breastfeeding for 6 months. From 6 months of age, gradual introduction of a diversified diet is recommended. Breast milk as part of the diet is recommended throughout the child's first year, and partial breastfeeding can be continued for as long as it suits the mother and child.

References

1. Horta B, Bahl R, Martines JC, Victora CG. Evidence on the long-term effects of breastfeeding: systematic review and meta-analyses: WHO 2007.
2. Ogra PL, Greene HL. Human milk and breast feeding: an update on the state of the art. *Pediatr Res*. 1982 Apr;16:266–71.
3. Ip S, Chung M, Raman G, Trikalinos TA, Lau J. A summary of the Agency for Healthcare Research and Quality's evidence report on breastfeeding in developed countries. *Breastfeed Med*. 2009 Oct;4 Suppl 1:S17–30.
4. Bernt KM, Walker WA. Human milk as a carrier of biochemical messages. *Acta Paediatr Suppl*. 1999 Aug;88(430):27–41.
5. Wagner CL, Anderson DM, Pittard WB, 3rd. Special properties of human milk. *Clin Pediatr (Phila)*. 1996 Jun;35(6):283–93.
6. Sellen DW. Evolution of infant and young child feeding: implications for contemporary public health. *Annu Rev Nutr*. 2007;27:123–48.
7. Cuthbertson WF. Evolution of infant nutrition. *Br J Nutr*. 1999 May;81(5):359–71.
8. Nordic Nutrition Recommendations 2004. Integrating nutrition and physical activity. 4th ed. Arhus, Denmark: Nordic Council of Ministers; 2005.
9. Hornell A, Lagstrom H, Lande B, Thorsdottir I. Breastfeeding, introduction of other foods and effects on health: a systematic literature review for the 5th Nordic Nutrition Recommendations. *Food Nutr Res*. 2013;57.
10. Duijts L, Ramadhani MK, Moll HA. Breastfeeding protects against infectious diseases during infancy in industrialized countries. A systematic review. *Matern Child Nutr*. 2009 Jul;5(3):199–210.
11. Kramer MS, Kakuma R. Optimal duration of exclusive breastfeeding. *Cochrane Database Syst Rev*. 2012;8:CD003517.
12. Fisk CM, Crozier SR, Inskip HM, Godfrey KM, Cooper C, Roberts GC, et al. Breastfeeding and reported morbidity during infancy: findings from the Southampton Women's Survey. *Matern Child Nutr*. 2011 Jan;7(1):61–70.
13. Ladomenou F, Moschandreas J, Kafatos A, Tselentis Y, Galanakis E. Protective effect of exclusive breastfeeding against infections during infancy: a prospective study. *Arch Dis Child. [Journal Article]*. 2010 Dec;95(12):1004–8.
14. Nielsen GA, Thomsen BL, Michaelsen KF. Influence of breastfeeding and complementary food on growth between 5 and 10 months. *Acta Paediatr*. 1998 Sep;87(9):911–7.
15. Atladottir H, Thorsdottir I. Energy intake and growth of infants in Iceland—a population with high frequency of breast-feeding and high birth weight. *European Journal of Clinical Nutrition*. 2000;54(9):695–701.
16. Singhal A, Farooqi IS, O'Rahilly S, Cole TJ, Fewtrell M, Lucas A. Early nutrition and leptin concentrations in later life. *Am J Clin Nutr*. 2002 Jun;75(6):993–9.
17. Das UN. Is obesity an inflammatory condition? *Nutrition*. 2001 Nov-Dec;17(11–12):953–66.
18. Dewey KG. Growth characteristics of breast-fed compared to formula-fed infants. *Biol Neonate*. 1998;74(2):94–105.
19. Butte NF, Wong WW, Ferlic L, Smith EO, Klein PD, Garza C. Energy expenditure and deposition of breast-fed and formula-fed infants during early infancy. *Pediatr Res*. 1990 Dec;28(6):631–40.
20. Davies PS. Energy requirements and energy expenditure in infancy. *Eur J Clin Nutr*. 1992 Dec;46 Suppl 4:S29–35.
21. Escribano J, Luque V, Ferre N, Mendez-Riera G, Koletzko B, Grote V, et al. Effect of protein intake and weight gain velocity on body fat mass at 6 months of age: the EU Childhood Obesity Programme. *Int J Obes (Lond)*. 2012 Apr;36(4):548–53.

22. Hornell A, Lagstrom H, Lande B, Thorsdottir I. Protein intake from 0 to 18 years of age and its relation to health: a systematic literature review for the 5th Nordic Nutrition Recommendations. *Food Nutr Res.* 2013;57.
23. Kramer MS, Moodie EE, Dahhou M, Platt RW. Breastfeeding and infant size: evidence of reverse causality. *Am J Epidemiol.* 2011 May 1;173(9):978–83.
24. Wells JC, Jonsdottir OH, Hibberd PL, Fewtrell MS, Thorsdottir I, Eaton S, et al. Randomized controlled trial of 4 compared with 6 mo of exclusive breastfeeding in Iceland: differences in breast-milk intake by stable-isotope probe. *Am J Clin Nutr.* 2012 Jul;96(1):73–9.
25. The influence of maternal, fetal and child nutrition on the development of chronic disease in later life Scientific Advisory Committee on Nutrition (SACN)2011.
26. WHO Multicentre Growth Reference Study Group. WHO Child Growth Standards: Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: Methods and development Geneva: World Health Organization (WHO)2006.
27. Coombes R. UK adopts growth charts based on data from breastfed babies. *BMJ.* 2009;338:b1892.
28. Singhal A, Cole TJ, Lucas A. Early nutrition in preterm infants and later blood pressure: two cohorts after randomised trials. *Lancet.* 2001 Feb 10;357(9254):413–9.
29. Innis SM. Human milk: maternal dietary lipids and infant development. *Proc Nutr Soc.* 2007 Aug;66(3):397–404.
30. Koletzko B, Lien E, Agostoni C, Bohles H, Campoy C, Cetin I, et al. The roles of long-chain polyunsaturated fatty acids in pregnancy, lactation and infancy: review of current knowledge and consensus recommendations. *J Perinat Med.* 2008;36(1):5–14.
31. Gibson RA, Muhlhauser B, Makrides M. Conversion of linoleic acid and alpha-linolenic acid to long-chain polyunsaturated fatty acids (LCPUFAs), with a focus on pregnancy, lactation and the first 2 years of life. *Matern Child Nutr.* 2011 Apr;7 Suppl 2:17–26.
32. Virtanen SM, Kenward MG, Erkkola M, Kautiainen S, Kronberg-Kippila C, Hakulinen T, et al. Age at introduction of new foods and advanced beta cell autoimmunity in young children with HLA-conferred susceptibility to type 1 diabetes. *Diabetologia.* 2006;49(7):1512–21.
33. Scientific Opinion on the appropriate age for introduction of complementary feeding of infants. EFSA Journal [serial on the Internet]. 2009; 7(12).
34. Joint statement. Timing of introduction of gluten into the infant diet: Scientific Advisory Committee on Nutrition (SACN) and COT2011 Report No.: 2011/01.
35. Oken E, Osterdal ML, Gillman MW, Knudsen VK, Halldorsson TI, Strom M, et al. Associations of maternal fish intake during pregnancy and breastfeeding duration with attainment of developmental milestones in early childhood: a study from the Danish National Birth Cohort. *American Journal of Clinical Nutrition.* 2008;88(3):789–96.
36. Jedrychowski W, Perera F, Jankowski J, Butscher M, Mroz E, Flak E, et al. Effect of exclusive breastfeeding on the development of children's cognitive function in the Krakow prospective birth cohort study. *Eur J Pediatr.* 2012 Jan;171(1):151–8.
37. Whitehouse AJ, Robinson M, Li J, Oddy WH. Duration of breast feeding and language ability in middle childhood. *Paediatr Perinat Epidemiol.* 2011 Jan;25(1):44–52.
38. Kramer MS, Aboud F, Mironova E, Vanilovich I, Platt RW, Matush L, et al. Breastfeeding and child cognitive development: new evidence from a large randomized trial. *Arch Gen Psychiatry.* 2008 May;65(5):578–84.
39. Kramer MS, Matush L, Bogdanovich N, Aboud F, Mazer B, Fombonne E, et al. Health and development outcomes in 6.5-y-old children breastfed exclusively for 3 or 6 mo. *Am J Clin Nutr.* 2009 Oct;90(4):1070–4.
40. Zhou SJ, Baghurst P, Gibson RA, Makrides M. Home environment, not duration of breast-feeding, predicts intelligence quotient of children at four years. *Nutrition.* 2007 Mar;23(3):236–41.
41. Oddy WH, Robinson M, Kendall GE, Li J, Zubrick SR, Stanley FJ. Breastfeeding and early child development: a prospective cohort study. *Acta Paediatr.* 2011 Jul;100(7):992–9.

42. Akobeng AK, Ramanan AV, Buchan I, Heller RF. Effect of breast feeding on risk of coeliac disease: a systematic review and meta-analysis of observational studies. *Arch Dis Child*. Jan;91(1):39–43.
43. Agostoni C, Decsi T, Fewtrell M, Goulet O, Kolacek S, Koletzko B, et al. Complementary feeding: a commentary by the ESPGHAN Committee on Nutrition. *J Pediatr Gastroenterol Nutr*. 2008 Jan;46(1):99–110.
44. Klement E, Cohen RV, Boxman J, Joseph A, Reif S. Breastfeeding and risk of inflammatory bowel disease: a systematic review with meta-analysis. *Am J Clin Nutr*. 2004 Nov;80(5):1342–52.
45. Gdalevich M, Mimouni D, Mimouni M. Breast-feeding and the risk of bronchial asthma in childhood: a systematic review with meta-analysis of prospective studies. *J Pediatr*. 2001 Aug;139(2):261–6.
46. Factors influencing the development of asthma and allergies in children. scientific background [In Swedish. Faktorer av betydelse för uppkomsten av astma och allergisjukdom hos barn. Vetenskaplig bakgrund.] Swedish Paediatric Society (Svenska barnläkarföreningen) 2010.
47. Greer FR, Sicherer SH, Burks AW, American Academy of Pediatrics Committee on N, American Academy of Pediatrics Section on A, Immunology. Effects of early nutritional interventions on the development of atopic disease in infants and children: the role of maternal dietary restriction, breastfeeding, timing of introduction of complementary foods, and hydrolyzed formulas. *Pediatrics*. 2008;121(1):183–91.
48. Hesselmar B, Saalman R, Rudin A, Adlerberth I, Wold A. Early fish introduction is associated with less eczema, but not sensitization, in infants. *Acta Paediatr*. 2010 Dec;99(12):1861–7.
49. Nwaru BI, Erkkola M, Ahonen S, Kaila M, Haapala AM, Kronberg-Kippila C, et al. Age at the introduction of solid foods during the first year and allergic sensitization at age 5 years. *Pediatrics*. 2010 Jan;125(1):50–9.
50. Virtanen SM, Kaila M, Pekkanen J, Kenward MG, Uusitalo U, Pietinen P, et al. Early introduction of oats associated with decreased risk of persistent asthma and early introduction of fish with decreased risk of allergic rhinitis. *Br J Nutr*. 2010 Jan;103(2):266–73.
51. Yang YW, Tsai CL, Lu CY. Exclusive breastfeeding and incident atopic dermatitis in childhood: a systematic review and meta-analysis of prospective cohort studies. *Br J Dermatol*. Aug;161(2):373–83.
52. Tarini BA, Carroll AE, Sox CM, Christakis DA. Systematic review of the relationship between early introduction of solid foods to infants and the development of allergic disease. *Arch Pediatr Adolesc Med*. 2006 May;160(5):502–7.
53. Standl M, Sausenthaler S, Lattka E, Koletzko S, Bauer CP, Wichmann HE, et al. FADS gene cluster modulates the effect of breastfeeding on asthma. Results from the GINIplus and LISAplus studies. *Allergy*. 2012 Jan;67(1):83–90.
54. Olafsdottir AS, Wagner KH, Thorsdottir I, Elmadfa I. Fat-soluble vitamins in the maternal diet, influence of cod liver oil supplementation and impact of the maternal diet on human milk composition. *Ann Nutr Metab*. 2001;45(6):265–72.
55. Markestad T. Effect of season and vitamin D supplementation on plasma concentrations of 25-hydroxyvitamin D in Norwegian infants. *Acta Paediatr Scand*. 1983 Nov;72(6):817–21.
56. Jonsdottir OH, Thorsdottir I, Hibberd PL, Fewtrell MS, Wells JC, Palsson GI, et al. Timing of the introduction of complementary foods in infancy: a randomized controlled trial. *Pediatrics*. 2012 Dec;130(6):1038–45.
57. Pizarro F, Yip R, Dallman PR, Olivares M, Hertrampf E, Walter T. Iron status with different infant feeding regimens: relevance to screening and prevention of iron deficiency. *J Pediatr*. 1991 May;118(5):687–92.
58. Domellöf M, Lönnerdal B, Abrams SA, Hernell O. Iron absorption in breast-fed infants: effects of age, iron status, iron supplements, and complementary foods. *Am J Clin Nutr*. 2002 Jul;76(1):198–204.
59. Thorisdottir AV, Ramel A, Palsson GI, Tomasson H, Thorsdottir I. Iron status of one-year-olds and association with breast milk, cow's milk or formula in late infancy. *Eur J Nutr*. 2013 Sep;52(6):1661–8.
60. Thorisdottir AV, Thorsdottir I, Palsson GI. Nutrition and Iron Status of 1-Year Olds following a Revision in Infant Dietary Recommendations. *Anemia*. 2011;2011:986303.

61. Andersson O, Hellstrom-Westas L, Andersson D, Domellof M. Effect of delayed versus early umbilical cord clamping on neonatal outcomes and iron status at 4 months: a randomised controlled trial. *BMJ*. 2011;343:d7157.
62. Christensen AM, Sjöberg Brixval C, Svendsen M, Laursen B, Holstein BE. Årsrapport for børn født i 2008 og 2009 fra Databasen Børns Sundhed: Amning i 14 kommuner. København: Region Hovedstaden 2011.
63. Uusitalo L, Nyberg H, Pelkonen M, Sarlio-Lähteenkorva S, Hakulinen-Viitanen T, Virtanen S. Imeväisikäisten ruokinta Suomessa vuonna 2010 (Infant feeding in Finland in 2010) [In Finnish, short summary in Swedish]. Helsinki 2012.
64. Sigbjörnsdóttir HB, Gunnarsdóttir BE. Newsletter. Breastfeeding statistics from Iceland [in Icelandic] 2012.
65. Øverby NC, Kristiansen AL, Andersen LF, Lande B. Spedkost 6 måneder – Norwegian national dietary survey among infants at 6 months (in Norwegian). Oslo, Norway: Norwegian Directorate of Health 2008.
66. Øverby NC, Kristiansen AL, Andersen LF, Lande B. Spedkost 12 måneder – Norwegian national dietary survey among infants at 12 months (in Norwegian). Oslo, Norway: Norwegian Directorate of Health 2009.
67. Statistics – Health and Medical Care. Amning och föräldrars rökvanor – Barn födda 20010 [Breast-feeding and smoking habits among parents of infants born in 2010] 2012.
68. Michaelsen KF, Larsen PS, Thomsen BL, Samuelson G. The Copenhagen cohort study on infant nutrition and growth: duration of breast feeding and influencing factors. *Acta Paediatr*. 1994 Jun;83(6):565–71.
69. Hasunen K. Infant feeding in Finland 2000 [In Finnish: Imeväisikäisten ruokinta Suomessa vuonna 2000]. Helsinki 2002. Report No.: 2001:12.
70. Lande B, Andersen LF, Baerug A, Trygg KU, Lund-Larsen K, Veierod MB, et al. Infant feeding practices and associated factors in the first six months of life: the Norwegian infant nutrition survey. *Acta Paediatr*. 2003;92(2):152–61.
71. Infant and young child nutrition, Resolution No WHA 54.2. (2001).
72. Amning av barn födda 2000: Epidemiologiskt centrum 2002 Report No.: 2002:7.
73. Complementary feeding: report of the global consultation, and summary of guiding principles for complementary feeding of the breastfed child. Geneva, Switzerland: World Health Organization (WHO) 2002.
74. Kramer MS, Kakuma R. The optimal duration of exclusive breastfeeding: a systematic review. Geneva: World Health Organization 2001 Report No.: (WHO/NHD/01.09, WHO/FCH/CAH 01.24).
75. Global strategy for infant and young child feeding. Geneva: WHO/UNICEF 2003.
76. Breastfeeding and the use of human milk. *Pediatrics*. 2012 Mar;129(3):e827–41.
77. Agostoni C, Braegger C, Decsi T, Kolacek S, Koletzko B, Michaelsen KF, et al. Breast-feeding: A commentary by the ESPGHAN Committee on Nutrition. *J Pediatr Gastroenterol Nutr*. 2009 Jul;49(1):112–25.

6

Sustainable food consumption – Environmental issues

Introduction

For food consumption to be sustainable it has to be safe and healthy in both amount and quality, and this has to be achieved through means that are economically, socially, culturally, and environmentally sustainable. In addition, waste and pollution need to be reduced, and the changes in food consumption at e.g. individual, national or regional level, should not jeopardize the needs of others (1). According to the international Susfood project (2), the changes need to operate within the biological limits of natural resources, especially with regard to soil, water, and biodiversity. The way we choose to consume food has an effect on the environment as measured in terms of climate change, toxic impact, biodiversity, eutrophication, acidification, land use and change, and water use.

This chapter gives a short overview of the major issues recognized in connection with food consumption and its environmental impact. This field is new, the measurement tools are not always agreed upon, and new aspects are continuously being added. In this overview, the literature search has not been systematic and the discussion is more exploratory.

Among all dietary guidelines, the following are pivotal: Eat a varied diet! Choose food from a variety of food groups over the day and do not overeat. This advice ensures that diet that covers the necessary nutrients and maintains a healthy body weight. The question, however, is whether this will still be true when the diet is altered to a more environmentally sustainable one. Is it possible to eat a nutritionally adequate diet in a sustainable way? This overview seeks to describe some of the issues around such dietary changes.

Environmental factors

Planetary boundaries

In general, current sustainable food consumption issues have been focused on climate impact, i.e. in terms of greenhouse gas emission (carbon dioxide equivalents), and less on the effect of toxic impact, biodiversity, eutrophication, acidification, land use, land use change, and water use. Rockström et al. (3) propose a set of nine planetary boundaries within which humanity can continue to develop and thrive for generations to come. Crossing these boundaries, however, could generate abrupt or irreversible environmental changes, and respecting the boundaries reduces the risks to human society of crossing these thresholds. (3). The authors' analysis suggest that three areas are already beyond safe planetary boundaries; the nitrogen cycle, climate change, and biodiversity.

The Millennium Ecosystem Assessment of 2005 (4) concluded that changes in *biodiversity* due to human activities were more rapid in the past 50 years than at any time in human history and that this has increased the risks of abrupt and irreversible changes to ecosystems.

Nitrogen and phosphorus are both essential elements for plant growth so fertilizer production and application is the main concern. Much of this nitrogen is emitted into the atmosphere in various forms rather than being taken up by plants. When nitrogen is washed out of the soil by rain, it pollutes waterways and coastal zones or accumulates in the terrestrial biosphere. Similarly, only a relatively small proportion of phosphorus fertilizers applied to food production systems is taken up by plants, and much of the phosphorus ends up in aquatic systems. These systems can then become oxygen-starved in the process of eutrophication in which bacteria consume the blooms of algae that grow in response to the high nutrient supply. A significant fraction of the nitrogen and phosphorus applied as fertilizer makes its way to the sea and can push marine and aquatic systems across ecological thresholds of their own.

Land is converted to human use across the planet. Forests, wetlands, and other vegetation types have primarily been converted to agricultural land. This *land-use change* is one driving force behind the serious reductions in biodiversity, and it has impacts on water flows and on the cycling of carbon, nitrogen, phosphorus, and other important elements.

Fresh water is becoming increasingly scarce – by 2050 about half a billion people are estimated to be subject to water-stress increasing the pressure to intervene in water systems.

Emissions of *toxic compounds* represent some of the key human-driven changes to the planetary environment. Many of these compounds can persist in the environment for a very long time, and their effects are potentially irreversible. The effects of reduced fertility and the potential of permanent genetic damage can have severe effects on ecosystems.

Recent evidence suggests that the Earth, now passing 387 ppm CO₂ in the atmosphere, has already transgressed the planetary boundary and is approaching several Earth system thresholds. We have reached a point at which the loss of summer polar sea ice is almost certainly irreversible. The weakening or reversal of terrestrial carbon sinks, for example, through the on-going destruction of the world's rainforests, is a primary cause of increased CO₂ levels (3).

Greenhouse emissions

The most widely used measure describing environmental impact is the effect on the climate. The impact on climate is estimated by computing and converting the greenhouse gases into carbon dioxide equivalents (CO₂e) which is the summary measurement of the emissions of carbon dioxide (CO₂), methane (CH₄), nitrous oxide (N₂O), and the extremely powerful refrigerants used to keep fish cold on ships at sea. Greenhouse gases trap global heat and increase the temperature in the planet's atmosphere, and the ability of certain gases to trap this heat is referred to as the compound's global warming potential. Food systems, including food production, food consumption, export, import, transport, storage, and retail, account for about 20%–25% of all greenhouse gas emissions in European countries (5, 6). Emissions of CO₂ are tied to the use of fossil fuels in the production and transport of food. Lately, CO₂ emissions generated from land-use change are an emerging issue (7, 8).

Emissions of CH₄ are tied to production of meat, milk, and rice. CH₄ is generated by decomposing organic material under anaerobic conditions such as digestion by ruminant livestock, storage of manures, and cultivation of rice under flooded conditions. Emissions of N₂O are tied to all crops and animal products. N₂O is generated by the microbial transformation

of nitrogen in soils and manures, and emissions also occur during the production of synthetic fertilizers.

In northern European countries, each person currently contributes an average of at least 10 tonnes of greenhouse gas emissions per year through their food consumption, housing, transport, and shopping habits. Food consumption is estimated to account for approximately 2 tonnes in the Nordic countries (9, 10). The transition toward a competitive low-carbon emission economy means that the EU should prepare for reductions in its domestic emissions by 80% by 2050 compared to 1990. The global increase in temperature might then stay below 2°C. In short, total climate impact should not exceed 2 tonnes of greenhouse gas emissions per person per year (11).

A complex web

Environmental impact is a complex web in which each factor is related to the others in a complicated arrangement of positive and negative effects. Rearing ruminant livestock adds to the production of CH₄ and N₂O emissions and eutrophication (excess nitrogen and/or phosphorous in the water). However, grazing livestock contribute to the maintenance of a rich biodiversity in countries with an abundance of land. It is calculated that greenhouse gas emissions in the European Union from the livestock sector correspond to close to 13% of the total greenhouse gases emitted, including the effects of land use and land-use change. Approximately half of the emissions originate from enteric fermentation (CH₄). Corresponding global emissions from livestock amount to 18% of all the greenhouse gases (12, 13).

Promotion of biodiversity is connected to reduced use of pesticides in agriculture and, when land is abundant, to the grazing of animals. In countries with less land, however, overgrazing threatens biodiversity. When biodiversity is rich, fluctuations and stress are absorbed in the ecosystem – the resilience is good – and biodiversity is a crucial endpoint in many of the environmental measurements. Globally, around 30% of the total human-induced biodiversity loss is related to livestock production. Crop-land and grassland areas are expected to expand by 10%–20% over the coming decades in low-income countries leading to significant losses of terrestrial biodiversity (4). Globally, 80% of commercial fish populations are being fully exploited or overexploited (14).

Food consumption and climate impact

A balanced diet is achieved by choosing foods from several different food groups and not overeating. Foods in the same food group have certain nutritional and other characteristics in common. From a nutritional point of view, this gives many options for choosing similar products within the same food group. However, from the viewpoint of sustainability the choice can have a marked impact.

The value of CO₂e generated during the production-cycle of a food differs widely between foodstuffs but also within each food depending on how it is produced and what energy mix is used. For example, the climate impact of milk varies from 1 kg CO₂e per kg milk in the Nordic countries up to 7 kg CO₂e per kg milk in sub-Saharan Africa (15). Thus milk production is more efficient in the Nordic countries and a larger volume of milk can be produced per the same amount of greenhouse gases emitted from the cows. In Table 1, foods are roughly divided into three groups depending on their climate impact in terms of its CO₂e value or carbon footprint.

The climate impact of food can be related to other units than per weight, for example, per MJ, per gram protein, or per unit of weight of any other nutrient. Combinations of several key nutrients can be added together in an index as illustrated by the comparison between milk and other beverages by Smedman et al. (16).

Food waste

Globally, food waste accounts for roughly one third of the food produced for human consumption (17). The per capita food waste by consumers in Europe and North America is estimated to 95–115 kg per year, but this figure in Sub-Saharan Africa and South/Southeast Asia is only 6–11 kg per year. The average household food waste per person is estimated to about 95 kg in Denmark (18) and to about 75 kg in Sweden (19). Approximately one third of the Swedish waste in the household is potentially edible, unnecessary waste, and the rest of the waste is inedible (banana skins, trimmings, bones, potato peels). A larger proportion of vegetables, fruits, and bread is wasted in comparison to other food groups (WRAP 2008). The climate impact of the 25 kg of unnecessary waste corresponds to approximately 50 kg CO₂e per year or 2.5% of the climate impact of the food used in the household (19).

All values are combined estimates based on Macdiarmid et al (20); Carlsson et al (8); Cederberg et al (13); and various Life Chain Analyses reports on food items (21–24).

Nutritional benefits from a sustainable diet

It has been shown that no food group affects the environment as much as the production of meat and dairy products (25), and their effect on the climate contributes to almost half of the present climate impact from food consumption in the Nordic countries. This is seen in Denmark where calculations on the average diet show that combined meat and dairy products contribute to 53% of the climate impact from the diet (10). The corresponding figure for Sweden is 40% from the national calculations by the Environmental Protection Agency (9). Meat and dairy contribute to almost 2/3 of the intake of protein among adults in the Nordic countries according to recent dietary surveys (26, 34), and the average EU consumption of animal protein per capita is about twice the global average (50).

One way to measure the climate efficiency of protein production is to calculate how much protein is produced per kilogram CO₂e from various food sources. The most efficient protein source is wheat and legumes giving at least 160 g protein per kg CO₂e. Corresponding amounts of protein for other foods are 145 g/kg CO₂e for herring, 53 g/kg CO₂e from eggs, 50 g/kg CO₂e from chicken, and 10 g/CO₂e from beef. These calculations are based on data from life-cycle analyses performed by the Swedish Institute for Food and Biotechnology (SIK) and on nutritional data from the food database of the Swedish National Food Agency (NFA).

For health reasons, a limitation of the consumption of meat products and meat, especially red meat from cows, lambs, goats and pigs, is recommended by the World Cancer Research Fund (WCRF). Few countries achieve the population goal set by WCRF of a maximum of 300 g (400–450 g raw meat) of prepared red meat per week (27). The Nordic consumption of red meat amounts to an average of up to 400 g prepared meat per week. Women in all countries tend to have an average intake closer to the recommended amount than men (28). Red meat is a source of iron, and iron deficiency is estimated to be the most common cause of anaemia in all countries of the world (29). A British report on iron and health emphasizes the importance of a healthy, balanced diet that includes a variety of foods containing iron, and this is more important than focusing primarily on iron from meat sources (30). In a Dutch study, replacement of meat and dairy

by plant-based foods benefited the environment by decreasing land use. On average, total iron intake increased by 2–5 mg/d, although most of the iron intake was from less bioavailable sources (31). In a Nordic study, it was shown that reducing the intake of red meat to the recommended level of 300 g prepared red meat per week (27) did not reduce the intake of iron to any substantial degree. However, the bioavailability of iron in the diet may be affected with a decreased meat intake, depending on which foods replace the meat, an aspect that was not studied. The study also showed that there would be no significant nutritional consequences from reducing the intake of red meat or processed meat to the recommended levels (28).

A shift in the consumption of proteins from animal to vegetable sources would also likely result in reduced consumption of saturated fat. A study from the UK estimated that reducing the overall intake of meat, which also reduced the overall consumption of saturated fat, would lead to a 17% decrease in the number of premature deaths from heart disease among adults (32). Furthermore, it was calculated that a 30% reduction in meat production and subsequent decline in consumption would match nutritional guidelines. In Sweden, 40% of the saturated fat in the diet comes from animal sources excluding fish. In the UK, the intake of saturated fatty acids from meat is 40% higher than in the Nordic countries (26, 33–35).

An increased consumption of dried legumes increases the intake of dietary fibre, folate, carbohydrates (starch), and several other nutrients. The production of pulses has a low climate impact, they are beneficial in crop rotation systems, and they store well. The amino acid content in legumes is not fully balanced, but when combined with the amino acids in cereals or animal sources a satisfactory amino acid combination can be achieved.

Field and root vegetables contribute with vitamins and minerals and are rich in dietary fibre. They tend to store well and their climate impact is low, around 100 g CO₂e or less per kilogram of vegetable. Vegetables grown in heated greenhouses have a higher climate impact and are often more perishable, but positive developments in greenhouse heating technology have been seen, for example, in the Netherlands, Sweden (36), and Finland (37). The climate impact from producing peppers and tomatoes has dramatically declined with the introduction of residual heat use from nearby industries and the use of renewable energy sources. In Iceland, a substantial portion of the tomatoes consumed comes from greenhouses heated by geothermal power.

The climate impacts of reducing body weight and of the extra burden of obesity on the climate have been calculated. A reduction of ten kilogram in

all obese and overweight people would result in a decrease in CO₂ production equal to 0.2% of the CO₂ emitted globally in 2007 (38). Compared with a normal population distribution of BMI, a population with 40% obese requires 19% more food energy for its total energy expenditure (39). Greenhouse gas emission from food production and car travel due to increases in obesity in a population of one billion are estimated to be between 0.4 gigatonnes (GT) and 1.0 GT of CO₂e per year (39).

Healthy and sustainable diets – potential conflicts

Presently there are some areas of potential conflict concerning dietary recommendations and sustainability. One concerns the recommendation of increased fish consumption, the second the restriction of butter, and the third concerns iodine.

An increase in *fish* consumption is recommended because fish in general is a valuable source of vitamin D, selenium, iodine, and long-chain n-3 fatty acids. There are several nutritional reasons to promote increased fish consumption, but from a sustainability point of view there are doubts. Wild fish in general are overexploited, and currently 80% of the fish populations are fully exploited or overexploited (14). Aquaculture is steadily compensating for wild fish, but to an increasing degree plant-based feed is added to the feed given to farmed fish, including predatory fish. Eutrophication is a significant environmental problem associated with aquaculture, and in tropical waters coastal deforestation is a growing issue. In addition, some populations of wild fish, such as herring, are contaminated and restricted consumption is advised despite the availability of strong stocks.

There are few nutritional benefits to the use of butter. The use of palm oil as an ingredient in industrial food products instead of butter or hydrogenated oils has increased but is generally not a sustainable solution. Actors in the Norwegian government and food industries in Nordic countries have taken a firm position against the use of palm oil in ice creams, cookies, spreads, and for frying and this has resulted in a considerably reduced consumption of the oil (40). Other fat sources could be considered and use of more sustainably produced palm oil should be prioritised.

In the Nordic countries, milk and milk products contribute substantially to the intake of *iodine*. Over half of the iodine intake comes from the current intake of milk and milk products in Norway (41), and milk contributes 40% of the daily intake of iodine in Finnish adults (33). To compensate for a lower intake of dairy products, an increased consumption of fish

and shellfish is an option, which is in line with dietary guidelines. Other management options, including fortification, could also be considered.

Experiences of healthy and sustainable diets and potential positive climate impact

How low can we go in terms of climate impact by changing our consumption and still eat a nutritionally adequate diet? Substantial decreases in climate impact – by up to a factor of 10 – have been seen when comparing single meals (8), but more realistic differences are shown when whole diets are compared over time. In Denmark, a diet following the Danish dietary guidelines showed a 4% decrease in climate impact compared to the average diet. If foods with low carbon footprint were chosen from the meat, vegetable, and fruit food groups, the climate impact was reduced by up to 23% in comparison to the average diet, including beverages (10). The total household food waste was estimated to be around 20% and accounted for 12% of the climate impact of food production. The authors of that study concluded that there is a potential synergy between the goal of a healthier diet and the goal of reduced carbon footprint.

In a similar Swedish calculation, a diet including 86 different food items over one week and fulfilling all dietary guidelines would lead to a reduction in climate impact from approximately 1.7–2 tonnes CO₂e for the average diet to 1.4–1.7 tonnes CO₂e per person per year. In addition, choosing foods with a lower climate impact from all food groups and eating less meat and dairy would lead to a climate impact of 0.9–1.3 tonnes CO₂e per person per year. This diet was based on a one-week menu in March containing 110 food items (42). A Dutch study showed that if diets became more plant based it would be possible to achieve a 25% to 50% reduction in climate impact from the diet. In addition, reducing all food waste would lower the climate burden of food by 15% (43). Furthermore, a 25% reduction in climate impact was seen when theoretically shifting the entire Dutch population to a diet with reduced consumption of meat, fish, and eggs. Only a 13% reduction in climate impact was seen when consumption of beef and pork was cut by about half in the entire population (43). In another Dutch study, the potential reduction in climate impact of changing to a healthy diet was greater than that of having a meat-free day. The reduction potential was three times higher if the only meat consumed was chicken and six times higher for a switch to a vegan diet (44).

A 36% reduction in climate impact was seen when a British seven-day

sample menu for adult women meeting the dietary requirements was set (20). The diet contained 52 food items and included meat products in smaller quantities than in the current UK diet. High-fat and sweetened foods were also included, but no alcoholic beverages.

The climate impact from a vegan diet was almost half that of the average German diet, and an ovo-lacto vegetarian diet had 25% lower impact compared to the average diet. The vegan and the lacto-ovo diets followed the dietary patterns issued by US Department of Agriculture/Department of Health and Human Services. A diet that met German recommendations reduced climate impact by 16% compared to the average German diet (45). Participants following a diet with lower climate impact tended to have intakes closer to the Nordic nutrition recommendations than those with a higher climate impact diet. These were results from a 7-day weighed food record from 177 Swedish participants linked to life-cycle assessment data on greenhouse gas emissions for 80 food groups or products (46).

The European Commission's Roadmap 2050 outlines various paths to a decarbonised Europe by 2050 (11). A Swedish study investigated predicted food consumption in the year 2050 in a society not using fossil fuels and calculated that the climate impact of food consumption would be 30% lower than with fossil fuels (47). Further calculations showed that very low meat consumption or a vegan diet together with further improvements in agricultural efficiency could reach a climate impact of 0.3–0.4 tonnes CO₂e per person per year.

Conclusions

Environmental and public health scientists agree that a predominantly plant-based diet is preferable to one largely based on animal sources. The above calculations show that if the climate impact from food production could be reduced by 40%–50% in combination with changes in consumption and production then each individual's contribution could be reduced to as low as 0.5 tonnes CO₂e per year. However, the uncertainty in the calculations is large and comparisons between the studies should be undertaken with caution.

The question, then, becomes whether people will be able to maintain nutritional balance if they alter their diet to a more sustainable one. The change must be made to the entire diet to reflect increases in some foods in response to decreases in others (6). Studies have shown that those following a diet close to the dietary guidelines had a diet with a lower climate

impact. Those who choose a diet with less meat and dairy products have even less of an impact on the climate than those eating the present average diet. With a general vegan diet, it is possible to halve the climate impact from what we eat.

The food choices necessary to reach a more sustainable diet are summarized in Table 1. Existing official dietary guidelines almost all coincide with the dietary changes necessary to achieve an environmentally sustainable diet. However, to reach a more sustainable diet requires more plant-based foods and less animal-based food; choosing primarily meat and fish with low environmental impact; eating more dried beans, peas, lentils, and cereals; choosing mainly field vegetables, root vegetables, potatoes, fruits, and berries that store well; choosing perishable products when they are in season; and minimizing waste. There could be a conflict between nutritionally and environmentally sustainable diets regarding the advice for fish and seafood and for the use of dairy fat in the food industry. The overall conclusion is that there are promising possibilities to eat nutritionally adequate and varied diets in a sustainable way.

Table 1. Possible dietary changes from present average consumption to reach a sustainable diet: Health and environmental impact (10, 48, 49)

Consumption challenges. Change in present average consumption	Health effects		Environmental effects		Comments
	Positive	Negative	Positive	Negative	
Meat and eggs					
Less Meat – Ruminants (beef, sheep, game)	Less saturated fat Decreased cancer risk	Less iron and zinc	Less GHG* Less eutrophication	Lack of grazing animals where land is in abundance results in difficulty keeping the landscape open and varied. This might have a negative impact on biodiversity.	2/3 of all grain produced goes to animal feed. Demand for soy drives deforestation in the Amazon. Combined meat and milk production is more efficient than only meat production.
Less Meat – Pork, poultry	Less saturated fat Decreased cancer risk	Less iron and zinc	Less GHG	Pork and fowl production does not affect biodiversity.	2/3 of grain produced used in animal feed. Demand for soy drives deforestation in the Amazon. Fowl production is very efficient
More eggs	Source of many nutrients		Climate effective	Soy in feed drives deforestation in Amazonas.	Soy can be replaced by domestic legumes.
Dairy products					
Less Dairy Milk	Less saturated fat (if consumption of high-fat dairy is reduced)	Less calcium Less iodine	Less GHG* Less eutrophication	Lack of grazing animals where land is in abundance results in difficulty keeping the landscape open and varied. This might have a negative impact on biodiversity.	Difficult to meet calcium recommendations without milk and dairy products.
Less Dairy Cheese	Less saturated fat Less salt	Less calcium Less iodine	Less GHG* Less eutrophication	Lack of grazing animals where land is in abundance results in difficulty keeping the landscape open and varied. This might have a negative impact on biodiversity.	Difficult to meet calcium recommendations without milk and dairy products.

Fish and shellfish				
More fish and seafood Wild	More unsaturated long-chain fatty acids More vitamin D More selenium More iodine	Risk of being exposed to contam-inants	Increased pressure on fish stocks if not handled with caution following scientific advice. Leaking refrigerants on fishing vessels are strong greenhouse gases. Some fishing methods damage the ocean floor. Fishing methods are not always selective and large amounts of unwanted fish are caught and discarded.	Overexploitation of fish populations is a concern. Scientific advice and policies need to be followed.
More fish and seafood Farmed	More unsaturated long-chain fatty acids More vitamin D More selenium More iodine	More vegetable-based fodder is used, including for predatory fish	Farmed predators need large amount of fodder based on fish. Farmed fish that escape might weaken the wild population's genetic pool. Disease might spread to wild fish. Eutrophication from concentration of fish.	Aquaculture is rapidly increasing. Herbivorous fish species and filtering organisms are interesting options. Microalgae oil with DHA and EPA could be an alternative to consuming fish, but supplements have not been shown to perform as well as the naturally occurring nutrients in fish.
Plant food				
More fruits and berries	More dietary fibre More folate More nutrients overall	Low climate impact	Many fruits and berries are treated with pesticides. Large amounts are imported and transported long distances under climatised conditions.	Numerous foods in this category are transported by air.
More field vegetables including root vegetables	More dietary fibre More folate More nutrients overall	Low climate impact	Many field vegetables are treated with pesticides	Choose those that store well. Many of these vegetables store well.

Consumption challenges. Change in present average consumption	Health effects		Environmental effects		Comments
	Positive	Negative	Positive	Negative	
More, but less fossil fuelled greenhouse-grown, vegetables	More dietary fibre More folate More nutrients overall		Fewer pesticides are used and are often replaced with biological methods in greenhouses.	Fossil fuel is used for heating and cooling greenhouses.	Many greenhouse-grown vegetables are sensitive and require care-ful handling and certain tem-pe-ra-tures during transport. Many do not store well. More fossil free greenhouses are seen.
More potatoes	More dietary fibre More vitamins and minerals		Low climate impact	Potatoes are exposed to many diseases and pesticides are often used.	Store well
More dried legumes	Protein-dense plant food Low fat More dietary fibre More nutrients Overall health-promoting benefits	Might contain anti-nutritional substances and flatulence-promoting oligosaccharides	Nitrogen fixator Beneficial for crop rotation	Pesticide use	Store well Acceptability problems among some consumers
More nuts and seeds	Unsaturated fat Dietary fibre Protein Vitamins and minerals	Prone to mould.			Toxins, e.g. aflatoxins
More cereals and grains	More dietary fibre/whole grain (if the intake of whole-grain cereals is increased) More vitamins and minerals	Increased intake of rapidly absorbed starch, with only minimal amounts of fibre and nutrients (if the intake of refined grain is increased). Prone to mold	Low climate impact for cereals, higher impact for rice.	Production of fertilizers causes emission of N ₂ O. Fertilizer used on fields cause eutrophication and emission of N ₂ O. Pesticide use	Rice production leads to CH ₄ emissions due to anaerobic fermentation under water. Toxins, e.g. DON in cereals.
More organic	None	None	Less or no pesticide is used		Lower production per hectare

Beneficial for soil conditions Promotes biodiversity				
Dietary fats				
More vegetable oils	More unsaturated fats	Rapeseed production is part of agricultural rotation	Pesticide use	
Less butter	Less saturated fat	Less GHG* Less eutrophication	Lack of grazing animals where land is in abundance results in difficulty keeping the landscape open and varied. This might have a negative impact on biodiversity.	
Less palm oil	Less saturated fat	Less pesticide use Less deforestation Biodiversity increases		Palm oil production is very efficient per hectare. Land use change from deforestation is not included in the evaluation. Production of certified (sustainable production) palm oil is small but increasing. Trans fatty acid intake decreased dramatically when palm oil replaced partially hydrogenated fats.
Other				
Less savoury snacks	Less salt and fat			
Less sweets	Less energy (sugar and fat)		Some sweets have high climate impact	This is a large group consisting of a variety of sugary and fatty foods. The nutritional benefit is very low.
More water as drink	Contains no extra energy		The packaging and transport of bottled water has a significant climate impact.	

GHG = greenhouse gases.

Table 2. Climate impact from primary production of food: Low, Medium, and High CO₂e values per kg edible weight

Low < 1 kg CO ₂ e/kg	Medium 1–4 kg CO ₂ e/kg	High > 4 kg CO ₂ e/kg
Field vegetables	Poultry	Beef
Root vegetables	Greenhouse vegetables (heated with fossil fuels)	Lamb
Greenhouse vegetables (heated with renewable resources)	Rice	Pork
Potatoes	Fish	Cheese
Beans, peas, lentils	Vegetable oil (olive, rape)	Tropical fruits and vegetables transported by air
Cereals	Sweets	Butter
Pasta	Snacks	
Bread	Fruits (bananas, melons)	
Fruits, local (apples, pears)	Vegetables imported from a far distance	
Vegetable oil (palm, coconut)	Wine	
Sugar	Eggs	
	Milk, yoghurt	

References

1. Reisch LA. A Definition of “Sustainable Food Consumption”: Copenhagen Business School 2010.
2. SUSFOOD (Sustainable Food production and consumption). Available from: <https://www.susfood-era.net/>
3. Rockstrom J, Steffen W, Noone K, Persson A, Chapin FS, 3rd, Lambin EF, et al. A safe operating space for humanity. *Nature*. 2009 Sep 24;461(7263):472–5.
4. Millennium Ecosystem Assessment. Ecosystems and Human Well-being: Biodiversity Synthesis. Washington, DC: World Resources Institute 2005.
5. The Climate Impact of Swedish Consumption Report. Stockholm: Swedish environmental protection agency (Naturvårdsverket) 2010 Report No.: 5992.
6. Audsley E, Brander M, Chatterton J, Murphy-Boken D, Webster C, Williams A. How low can we go? An assessment of greenhouse gas emissions from the UK food system and the scope reduction by 2050: WWF-UK 2010.
7. Flysjö A, Cederberg C, Henriksson M, Ledgard S. The interaction between milk and beef production and emissions from land use change – critical considerations in life cycle assessment and carbon footprint studies of milk. *Journal of Cleaner Production*. 2012;28(0):134–42.
8. Carlsson-Kanyama A, Gonzalez AD. Potential contributions of food consumption patterns to climate change. *Am J Clin Nutr*. 2009 May;89(5):1704S–9S.
9. Konsumtionens klimatpåverkan. Stockholm: Naturvårdsverket 2008 Report No.: 5903.
10. Thorsen AV, Morgensen L, Jørgensen MS, Trolle E. Klimatorienterede kostråd. Søborg, Denmark: Danmarks Tekniske Universitet (DTU), Fødevareinstituttet 2013.
11. A Roadmap for moving to a competitive low carbon economy in 2050. Brussels 2011 Report No.: COM 112 final.

12. Steinfeld H, Gerber P, Wassenaar T, Castel V, Rosales M, de Haan C. *Livestock's Long Shadow. Environmental Issues and Options*. Rome: FAO, Food and Agriculture Organization of the United Nations 2006.
13. Cederberg C, Berglund M, Gustavsson J, Wallman M. *Environmental impact from livestock production with different animal welfare potentials – a literature review (SIK report number 844)* 2012.
14. *The State of World Fisheries and Aquaculture*. Rome: FAO (Food and Agriculture Organization of the United Nations), Fisheries and Aquaculture Department; 2012.
15. *Greenhouse gas emissions from the dairy sector. A life cycle assessment*. Rome: FAO (Food and Agriculture organization of the United Nations), Animal production and health 2010.
16. Smedman A, Lindmark-Mansson H, Drewnowski A, Edman AK. Nutrient density of beverages in relation to climate impact. *Food Nutr Res*. 2010;54.
17. Gustavsson J, Cederberg C, Sonesson U, van Otterdijk R, Meybeck A. *Global food losses and food waste. Extent, causes and prevention*. Rome, Italy: FAO 2011.
18. *Kortläggning av dagrenovation i enfamilieboliger med särskilt fokus på matspild, batterier och småt elektronikavfald*. Köpenhamn: Miljöministeriet 2012 Report No.: 1414.
19. *Matavfall 2010 från jord till bord: SMED Svenska Miljöemissionsdata 2011* Report No.: 2011–99.
20. Macdiarmid JJ, Kyle J, Horgan GW, Loe J, Fyfe C, Johnstone A, et al. Sustainable diets for the future: Can we contribute to reducing greenhouse gas emissions by eating a healthy diet? *Am J Clin Nutr*. 2012 Sep;96(3):632–9.
21. Nilsson K, Sund V, Florén B. *The environmental impact of the consumption of sweets, crisps and soft drinks* Nordiska Ministerrådet and Livsmedelsverket 2011. Report No.: 509.
22. Lagerberg Fogelberg C. *På väg mot miljöanpassade kostråd: Vetenskapligt underlag inför miljökonsekvensanalysen av Livsmedelsverkets kostråd*. Uppsala: Livsmedelsverket 2008. Report No.: 9.
23. Ziegler F. *På väg mot miljöanpassade kostråd: Delrapport fisk*. Uppsala: Livsmedelsverket 2008. Report No.: 10.
24. Rööf E. *Mat-klimat-listan. Version 1.0*. Uppsala: Institutionen för energi och teknik. SLU (Sveriges Lantbruksuniversitet) 2012. Report No.: 040.
25. *Livestock's long shadow – environmental issues and options*. Rome: FAO 2006.
26. Amcoff E, Edberg A, Enghardt Barbieri H. *Riksmaten vuxna 2010–11. Livsmedels- och näringsintag bland vuxna i Sverige. Resultat från matvaneundersökningen utförd 2010–11 (Food and nutrient intake in Sweden 2010–11. (In Swedish, summary, figures and tables in English)* Uppsala: Livsmedelsverket 2012.
27. *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective*. Washington DC: World Cancer Research Fund, American Institute for Cancer Research 2007.
28. Tetens I, Hoppe C, Frost Andersen L, Helldán A, Warensjö Lemming E, Trolle E, et al. *Nutritional evaluation of lowering consumption of meat and meat products in the Nordic context* Nordic Council of Ministers 2013 Report No.: 2013:506.
29. *Joint WHO/FAO expert consultation on Diet, nutrition and the prevention of chronic diseases*. Geneva: WHO/FAO 2002 Report No.: 916.
30. *Iron and Health*. London: SACN (Scientific Advisory Committee on Nutrition of the UK Department of Health) 2010.
31. Temme EH, van der Voet H, Thissen JT, Verkaik-Kloosterman J, van Donkersgoed G, Nonhebel S. Replacement of meat and dairy by plant-derived foods: estimated effects on land use, iron and SFA intakes in young Dutch adult females. *Public Health Nutr*. 2013 Oct;16(10):1900–7.
32. Friel S, Dangour AD, Garnett T, Lock K, Chalabi Z, Roberts I, et al. Public health benefits of strategies to reduce greenhouse-gas emissions: food and agriculture. *Lancet*. 2009 Dec 12;374(9706):2016–25.
33. Helldán A, Kosonen M, Tapanainen H. *The National FINDIET 2012 Survey. (In Finnish, summary, figures and tables in English)* Helsinki: National Institute For Health and Welfare 2013 report No.: 16/2013.
34. Pedersen AN, Fagt S, Velsing Groth M. *Danskernes kostvaner 2003–2008. Hovedresultater (Dietary habits of Danes 2003–2008. Main results)*: DTU Fødevareinstituttet 2010.

35. Totland TH, Kjerpeseth Melnæs B, Lundberg-Hallén N. En landsomfattende kostholdsundersøkelse blant menn og kvinner i Norge i alderen 18–70 år 2010–11. Oslo: Helsedirektoratet 2012 Report No.: 06/2000.
36. Statistik från Jordbruksverket. Energianvändning i växthus 2011. Tomat, gurka och prydnadsväxter (Greenhouse energy use in 2011): Jordbruksverket 2012 Report No.: 2012:05.
37. Yrjänäinen H, Silvenius F, Kaukoranta T, Näkkilä J, Särkkä L, Tuhkanen E-M. Kasvihuonetuotteiden ilmastovaikutuskanta: Loppuraportti. (Greenhouse, environmental impacts, carbon footprint, tomato, cucumber, salad, tulip, pot plant) 2013 Report No.: 83.
38. Gryka A, Broom J, Rolland C. Global warming: is weight loss a solution? *Int J Obes (Lond)*. 2012 Mar;36(3):474–6.
39. Edwards P, Roberts I. Population adiposity and climate change. *Int J Epidemiol*. 2009 Aug;38(4):1137–40.
40. Hermansen JE, Knudsen MT, Sörensen J. Soja og palmeolie. Certificeringsordninger til dokumentation af bæredygtighed i forbindelse med produktion. Nationelt Center for Fødevarer og Jordbrug, Aarhus universitet 2013 Report No.: 020 marts 2013.
41. Brantsæter A, Abel M, Haugen M, Meltzer H. Risk of Suboptimal Iodine Intake in Pregnant Norwegian Women. *Nutrients*. 2013;5(2):424–40.
42. Hur liten kan livsmedelsproduktionens klimatpåverkan vara år 2050? Ett diskussionsunderlag om vad vi äter 2050: Jordbruksverket 2013.
43. Milieuanalyses Voedsel and Voedselverliezen. Ten behoeve van prioritaire stromen ketengericht afvalbeleid, Delft (Environmental analysis of food and food waste. LCA-based guidelines for waste management): CE and Blonk Milieu Advies 2010.
44. Blonk H, Kool A, Luske B, S dW. Environmental effects of protein rich food products in the Netherlands: Consequences of animal protein substitutes. Gouda: Blonk Milieu Advies 2008.
45. Meier T, Christen O. Environmental impacts of dietary recommendations and dietary styles: Germany as an example. *Environ Sci Technol*. 2013 Jan 15;47(2):877–88.
46. Ekström S. Greenhouse gas emissions and food consumption: a study of sustainable food habits in Sweden: Karolinska Institute and Stockholm University; 2012.
47. Bryngelsson D, Hedenus F, J. L. Scenarier för klimatpåverkan från matkonsumtionen 2050. Göteborg: Chalmers 2013.
48. Lagerberg Fogelberg C. På väg mot miljöanpassade kostråd. Uppsala, Sverige 2008 Report No.: 9/2008.
49. Ziegler F. På väg mot miljöanpassade kostråd – fisk. Uppsala, Sverige 2008 Report No.: 10/2008.
50. <http://faostat.fao.org/site/368/default.aspx#ancor7> Fluid and water balance.

14 Dietary antioxidants

Introduction

Free radicals and other reactive oxygen and nitrogen species (ROS and RNS) are formed continuously in the body, partially through the normal cellular oxidative metabolic reactions, that are required to maintain homeostasis. Such reactive species also develop as the result of diseases (e.g., during inflammation) as well as exposure to tobacco smoke, environmental pollutants, drugs, radiation, excessive alcohol consumption, and other unknown factors. Antioxidants are substances that delay or prevent (quench) the oxidation of oxidisable substrates such as lipids, carbohydrates, proteins, and DNA. If not adequately quenched by endogenous or exogenous antioxidants, the free radicals can react with and potentially alter the structure and function of cellular components such as lipid-containing cell membranes, lipoproteins, proteins, carbohydrates, RNA, and DNA. Many substances show antioxidative properties *in vitro* but do not necessarily show such activity *in vivo*.

Oxidative stress results when the critical balance between the endogenous generation of ROS or RNS and antioxidant defences is severely disrupted, rather than when the balance is only mildly affected for a short time period (1–5). Studies from the past three decades have shown that such oxidative damage or oxidative stress is involved in the pathophysiology of many otherwise unrelated types of disease. Oxidative stress has been associated with inflammatory diseases, ischemic diseases (heart disease, stroke, and intestinal ischemia), cancer, obesity-related diseases (dyslipidaemia, vascular inflammation, hypertension, and diabetes), hypertension and preeclampsia, and neurological diseases (multiple sclerosis, Alzheimer's disease, and Parkinson's disease) (1, 2, 5–8).

A complex endogenous antioxidant defence system has evolved in all living organisms to counteract oxidative stress and is essential for all aerobic cells. Both enzymatic and non-enzymatic processes prevent radical forma-

tion, remove radicals before damage can occur, repair oxidative damage, eliminate damaged molecules, and prevent mutations (1, 8, 9).

In addition to the endogenous antioxidants, diet might also contribute to the antioxidant defence system of the body. The roles of vitamin C, vitamin E, and selenium as antioxidants are discussed in their respective chapters. Recent research has shown, however, that whole plants and plant extracts contain numerous known and unidentified compounds with antioxidant properties (10). The role of these dietary antioxidants is not well understood because evaluation of their impact on health and disease is difficult, but they are potentially important modulators of several diseases related to oxidative stress.

Types and food sources of compounds with antioxidative properties

Foods containing high levels of total antioxidants include several berries (blueberries, blackberries, strawberries, and raspberries), fruits (pomegranates, grapes, and oranges), nuts and seeds (walnuts and sunflower seeds), vegetables (kale, red cabbage, and pepper), and drinks (green tea and red wine) (11, 12). The dietary antioxidants in fruits and vegetables might contribute to two important components of the antioxidant defence system: 1) the ability to scavenge or neutralize free radicals directly and 2) the ability to induce endogenous antioxidants. In this context, the redox process, where both oxidation and reduction occur simultaneously, is system dependent. The redox-active compound might be an antioxidant in one system (such as a plant subcellular system or an *in vitro* system) but inactive or a pro-oxidant in another biological system (13).

Compounds with ability to scavenge or neutralize free radicals

Carotenoids are ubiquitous in the plant kingdom, and more than 1000 naturally occurring variants have been identified. Carotenoids are good examples of compounds that both inhibit and enhance oxidation depending on the biological system (14). About 600 different carotenoids have been characterised in plants, and our diet contains at least 60 of these (9). The main carotenoids present in the diet are the provitamin A carotenoids α - and β -carotene and β -cryptoxanthin; lycopene; and the hydroxy-carotenoids (xanthophylls) lutein and zeaxanthin. In plants, carotenoids are auxiliary light harvesting components, and they quench excited molecules formed during photosynthesis (1, 8, 9).

Phenolic compounds are also ubiquitous in many edible plants (9). They are synthesised as various types of molecular families such as benzoic acid derivatives, flavonoids, proanthocyanidins, stilbenes, coumarins, lignans, and lignins. Over 8,000 plant phenols have been isolated. Plant phenols are antioxidants by virtue of the hydrogen-donating properties of the phenolic hydroxyl groups (9).

Glutathione, the major cellular antioxidant, is present in abundant amounts in the diet, although it is not absorbed as such from the diet but is broken down into its constituent amino acids during digestion. However, the dietary availability of sulfur amino acids can modulate cellular glutathione production (1, 8, 9, 15, 16).

Antioxidants from the different molecular families with different chemical and biochemical properties likely activate each other through complex and integrated processes. Packer and colleagues (16) have shown such in vitro interactions for α -tocopherol, α -tocotrienol, ascorbic acid, lipoic acid, and thiols, but Buettner (15) has suggested that the concept could have much broader applicability. This implies that a variety of antioxidants is necessary to maintain the proper endogenous antioxidant defence system. Thus, incorporating the appropriate amount and balance of antioxidants (i.e., electron- or hydrogen-donating reductants) in the diet might be a better concept than focusing on individual dietary antioxidants.

The antioxidant defence system in humans consists of both antioxidants obtained from the diet and endogenous antioxidants generated in the body. However, the antioxidant defence capacity in a plant is not necessarily relevant to human health (17). The methods used to assess the total antioxidant capacity of dietary plants could produce different results when applied to plasma or bodily fluids in humans (17–22). These methods are based on the different chemical properties of the antioxidants, and the ability to detect both water- and fat-soluble antioxidants varies with the substrate.

Compounds with ability to induce antioxidant enzymes

An additional antioxidant defence mechanism involves the induction of several classes of detoxification enzymes.

Phase 2 enzymes. One family of enzymes includes γ -glutamylcysteine synthetase, which is required for the synthesis of the endogenous antioxidant glutathione, glutathione reductase for glutathione reformation, and NAD(P)H:quinone reductase for inhibition of redox cycling (23, 24). These enzymes

are generally referred to as phase 2 enzymes because they are regulated along with enzymes that catalyse the conversion of xenobiotics, which are mutagenic metabolites, or their precursors into compounds that are more readily excreted. It is thought that if benign or non-damaging plant compounds induce the phase 2 enzymes, cells are better able to 'neutralize' toxic agents such as free radicals and other toxic electrophiles when they appear.

Glucosinolates and several other sulfur-containing plant compounds are the major plant compounds believed to support antioxidant defence through this mechanism. Glucosinolates are common components of plants, and glucosinolate breakdown products (such as the isothiocyanate sulforaphane) are thought to induce phase 2 enzymes and are, therefore, responsible for the protective effects shown by Brassica vegetables (9, 23, 24). Dietary plants rich in compounds that induce phase 2 detoxification enzymes include broccoli, Brussels sprouts, cabbage, kale, cauliflower, carrots, onions, tomatoes, spinach, and garlic. However, the evidence for phase 2 enzyme induction at ordinary intake levels of plant foods in humans is limited, and the importance of this defence mechanism in the overall protection against oxidative damage is still uncertain.

Catalase, glutathione peroxidases, superoxide dismutases, paraoxonase, and several other enzymes are included in another important but heterogeneous class of antioxidant enzymes that are directly involved in the removal of reactive oxygen species. Several of the enzymes in this class are inducible by factors in food. For example, glutathione peroxidase 1 is induced by increased intakes of fruit, berries, vegetables, and many other polyphenol-rich foods (25).

Both of the above enzyme classes include selenoenzymes, and the control of their regulation is related to selenium homeostasis. Selenium (see Selenium chapter) is sometimes referred to as an antioxidant because it is present in these enzymes. Several genetic variants of these enzymes have been shown to alter the risk of disease (26, 27) suggesting that enzymes from both groups are important for the defence against oxidative stress.

Effects of antioxidant-rich diets in experimental animal and human studies

The suggested role of dietary antioxidants in protection against oxidative damage is often based on extensive studies in cell culture systems. However, it is uncertain whether the effects of antioxidants observed in cell cultures, often with high doses of single compounds, can be extrapolated to humans. For example, cell culture studies do not usually show how the phytochemicals are processed *in vivo*, how they are absorbed and metabolised in the body, or whether they are available to the tissues of interest. Antioxidant-rich diets also contain many compounds that are not redox active, so other mechanisms might also provide benefits. These mechanisms might be involved in interventions with plant compounds, but direct proof is still lacking. Direct proof would be obtained only from studies in which different and well-defined preparations of antioxidants are shown to protect a specific molecular target from oxidation and that this target is shown to be beneficial to health. In these complex circumstances, long-term adequate clinical studies on intake of antioxidant-rich diets are necessary. Further, suitable gold-standard biomarkers of oxidative stress *in vivo* are needed to clarify the health benefits of antioxidants.

Initial experimental dietary studies have confirmed the beneficial effects of dietary plants rich in either phytochemicals that scavenge free radicals or phytochemicals that induce phase 2 enzymes. A diet of strawberries, spinach, and blueberries retarded and reversed age-related neurodegeneration in rats (28, 29). Antioxidant-rich raspberries and strawberries (30, 31) also efficiently inhibited carcinogenesis in experimental animals. Walnuts (32, 33) and pomegranates (34, 35), which are exceptionally rich in scavenging antioxidants, reduced LDL oxidation and atherosclerosis-related processes in animals and humans. Brussels sprouts, onions, and tomatoes have been shown to reduce the excretion of 8-oxo-deoxyguanosine (8-oxo-dG), a biomarker for oxidative free radical DNA damage, into urine and to reduce the level of DNA damage in lymphocytes in animals and humans (36–40). However, in some rigidly controlled studies, little effect was observed after intervention with plant food items rich in antioxidants (41, 42). Low bio-availability and extensive metabolism of some plant-derived antioxidants might explain some of these discrepancies.

Many of the reviewed studies were performed with animals, and relating these beneficial findings to human studies is complicated because of the differences in absorption, distribution, metabolism, excretion, gut

microbiota, endogenous antioxidant systems, and inappropriate doses. In addition, interpreting studies of complex foods such as berries, fruits, and vegetables is difficult due to the lack of comparable controls. With these considerations in mind, detailed clinical studies of longer duration and inclusion of new biochemical technologies (including metabolomics, proteomics, genomics, etc.) are needed to confirm the beneficial effects of antioxidants and polyphenols (43).

Intervention trials with antioxidant supplements

A protective effect of antioxidant supplements such as vitamin E, vitamin C, or β -carotene has not been conclusively shown in intervention trials. Indeed, supplementation with antioxidants has often resulted in no effect or even adverse disease outcomes in clinical trials. A review concluded that no studies so far have convincingly shown that giving vitamin C, vitamin E, or β -carotene supplements to non-depleted humans affects biomarkers of oxidative stress (44).

The majority of such clinical trials have examined effects of treatment with high doses of supplements containing antioxidant vitamins, and study participants have often been at high risk for, or have been recovering from, acute chronic disease events. Therefore, less is known about the health protection of low dose antioxidant supplements (similar to doses found in everyday diets) in essentially healthy individuals. However, a recent study in which different antioxidant supplements extracted mainly from fruit, berries, and vegetables were given to overweight men for six weeks and to type 2 diabetes patients for twelve weeks did not show any effect on F_2 -isoprostanes or 8-OHdG, the two currently reliable biomarkers of in vivo oxidative stress (45, 46).

Results from the Finnish Alpha-Tocopherol, Beta-Carotene Cancer Prevention (ATBC) Study of smoking men who received a daily supplement of α -tocopherol (50 mg), β -carotene (20 mg), both α -tocopherol and β -carotene, or a placebo for 5–8 years (median 6.1 years) showed that β -carotene supplementation was associated with about a 20% increase in lung cancer risk (47). Rapola et al. (48) found significantly higher coronary heart disease mortality among the men in the β -carotene groups who had a previous myocardial infarction. Similar findings were observed in the Beta-Carotene and Retinol Efficacy Trial (CARET) by Omenn et al. (49) who tested a combination of 30 mg β -carotene and 25,000 IU retinyl palmitate taken daily against a placebo in 18,314 men and women at high risk of developing

lung cancer. The CARET intervention was stopped 21 months early because of clear evidence of no benefit and substantial evidence of possible harm; there were 28% more lung cancers and 17% more deaths in the active intervention group that was administered the combination of β -carotene and retinyl palmitate. The CARET study also found that the active treatment group had a 26% increase in relative risk of death from cardiovascular disease (49). In a study that included 160 patients with coronary disease, Brown et al. (50) observed that antioxidants (100 mg selenium, 1 g vitamin C, 800 IU vitamin E, and 25 mg β -carotene) had no effect alone, but they decreased the protective effects of simvastatin on both lipid markers and clinical endpoints. Waters et al. (51) also observed potential adverse effects of antioxidants (800 IU vitamin E and 1 g vitamin C per day) in a study of coronary atherosclerosis in 423 postmenopausal women with coronary stenosis. Finally, Graat et al. (52) studied the effect of antioxidant supplement on immune response in 652 non-institutionalised individuals aged 60 years or older. They observed that the individuals treated with 200 mg vitamin E per day had an increased severity of infections compared to the controls.

Several studies have shown no clinical effects of antioxidant treatment. For example, the MRC/BHF Heart Protection Study (53), which included 20,536 adults with coronary disease, observed that an antioxidant mixture (600 mg vitamin E, 250 mg vitamin C, and 20 mg β -carotene) improved plasma biomarkers but had no effects on clinical endpoints. Furthermore, the Heart Outcomes Prevention Evaluation (HOPE) Study (54), in which 9,541 men and women 55 years of age and older who were at high risk for cardiovascular events were enrolled, observed no significant effects of a daily 400 IU vitamin E supplement given for a mean of 4.5 years.

There have, however, been studies that have shown positive effects of antioxidants on clinical endpoints. The Cambridge Heart Antioxidant Study (CHAOS) (55), which consisted of 1,035 patients with coronary atherosclerosis who received either 800 IU vitamin E or a placebo, observed that vitamin E reduced the rate of non-fatal myocardial infarction after one year of treatment. A non-significant increase in all-cause mortality was also seen. Increased mortality was suggested by a study of subjects who received vitamin E in doses of 400 IU per day or higher (135,967 participants in 19 clinical trials; (18). In addition, in the Antioxidant Supplementation in Atherosclerosis Prevention (ASAP) Study (n = 520), retarded progression of carotid atherosclerosis was observed in men, but not in women, after treatment with 182 mg α -tocopherol and 500 mg vitamin C per day for three years (56).

Several meta-analyses of RCTs with supplements with high doses of nutrients with antioxidant properties have shown no protective effects on CVD, gastrointestinal cancer or mortality (18, 57–59). Analysis of 47 high-quality studies included in the meta-analysis by Bjelakovic et al. (58) showed a significant increased risk of total mortality for β -carotene (7%), retinol (16%) and tocopherol (4%). Results from subsequent studies have failed to show any protection against cancer or CVD (60–62).

Dietary antioxidants and health

There is a large body of evidence that a diet rich in fruits, berries, vegetables, pulses, nuts, and seeds reduces the risk of cardiovascular disease, cancer, and other chronic diseases associated with major oxidative stress (11, 12, 63, 64). However, the NNR systematic review on foods graded the evidence for a protective effect of berries per se as limited-inconclusive due to too few studies (65). There is insufficient scientific evidence to show that the antioxidative mechanisms are specifically involved in the protective effects of fruits, berries, and vegetables. Severe difficulties to reliably determine oxidative stress in vivo still remain because of the complexities associated with measuring free radical reactions and defining them correctly for different biological conditions. For instance, the variable effects of fruit, berry, and vegetable intake on oxidative stress markers seen in many observational studies possibly depend on the different assays used (25, 66, 67). Therefore, recommendations for specific antioxidant-rich fruits and vegetables beyond the ordinary dietary recommendations cannot be given at this time. Several governmental and non-governmental organizations (68–72) do not recommend the intake of supplemental antioxidants either individually or in combination.

Conclusion

There is a large body of evidence suggesting that elevated intakes of certain supplements, mainly vitamins with antioxidative properties, might increase the risk of certain adverse health effects, including mortality. Thus, there is no scientific justification for using supplements as a tool for adjusting an unbalanced diet.

References

1. Gutteridge JM, Halliwell B. Free radicals and antioxidants in the year 2000. A historical look to the future. *Ann N Y Acad Sci*. 2000;899:136–47.
2. Sies H. Oxidative stress: oxidants and antioxidants. *Exp Physiol*. 1997 Mar;82(2):291–5.
3. Halliwell B. Free radicals, antioxidants, and human disease: curiosity, cause, or consequence? *Lancet*. 1994 Sep 10;344(8924):721–4.
4. Sies H. Oxidative stress: introductory remarks. *Oxidative stress*. London, UK: Academic Press; 1985. pp. 1–8.
5. Basu S. F2-isoprostanes in human health and diseases: from molecular mechanisms to clinical implications. *Antioxid Redox Signal*. 2008 Aug;10(8):1405–34.
6. McCord JM. The evolution of free radicals and oxidative stress. *Am J Med*. 2000 Jun 1;108(8):652–9.
7. Beckman KB, Ames BN. The free radical theory of aging matures. *Physiol Rev*. 1998 Apr;78(2):547–81.
8. Halliwell B. Antioxidants in human health and disease. *Annu Rev Nutr*. 1996;16:33–50.
9. Lindsay DG, Astley SB. European research on the functional effects of dietary antioxidants – EUROFEDA. *Mol Aspects Med*. 2002 Feb-Jun;23(1–3):1–38.
10. Halliwell B. Reactive species and antioxidants. Redox biology is a fundamental theme of aerobic life. *Plant Physiol*. 2006 Jun;141(2):312–22.
11. Blomhoff R, Lande G, Ose T. Nye anbefalinger for inntak av frukt og grønnsaker. Oslo: Statens Ernæringsråd 1995.
12. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: World Cancer Research Fund, American Institute for Cancer Research 2007.
13. Rietjens IM, Boersma MG, Haan L, Spenkelink B, Awad HM, Cnubben NH, et al. The pro-oxidant chemistry of the natural antioxidants vitamin C, vitamin E, carotenoids and flavonoids. *Environ Toxicol Pharmacol*. 2002 Jul;11(3–4):321–33.
14. El-Agamey A, Lowe GM, McGarvey DJ, Mortensen A, Phillip DM, Truscott TG, et al. Carotenoid radical chemistry and antioxidant/pro-oxidant properties. *Arch Biochem Biophys*. 2004 Oct 1;430(1):37–48.
15. Buettner GR. The pecking order of free radicals and antioxidants: lipid peroxidation, alpha-tocopherol, and ascorbate. *Arch Biochem Biophys*. 1993 Feb 1;300(2):535–43.
16. Packer L, Weber SU, Rimbach G. Molecular aspects of alpha-tocotrienol antioxidant action and cell signalling. *J Nutr*. 2001 Feb;131(2):369S–73S.
17. Sies H. Total antioxidant capacity: appraisal of a concept. *J Nutr*. 2007 Jun;137(6):1493–5.
18. Miller ER, 3rd, Pastor-Barriuso R, Dalal D, Riemersma RA, Appel LJ, Guallar E. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. *Ann Intern Med*. 2005 Jan 4;142(1):37–46.
19. Benzie IF, Strain JJ. The ferric reducing ability of plasma (FRAP) as a measure of “antioxidant power”: the FRAP assay. *Anal Biochem*. 1996 Jul 15;239(1):70–6.
20. Cao G, Alessio HM, Cutler RG. Oxygen-radical absorbance capacity assay for antioxidants. *Free Radic Biol Med*. 1993 Mar;14(3):303–11.
21. DeLange RJ, Glazer AN. Phycoerythrin fluorescence-based assay for peroxy radicals: a screen for biologically relevant protective agents. *Anal Biochem*. 1989 Mar;177(2):300–6.
22. Halvorsen BL, Holte K, Myhrstad MC, Barikmo I, Hvattum E, Remberg SF, et al. A systematic screening of total antioxidants in dietary plants. *J Nutr*. 2002 Mar;132(3):461–71.
23. Fahey JW, Zhang Y, Talalay P. Broccoli sprouts: an exceptionally rich source of inducers of enzymes that protect against chemical carcinogens. *Proc Natl Acad Sci U S A*. 1997 Sep 16;94(19):10367–72.
24. Talalay P. Chemoprotection against cancer by induction of phase 2 enzymes. *Biofactors*. 2000;12(1–4):5–11.
25. Dragsted LO, Pedersen A, Hermetter A, Basu S, Hansen M, Haren GR, et al. The 6-a-day study: effects of fruit and vegetables on markers of oxidative stress and antioxidative defense in healthy nonsmokers. *The American journal of clinical nutrition*. 2004 Jun;79(6):1060–72.

26. Seow A, Yuan JM, Sun CL, Van Den Berg D, Lee HP, Yu MC. Dietary isothiocyanates, glutathione S-transferase polymorphisms and colorectal cancer risk in the Singapore Chinese Health Study. *Carcinogenesis*. 2002 Dec;23(12):2055–61.
27. Ravn-Haren G, Olsen A, Tjønneland A, Dragsted LO, Nexø BA, Wallin H, et al. Associations between GPX1 Pro198Leu polymorphism, erythrocyte GPX activity, alcohol consumption and breast cancer risk in a prospective cohort study. *Carcinogenesis*. 2006 Apr;27(4):820–5.
28. Joseph JA, Shukitt-Hale B, Denisova NA, Bielinski D, Martin A, McEwen JJ, et al. Reversals of age-related declines in neuronal signal transduction, cognitive, and motor behavioral deficits with blueberry, spinach, or strawberry dietary supplementation. *J Neurosci*. 1999 Sep 15;19(18):8114–21.
29. Youdim KA, Joseph JA. A possible emerging role of phytochemicals in improving age-related neurological dysfunctions: a multiplicity of effects. *Free Radic Biol Med*. 2001 Mar 15;30(6):583–94.
30. Xue H, Aziz RM, Sun N, Cassady JM, Kamendulis LM, Xu Y, et al. Inhibition of cellular transformation by berry extracts. *Carcinogenesis*. 2001 Feb;22(2):351–6.
31. Kresty LA, Morse MA, Morgan C, Carlton PS, Lu J, Gupta A, et al. Chemoprevention of esophageal tumorigenesis by dietary administration of lyophilized black raspberries. *Cancer Res*. 2001 Aug 15;61(16):6112–9.
32. Sabate J. Nut consumption, vegetarian diets, ischemic heart disease risk, and all-cause mortality: evidence from epidemiologic studies. *Am J Clin Nutr*. 1999 Sep;70(3 Suppl):500S–3S.
33. Anderson KJ, Teuber SS, Gobeille A, Cremin P, Waterhouse AL, Steinberg FM. Walnut polyphenolics inhibit in vitro human plasma and LDL oxidation. *J Nutr*. 2001 Nov;131(11):2837–42.
34. Aviram M, Dornfeld L, Rosenblat M, Volkova N, Kaplan M, Coleman R, et al. Pomegranate juice consumption reduces oxidative stress, atherogenic modifications to LDL, and platelet aggregation: studies in humans and in atherosclerotic apolipoprotein E-deficient mice. *The American Journal of Clinical Nutrition*. 2000 May 1, 2000;71(5):1062–76.
35. Kaplan M, Hayek T, Raz A, Coleman R, Dornfeld L, Vaya J, et al. Pomegranate juice supplementation to atherosclerotic mice reduces macrophage lipid peroxidation, cellular cholesterol accumulation and development of atherosclerosis. *J Nutr*. 2001 Aug;131(8):2082–9.
36. Verhagen H, de Vries A, Nijhoff WA, Schouten A, van Poppel G, Peters WH, et al. Effect of Brussels sprouts on oxidative DNA-damage in man. *Cancer Lett*. 1997 Mar 19;114(1–2):127–30.
37. Verhagen H, Poulsen HE, Loft S, van Poppel G, Willems MI, van Bladeren PJ. Reduction of oxidative DNA-damage in humans by brussels sprouts. *Carcinogenesis*. 1995 Apr;16(4):969–70.
38. Lampe JW. Health effects of vegetables and fruit: assessing mechanisms of action in human experimental studies. *Am J Clin Nutr*. 1999 Sep;70(3 Suppl):475S–90S.
39. Lean ME, Noroozi M, Kelly I, Burns J, Talwar D, Sattar N, et al. Dietary flavonols protect diabetic human lymphocytes against oxidative damage to DNA. *Diabetes*. 1999 Jan;48(1):176–81.
40. Boyle SP, Dobson VL, Duthie SJ, Kyle JA, Collins AR. Absorption and DNA protective effects of flavonoid glycosides from an onion meal. *Eur J Nutr*. 2000 Oct;39(5):213–23.
41. van het Hof KH, de Boer HS, Wiseman SA, Lien N, Weststrate JA, Tijburg LB. Consumption of green or black tea does not increase resistance of low-density lipoprotein to oxidation in humans. *Am J Clin Nutr*. 1997 Nov;66(5):1125–32.
42. Young JF, Dragsted LO, Haraldsdottir J, Daneshvar B, Kall MA, Loft S, et al. Green tea extract only affects markers of oxidative status postprandially: lasting antioxidant effect of flavonoid-free diet. *Br J Nutr*. 2002 Apr;87(4):343–55.
43. Biesalski HK. Polyphenols and inflammation: basic interactions. *Current opinion in clinical nutrition and metabolic care*. 2007 Nov;10(6):724–8.
44. Dragsted LO. Biomarkers of exposure to vitamins A, C, and E and their relation to lipid and protein oxidation markers. *Eur J Nutr*. 2008 May;47 Suppl 2:3–18.

45. Rytter E, Johansson C, Vessby B, Sjodin A, Moller L, Akesson B, et al. Biomarkers of oxidative stress in overweight men are not influenced by a combination of antioxidants. *Free Radical Research*. 2010 May;44(5):522–8.
46. Rytter E, Vessby B, Asgard R, Ersson C, Moussavian S, Sjodin A, et al. Supplementation with a combination of antioxidants does not affect glycaemic control, oxidative stress or inflammation in type 2 diabetes subjects. *Free Radic Res*. 2010 Dec;44(12):1445–53.
47. Albanes D, Heinonen OP, Taylor PR, Virtamo J, Edwards BK, Rautalahti M, et al. Alpha-Tocopherol and beta-carotene supplements and lung cancer incidence in the alpha-tocopherol, beta-carotene cancer prevention study: effects of base-line characteristics and study compliance. *J Natl Cancer Inst*. 1996 Nov 6;88(21):1560–70.
48. Rapola JM, Virtamo J, Ripatti S, Huttunen JK, Albanes D, Taylor PR, et al. Randomised trial of alpha-tocopherol and beta-carotene supplements on incidence of major coronary events in men with previous myocardial infarction. *Lancet*. 1997 Jun 14;349(9067):1715–20.
49. Omenn GS, Goodman GE, Thornquist MD, Balmes J, Cullen MR, Glass A, et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. *N Engl J Med*. 1996 May 2;334(18):1150–5.
50. Brown BG, Zhao XQ, Chait A, Fisher LD, Cheung MC, Morse JS, et al. Simvastatin and niacin, antioxidant vitamins, or the combination for the prevention of coronary disease. *N Engl J Med*. 2001 Nov 29;345(22):1583–92.
51. Waters DD, Alderman EL, Hsia J, Howard BV, Cobb FR, Rogers WJ, et al. Effects of hormone replacement therapy and antioxidant vitamin supplements on coronary atherosclerosis in postmenopausal women: a randomized controlled trial. *JAMA*. 2002 Nov 20;288(19):2432–40.
52. Graat JM, Schouten EG, Kok FJ. Effect of daily vitamin E and multivitamin-mineral supplementation on acute respiratory tract infections in elderly persons: a randomized controlled trial. *JAMA*. 2002 Aug 14;288(6):715–21.
53. MRC/BHF Heart Protection Study of antioxidant vitamin supplementation in 20,536 high-risk individuals: a randomised placebo-controlled trial. *Lancet*. 2002 Jul 6;360(9326):23–33.
54. Yusuf S, Dagenais G, Pogue J, Bosch J, Sleight P. Vitamin E supplementation and cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. *N Engl J Med*. 2000 Jan 20;342(3):154–60.
55. Stephens NG, Parsons A, Schofield PM, Kelly F, Cheeseman K, Mitchinson MJ. Randomised controlled trial of vitamin E in patients with coronary disease: Cambridge Heart Antioxidant Study (CHAOS). *Lancet*. 1996 Mar 23;347(9004):781–6.
56. Salonen RM, Nyyssonen K, Kaikkonen J, Porkkala-Sarataho E, Voutilainen S, Rissanen TH, et al. Six-year effect of combined vitamin C and E supplementation on atherosclerotic progression: the Antioxidant Supplementation in Atherosclerosis Prevention (ASAP) Study. *Circulation*. 2003 Feb 25;107(7):947–53.
57. Huang HY, Caballero B, Chang S, Alberg AJ, Semba RD, Schneyer CR, et al. The efficacy and safety of multivitamin and mineral supplement use to prevent cancer and chronic disease in adults: a systematic review for a National Institutes of Health state-of-the-science conference. *Ann Intern Med*. 2006 Sep 5;145(5):372–85.
58. Bjelakovic G, Nikolova D, Gluud LL, Simonetti RG, Gluud C. Mortality in randomized trials of antioxidant supplements for primary and secondary prevention: systematic review and meta-analysis. *JAMA*. 2007 Feb 28;297(8):842–57.
59. Bjelakovic G, Nikolova D, Simonetti RG, Gluud C. Antioxidant supplements for preventing gastrointestinal cancers. *Cochrane Database Syst Rev*. 2008(3):CD004183.
60. Sesso HD, Buring JE, Christen WG, Kurth T, Belanger C, MacFadyen J, et al. Vitamins E and C in the prevention of cardiovascular disease in men: the Physicians' Health Study II randomized controlled trial. *JAMA*. 2008 Nov 12;300(18):2123–33.

61. Lippman SM, Klein EA, Goodman PJ, Lucia MS, Thompson IM, Ford LG, et al. Effect of selenium and vitamin E on risk of prostate cancer and other cancers: the Selenium and Vitamin E Cancer Prevention Trial (SELECT). *JAMA*. 2009 Jan 7;301(1):39–51.
62. Gaziano JM, Glynn RJ, Christen WG, Kurth T, Belanger C, MacFadyen J, et al. Vitamins E and C in the prevention of prostate and total cancer in men: the Physicians' Health Study II randomized controlled trial. *JAMA*. 2009 Jan 7;301(1):52–62.
63. Wirfält E, Drake I, Wallström P. What do review papers conclude about food and dietary patterns? *Food & Nutrition Research*. 2013.
64. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer i Norge – Metodologi og vitenskapelig kunnskapsgrunnlag. Oslo: Nasjonalt råd for ernæring 2011.
65. Åkesson A, Frost Andersen L, Kristjánsdóttir Å, Roos E, Trolle E, Voutilainen E, et al. Health effects associated with foods characteristics of the Nordic diet – A Systematic Literature Review. *Food & Nutrition Research* 2013.
66. Helmersson J, Arnlov J, Larsson A, Basu S. Low dietary intake of beta-carotene, alpha-tocopherol and ascorbic acid is associated with increased inflammatory and oxidative stress status in a Swedish cohort. *Br J Nutr*. 2009 Jun;101(12):1775–82.
67. Helmersson J, Arnlov J, Vessby B, Larsson A, Alfthan G, Basu S. Serum selenium predicts levels of F2-isoprostanes and prostaglandin F2alpha in a 27 year follow-up study of Swedish men. *Free Radic Res*. 2005 Jul;39(7):763–70.
68. Routine vitamin supplementation to prevent cancer and cardiovascular disease. *Nutr Clin Care*. 2003 Oct-Dec;6(3):102–7.
69. Dietary Reference Intakes. Proposed Definitions and Plan for Review of Dietary Antioxidants and Related Compounds. Washington DC: Institute of Medicine, Food and Nutrition Board 1998.
70. Dragsted L. Antioxidants in Fruits and Vegetables Søborg: Veterinær- og Fødevarerdirektoratet 1998.
71. Tribble DL. AHA Science Advisory. Antioxidant consumption and risk of coronary heart disease: emphasis on vitamin C, vitamin E, and beta-carotene: A statement for healthcare professionals from the American Heart Association. *Circulation*. 1999 Feb 2;99(4):591–5.
72. Routine vitamin supplementation to prevent cancer and cardiovascular disease: recommendations and rationale. *Ann Intern Med*. 2003 Jul 1;139(1):51–5.