Diet, Physical Activity and Cardiovascular Disease Prevention in Europe

November 2011











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European Heart Network

The European Heart Network (EHN) is a Brussels-based alliance of heart foundations and like-minded non-governmental organisations throughout Europe.

EHN plays a leading role in the prevention and reduction of cardiovascular diseases, in particular heart disease and stroke—through advocacy, networking, education and patient support—so that they are no longer a major cause of premature death and disability throughout Europe.

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Contributing experts

Marleen A. van Baak
NUTRIM School for Nutrition, Toxicology and Metabolism
Department of Human Biology
Maastricht University Medical Centre+
Maastricht, THE NETHERLANDS

Lydia Collingridge MBBS
London School of Hygiene and Tropical Medicine
London, UK

Simona Costanzo ScD
Laboratori di Ricerca
Università Cattolica del Sacro Cuore
Campobasso, ITALY

Augusto Di Castelnuovo ScD, PhD
Laboratori di Ricerca
Università Cattolica del Sacro Cuore
Campobasso, ITALY

Vardis Dilis MSc
Hellenic Health Foundation,
Tetrapoleos 10-12, 115 27
Athens, GREECE

Maria Benedetta Donati, MD, PhD
Laboratori di Ricerca
Università Cattolica del Sacro Cuore
Campobasso, ITALY

Giovanni de Gaetano, MD, PhD
Laboratori di Ricerca
Università Cattolica del Sacro Cuore
Campobasso, ITALY

Licia Iacoviello, MD, PhD
Laboratori di Ricerca
Università Cattolica del Sacro Cuore
Campobasso, ITALY

Per Ole Iversen, MD
Professor of Medicine, Department of Nutrition
Institute of Basic Medical Sciences
University of Oslo
Oslo, NORWAY

Rachel Jackson Leach MSc, BA
International Association for the Study of Obesity (IASO)
London, UK

Tim Lobstein PhD
International Association for the Study of Obesity (IASO)
London, UK

Karen Lock BMBCh, MA, MSc, PhD, FFPH
London School of Hygiene and Tropical Medicine
London, UK

Professor Helene McNulty RD, PhD
Northern Ireland Centre for Food and Health (NICHE)
School of Biomedical Sciences
University of Ulster, NORTHERN IRELAND

Jan I. Pedersen MD, PhD
Department of Nutrition
Institute of Basic Medical Sciences
University of Oslo
Oslo, NORWAY

Robert Pederson MSc, BSc, BA
European Public Health and Agriculture Consortium (EPHAC)
Brussels, BELGIUM

Caroline Small BSc (Hons), PhD, MBA
(Executive Director of IASO until March 2010)
Honorary Senior Lecturer at Imperial College
London, UK

Riitta Törrönen PhD
Institute of Public Health and Clinical Nutrition
University of Eastern Finland
Kuopio, FINLAND

Professor Emeritus Ilkka Vuori MD, PhD, FACSM, FECSS
The Finnish Bone and Joint Federation
Tampere, FINLAND

Mary Ward RD, PhD
Northern Ireland Centre for Food and Health (NICHE)
School of Biomedical Sciences
University of Ulster, NORTHERN IRELAND

Nutrition expert group

Mike Rayner MA, DPhil
Chair of the EHN nutrition expert group
Director, British Heart Foundation Health Promotion Research Group
Department of Public Health
University of Oxford
Oxford, UK

Ineke van Dis MSc
Nutritional epidemiologist
Dutch Heart Foundation
The Hague, THE NETHERLANDS

Mirjana Gurinovic MD, PhD Nutrition
Scientific Research Advisor
Centre of Research Excellence in Nutrition and Metabolism
Institute for Medical Research, University of Belgrade
Belgrade, SERBIA

Professor W. Philip T. James CBE, MD, FRCP, FRSE
President
International Association for the Study of Obesity (IASO)
London, UK

Professor em. Kaare R. Norum MD, PhD
Department of Nutrition
Institute of Basic Medical Sciences
Faculty of Medicine
University of Oslo
Oslo, NORWAY

Ursula Schwab PhD, Adjunct Professor
University of Eastern Finland, Kuopio Campus
School of Medicine, Institute of Public Health and Clinical Nutrition, Department of Clinical Nutrition
Kuopio, FINLAND

Professor Antonia Trichopoulou MD, PhD
Director, WHO Collaborating Center for Food and Nutrition Policies
Department of Hygiene and Epidemiology and Medical Statistics
School of Medicine, University of Athens
Athens, GREECE

Authors

The publication was coordinated by:

Susanne Løgstrup Cand Jur, MBA, FESC
Director, European Heart Network
Brussels, Belgium

Other sections of the report were drafted by:

Karen McColl MPH
Independent consultant (public health)
Savoie, France

Some key definitions

Europe – In this report Europe refers to the 53 member states of the World Health Organization's European region, stretching from Iceland in the west to Kazakhstan in the east, and including the 27 member countries of the European Union.

Cardiovascular disease (CVD) refers to diseases of the heart and circulatory system. The main forms of cardiovascular disease are coronary heart disease and stroke. It also includes, among other things, heart failure, peripheral artery disease and arrhythmia.

Population dietary goals represent a recommended average intake or level. Sub-groups of the population—such as babies and children, pregnant women and older people—will have different needs. The goals are intended primarily for informing and monitoring food policy and are not dietary guidelines for individuals. For nutrition education purposes these goals need to be translated into appropriate food-based dietary guidelines.

Fat and dietary lipids: in this report we use the term "fat" to refer to dietary lipids. This includes both solid fats and oils.

Fruit and vegetables: in general, the term "fruit and vegetables" includes fresh, frozen, dried and canned fruits and vegetables. Potatoes are usually excluded because in most European diets these are consumed as starchy staples and are major sources of complex carbohydrates. Beans and pulses may be included, although in some countries these are eaten as a non-animal protein source. Different studies examined in the course of the expert review on fruit and vegetables use a variety of definitions—these are outlined in the detailed scientific review paper.

Executive Summary

In May 2002, the European Heart Network published *Food, Nutrition and Cardiovascular Disease Prevention in the European Union: Challenges for the New Millennium*. This paper reviews the scientific and policy developments since 2002 and goes on to propose a revised series of population goals and policy recommendations for action.

The case for taking action on diet and physical activity to prevent cardiovascular disease remains clear and compelling. Despite improvements in many countries, cardiovascular disease (CVD) continues to be the main cause of death in Europe, accounting for nearly half of all deaths in the region. Coronary heart disease and stroke are both also major causes of ill-health and disability. The economic burden is heavy, costing the European Union an estimated 192 billion euro in 2006. Not every one has benefited from the recent progress—there remains a startling gap of 21 years in healthy life expectancy for men and a gap of 19 years in women between European countries and stark inequalities in health within countries persist. (*Chapter 1.1*)

The good news is that cardiovascular disease is largely preventable and experience has shown that prevention does work and is cost-effective. Diet and physical activity play an important role—seven of the eight risk factors which collectively account for 61% of cardiovascular deaths globally are related to diet and physical activity. (*Chapter 1.2*)

For this report, EHN commissioned a series of scientific reviews to examine the latest evidence and two sets of population goals are proposed:

Goal	Intermediate population goal	Longer-term goal
Total fat	Less than 30% of food energy	Between 20-25% of food energy
Saturated fat	Less than 10% of energy	Less than 7% of energy
Trans fats	Less than 1% of energy	Less than 0.5% of energy.
Polyunsaturated fatty acids (PUFA)	Between 6 and 11% of food energy for total PUFA Alpha-linolenic acid: between 1 and 2% of food energy Very long chain n-3 PUFAs (such as EPA and DHA): 250 - 500 mg per day	Between 5 and 8% of food energy Alpha-linolenic acid: at least 2% of food energy Very long chain fatty acids (such as EPA and DHA): 250-500 mg per day
Monounsaturated fatty acids	Between 8% and 13% of energy	Between 7.5% and 9.5%
Fruit and vegetables	More than 400 g/day	More than 600 g/day.
Salt	Less than 5 g/day	Less than 4 g/day
Physical activity	150 minutes of at least moderate intensity endurance or aerobic activity per week for cardiovascular health. One hour of moderate activity on most days (around 300 minutes per week) is recommended for additional health benefits and to prevent overweight and obesity	Higher, still attainable, amounts of at least moderate-intensity physical activity, say 60 minutes a day, will bring additional health benefits.
Body mass index	Average BMI of less than 23 for adults	Average BMI of 21 for adults
Total carbohydrate	More than 55% of energy	Up to 60%-70% of energy
Added sugars	Less than 10% of energy	Goal tentatively set at 5% of energy
Sugar-sweetened drinks	To reduce consumption, as much as possible	Zero consumption of sugar-sweetened drinks
Fibre [1]	More than 20 g non-starch polysaccharides per day (>1.6 g/day of non-starch polysaccharides/MJ) or 27 g AOAC fibre	More than 25 g non-starch polysaccharides (>2 g non-starch polysaccharides/MJ) or >35 g AOAC fibre (2.8 g fibre/MJ)

1 The proposed goals include figures for fibre according to two different definitions in widespread use: **Non-starch polysaccharides** (NSP) of plant cell wall origin and fibre as measured by the **Association of Official Analytical Chemists (AOAC) method** which includes indigestible material and tends to give a higher fibre values.

- Intermediate targets which include pragmatic considerations of what might realistically be aimed for in the next five to 10 years.

- Ambitious longer-term goals which highlight the levels we should ultimately be aiming for, if pragmatic constraints can be overcome. (*see Chapter 1.3 for the detailed scientific reviews and a full explanation of the rationale for these goals*)

Alcohol: If alcohol is consumed at all, it should be limited to not more than 20 g alcohol (two drinks) per day for men and not more than 10 g alcohol (one drink) per day for women. It is not recommended that adult abstainers begin drinking.

Folate: The evidence is not currently sufficient to justify a population dietary goal in relation to cardiovascular disease prevention.

Antioxidants and polyphenols: There is not currently sufficient conclusive evidence to justify making a public health recommendation for antioxidants.

Breastfeeding: No population goal is included in the table, but on a population basis, we recommend exclusive breastfeeding for the first six months, followed by continued breastfeeding with appropriate complementary foods for up to one year of age.

Water: Although it is not included in the table, water is essential for adequate hydration of the body and an adequate water intake is vital.

Nutrient-dense vs energy-dense foods: A diet rich in nutrient-dense foods—rather than foods which are energy-dense and nutrient poor—is important to ensure that the diet can provide essential nutrients and is appropriate for healthy weight maintenance.

Towards a plant-based diet: A diet that meets these population goals favours a shift towards a predominantly plant-based diet from an animal-based diet.

Fish consumption: The 250-500mg/day goal for very long chain n-3 polyunsaturated fatty acids is consistent with twice weekly consumption of oily fish. Despite good nutritional reasons for this recommendation, the European Heart Network is aware of the potential consequences for the current state of the world's fish stocks.

Public health approach: Despite recent advances in medicine, including the widespread use of statins, EHN continues to advocate a population-wide approach to preventing cardiovascular disease through diet and physical activity.

Examination of current trends in food supply, food consumption and physical activity patterns suggest that there remains a great deal of work to be done. Greater opportunities for food purchasing and falling prices for many foods have helped stimulate the demand for food and thereby raised the levels of intake. (*Chapter 1.4*)

The majority of the population is failing to meet the interim goals proposed above for salt and (with the exception of Italy and Portugal) saturated fat. Despite real progress in reducing trans fatty acid levels in recent years, popular foods with high amounts of trans fats are still easily available in eastern Europe and there are concerns about potentially high intakes among some population sub-groups. (*Chapter 1.4*)

While fruit and vegetable consumption has increased in most countries, the nutritional quality may have deteriorated and Mediterranean countries continue to have much higher intakes. Northern Europe has continued to have high intakes of Common Agricultural Policy-supported meat and dairy foods, while some southern European countries have seen meat supplies increase nearly four-fold in a generation. (*Chapter 1.4*)

Market data show significant growth in sales of snacks, confectionery and soft drinks throughout the region. Similarly, company information illustrates the dramatic rise in fast food outlets throughout the region over recent decades. Breastfeeding levels continue to disappoint, but it is encouraging that rates in all countries surveyed are either increasing or stable. A significant generation shift is apparent, especially in southern Europe, where traditional foods are increasingly being replaced by a more 'modern' diet with a greater proportion of calories from oils and fats, sugars and processed starches, and for some sections of the population, alcohol. (*Chapter 1.4*)

The recent major food, nutrition and physical activity policy initiatives at the global, European and EU levels go some way toward answering EHN's call for"*a comprehensive and integrated European food and nutrition policy*". Further progress is needed, howevever, in translating these strategic documents into concrete and effective action on the ground.

Section 2 of this paper presents a series of recommendations for policy actions in the following areas:

- Reformulation of food products to reduce the salt, saturated fat, and added sugar content of foods and portion size
- Legislation to ban industrially produced trans fatty acids
- Ensuring that consumers have easy access to meaningful information about the nutritional quality of foods
- Ensuring availability of fresh drinking water
- Controlling advertising of unhealthy foods aimed at children
- Mass media educational campaigns to increase demand for healthy foods and to promote physical activity

- Promoting healthy options
- Rules on nutrition and health claims
- Promotion of breastfeeding and ensuring appropriate marketing of breastmilk substitutes
- The use of economic tools (taxes and subsidies) to make healthier foods more affordable and less healthy foods more expensive
- Pricing strategies to promote healthier food choices
- Use of the Common Agricultural Policy to promote a healthy diet across Europe
- Improving access to affordable healthy foodstuffs for vulnerable and disadvantaged groups
- Economic tools (taxes, subsidies and pricing strategies) to promote physical activity
- Improving access to affordable healthy food and physical activity opportunities
- Improving the nutritional quality of food served and/or sold in public institutions (sports and leisure facilities, government offices, universities, facilities for older people, prisons, detention centres, hospitals and other public services)
- Schools and pre-school facilities as health-promoting environments that encourage and facilitate healthy eating and active living
- Measures to enable people to make healthier choices when they eat out (in the commercial catering sector)
- Actions in the workplace to improve diet and physical activity
- Creation of environments that promote active living
- Health service involvement in promoting healthy lifestyles

It is clear that it is no longer possible to consider food, nutrition and physical activity in isolation of today's major social and environmental challenges. There is growing interest in defining what constitutes a sustainable diet and level of physical activity. The population goals proposed in this paper are formulated from the perspective of preventing cardiovascular diseases but we have sought to take some note of environmental perspectives e.g. in setting the goals for n-3 fatty acids and making recommendations about fish. We anticipate that future editions of this report will need to take more note of this evidence base. (Chapter 2.5)

Summary of Recommendations

Reformulation of food products to reduce the salt, saturated fat, and added sugar content of foods and portion size

- Efforts to reduce the fat, sugar and salt content and portion sizes of mainstream food and drink products should be a key priority for Europe.
- EHN has supported the European Commission's efforts in this area, but questions whether the proposed voluntary approach will be adequate. There are particular concerns that some sectors of the population may miss out on the benefits if reformulation is limited to voluntary private sector efforts.
- The Commission and national governments should, therefore, set firm targets for progress in product reformulation and should set a priority list of products for reformulation (e.g. bread and bakery, ready prepared meals, breakfast cereals) and progressive and time-bound levels. If the collaborative voluntary approach does not deliver results within that timescale, the Commission or national governments should introduce legislation setting maximum levels of these nutrients/ingredients for different foodstuffs.
- Reformulation efforts should be accompanied by government information campaigns that help to create consumer demand for lower salt/fat/sugar products with a higher fibre (non-starch polysaccharide) content.
- Agricultural production methods that improve the nutritional quality of foods should be promoted with specific measures being taken to increase the production of vegetables and fruit and their ease of access to publicly-funded facilities.
- The food manufacturing, retail and catering industries should build on the progress that has already been made, and collaborate fully with national and European authorities to reduce the fat, sugar and salt content of foods and to reduce portion sizes.

Legislation to introduce strict limits on industrially produced trans fatty acids

- A legislative approach to reducing industrially-produced trans fatty acids has been shown to be effective. Given the variations in trans fat intakes, policy should be guided by actual intake data of vulnerable groups and not on population averages. Measures are also needed to ensure that trans fats are not replaced with saturated fat.
- The European Commission should bring forward a proposal for an EU-wide regulation to eliminate industrially produced trans fats in foodstuffs marketed in the EU. Action at the EU level will support the functioning of the internal market while at the same time ensuring a high level of health protection for all.

- In the absence of proposed EU legislation, or for countries outside the EU, national legislation to limit the presence of industrially-produced trans fatty acids in foodstuffs is recommended and consideration should be given to using the public health provisions in international trade law to limit trade in products containing industrially produced trans fats.
- In many countries, food sold in catering outlets is regulated and inspected at the local level. In the absence of national legislation, this presents the opportunity for local authorities to ban trans fats from foods served in restaurants, fast food chains, pub, cafés and other catering outlets.
- EHN favours the regulatory approach in this case, as it has been shown to be more effective. Nonetheless, voluntary approaches in a few countries (UK, The Netherlands, Norway) have yielded some progress and—pending any national or European legislation—the food industry should work collaboratively with food authorities to reduce the presence of trans fatty acids in foods. In particular, multi-national companies have a responsibility to ensure and demonstrate that the low levels of trans fats that they can achieve in countries where there is regulatory or policy pressure are applied uniformly across the countries where they operate.

Ensuring that consumers have easy access to meaningful information about the nutritional quality of foods

- Mandatory, legible, back-of-pack and front-of-pack labelling should receive first priority at the European level.
- EHN continues to call for a simplified front of pack scheme. This should be a "sign posting" scheme that would enable consumers to identify healthier choices at a glance. It should only contain key elements: energy, fat, saturated fat, sugars and salt. The front-of-pack scheme should be colour coded with red, yellow and green indicating high, medium and low levels of the four elements (based on percentage GDAs per serving).
- National governments must be able to provide meaningful nutrition labelling to consumers in their countries over and above the minimum guarantees stipulated in EU legislation.
- Urgent action is needed to improve the provision of nutrition information in restaurants, cafés and other catering outlets. Authorities should consider a mandatory requirement to provide nutrition information (in the form of a traffic light labelling scheme covering key nutrients) for all chains with more than 10 outlets.

Ensuring availability of fresh drinking water

- Policies to ensure the provision of safe unsweetened normal drinking water, particularly in schools, workplaces, public places and public institutions should be introduced.

Controlling advertising of unhealthy foods aimed at children

- Measures to protect children from audiovisual commercial communication concerning unhealthy food and drinks should be a priority at European level.
- There should be no audiovisual commercial communication for foods high in fat, sugar or salt broadcast between 06h00 to 21h00. Nutrient profiling schemes can be used to identify affected food products. Audiovisual commercial communication includes surreptitious advertising, sponsorship and product placement.
- Restrictions on marketing of unhealthy foods to children should also encompass non-broadcast marketing techniques (eg online media etc) and the use of toy promotions to promote unhealthy foods. Controls should be introduced to prevent the promotion of links between sports or celebrities and unhealthy foods.
- An international code should be developed—under the auspices of WHO—to reduce substantially the extent and impact of marketing of unhealthy foods and beverages, particularly to children.
- Pending the introduction of an international code or EU legislation, national governments should introduce their own restrictions to protect children from advertising for unhealthy foods.
- Local authorities—including, for example, health, education and recreation departments—can also take action to protect children from commercial messages about unhealthy foods in or near schools, play areas and other places where children gather.

Mass media educational campaigns to increase demand for healthy foods and to promote physical activity

- There is a role for the national and local governments in developing campaigns to increase demand for healthy foods and to promote physical activity. There may also be a role for the European Union to support government initiatives. It is important that any campaigns to promote healthy eating or physical activity are:
  - solidly embedded in a broader, multi-sectoral food and nutrition policy;
  - take into account research identifying criteria associated with campaigns that have been effective; and
  - are backed by supportive policies and broader environmental changes.

Promoting healthy options

- At the European level, sufficient funds should be made available for interventions to increase fruit and vegetable consumption. The agriculture and health sectors, which both stand to benefit from increases in fruit and vegetable consumption, should coordinate

efforts to promote fruit and vegetables.
- CAP promotion budgets should give preference to foods of high nutritional value and incentive given to create synergies with the EU School Fruit Scheme and Most Deprived Persons Scheme. Unused promotion funds should be made available for public campaigns focusing on healthy diets. Funds should not be used to promote products that are not otherwise promoted as part of healthy eating.
- Authorities at the national and local levels should explore options to promote healthier food and drink products. This should include working with the food manufacturing, retailing and catering sectors, as well as the creative industries, to harness the potential of marketing and promotional techniques to promote health.
- The food manufacturing, retail and catering sectors should use their know-how and the promotional techniques at their disposition—including product position and price promotions—to promote healthier foods. This private sector involvement should only follow on after the public health messages have been developed by the competent authorities/government.

Rules on nutrition and health claims

- EHN proposes that the Commission stop seeking to develop its own model and adopts the model developed by Food Standards Australia New Zealand—this model would be fit for purpose in the European context. A model with fewer food categories, more nutrients and nutrient scoring is recommended.

Promotion of breastfeeding and ensuring appropriate marketing of breastmilk substitutes

- The EU must ensure that international conventions on infant feeding are adhered to.
- Europe can also play an important role in supporting member states in the implementation of sound infant and young child feeding strategies. There is also an important role for Europe in setting a framework for social and employment policies that protect breastfeeding by improving parental leave provision, requiring facilities for breastfeeding etc.
- EU policies must be informed by and based on evidence, which includes independently reviewed and independently funded research. Notably, EU policy should support member states' right to adopt legislative measures setting the highest levels of protection, including, but not restricted to, banning the promotion of breastmilk substitutes (BMS) (including follow-on formula and specialised formulas) and banning all health and nutrition claims on foods for infants, young children, pregnant women and nursing mothers.
- Governments should devise and implement national strategies for infant and young child feeding, which incorporate wide-ranging measures to protect, support and promote breastfeeding and reviewing on a regular basis national implementation of the International Code of Marketing of Breast Milk Substitutes and subsequent relevant WHA Resolutions.

The use of economic tools (taxes and subsidies) to make healthier foods more affordable and less healthy foods more expensive

- Economic instruments, such as taxes and subsidies, could play an important role in promoting healthier eating. In relation to taxes, further work is needed to explore ways of mitigating the impact on poorer groups, while maximising the public health benefits. Mechanisms to explore include the combined use of taxes and subsidies, as well as compensatory welfare payments or tax exemptions. The price elasticity of food operates in all socioeconomic groups except the richest, so changing the relative cost of healthier vs unhealthy products still has an impact, even when measures are taken to reduce the economic differences between social groups in society.
- The introduction of subsidies on healthy foods should be a priority for European action. The options for use of value added tax (VAT) as an economic instrument to improve health should also be explored—such as allowing member states the possibility of levying a negative VAT rate.
- Governments should explore the potential for subsidies on healthy foods, which may be used in conjunction with higher taxes on unhealthy foods. Although EU member states are bound by EU rules on Value Added Tax (VAT), there is still considerable scope for action.

Pricing strategies to promote healthier food choices

- There is considerable potential for pricing strategies to be used to promote healthier eating at both macro and micro levels—from Europe-wide to local education or health authorites to individual schools or hospitals.

Use of the Common Agricultural Policy to promote a healthy diet across Europe

- The objectives of the Common Agricultural Policy urgently need to be brought into line with EU-wide nutritional population goals. This should be one of the key priorities for action.
- Food surpluses should be targeted for human consumption, especially disadvantaged communities, in line with nutritional requirements. Market intervention for saturated fat (butter) should be phased out and intervention stocks should not be sold at submarket prices to commercial operators.
- The future CAP should be based on a sound impact assessment, as a precondition for meeting future

objectives. Health, social and environmental impact should be a central measure of the assessment process and identify policy measures that can improve diets and public health across Europe.
- An integrated European Food and Agriculture Policy, which works towards improving European diets in a sustainable way, should be developed. This policy should provide incentives for production of more plant-based foods and promote a shift towards more plant-based diets, with reduced consumption of meat and saturated fat and increased consumption of fruit, vegetables and whole grains.

Improving access to affordable healthy foodstuffs for vulnerable and disadvantaged groups

- European strategy and national action plans to improve diets should place particular emphasis on improving access to affordable healthy foodstuffs for lower socioeconomic groups. There may be a role for the Commission to support member states by mapping retailers' locations.

Economic tools (taxes, subsidies and pricing strategies) to promote physical activity

- Governments—at national, local and regional levels—should pursue policies and interventions which remove financial barriers to sports and leisure facilities, and to forms of active travel. These may include reducing prices, subsidising facilities, providing vouchers and/or prescribing physical activity. It should also include ensuring that appropriate facilities are provided in areas of disadvantage, built and natural environments are designed to promote physical activity and that useable and affordable transport to travel to physical activity opportunities is offered.

Improving access to affordable healthy food and physical activity opportunities

- EU, national and local policies should encourage and facilitate retailers to locate in under-served areas. Planning and zoning rules and incentives—such as tax benefits, loan guarantees, grants to cover start-up costs—should be used.
- Strategies that include creation of incentives for existing retailers to offer healthier choices in areas that have poor access should be explored.
- Health considerations should also be taken into account in planning decisions relating to the location of and density of fast-food outlets and other catering establishments.
- European agricultural policy, along with national and local governments, should aim to increase access to mechanisms for people to buy directly from farms and to support community food growing. Options include farmers' markets, community-supported agriculture, pick your own, farm-to-school initiatives, community allotments, school growing schemes, temporary leases to allow growing on unused land and a community land bank to act as a broker between landowners and groups wanting land for growing food. Urban agriculture has many social as well as economic and health benefits and has been seldom explored in Europe.
- The EU should also facilitate, encourage and support policies and interventions to ensure access to affordable physical activity opportunities.

Improving the nutritional quality of food served and/or sold in public institutions (sports and leisure facilities, government offices, universities, facilities for older people, prisons, detention centres, hospitals and other public services)

- Ensuring that food sold or served in public institutions is coherent with population-wide dietary goals should be a key priority. This should apply to *all* foods served in public institutions, whether it is sold directly or by an external provider under contract. These efforts should be consistent with other policies towards a sustainable food supply—promoting procurement of local and sustainable produce—and could be an important economic driver for an improved food supply.
- The Commission should support and encourage member states in the application of nutritional standards for food sold or served in public institutions. A first step could be a Europe-wide review of the existing quality standards and procurement practices for food provided in public institutions. This could be done, for example, under the auspices of the High Level Group as a cooperative measure.
- Authorities at the national and/or local level should act to introduce nutritional standards for foods sold or served in public institutions. Public procurement guidelines should ensure that health criteria are taken into account.
- The application of nutritional standards should be accompanied by pricing strategies to improve the relative affordability of healthier items, introduction of smaller portion sizes, restrictions on commercial information relating to foods high in fat, sugar or salt and rules requiring public institutions to provide safe drinking water.

Schools and pre-school facilities as health-promoting environments that encourage and facilitate healthy eating and active living

- The creation of health-promoting schools, that adopt a whole-school approach to health, should be encouraged.

- In relation to food, this should include:
  - ensuring that children have the necessary education and practical skills by including both nutrition and food skills in the school curriculum;
  - ensuring that food provided in schools complies with high nutritional quality standards (whether meals, or from vending machines or other outlets);
  - ensuring that energy dense and high-salt snacks, high-sugar soft drinks and confectionery that is high in fat or sugar are not available in schools;
  - the development of policies (whether by schools, education departments or local authorities) which limit children's access to outlets selling unhealthy foods during the school day;
  - ensuring access to free drinking water for all pupils;
  - a prohibition on commercial communications for foods high in fat, sugar or salt aimed at children and young people in schools, as elsewhere.
- The impact of these measures could be greatly enhanced by introducing policies to provide free school meals to all pupils.
- In terms of promoting physical activity, the whole-school approach should encompass:
  - increasing the number of hours of physical education and activity in the school day, with a recommended minimum number of hours per week;
  - establishing guidance and incentives for schools and local government to improve the environment around schools to encourage walking and cycling;
  - establishing safe routes to schools from neighbouring communities;
  - facilitating and improving the quality of physical education;
  - promoting opportunities and practices to build activity in and around the school day (including before and after school clubs);
  - establishing safe zones around all schools where walking and cycling are prioritised and car travel is much more difficult.

Measures to enable people to make healthier choices when they eat out (in the commercial catering sector)

- Improving provision of nutrition information on food eaten outside the home should be a priority for action in European countries.
- National and/or local authorities throughout Europe should work with the catering sector—from major fast-food chains to small local restaurants—to encourage and support them in the provision of healthier choices, to encourage the use of oils high in polyunsaturated or monounsaturated fats rather than hard fats high in saturated fats, introducing smaller serving sizes and providing clear nutrition information. National governments should consider requiring catering establishments to follow the Finnish example of making vegetables and a salad bar an intrinsic cost of the main meal.
- For smaller catering establishments (with fewer than 10 outlets) where a regulatory approach may not be appropriate, incentive approaches (such as award schemes) should be used to encourage them to provide healthier menu items and to provide nutrition information.

Actions in the workplace to improve diet and physical activity

- WHO, the European Commission and national governments should continue to encourage and support employers, trade unions and other employees' organisations to work together to promote and facilitate healthy eating and physical activity in the workplace.
- Every workplace should have a healthy work-life balance policy, developed collaboratively with employees and their representatives. This policy should ensure that food served or sold in the workplace is of a high nutritional quality and that employees have access to clear nutrition information for all food sold or served in the workplace.
- Employers should introduce measures to encourage employees to incorporate physical activity into their daily life. These could include:
  - encouraging employees to walk, cycle or use other forms of active transport to travel part or all of the way to and from work;
  - helping employees to be physically active during the day (eg providing prompts to encourage stair use, encouraging short walk breaks etc);
  - providing information about cycling and walking routes.

Creation of environments that promote active living

- The EU should ensure that European development or structural funding is conditional on projects being able to enhance the infrastructure for promoting active living.
- Authorities at the national and local levels should implement a wide range of measures to encourage and facilitate walking, cycling and other forms of physical activity as part of people's daily routines. These can include:
  - development of an integrated transport strategy that emphasises walking and cycling;
  - improving provision of local parks and green places;
  - provision of maps and guides on good places to walk and cycle;
  - provision of clear signs to enable people to measure the distance they walk/cycle;

- improving access to sports and leisure facilities for all sectors of the community (eg free childcare facilities, discounted access, late night sessions etc);
- ensuring stairs are prominent, accessible and well-lit in new buildings;
- encouraging town planners to provide facilities that can be walked to and around, and to promote development of areas which have a mixture of residential, commercial and public services;
- taking action to enhance personal safety in areas where people could be physically active.

Health service involvement in promoting healthy lifestyles

- The European Commission should take steps to improve and enhance the role of health services in prevention and early detection of cardiovascular diseases, including a review of how effective health services across the EU are in promoting prevention and disease detection and a review of the inclusion of healthy lifestyle promotion in the curricula of health professionals across the EU.
- Developing the training and further education opportunities for health professionals is an important aspect of capacity building in central and eastern European countries, where such opportunities are sparse currently.

Introduction

"Every child born in the new millennium has the right to live until the age of at least 65 without suffering from avoidable cardiovascular disease"

St Valentine's Declaration, 14 February 2000

Background

In May 2002, the European Heart Network published *Food, Nutrition and Cardiovascular Disease Prevention in the European Union: Challenges for the New Millennium.*[1] This policy position paper updated a previous report, published in 1998, in which the European Heart Network first set out a summary of the relationship between diet and cardiovascular disease (CVD).[2]

The 2002 policy position paper summarised the consensus of scientific thinking on diet and the prevention of CVD. It presented population dietary goals for the European region based on the Core Report of the European Commission's Eurodiet project.[3] The paper also set out a series of policy recommendations and, in particular, examined what national heart foundations could do to help.

The scientific consensus and policy environment have continued to evolve in the nine years since we published our position paper. There have been important changes in the patterns of certain diet-related cardiovascular risk factors. There have also been developments in the scientific consensus on the relationship between diet, physical activity and cardiovascular disease, and a much larger body of evidence now exists. The dietary goals outlined in the 2002 report were intermediate goals, which were a pragmatic response based on both the state of scientific thinking at that time and on assessments of how much the prevailing dietary patterns could realistically be expected to change. As the relationship between diet and health has evolved a review of the population dietary goals, and the policy changes that are needed to help achieve these population goals, is timely.

A rapidly changing context

There have been important changes in the science, policy and general context in relation to diet, physical activity and cardiovascular disease over the last decade. Many of those changes have been encouraging, and give grounds for optimism that we will be able to achieve the vision set out on Valentine's Day in the year 2000, of every child born in the new millennium being able to live free from avoidable cardiovascular disease until at least the age of 65.

In policy terms, prevention of chronic diseases, including cardiovascular disease, has continued to rise up the global and European health agenda—non-communicable diseases cause 86% of deaths and 77% of the disease burden in the European region[4]. World Health Organization chronic disease strategies are now in place, both at the global and European levels.[5,6] In May 2010 the UN General Assembly adopted a resolution on the prevention and control of non-communicable diseases and, as part of the follow-up a special session of the UN General Assembly was held on this issue in September 2011.[7]

There has also been increasing understanding of the common risk factors involved in major chronic diseases and the potential of a joint approach to prevention. In 2010, 10 not-for-profit organisations issued a joint appeal, under the umbrella of the European Chronic Disease Alliance, for political action to tackle the growing burden of chronic non-communicable diseases which affects more than a third of Europe's population.[8]

Working towards a heart healthy diet in Europe: Reasons to be cheerful

- Health gains which show that prevention can work: a large proportion of the dramatic decline in coronary heart disease mortality rates witnessed in most western European countries in recent decades was achieved by reducing risk factors linked to diet, physical inactivity and smoking. Real experience now shows that science and policy can come together to save lives—and diet can play an important role.
- There is now much greater awareness—in terms of both the general public and political decision-makers—of the importance of nutrition, physical activity and cardiovascular disease than there was at the start of the decade. When the 2002 policy paper was published there was widespread public concern about food safety and it was these issues, rather than nutritional matters, which dominated the European policy environment. Today, growing public alarm about childhood obesity levels in particular has pushed nutritional issues higher up the agenda.
- There has been a sea change in thinking about why we eat what we eat. There is now much greater recognition of the influence of external factors (such as easy and plentiful access to inexpensive, energy-dense foods or urban environments) on what we eat and how active we are. This means that policy makers need to address a wide range of issues to be able to change diets and physical activity levels. There has been progress on a range of issues that would have been largely unthinkable a decade ago—product reformulation, controls on advertising to children, legislation to ban industrially produced trans fatty acids, restrictions on access to snack foods and drinks in and around schools to name a few.

Our 2002 policy paper called for establishment of "*an integrated European food and nutrition policy*" and there have been encouraging developments. The World Health Assembly endorsed a global strategy on diet, physical activity and health in 2004,[9] while in the European region a second food and nutrition action plan was endorsed by WHO's Regional Committee in 2007.[10] Within the European Union—which has expanded from 15 to 27 members since 2002—the European Commission adopted a White Paper on "*A Strategy for Europe on Nutrition, Overweight and Obesity related health issues*" in 2007.[11]

In November 2008, the Council of the European Union endorsed guidelines on physical activity (as part of the white paper on sports adopted in July 2007). In January 2011, the Commission adopted a communication entitled *Developing the European Dimension in Sport*.[12] WHO published *Global Recommendations on Physical Activity for Health* in 2010.[13]

Improving diets and physical activity levels towards a heart healthy Europe

The case for taking action on diet and physical activity to prevent cardiovascular disease is clear and compelling. Cardiovascular disease is Europe's biggest killer, and every year it causes death and disability on a massive scale across the region. For families and individuals, the personal costs are all too clear. Too many Europeans are dying before their time—as well as being the main cause of death overall, cardiovascular disease is the main cause of death before the age of 65 in Europe.[14] For those left behind, this may mean facing a lonely old age.

As well as being concerned about cardiovascular deaths, however, we also need to tackle the illness and disability caused by heart disease and stroke. Only a minority of individuals under the age of 75 now die when they have a heart attack or stroke and they are often left with a long-term disability, particularly after a stroke. As a result of this ill health and disability millions of people are unable to enjoy a happy and active retirement. And many families are left to care for partners or relatives who have been incapacitated by cardiovascular disease.

The collective burden of cardiovascular disease on society is also evident. It is clear that chronic diseases cost economies dearly—through the medical and social care needs, as well as the economic impact of premature retirement and reduced work force productivity. WHO estimates that, by 2015, GDP in most countries will be 1% lower than it would have been in the absence of chronic diseases and for the Russian Federation the predicted loss of GDP as a result of chronic diseases is $303 billion, while the income loss in the UK is predicted to be almost $33 billion.[12] Cardiovascular disease is estimated to have cost the European Union just over 192 billion euro in 2006[2], including almost 110 billion euro on health care and 82 billion in lost productivity and informal care costs.[14] The social costs of early death among the middle-aged, combined with the costs of formal and informal care for people living with chronic disease, are considerable. As Europe's population grows older—and the ratio of retired people to people of working age increases—it is fundamentally important we do everything that we can to ensure that people remain healthy and independent for as long as possible.

Cardiovascular disease is largely preventable and modifying known risk factors can achieve enormous

2 Latest figures available at time of publication.

public health gains. Many western European countries have seen impressive improvements in recent decades and CVD death rates have fallen dramatically. Diet and lifestyle changes have played a major role.

There is, however, no room for complacency (see Box).

Although progress has been made, not everyone has benefited from it. Some worrying trends have emerged which put this vision of a heart healthy future at risk. Some of these changes threaten to undo some of the gains made in some countries and to hinder progress in others.

No grounds for complacency

- Cardiovascular disease remains Europe's biggest killer. The fact that coronary heart disease death rates have halved in many countries over the last three decades must not detract from the 4.3 million deaths caused by cardiovascular disease in the European region every year and the enormous individual and societal burden of cardiovascular ill-health throughout the region.
- Recent health gains have not been consistent across the region: some European countries continue to have among the highest cardiovascular death rates in the world. Within the European region there is a gap of over 20 years in healthy life expectancy for men between countries and cardiovascular disease is a key contributor. The health inequalities that exist between different socio-economic groups within countries continue to persist—and in many countries the gap continues to widen.
- Some risk factors are on the increase: the dramatic increase in obesity, overweight and diabetes in Europe threatens to undo the gains that have been made. The signs of a levelling off in the decline in coronary heart disease mortality in younger age groups that have been seen in some countries may be due to these adverse risk trends.
- Countries throughout Europe are still far from achieving the population goals set out in the EHN's 2002 policy paper. Although progress has been made in some areas, much more is needed. There are worrying signs of a generation shift in southern Europe as traditional foods are increasingly replaced by a more 'modern' diet with a greater proportion of energy from oils and fats, sugars and processed starches.

Public health advocates—including national heart foundations—have continued to emphasise that wide-reaching policy action is needed to bring about dietary change. The existence of global and European nutrition strategies is testament to their efforts. Strategies based purely on nutrition education and consumer information will not be adequate. There remains considerable scope for further action—at the international, national and local levels—to improve the diets and physical activity levels of European populations. It is a good time to outline some of these policy options and to feed into the future of European agriculture and inform the continuing implementation of the EU nutrition strategy, as well as into national policy making.

Experience now tells us that prevention can work. Around the world we've seen that science and policy can come together to create public health measures that bring about quick and dramatic changes. It is time to build on the progress which has been made, and to harness the growing public concern about nutrition issues, to ensure the heart health of future generations across Europe.

The aim of this paper

The aim of this new policy position paper, therefore, is to review the evidence and to set out the scientific and policy rationale for change.

Section 1 of this report presents an updated synopsis of the evidence on the relationship between nutrition, physical activity, dietary patterns and cardiovascular disease. Leading experts have been commissioned to prepare an overview of recent review studies (ie those published since 2002) and the EHN's Nutrition Expert Group examined the new evidence presented with a view to revising the population dietary goals proposed in the 2002 policy paper. This section also includes a summary of how dietary patterns in Europe are changing.

Section 2 examines the current policy context, including major developments at the global and European levels since 2002. This section also sets out an argument why an integrated and comprehensive policy response is required to improve European diets and physical activity levels. Section 2 goes on to examine dietary advice in the context of the current major global challenges of climate change, water, and food security. Finally, the section outlines what needs to happen for these dietary goals and policy recommendations to become a reality and what heart foundations, other non-governmental organisations and health professionals can do to help.

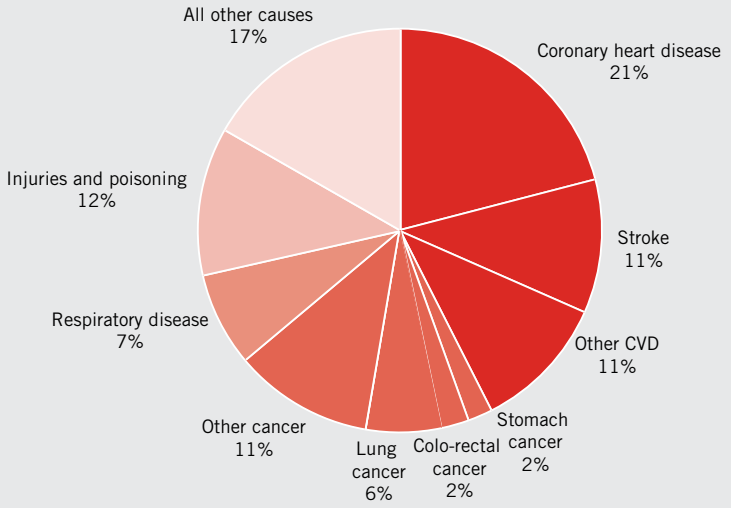
1 Diet, Physical Activity and Cardiovascular Disease - The evidence

1.1 CARDIOVASCULAR DISEASE MORTALITY IN EUROPE

Cardiovascular disease (CVD) continues to be the main cause of death in Europe,[3] accounting for nearly half (48%) of all deaths in the region, equivalent to over 4.3 million deaths each year. It is also the main cause of death in the European Union, where it leads to over two million deaths a year. Just over half of all cardiovascular disease deaths are caused by coronary heart disease (CHD) and nearly a third are from stroke.

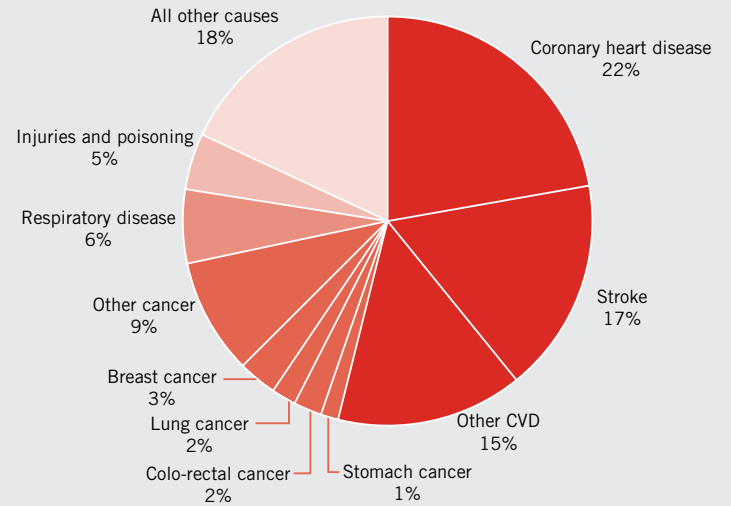
Not only is cardiovascular disease the biggest cause of death in Europe, it is also the main cause of *premature* death (before the age of 65). Coronary heart disease alone is the single most common cause of premature death in Europe, accounting for 17% of deaths in men under 65 and 12% of deaths before the age of 65 in women. Stroke, the other main form of cardiovascular disease, accounts for 6% of deaths in men and 9% of deaths in women before the age of 65 in Europe.

Figure 1 Deaths by cause, men, latest available year, Europe



Note: Data from between 1997 and 2006. No national mortality data available for Andorra, Bosnia and Herzegovina, Monaco and Turkey. Source: European Cardiovascular Statistics 2008. www.ehnheart.org

Figure 2 Deaths by cause, women, latest available year, Europe



Note: Data from between 1997 and 2006. No national mortality data available for Andorra, Bosnia and Herzegovina, Monaco and Turkey. Source: European Cardiovascular Statistics 2008. www.ehnheart.org

3 Unless stated otherwise, the statistics and figures in the following sections are taken from European Cardiovascular Disease Statistics 2008, published by the European Heart Network. These are the most recent data available at the time of publication.

Although cardiovascular disease remains Europe's biggest killer, parts of Europe have witnessed dramatic improvements in recent decades. In most industrialised countries coronary heart disease mortality rates have fallen by as much as 50%-60% since the late 1970s.[16]

Researchers have sought to try and unravel the different reasons for this dramatic decline in CHD death rates and, in particular, to identify how much of the decline is because people with CHD have access to better care and treatment, and how much is because people have been less exposed to CHD risk factors. Analysis of data from the WHO MONICA project attributed more than two-thirds of the decline in coronary heart disease deaths between the early 1980s and the 1990s to lower incidence of CHD because of reduced exposure to risk factors, such as smoking or high blood cholesterol. The remaining third of the decline in CHD mortality was estimated to be due to better survival because of improved care and treatment.[17] Similarly, modelling studies have found that a large proportion of the decline—from 44% in the USA and Italy, for example, to as much as 72% in Finland—can be attributed to reduced exposure to risk factors.[17-21]

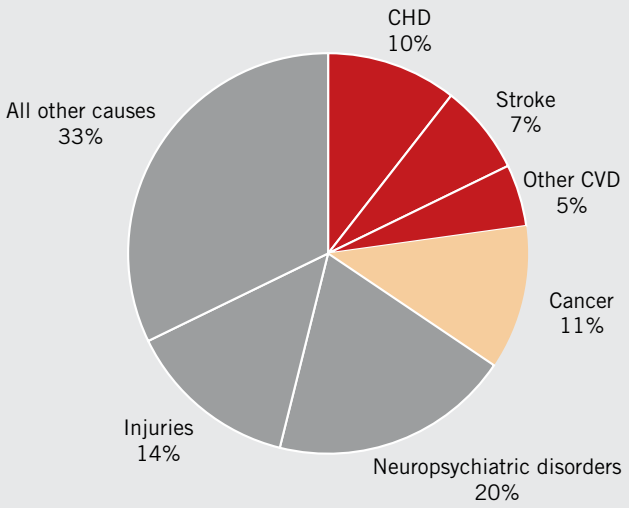
In eastern Europe cardiovascular death rates have not followed the same pattern. Male cardiovascular death rates rose steadily and female death rates remained stable between 1965 and 1990. Since 1990, there have been different patterns in cardiovascular disease mortality among former socialist countries. Of the 10 former socialist countries of the EU, five (Slovenia, Poland, Czech Republic, Slovakia and Hungary) saw dramatic reductions in CVD death rates between 1990 and 2002, the three Baltic states (Latvia, Lithuania and Estonia) saw rates rise steeply until 1994 and then drop back to previous levels, while levels continued to increase until the end of the 1990s in Romania and Bulgaria.[22]

Despite the welcome decline in CHD mortality rates experienced by many European countries in recent years, there is no cause for complacency. The improvements in coronary heart disease mortality have occurred despite the fact that there are some worrying adverse risk factor trends. Namely, the dramatic increase in the prevalence of obesity—which has tripled in the European region since the 1980s[23]—and diabetes. Without such adverse risk trends CHD death rates would have fallen even further, saving more lives. There are now signs that the decline in CHD mortality is slowing down, particularly in younger age groups, in the US, Australia and the UK (England and Wales).[18,24] The increase in obesity and overweight is a possible reason for this levelling off in the CHD mortality decline in some age groups.

1.1.1 The massive burden of cardiovascular ill-health

When we consider the burden of cardiovascular disease in Europe it is important not to focus solely on deaths. Coronary heart disease and stroke are both major causes of ill-health and disability. In Europe, 22% of all the years lost to premature death and years of healthy life lost to disability (Disability Adjusted Life Years or DALYs) are lost to cardiovascular disease each year. In developed European countries this is the second largest single cause of DALYs lost (after neuropsychiatric disorders) and in less developed European countries CVD is the largest cause of loss of DALYs.

Figure 3 Disability adjusted life years lost by cause, 2002, Europe



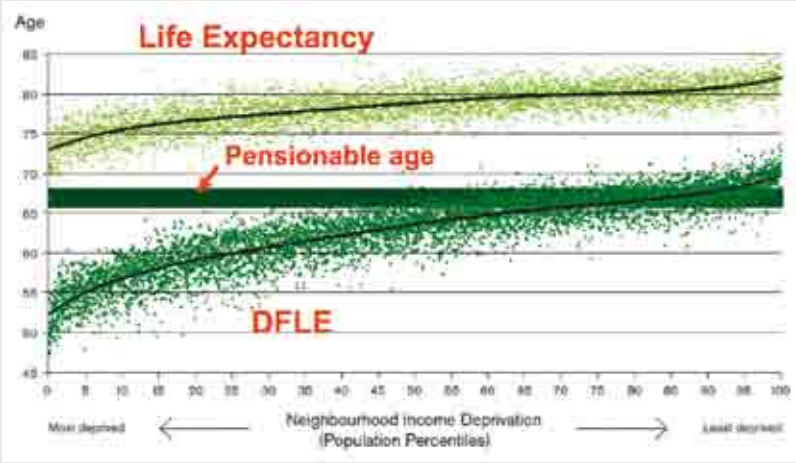
Source: European Cardiovascular Statistics 2008, published by the European Heart Network. www.ehnheart.org

The individual and societal costs of the burden of cardiovascular ill-health present one of the most profound policy challenges facing European governments today. The scale of the problem is mind-blowing: according to the most recent data available cardiovascular disease is estimated to have cost the European Union over 192 billion euro in 2006, including almost 110 billion euro on health care and 82 billion in lost productivity and informal care costs.[14] Cost calculations in England, for example, estimate that the costs of ongoing health service care for five years following a stroke or a heart attack are €22,300 and €2,300 respectively per patient.[25] As well as the health service costs and the lost productivity there are significant costs associated with the informal care provided by family members and others in the community (just under €42 billion in the EU in 2006).[14]

With the region's ageing population, the relative numbers of people of working age who will have to finance pensions and care for the growing numbers of older people are decreasing. In response, and in light of the improvements in life expectancy, governments are contemplating raising retirement ages to keep people in economic activity for longer. However, as Figure 4 illustrates on the basis of detailed data from England, for example, if we look at life-expectancy *free from disability* the picture is quite different. More than three quarters of the population do not have disability-free life expectancy at the age of 68 (the proposed retirement age which England is moving towards). The picture is notably worse in more deprived areas.

This is where the reality of the societal burden of cardiovascular disease really hits home: how will European societies be able to pay for the care for people living with cardiovascular ill-health? It is fundamentally important we do everything that we can to ensure that people remain healthy and independent for as long as possible.

Figure 4 Life expectancy and disability-free life expectancy (DFLE) at birth, by neighbourhood income level, England, 1999-2003



Note: the broad horizontal green band represents potential pension age increases between 2026 and 2046 on the basis of current policy discussions. Source: Adapted from The Marmot Review, 2010.[26]

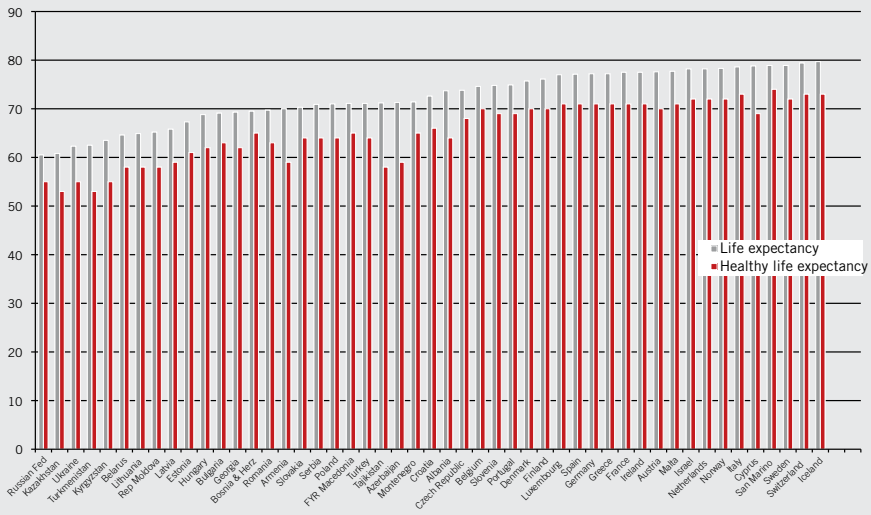
1.1.2 Health inequalities and cardiovascular disease

Although last year was the European Year against Poverty and Exclusion, policy makers still struggle to close the health gaps that exist in Europe—both between and within countries. Throughout Europe, people with higher incomes, more education and better jobs live longer, healthier lives. Cardiovascular disease is a leading contributor to the health gap between European countries and to the inequalities that exist within countries.

Within the European region there is a 19-year gap in life expectancy at birth between men born in Iceland and in the Russian Federation, according to 2007 figures. For women, the gap between the lowest and highest life expectancy at birth across the whole European region is almost 15 years.[25] Within the EU there is a 14-year gap in life expectancy at birth for men and an 8-year gap for women.

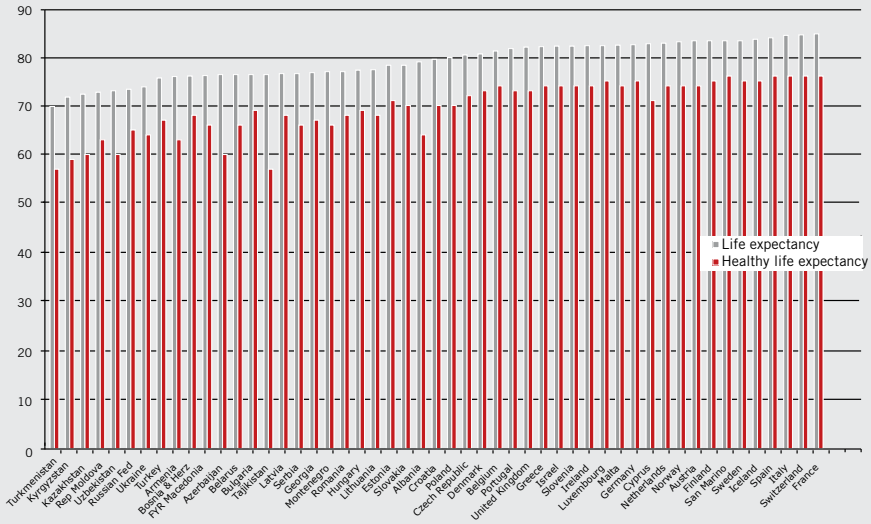
The differences in the number of years people can expect to live free of illness or disability (healthy life expectancy) are even greater—ranging from 74 years for men in San Marino to only 53 years for men in Tajikistan or Turkmenistan, according to 2007 estimates.[25] For women, this gap in healthy life expectancy is 19 years. There are also significant differences within the EU: a boy born in Italy today can expect to have 12 more years free from disability than a boy born in Estonia. For women, there is an eight-year gap between EU member states. Cardiovascular disease is a leading contributor to these differences: many European countries have among the lowest rates of cardiovascular disease deaths in the world, while some of the eastern European countries have among the highest.[22]

Figure 5 Male life expectancy and healthy life expectancy in Europe



Source: from data in World Health Organization Regional Office for Europe, European Health Report, 2009.[27]

Figure 6 Female life expectancy and healthy life expectancy in Europe



Source: from data in World Health Organization Regional Office for Europe, European Health Report, 2009.[27]

As well as these differences between countries, there are also significant health inequalities within countries. Across Europe, people with lower education, lower occupational class or lower income tend to die at a younger age. Differences in life expectancy at birth between lowest and highest socio-economic groups reach 10 years for men and six years for women in some countries.[28] Not only do people in higher socio-economic groups live longer, they are also healthier for longer—the differences are often as great as 15 years.[29] To the consternation of health policy makers, the gap between the health of the most disadvantaged and the most advantaged groups increased in the last decades of the 20th century in many European countries.

Differences in deaths and illness caused by cardiovascular disease play a significant role in national health inequalities. In all European countries for which data exists, mortality from cardiovascular disease is higher among men and women with lower socio-economic status.[30] This picture is constant for death from stroke, but is more complex for ischaemic heart disease—the strong social gradient for ischaemic heart disease mortality which exists in northern Europe is not so evident in southern European countries.[30] This may be because the traditional diets in those countries,[28] particularly among older generations, are protective (see Section 1.4.3 on disappearing traditional diets).

A 2006 review of inequalities in Europe concluded that cardiovascular disease accounts for almost 40 per cent of the difference in mortality rates between men in higher and lower educational groups and that, in women, the contribution of cardiovascular disease could be as high as 60 per cent.[30]

Cardiovascular disease has also played a role in the widening of the health gap; in general, in countries where cardiovascular disease death rates have fallen in recent decades, groups with lower socio-economic status have not benefited as much from the improvements in health behaviour or access to better treatment that have led to that decline. In some eastern European countries where cardiovascular disease mortality has increased, this increase has been more pronounced among more disadvantaged groups.

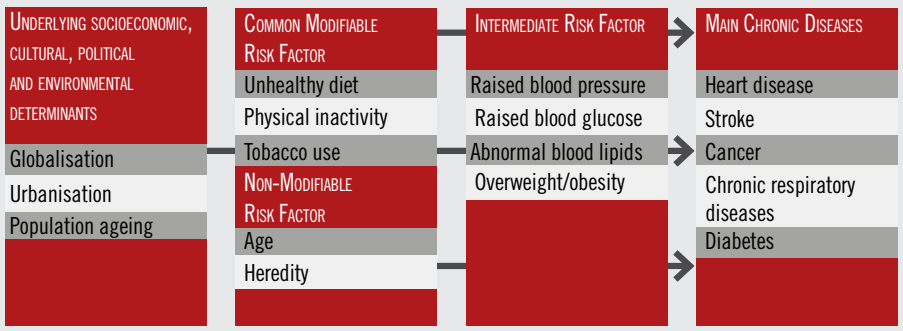
1.2 DIET, PHYSICAL ACTIVITY AND NON-COMMUNICABLE DISEASES

A small group of inter-related chronic diseases and key risk factors are responsible for a large part of the disease burden in Europe. Non-communicable diseases in Europe account for 77% of the disability adjusted life years (DALYs) lost in Europe every year. Taken together cardiovascular disease, cancer and diabetes account for almost half (46%) of the disease burden and 73% of all deaths in the region.[6]

Many of these conditions are inter-related—diabetes, for example, is a risk factor for cardiovascular disease—and have common risk factors. WHO estimates that almost 60% of the disease burden in Europe is caused by seven risk factors and each of these risk factors is linked to at least two major chronic diseases.[6] Each of the leading chronic diseases is linked with two or more risk factors.

As with other diseases, both genetic and environmental factors play a role in cardiovascular disease development. Figure 7 illustrates the causes of chronic disease—starting with the broad influences of urbanisation, globalisation and population ageing.

FIGURE 7 WHO MODEL OF CHRONIC DISEASES



Source: WHO

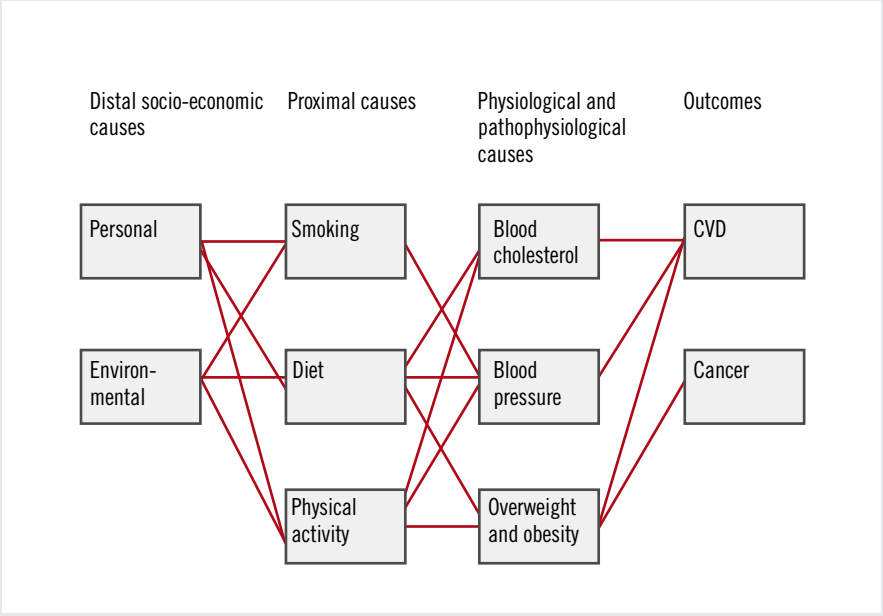
1.2.1 *Diet- and physical activity-related risk factors*

Diet and physical activity are key factors in the environmental side of the equation. WHO estimates that eight risk factors—alcohol use, tobacco use, high blood pressure, high body mass index, high blood cholesterol, high blood glucose, low fruit and vegetable intake and physical inactivity—collectively account for 61% of cardiovascular deaths globally and that reducing exposure to these eight risk factors would increase global life expectancy by almost five years.[30] The INTERHEART study—a case-control study of more than 15,000 heart attacks in 52 countries—confirmed that a relatively small number of potentially modifiable risk factors are responsible for the major proportion of cardiovascular risk.[32] Nine factors accounted for over 90% of the risk of an initial acute myocardial infarction (93% in men and 96% in women).

Seven of the eight risk factors highlighted by WHO are related to diet and physical activity levels. Other risk factors that play a role include stress and indoor or outdoor air pollution.

Figure 8 illustrates the web of complex inter-relationships that exists between different dietary factors and some common diseases.

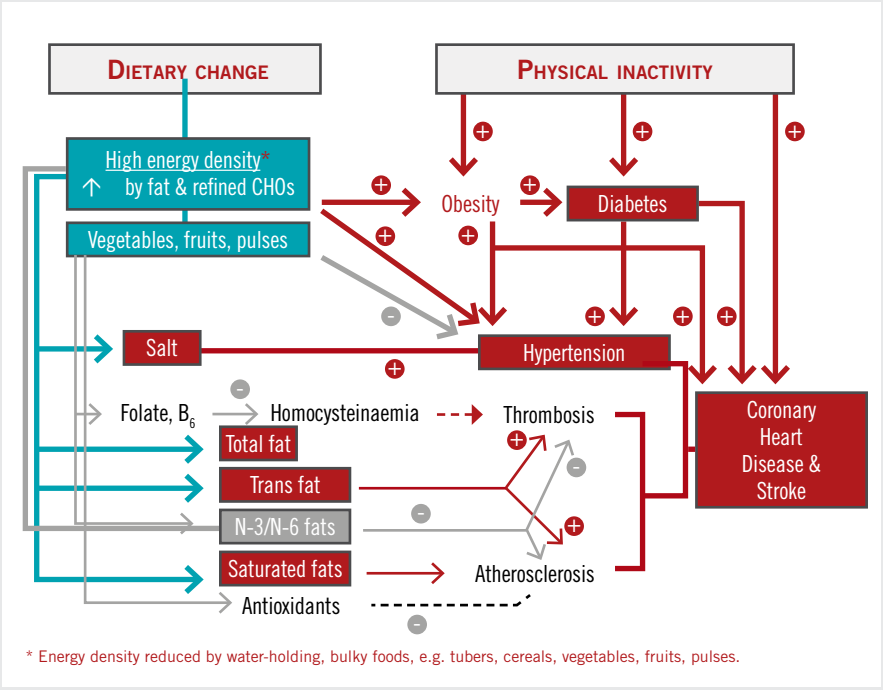
FIGURE 8 THE RELATIONSHIP BETWEEN ENVIRONMENT AND HEALTH CAN BE CONCEPTUALISED AS A 'CAUSAL WEB'



Source: Adapted from Murray C et al, Population Health Metrics 2003; 1:1.

Figure 9 illustrates in more detail the complex nature of the relationships between key risk factors and the development of coronary heart disease and stroke in individuals.

FIGURE 9 CHRONIC DISEASES AND RISK FACTORS - THE KEY ROLE OF DIET AND PHYSICAL INACTIVITY



Note: Factors that have a positive effect are shown with a green line; Factors that negatively impact on CHD and stroke are shown in pink. Dotted lines signify possible, but not proven, links. Source: Professor Philip James.

The fact that several risk factors are involved means that multiple interventions are needed to target heart disease and perhaps another set of risk factors to target the prevention of strokes. In addition, in practical terms there may be very many measures which need to be implemented to tackle just one of the risk factors. The challenge is, therefore, to find the most effective combination of measures that can be practically implemented for the key factors contributing to cardiovascular disease in Europe. Some elements in the chain, such as high blood pressure, cholesterol and tobacco, act as a relatively direct cause of disease. In contrast, other risks are located further back in the causal chain and act indirectly through intermediary factors.

The fact that many chronic conditions and their risk factors are inter-related also means that targeting key risk factors for cardiovascular disease may help to prevent cancer and diabetes. So, while the focus in this report is on preventing cardiovascular disease, the population dietary goals outlined, along with the policy measures recommended in Section 2, would also help reduce the risk of other major diet-related diseases.

Nutrigenomics and epigenetics—how genes and diet interact

One area of science relating to nutrition and cardiovascular disease that has developed rapidly since our 2002 policy paper is the field of nutrigenomics. It has long been known that a combination of genes and environmental factors determine a person's susceptibility to a particular disease. In fact, individual differences in how blood cholesterol responses to dietary changes were recorded as early as 1933[34] and have been confirmed by later research.[35,36] At least some of this individual variability is due to genetic differences. Research points to many other genetic differences in the way our bodies deal with dietary fats and other factors related to cardiovascular risk.

Improving understanding of interactions between genetics and diet is now the focus of intense research interest. Learning more about these interactions has potentially important implications for improving the success of public health measures and the development of individualised prevention and treatment approaches.

Within this field, another important topic has emerged—*epigenetics*. It has only been discovered more recently that, although our genes are determined by inheritance, environmental factors in early life can influence whether some particular genes are "switched on" or not.[37]

Increasingly, research suggests that nutritional factors affecting the foetus or young baby can cause epigenetic changes that affect whether genes are expressed. These changes, in turn, may go on to influence susceptibility to metabolic and cardiovascular disease later in life. Nutritional factors that have been identified include under-nutrition in pregnant women, over-nutrition in pregnant women and post-natal over-nutrition in the young infant.[37,38,39]

The evidence to date from this rapidly expanding area of scientific knowledge, taken together with observational data on the effects of early nutrition on health in later life, suggests that pre-natal and early post-natal nutrition are vitally important areas for nutritional policy.

While the inter-related nature of chronic diseases such as cardiovascular disease, cancer and diabetes has long been recognised, a relatively new area of interest is the relationship between cardiovascular health and dementia. There is growing evidence that Alzheimer's Disease (AD) and other forms of dementia share risk factors (e.g. hypertension, diabetes, diets high in saturated or trans fatty acids, high calorie diets) and protective factors (e.g. increasing intakes of omega-3 fatty acids, regular physical activity) with cardiovascular disease.[40,41] Intervention studies are underway to further explore these issues.

1.2.2 The burden of alcohol-related harm

One of the differences between this position paper and the earlier 2002 report is the inclusion of more detail on the issue of alcohol.

Alcohol is said to be a risk factor in 60 types of illness and injuries, and a component cause in 200 other conditions. An analysis of the contribution of major risk factors to mortality and morbidity found that, globally, 3.8% of all deaths and 4.5% of the global burden of disease and injury was attributable to alcohol in 2004.[42] This represented 6.2% of all male deaths and 1.1% of deaths in women.[42] Alcohol use is the third most important risk factor in terms of disability-adjusted life years (DALYs) lost globally and it is by far the leading risk factor in deaths among young men (15-29 years). The biggest contributors to the global burden of disease attributed to alcohol were alcohol use disorders (including harmful use of alcohol, alcohol dependence and alcohol psychoses) which account for 34% of alcohol-attributable DALYs, followed by road traffic accidents (11.6%), cirrhosis (9.6%), other unintentional injuries (8.9%) and violence (7.8%).[42]

The European region is the heaviest drinking region in the world—with an average per capita consumption of 9.24 litres per year and where over a fifth of the adult population are binge drinkers.[43] Within Europe, there are very different drinking patterns. The proportions of non-drinkers in the population and the prevalence of heavy episodic drinking (binge drinking), for example, vary dramatically between countries. As a result, the extent of alcohol-related harm varies across Europe. The proportion of alcohol-attributable mortality in the Russian Federation and neighbouring countries (10-14% of all deaths), for example, is the highest in the world. In western Europe, by comparison, deaths due to alcohol use (2-4.9% of all deaths) are relatively low despite the high level of alcohol consumption.[40] The burden is considerable across Europe—alcohol consumption has been estimated to be the third largest risk factor for ill health in the European Union, at an estimated economic cost of €125 billion.[44]

It is estimated that a quarter of the gap in male life expectancy (see Section 1.1.2) between western and eastern europe in 2002 could be attributed to alcohol.[41] Risky drinking patterns (heavy episodic drinking) are more prevalent in eastern Europe and mortality rates for liver cirrhosis, alcohol-related liver disease, road traffic accidents, alcohol poisoning and violence are higher for both men and women in eastern Europe than other parts of the region.[4,43,44]

Research has suggested that low to moderate alcohol consumption can be protective against cardiovascular disease (see chapter 1.3.8 for a detailed review on this topic). WHO's estimate of the burden of alcohol-related mortality, therefore, is adjusted to take into account the assumed cardiovascular benefits—resulting in a net loss of 2.25 million lives globally in 2004.[42]

In recognition of this burden of alcohol-related harm, the World Health Assembly adopted a *Global strategy to reduce the harmful use of alcoho*l in May 2010.[45]

1.2.3 Prevention is key

Most cardiovascular disease is preventable. The complexity of the causal webs shown in Figures 7, 8 and 9 illustrate effectively that there are many points along the causal chain where action can be taken—good news for policy makers.

International comparisons have shown us for a long time that, in theory, a large proportion of cardiovascular deaths are preventable. If, for example, the cardiovascular death rates in men in Ukraine (543 per 100,000) in 2002 were reduced to the same level as they were in Japan (47 per 100,000) this would amount to a reduction of over 90% in male cardiovascular mortality in Ukraine.[46] Experience has also shown us that these deaths are preventable in practice. As outlined above, research suggests that a large proportion of the dramatic reductions in coronary heart disease death rates in recent decades can be attributed to changes in exposure to risk factors. In other words, prevention has played a major role.

We also know that reducing exposure to risk factors can work in a relatively short timescale. In Poland in the 1990s, for example, there were significant—and rapid—reductions in cardiovascular disease mortality rates.[22] In younger adults (between 20 and 44) there was an average annual decrease of 10% in heart disease death rates, while in 45-64 years olds the rate of decline was nearly 7%.[47] Following the political and economic changes in 1991, the removal of price subsidies for products containing animal fat, mainly butter, resulted in greater consumption of vegetable fats and oils. This replacement of saturated fat with polyunsaturated fat is thought to be a major factor in the spectacular fall in cardiovascular disease mortality in the country.

Dietary and lifestyle changes can help prevent cardiovascular disease (primary prevention) and they can also help to prevent the progression of the disease in people who already have symptoms or have been identified as being at high risk (secondary prevention). Given the complex multi-factorial nature of cardiovascular disease, it is not possible to identify all those at risk and the risks are not confined to people in high-risk groups. In fact, the total burden of cardiovascular disease from those with a moderate level of risk factors is greater than the total burden from those at higher risk.[5] The potential to save lives and prevent ill-health is much greater if prevention efforts are also directed at the wider population, and not only those who show clinical signs of illness or risk factors. While diet and lifestyle changes are tremendously important for individuals with cardiovascular conditions or who have been identified as being at high risk, it is vital that prevention efforts also focus on the general population who do not have any CVD symptoms.

For policy makers, a fundamental point is that prevention is a cost-effective approach. The National Institute for Health and Clinical Excellence (NICE) in the UK has recently set out calculations on potential lives saved and associated cost savings of various prevention activities and policy options.[25]

4 Mortality rates were higher in the nine countries with low child mortality but high adult mortality classified as EUR-C by WHO namely, Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation and Ukraine.

Table 1 Potential lives saved and cost savings of public health interventions (based on UK calculations)[25]

Intervention and costs, where available	Estimates of lives saved	Potential savings
Salt reduction campaign to reduce average salt intake to 8.6g/person/day is estimated to cost €18.3 million.	Can avoid 6,000 deaths per year.	**Potential savings of around €1.8bn per year.** More ambitious targets (to a salt intake of no more than 6g/person/day) could lead to even greater savings
Legislation to ban trans fatty acids	Such legislation in England would save a total of 1.4 million life years for the population aged between 40 and 79.	**Such a measure would be cost-effective even it were to cost €2.4bn nationally.**
Local intervention to improve physical activity among people who are overweight in a high deprivation area (total population aged over 40 c.200,000).	If a modest 3% success rate is assumed for interventions, 2,508 people would achieve acceptable weight as a result of physical activity.	**The cost savings over a five-year period as a result of CVD events avoided are estimated to be €58,000 for that local area.**
Local dietary intervention to reduce cholesterol levels in a high deprivation area (total population aged over 40 c.200,000).	If a modest 3% success rate is assumed for interventions, 4,200 would sustain acceptable cholesterol levels as a result of compliance with dietary interventions.	**The cost savings over a five-year period as a result of CVD events avoided are estimated to be €240,000 for that local area.**

Prevention is key: main messages

- Cardiovascular disease is largely preventable.
- We know prevention can work: a large proportion of the fall in cardiovascular death rates in western Europe in recent decades can be attributed to primary prevention reducing exposure to risk factors.
- Small changes to risk factors can have a large public health benefit. And quickly.
- Prevention strategies should be directed at the whole population, and not only those who have symptoms or at high risk.
- A preventive approach is cost effective—from national policy changes to local interventions.

Although in broad terms we do know what works, it is still useful to have more information about the effectiveness of specific policies and interventions—the evidence base for prevention strategies can therefore be strengthened further. However, considering the scale of the problem, there is no time and no need to wait. Immediate action to implement recommended and proven policies and interventions is required.

The aim of population-based strategies is to shift the social norms in our societies—by encouraging an increase in healthy behaviour and by reducing health risks.[31] Population-based strategies take into account the social and economic determinants of health and, crucially, they recognise that the responsibility for tackling major risks lies with governments, as well as individuals.

1.3 DIET, PHYSICAL ACTIVITY AND CARDIOVASCULAR DISEASE PREVENTION – THE EVIDENCE

The population dietary goals in EHN's 2002 paper were based on the scientific recommendations of the Eurodiet Project published in 2001.[3] These recommendations and other current goals were appropriate and pragmatic in relation to the evidence available at the time. There is now a case, however, for taking a fresh look at the extended body of evidence and reviewing these goals.

The European Heart Network commissioned a series of reviews to examine the newest evidence in relation to the relationship between various nutrients and cardiovascular disease. For most of the nutrients or dietary components included in this report, an external expert review summarised relevant systematic reviews published since 2002. In a small number of cases, a member of the EHN's Nutrition Expert Group carried out the reviews.

The European Heart Network's Nutrition Expert Group then reviewed these expert contributions and agreed a series of intermediate and ambitious longer-term population goals.

1.3.1 Fats

This review was contributed by Jan I. Pedersen, Department of Nutrition, Institute of Basic Medical Sciences, University of Oslo, Oslo, Norway.

1.3.1.1 Total fat

Total fat includes all dietary lipids—both invisible fat from animal and vegetable sources as well as visible fat from vegetable oils, butter and margarine.

Both a too high and a too low fat intake may have health implications. Two main arguments have been used to justify a recommendation of a moderate total fat intake: a high fat diet may increase risk of coronary heart disease (CHD) and a high fat diet may promote overweight. How strong is the evidence for such associations? In the seven countries study a positive correlation was found between mortality from CHD in middle-aged men and the percentage of calories from fat in their diet.[48] The correlation was weak, however, and explained by the increase in saturated fat as total fat increases; the correlation with saturated fat was much higher than with total fat.[48,49] It is noteworthy that the men of Crete with the highest fat content in the diet also had the lowest mortality from CHD despite having the highest body weights. The fatty acid composition of this diet differed from the others with its high content of oleic acid derived from olive oil. These early observations suggest that the quality of the dietary fat is more important than the quantity of total dietary fat in determining the risk of cardiovascular disease. The fat composition of western diets with their high proportion of animal products is such that almost any increase in total fat will result in an increase in saturated fat, dietary cholesterol and energy density. This is one reason why IOM 2002[50] and The Dietary Guidelines for Americans 2005 (DGA 2005)[51] included a recommendation on total fat which they set as acceptable up to a level of 35% of energy (E%) from fat. The arguments were clearly set out in the Dietary Guidelines as follows: "A high intake of fat (greater than 35 percent of calories) generally increases saturated fat intake and makes it more difficult to avoid consuming excess calories. A low intake of fats and oils (less than 20 percent of calories) increases the risk of inadequate intakes of vitamin E and of essential fatty acids and may contribute to unfavourable changes in high-density lipoprotein (HDL) blood cholesterol and triglycerides."

A high fat diet has a high energy density, and physiological and metabolic studies on diets of different energy density fed *ad libitum* to adults show that the dietary energy density rather than the fat content *per se* is the factor conducive to weight gain through what has been termed "passive over-consumption." This unconscious over-consumption is much more likely if an individual is relatively inactive. This implies that when populations become progressively more inactive they need to consume a higher quality diet lower in energy density to avoid weight gain. This is why the WHO analyses on diet and physical activity in relation to chronic diseases[52] focused on energy density and physical inactivity as the two key features selectively determining societal weight gain. The report then emphasised the quality of the dietary fat as the principal issue to be considered when dealing with coronary heart disease. A low fat diet may also be favourable in relation to blood pressure. In an eight-month intervention study with 30 healthy young individuals it was shown that switching from an habitual Danish high fat diet (37 E% fat, 14 E% saturated fat, 45 E% carbohydrates) to a low fat diet (30 E% fat, 8 E% saturated fat, 58 E% high fibre carbohydrates) resulted in a significant reduction in total cholesterol, no change in triglycerides and in the men, a significant reduction in systolic blood pressure.[53] Meticulous metabolic studies have also shown that increases in total fat increase blood pressure when body weight, salt, fruit and vegetable intakes are held constant (DASH trials) but the increased risk of coronary heart disease induced by higher blood pressures is dominated by the impact of specific dietary fats on blood lipids and the process of atherosclerosis. Thus, there are populations with high blood pressure and a major problem of strokes but the development of coronary artery disease depends on increasing dietary saturated and trans fatty acid intakes with increases in the blood concentrations of total and low density cholesterol. Nevertheless, greater intakes of total fat (whether or not the fat is of a saturated kind) will increase the blood pressure and appreciably increase the risk of strokes.

The issue of total fat and its overall effect has for a long time been a controversial issue, in part because the reasons why children and adults gain excess weight are complex and relate to physical inactivity and several different aspects of dietary quality.[54] The amount of foods eaten depends on a marked variety of social, economic and physiological factors only some of which link to dietary quality. There are dietary factors which either promote or limit the likelihood of the individual eating an energy intake which is in

excess of their lower energy needs when they are physical inactive. The dominant dietary factor is the overall energy density of the diet and the additional likelihood of drinking energy-containing drinks.[52] A high fat diet is of high energy density[55] and therefore likely to promote overweight because of "passive over-consumption." The DGA 2005 also cite evidence that low fat diets tend to be hypo caloric compared to high fat diets and therefore might decrease the risk of obesity. They use this as an argument for the upper limit of 35 E% from fat. This level of fat intake is lower than many Americans consume but very high when related to the traditional diets of low and middle income countries where cardiovascular disease was unusual until comparatively recently.

A large number of studies on the relationship between fat intake and weight change have been published, both clinical and observational, but the results are conflicting. From two recent extensive reviews the conclusion was drawn that there is insufficient evidence for an association between total fat intake and body weight.[56,57] The fact that body weight is increasing in most European countries in spite of a decreasing trend in the fat content of the diet suggests that factors other than dietary fat *per se* are important determinants for the obesity epidemic. From the available literature one must conclude that in order to maintain or lose weight, calorie adjustment and physical activity are very important, and the calorie adjustment is influenced by the proportion of fat in the diet. However, to limit the likelihood of having a high energy density diet when one is relatively inactive requires the replacement of dietary fat with unrefined carbohydrates which, in general, have a high fibre content and are therefore bulkier. This is why the IOM and the American Dietary Guidelines 2005 advocate a dietary fat intake which can vary from 20 to 35% of energy depending on both the degree of physical activity and the other dietary components contributing to the energy density of the diet.

The IOM 2002 set a lower acceptable level of 20 E% from fat.[50] This was based on unfavourable effects of a low fat/high carbohydrate diet on blood lipids, i.e. increased triglycerides (TG) and lowering of HDL-cholesterol, increased total cholesterol/HDL-cholesterol ratio, and on the risk of insufficient intakes of polyunsaturated fatty acids (PUFA) and fat soluble vitamins.[50] In the DGA 2005 a modelling system based on dietary intakes in the US suggested, however, that 25 E% from fat was required to meet the desired level of PUFA given the nature of the fats and oils in the US.[51] A question that remains is to what extent increased physical activity and avoidance of overweight might counteract any unfavourable effects on blood lipids of a low fat/high refined carbohydrate diet.

The importance of considering the type of carbohydrate in association with changes in fat intake is fundamental. Studies with refined carbohydrate have suggested that there is no benefit in replacing saturated fat with carbohydrate, but these analyses relate to carbohydrates with a low fibre content, where the starch has been refined. The diets also often contain substantial amounts of free sugars. Thus, metabolic studies using refined carbohydrate diets have failed to show improvements in blood lipid levels, and some prospective studies assessing the impact of carbohydrates on cardiovascular disease have suggested that they may not be beneficial. This may not be the case, however, when fat is replaced by carbohydrates high in fibre.[53] Also, the remarkable reduction in cardiovascular death rates in Finland, with a marked fall in both total and saturated fat intakes and an increase in carbohydrate intakes, suggests that the type of carbohydrate may be of major public health importance. Finnish diets are recognised as having one of the highest non-starch polysaccharide contents in Europe and North America.[58]

Conclusion on total fat

The above data suggest that a population mean of 30 E% from fat is a reasonable interim target. The figure from the EHN 2002 report should thus be maintained. This is also in line with the recent European Guidelines on Cardiovascular Disease Prevention (ESC Guidelines).[61] European diets are high in animal products and a long-term goal to reduce saturated fat below the current recommendation will necessarily require further reductions in total fat. Reduction of total fat to 20 to 25 E% would thus be a reasonable long term goal and is in keeping with the IOM analyses provided that the quality of the fat is appropriate.

1.3.1.2 Saturated fat

Saturated fatty acids (SAFA) increase plasma total- and LDL-cholesterol which in turn are strongly related to the risk of CHD. The ESC Guidelines state that "There are strong, consistent, and graded relationships between saturated fat intake, blood cholesterol levels and mass occurrence of cardiovascular disease (CVD). The relationships are accepted as causal."[59] The LDL particle is considered the pathogenic factor for the development of atherosclerosis, the pathological basis of CHD. The rationale for the original recommendation to limit intake of saturated fat to 10% of energy was that at this level of intake mean total cholesterol will be around 5.2 mmol/L (200 mg/dL). In the 1970s and early 1980s an adult population mean of 5.2 mmol/L (200 mg/dL) or lower would contribute to a major reduction in CHD as a major public health problem.[60,61] However, based on both animal experiments and human epidemiological and physiological studies, it is now recognised that lower LDL cholesterol levels would be advantageous in limiting atherosclerosis. Thus, a total cholesterol of 3.88 mmol/l (150 mg/dL) or LDL-cholesterol of 2.58 mmol/L (100 mg/dL) would be desirable.[62]

It thus appears that there is no lower limit of cholesterol where the risk of CHD disappears. In the seven countries study CHD mortality was very low in the Japanese and Greek populations.[48] Mean total cholesterol was of the order of 3.6 – 4.4 mmol/L (140-170 mg/dL) and 5.2 mmol/L (200 mg/dL), and saturated fat intake below 5 E% and 7-8 E% respectively. Coronary heart disease has been shown to be almost non-existent in rural China when mean cholesterol levels are of the order of 3.5 mmol/L (135 mg/dL), with total

fat intakes about 15%E, and saturated fat intakes extremely low. This has raised the question of whether the goal for total cholesterol in the population should be set lower than 5.17 mmol/L (200 mg/dL). Based on the Chinese studies it has been suggested that in order to reduce CHD mortality to a minimum level the population mean should be reduced to 3.88 mmol/L (150 mg/dL).[63] This would probably require the amount of SAFA to be reduced to a level of around 5%E.[64] In the latest report from the American Heart Association the recommended level of SAFA intake is set at 7 E%[65] and the 2010 Dietary Guidelines for Americans[66] also recommends limiting saturated fat intakes to less than 7% of energy. A similar recommendation has been made for developing countries in Asia with total fat intake set at 21 E%, SAFA at 7 E%, MUFA at 7 E% and PUFA at 7 E%.[67] So far, no other expert groups have recommended such a low level of saturated fat intake.

The strong relationship between saturated fat intake and total- and LDL-cholesterol is based on a very large number of metabolic experiments reviewed in several papers and reports.[50,51,64] Increasing saturated fat also increases the ratio between LDL-cholesterol and HDL-cholesterol, a strong predictor of CHD risk. It should be mentioned, however, that even though HDL-cholesterol is a strong metabolic indicator of a reduced risk of CHD, there is so far no direct evidence that increasing HDL-cholesterol by drugs or diet lowers the risk of CHD.[68]

There is, as stated above, no lower limit for total cholesterol levels below which risk reduces no further. It therefore becomes a matter of judgment what the blood overall goal for total cholesterol level should be for the population. The DGA 2005 recommendations are based on the individuals' LDL-cholesterol level. For those with levels below 3.36 mmol/L (130 mg/dL) less than 10 E% from saturated fat is recommended and for those with elevated LDL-cholesterol (> 130 mg/dL) less than 7 E% from saturated fat is recommended.[51] This is consistent with the evidence-based recommendation for individuals made by the NCEP Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults,[69] and the Committee's meta-analysis of trials and a review of 33 more recent controlled trials on saturated fat intake and health.

In the more recent AHA Scientific Statement on Diet and Lifestyle Recommendations Revision 2006 a limitation on saturated fat intake to less than 7 E% is advocated for all individuals above two years of age.[70] No clear argument is given, however, for the 7 E% limit. It is simply stated: "In view of the positive relationship among dietary saturated fat, LDL cholesterol, and CVD risk, and current US intakes, the AHA now recommends a population-wide goal of <7% of energy."

As part of the Joint FAO/WHO Expert consultation on fat and fatty acids in human nutrition, Elmadfa and Kornsteiner reviewed the reports above and much of the background material.[56] They concluded that the recommendation should be for an intake not exceeding 10 E%, but this is based on a recognition that saturated fatty acid intakes in Europe are on average often appreciably above 10%. The final conclusion of the Expert Consultation is that dietary intakes of SAFAs should not exceed 10 E%.[71]

Recent dietary surveys in European countries have shown intake of saturated fat varying between 8.8 and 14.6 E% (after excluding Romania as an outlier with 26.3 E%).[72] In Scandinavian countries, for example, there has been a remarkable decline in CHD. In Finland and Norway in particular, a reduction in serum total cholesterol explains a large part of the decline and this has happened with a reduction in mean saturated fat intake from about 20% to about 12-13 E%.[73,74] The mean cholesterol levels in the European populations are still high, however. The MONICA study indicated a total blood cholesterol value near 5.8 mmol/L in the early 90s[75] while more recent data from a smaller selection of eight countries gave a mean of 5.3 mmol/L.[76] A further reduction is thus desirable. Whether the goal for saturated fat reduction should extend below 10 E% becomes as stated above, a matter of judgement. From the correlations between saturated fat intake and serum cholesterol given in the IOM 2002 it can be calculated that to reduce total blood cholesterol from 5.5 mmol/L (assumed to be near the population mean for the European population) to the proposed desirable level of 5.17 mmol/L (200 mg/dL) saturated fat intake has to be reduced by about 5 E%. This would favour a recommendation for a population mean closer to 8 E% than to 10 E%. However, the ESC Guidelines propose as one of the objectives of CVD prevention to achieve a total cholesterol level of < 5 mmol/L (190 mg/dL)[59] which implies an even lower saturated fatty acid intake.

Changing one nutrient will inevitably result in changes in other contributors to the diet's energy content and it is therefore necessary to investigate the effect of exchanging saturated fat for other nutrients. The question is what should replace SAFA? In metabolic experiments replacing SAFA by carbohydrates reduces total and LDL cholesterol.[64,77] In individuals with dyslipidemia and the metabolic syndrome, however, carbohydrates may actually result in increased triglycerides (VLDL) and lower HDL cholesterol which could, in theory, increase CVD risk. Large prospective studies in western populations have in fact shown increased risk when replacing SAFA by carbohydrate (see below).[78] This may appear a paradox since in many parts of the world, e.g. in China and in Japan during the time of the Seven Country studies, the population were consuming <15% total fat and well below 5% saturated fat at a time when CHD was considered a rare medical condition.[79] The reason for this discrepancy may be that these populations are characterised by consuming higher fibre, complex carbohydrates and having considerably higher energy expenditures and lower BMIs compared to affluent populations.[80]

Replacing saturated fat by PUFA in Western societies is the simplest way of reducing serum cholesterol on a population scale.[64,77] Prospective studies also show that replacing saturated fat by PUFA lowers the risk of CHD mortality.[78] Studies suggest that replacing SAFA by MUFA is less

efficient for cholesterol reduction[64,77] and epidemiological studies show that replacing SAFA by MUFA does not alter the risk.[78] There are, however, concerns that the older studies included in these analyses relate to a period when monounsaturated fatty acids in the diets featured in the studies were from industrial sources—rather than olive oil—and therefore accompanied by trans fatty acids which may have influenced the findings.

Considering the strong relationship between saturated fat intake and total cholesterol and risk of CHD one would expect an association between saturated fat intake and mortality from CHD. Such a relationship has not been consistently found, however. The recent extensive meta-analyses of a total of 28 cohort and 16 randomised control studies by Skeaff and Miller yielded disappointing results.[81] No association with intake of saturated fat was found. The most clear cut result was the increase in risk of CHD death and events with intake of trans fatty acids. Intake of very long-chain n-3 PUFA (VLCn-3 PUFA) was associated with decreased risk of CHD mortality and events. The results with PUFA intake were somewhat contradictory in that an increase in risk of CHD death was seen at the highest category of dietary PUFA while a 5% increase in PUFA intake was associated with a significant reduction in CHD events.[81] The authors discuss the shortcomings of this kind of study, in particular measurement errors, regression dilution bias, confounding etc.

A more recent meta-analysis of such studies also failed to find consistent associations between intake of saturated fat and risk of CHD.[82] This does not mean, however, that no such association exists. One problem with these studies is the adjustment for confounders, as seriously criticised in an editorial relating to this meta-analysis.[83] A major error in that analysis is the adjustment for serum cholesterol in six of the 16 studies that included CHD as the endpoint. SAFAs strongly affect blood cholesterol levels which means that adjustment for the cholesterol level removes the biomarker of the mechanism for generating CVD. This approach will, therefore, bias the results of the meta-analyses towards finding no association between dietary saturated fat intake and CVD. The marked individual variation in cholesterol responses to saturated fat intakes is well recognised to have a genetic origin. This variation also means that, in cohort studies within a single population with a relatively narrow range of SAFA intakes, it will be difficult if not impossible,[84] to discern the link between SAFAs and heart disease. Keys' multi-country studies allowed a much greater range of saturated fat intakes to be related to coronary heart disease and demonstrated the very clear relationship with blood cholesterol levels, a finding which was subsequently repeatedly confirmed.

Another main problem with studies on the effects of diet on health outcomes is the difficulty of obtaining reliable data for food intake. Most often food frequency questionnaires are used. With this method misclassification is considerable and it has been questioned whether it is at all reliable.[85,86] Any misclassification or random errors in the measurements will result in reduced association eventually to zero compared to any true association. Attempts to overcome this difficulty by selecting a biomarker for particular fat intakes—e.g. C17:0 as an index of dairy fat intake—are subject to substantial errors and do not necessarily provide an appropriate guide to dietary saturated fat intakes.[87]

An additional problem arises when attempts are made to relate animal food intakes to CVD, because different animal feeding systems will alter both the total amount of fat in the milk or carcass, and its fatty acid composition.

The recent meta-analysis[82] which failed to show a link between SAFA and CVD was also unable to study the effect of substituting one nutrient for another. This was the main focus of another recent pooled analysis of 11 cohort studies selected based on strict quality criteria, in particular the quality of dietary assessment.[78] Among the 344,696 persons included 5,249 coronary events and 2,155 coronary deaths occurred during a follow up of 4-10 years. The main finding was a significant decreased risk of CHD death and events when 5 E% of SAFA was replaced by PUFA in the diet. Replacement of SAFA by refined carbohydrate increased the risk slightly while no effect on the prevailing risk was observed when SAFA was replaced by MUFA.[78] In an editorial these results were discussed in relation to observations of earlier clinical trials with high P/S diets and the effects of PUFA on LDL-cholesterol and the ratio of total- to HDL-cholesterol and the conclusion was drawn that the studies suggest towards a risk reduction of PUFA substitution for SAFA of similar magnitude.[88] It seems reasonable, therefore, to conclude that there remains strong evidence that PUFA lowers risk of CHD when replacing SAFA in the diet. Interestingly, the findings of a recent large Danish prospective cohort study suggested that replacing SAFA with carbohydrates with high glycemic index (GI) was associated with increased risk of myocardial infarction while replacing SAFA with carbohydrates with low GI (i.e. fibre-rich) was associated with reduced risk.[89] This supports the contention that replacing SAFA with complex carbohydrates lowers the risk of coronary heart disease.

Conclusion on SAFA

Based on the above, a population mean of 8 to 10 E% (i.e. 9 E%) from SAFA keeps the cholesterol level at a level of < 5 mmol/L and this is compatible as an interim target for inducing a lower CVD risk in Europe. A longer term goal would be to reduce the risk further by reducing SAFA intake as much as possible, e.g below 7 E%. A reduction below a SAFA intake of 7 E% would, in practice, require an appreciable reduction in animal and vegetable SAFA fat intakes in many European diets.

1.3.1.3 Polyunsaturated fatty acids (PUFA)

This section summarises the latest evidence on polyunsaturated fatty acids, starting with linoleic acid (LA, 18:2 n-6), followed by alpha-linolenic acid (ALA, 18:3 n-3) and then very long chain n-3 fatty acids.

1.3.1.3.1 Linoleic acid

Linoleic acid and alphalinolenic acid cannot be formed in the body and are thus essential fatty acids. The estimated minimal daily requirement of LA to avoid overt clinical symptoms is probably of the order of 1 to 2% of energy intake.[56] Because of the low conversion of ALA to EPA a small amount of EPA (and DHA?) may also be required for optimal health. LA and ALA lower total- and LDL-cholesterol and already in the 1960s and 70s clinical trials indicated that increased intake of PUFA lowered risk of CHD. Based on reviews of the totality of studies performed since then[56,78,90,91] it is safe to conclude that there is now convincing evidence that increased intake of PUFA decreases the risk of CHD. The risk reduction is particularly important when PUFA are replacing SAFA in the diet and the reduction is seen over a wide range of PUFA intake.[78,90]

Based on the effects on total- and LDL-cholesterol and the results from observational and intervention studies a range from 6 E% to 11E% of PUFA has been proposed by FAO, following the FAO/WHO Expert Consultation in 2008, as acceptable for CHD prevention.[71] The AHA Science Advisory 2009 advocates an n-6 PUFA intake of at least 5% to 10%.[90] Adding about 1 E% as ALA to the proposed n-6 PUFA target then makes these two sets of recommendations essentially identical. As to the lower range it is notable that the reduction in CHD mortality in most European countries has been associated with an increase in PUFA intake.[73,74.92] Currently the PUFA intake is around 5 to 7% of energy in most European countries.[72] In the AHA Science Advisory 2009 report it is argued that there is little evidence to support a harmful effect of a omega-6 PUFA intake of up to 10% of energy and that even higher intakes may be safe and even beneficial.[90] In support of this conclusion are several studies including one from Israel where 25% of the population consumes >12% of energy as LA and where an inverse association was found between the concentration of LA in adipose tissue biopsies and the incidence of acute myocardial infarction.[93]

Recent analyses have been interpreted as suggesting that intake of LA is so beneficial that the upper recommended limit of 10E% linoleic acid should be reconsidered and increased.[90,94,95] The prospective studies cited above,[78] together with a re-evaluation of eight earlier randomised controlled trials (RCTs), are presented as support for such a view.[94] The aim of these trials was to test if a reduction in serum cholesterol by diet would lower the risk of CHD. At that time a low SAFA/high PUFA diet was known as the most efficient way of reducing serum cholesterol. PUFA intake in the trials varied between eight and 20 E%. Seven of these eight trials showed a reduction in CHD events. Based on the reported intake of PUFA the reduction in risk was estimated to be 13% for each 5 E% increase in PUFA. However, the use of the results of these RCTs as an argument for a high consumption of n-6 PUFA was recently strongly opposed.[96] The basis for the different perspective was that some of the trials had weak designs and should have been excluded (e.g. the two Helsinki mental hospital trials), whereas other trials with an unfavourable outcome should have been included, particularly those that used a high corn oil diet (which is very low in ALA). When dividing the trials into those that included both LA and ALA, rather than LA alone, then a reduction in risk was observed when an appreciable amount of ALA was included in the PUFA whereas those trials that used corn oil (containing LA but almost no ALA) showed an increase in risk.[96] Furthermore, in all the trials PUFA substituted not only SAFA but probably also an appreciable amount of trans fatty acids (TFA), so the lowering of risk may to a large extent be explained by a reduced intake of TFA rather than increase in PUFA.

It should be added that the aim of all these trials was to test if a reduction of serum cholesterol by diet would reduce the risk of CHD and they were not designed to test optimal intakes of any PUFAs. Unfortunately, the meta-analyses[94,96] of these different trials do not include data for effects on serum cholesterol and how this related to analysed changes in CHD risk.

We may conclude that at the present time there is insufficient evidence to advocate particularly high intake of linoleic acid and that care should be taken to ensure that PUFA in the diet includes ALA and the very long chain n-3 fatty acids in addition to LA. Given the potential, although much debated, concern for the proposed inflammatory and prooxidative properties of n-6 PUFA[97,98] their inhibitory effects on the metabolic conversion of the n-3 series of fatty acids to important biologically active leucotrenes, a prudent policy seems sensible. This cautious approach is also reasonable because no population has yet been exposed to life long very high intakes of PUFA. For these reasons one should not propose on the basis of current evidence unnecessarily large increases in n-6 PUFA intakes.

<u>Conclusion on PUFA</u>

The evidence is in keeping with the FAO/WHO Expert Consultation recommendation that the intake of PUFA (LA plus ALA) should be between six and 11% of energy based on a total fat content of 30 E%. A longer-term goal, which takes account of lower total fat and saturated fatty intakes, would require a PUFA intake of between five and eight per cent of energy, based on a proportionate reduction in PUFA intakes.

1.3.1.3.2 Alphalinolenic acid (ALA)

Alphalinolenic acid (ALA) is an essential fatty acid and an intake of 0.5 to 1 E% will cover the minimum requirement.[56] ALA reduces total- and LDL-cholesterol in a manner similar to that of LA. It may therefore be inferred that it also reduces the risk of CHD when replacing saturated fat. Epidemiological studies have, however, given conflicting results. While some have demonstrated a beneficial association between ALA intake and risk of CHD[99] a review of prospective cohort and randomised trials could not demonstrate a significant association between ALA intake and risk of CHD events or CHD deaths.[81]

The n-6 and n-3 fatty acids use the same series of enzymes for their metabolism—in fact they compete for them. In-vitro research suggests that the important Δ6 desaturase enzyme prefers alphalinolenic acid to linoleic acid, followed by oleic acid.[100]

In view of the essential biological need for ALA, its cholesterol lowering effect and to allow for competition between the metabolic pathways of the n-3 and n-6 fatty acids in generating important metabolites of major importance for cell regulation, it is recommended that ALA makes up at least 1 to 2%E so that over the range of total PUFA intake the ratio of n-6 to n-3 does not exceed five. However, it should be noted that there is no consensus in the literature about any optimal level or the usefulness of such a ratio, and for planning purposes a ratio of n-6 to n-3 PUFA between three and nine has been proposed to be adequate.[101]

1.3.1.3.3 Very long-chain n-3 fatty acids (VLCn-3 PUFA)

From several recent reviews one can draw the conclusion that there is convincing evidence that intake of VLCn-3 PUFA in the form of fish or as supplements of EPA and DHA reduces the risk of CHD death and sudden cardiac death and possibly CHD events in patients with heart disease.[56,81,102-104] A pooled analysis of both prospective cohort studies and randomised clinical studies in individuals with pre-existing CHD demonstrated a 36% lower risk of CHD death at an estimated intake of 250 mg/d of EPA + DHA with hardly any further decrease above this level.[103] It should be noted, however, that when related to fish consumption one to two servings of (oily) fish per week, i.e. approximately 250 to 500 mg per day (mg/d) was associated with near maximal reduction in risk.[105] After having reviewed the available quantitative evidence a recent symposium came to the following conclusion: "Consumption of 250 mg DHA and EPA per day, from either dietary or supplement sources, should be part of management for primary prevention of CHD death and after a coronary event to reduce risk of CHD death. Given the uncertainty of this estimated target intake and no evidence for harm at higher intakes, a target of 250–500 mg/d EPA plus DHA is reasonable. Differences based on relative amounts of EPA versus DHA, if any, still need to be determined".[106]

Conclusion on VLCn-3 PUFA

There is convincing evidence that intake of VLCn-3 PUFA reduces the risk of CHD. The current state of knowledge supports a recommendation of a minimum intake of 250 mg/d of VLCn-3 PUFA as a population goal for the prevention of CHD. This is equivalent to two portions of fish per week.

1.3.1.4 Monounsaturated fatty acids (MUFA)

Oleic acid (18:1 n-9) is the most abundant fatty acid in food and in the organism. Meat and dairy fat are the most abundant sources in western diets. Vegetable oils like olive oil and rape seed oil are also important sources. Olive oil may be a dominating source of oleic acid in olive growing Mediterranean countries. Small amounts of 16:1 and 18:1 n-7 are also present in small amounts in foods. However, very long chain monoenes like erucic acid (22:1n-9) in rape seed oil and mustard oil and cetoleic acid (22:1 n-11) in marine oils have been suspected of having cardiotoxic effects in animals. In view of these concerns, the rape seed oil now on the market as canola oil is practically devoid of erucic acid.

There is no physiological need for oleic acid since it can be rapidly formed by desaturation of stearic acid by an omnipresent steaoryl-CoA desaturase.

Metabolic experiments have shown that oleic acid when replacing carbohydrates in the diet has practically no effect on total cholesterol or LDL-cholesterol but increases slightly HDL cholesterol and decreases triglycerides.[78,107] When replacing SAFA and trans fatty acids LDL-cholesterol is reduced while HDL-cholesterol remains unchanged.[78,108] A diet high in oleic acid appears to have a more favourable effect on HDL-cholesterol and triglycerides than a diet high in complex carbohydrates.[109] Prospective cohort studies have given more mixed results. This is understandable since the results might be confounded by changes in the intake of saturated fatty acids where the main sources are meat and milk fat and by different polyunsaturated fat intakes where the source of MUFAs is mainly vegetable oils. In the most recent meta-analysis no association with risk of CHD death was found when SAFA were replaced by MUFA.[78]

Conclusion on Monounsaturated fatty acids

Since there is no physiological need for oleic acid and the risk reduction linked to MUFA *per se* is, if present, rather modest then any recommendation has to be set on the basis of simply providing the fat which accounts for the difference between total fat and the sum of SAFA, PUFA and TFA intakes. A distribution of <10 E% SAFA, 6-11 E% PUFA and <1 E% TFA and a total of 30 E% would leave 8 to 13 E% for MUFA. In most European countries the intake of MUFA is around 12-13 E% (Greece is an exception with 22 E%) when based on food availability data[72] while around 11 E% when based on surveys.[101] A longer-term goal for MUFA intakes, taking into account the reductions in total, saturated and trans fatty intakes, is between 7.5 and 9.5 E%.

1.3.1.5 Trans fatty acids (TFA)

Trans fatty acids (TFA) are formed by bio-hydrogenation in the rumen or industrially by partial hydrogenation of vegetable and marine oils. During heat treatment of fats and oils by processes such as deodorisation, cooking, frying etc a small amount of trans bonds may be formed.[110] These processes can affect the chain length, number of double bonds and the unusual position of cis bonds and the insertion of trans double bonds means that the resulting fats can have an almost uncountable number of

isomers with a number of unnatural cis-isomers and a wide array of, often unknown, metabolic effects.[111] Any specific health effects of these different isomers are, therefore, also unknown. Because of their effect on total cholesterol, TFAs were for long considered similar to saturated fatty acids, while analytically they were traditionally included in the monounsaturated fatty acids category. This, in practice, meant that there was an overestimation of the content of monounsaturated fatty acids in the diet. This situation changed with the publication of Mensink and Katan who demonstrated that TFAs increased LDL-cholesterol but also decreased HDL cholesterol with a magnitude of effect on a weight-to-weight basis which implied they could be more harmful than SAFA.[109] Since then a large number of publications have appeared demonstrating that in fact the intake of trans fatty acids is associated with an increased risk of CHD and this risk may be two to three times higher than that of an equivalent intake of SAFA.[112,113] Replacement of TFA by any other fatty acids may thus be potentially favourable. It should be noted that the comparison between TFA and SAFA is done with a mixture of SAFA in an ordinary diet i.e. including fatty acids that may not be associated with CHD risk. No attempt has been made to compare with palmitic acid, an important cholesterol increasing fatty acid in the diet. The widespread replacement of trans fat by palm oil with increasing intake of palmitic acid is thus of limited benefit.[114]

A WHO Scientific Update on Trans Fatty Acids was presented at a meeting in Geneva in 2007 and extensive scientific reviews of the participating experts have recently been published.[115] The conclusions from this Update are quite clear: there is convincing evidence that the intake of trans fatty acids is associated with an increased risk of CHD. TFA produced by partial hydrogenation should be considered as an industrial food additive, having no demonstrable health benefits and posing a clear risks to human health. The evidence on the effects of TFA and disease outcomes strongly supports the need to remove partially hydrogenated vegetable oil from the human food supply.

Ruminant TFA and industrially produced TFA have similar metabolic effects.[116,117] The ruminant TFAs cannot, however, be removed from the diet unless one avoids all ruminant-sourced fats. Their intake is low in most populations, generally below 0.5 E%[117] and therefore probably not of any significant health concern. Small amounts of TFA also result from refining of oils, particularly during deodorisation. A realistic upper limit of total intake of TFA has been set at 1 E% by WHO.[52] Removal of all partially hydrogenated fat from the food chain, selecting optimal conditions for refining of oils and some reduction in ruminant meat and dairy fat makes it possible to set an upper limit of 0.5 E%.

Conclusion on trans fatty acids

There is convincing evidence that intake of trans fatty acids is associated with increased risk of CHD and more so than the intake of SAFA. The intake should be limited to <0.5 E%.

1.3.2 Salt

This review was contributed by Professor Per Ole Iversen of the University of Oslo, Norway, and Stellenbosch University, South Africa.

1.3.2.1 Brief background—the salt issue up to 2002

Salt and its effect on human health has been a debated issue for centuries. The possible detrimental effect of salt on hypertension and blood pressure–related diseases—the "Salt hypothesis"—became of considerable interest when Kempner introduced his rice diets with exceptionally low salt intakes as a method of coping with patients with very high blood pressure.[118] Then after the second world war, Dahl and colleagues[119,120] showed that animals could be bred to reveal marked salt sensitivity, therefore suggesting that salt sensitivity was genetically inherited. Given the differential response of patients to reduced intakes of salt this also led to the concept that in humans there was a subgroup with salt sensitivity who would need to be advised to reduce their salt intake. This led to a great debate between Pickering[121] and Platt[122] as to whether one could readily distinguish a separate group in the population with higher blood pressures as a means of providing separate advice and management. Miall[123] then showed, as have many others, that there is a continuous spectrum of blood pressures within the population with a progressive increase in cardiovascular events as the systolic blood pressure rises from 115 mmHg.[124] This was traditionally considered a low-normal value. Rose then highlighted that if one sought to limit the hazards associated with high blood pressure (or indeed high blood cholesterol levels) then there is greater benefit in reducing the average blood pressure or cholesterol level of the whole population rather than focusing on a smaller group with particularly high blood pressure or cholesterol levels.[125]

Until the late 1980s progress in the salt field was hampered by small, and usually insufficiently powered, studies in humans. In addition, the estimates of salt intake in different population groups were challenged on methodological grounds. Many studies were based on interviews using food frequency questionnaires, a method with several recognised pitfalls. The gold standard remains the 24-hour sampling of urine to determine sodium excretion, a cumbersome method that is not always applicable. It was also realised that the major part of salt consumption (perhaps 70-80%) stems from processed foods[126] and often with insufficient reporting of salt content by the manufacturers.

Closer to 2002, some larger and important studies were published. The INTERSALT-study from 1988 remains the largest single study (of 10,079 individuals from 32 countries) to date reporting on the association between sodium excretion and blood pressure levels.[127] The authors reported a significant, albeit low, correlation between sodium excretion and blood pressure across populations. Similar findings based on integrated analyses were later published by Law and colleagues based on sodium

excretion data from about 47,000 individuals sampled from 24 different populations.[128-130] Both studies have, however, been criticised in terms of the complex methods used in taking account of country and other differences.[131-133]

The relevance of these studies which found correlations between salt intake and blood pressure in population groups was then amplified in 2001 by meticulous dietary studies involving testing the impact of altering specific components of the diet without weight loss in two groups with normal blood pressure and high blood pressure.[134] In these DASH trials involving 412 normotensive and pre-hypertensive individuals it was shown that in the whole group reducing the salt intake to intermediate levels led to a fall in systolic blood pressure of 2.1 mmHg with a further fall by 4.6 mmHg when the salt intake was reduced to the lowest level i.e. 3g salt/day. These reductions occurred when the diets were relatively low in fruit and vegetables but high in total and saturated fat. This full 6.7 mmHg fall compared with a total fall of 3.0 mmHg when equivalent reductions in salt intake were made but when diets were higher in fruit and vegetables and low in fat. Thus salt reduction produced greater falls in blood pressure in those on less healthy diets but on each diet there was a statistically significant reduction in blood pressure. Among non-hypertensive participants who received the control diet, lower (vs. higher) sodium intake decreased blood pressure by 7.0/3.8 mm Hg in those older than 45 years of age ($P < 0.001$) and by 3.7/1.5 mmHg in those 45 years of age or younger ($P < 0.05$).[135] All subjects with higher blood pressures when starting the DASH trials showed an even greater fall of 11.5 mmHg in their systolic blood pressures i.e. equivalent to that achieved by an effective drug for high blood pressure. This implies that the more susceptible groups in society would gain even greater benefit from consuming a lower salt diet.

These changes may seem modest but were statistically very significant because of the care taken with both the dietary changes and blood pressure measurements. The blood pressure changes are observed most readily in those where no other dietary changes are made,[136] so can be taken to apply to the effects of reducing salt intake in an otherwise unchanged European diet. Although the DASH trials were short term interventions with dietary changes similar to those of the DASH trial but also involving a modest reduction in body weight for several years were already recognised as also reducing the incidence of hypertension over a five year period.[137]

Numerous major intervention studies have shown that lowering the average blood pressure of a group of adults leads to marked falls in both the incidence of myocardial infarction and strokes and in cardiovascular mortality.[138] Thus, for example, a 5 mmHg reduction in diastolic blood pressure reduces the risk of stroke by at least 34% and of myocardial infarcts by 21% and this is seen in blood pressure ranges as low as 120-130 mmHg systolic and 70-80 mmHg diastolic.

1.3.2.2 Important questions addressed 2002—2009

This review will focus on four key questions about the association between salt intake and blood pressure.

Is there a relation between salt intake and blood pressure in community studies?

Concerns were raised about whether the INTERSALT study really provided evidence for a relation between salt intake and blood pressure across different populations. Several ecological analyses have reported conflicting results as to the relation between salt intake and blood pressure levels in various population groups, but as always there are many different factors in addition to salt that may have complicated the interpretation.[139-141] Three Cochrane reviews of intervention studies came to somewhat conflicting conclusions. He and MacGregor based their findings on a meta-analysis of 17 trials with hypertensives and 11 trials with normotensives, and concluded that lowering of salt intake for four weeks or more would lower blood pressure in both normotensive and hypertensive subjects.[142] Hooper *et al* assessed a small number of trials (five with hypertensives and three with normotensives), while Jurgens and Graudal studied 57 trials with hypertensives and 58 trials with normotensives.[143,144] Jurgens and Graudal found that in normotensive subjects reducing salt intake induced a fall in systolic blood pressure of 1.27 mmHg, whereas in those with an elevated blood pressure the low salt diet reduced systolic blood pressure by 4.18 mmHg. Both these effects were highly significant ($P <0.0001$). The authors concluded that these changes in blood pressure did not warrant a general recommendation to reduce salt intake in people with normal blood pressure, but their analyses were not concerned with the impact of small changes in blood pressure on cardiovascular morbidity and mortality at the population level.

Although the meticulously organised DASH trial showed a clear dose response relationship between salt intake and blood pressure in adults, a meta-analysis of 10 trials of children and three trials of infants by He and MacGregor in 2006, showed that a dose response was not evident. However, different degrees of salt reduction invariably reduced blood pressure.[145]

Additional support for the induction of higher blood pressure by salt in a graded dose dependent manner was shown in careful feeding studies in chimpanzees, a species phylogenetically close to humans.[146] The lowest and highest doses of salt used were equivalent to 0.1-1.5 g/d and 15 g/day, values equivalent to those observed in some tribal groups with practically no salt sources and in Japan where traditionally salt intakes were very high. Intakes of 15 g used to be common in Europe but further confirmation of the important graded relationship between salt intake and blood pressure came in a subsequent chimpanzee study where smaller amounts of salt ranging from the human equivalent of 2 to 12 g salt/day were used. Blood pressures returned to normal on withdrawal of the intervention with salt.[147]

In summary, reducing salt intake lowers systolic blood pressure by 3-7 mmHg (depending on age) in adults with a normal blood pressure and weight (BMI between 20 and 25 kg/m^2). There is also clear evidence that individuals with higher blood pressures reduce their systolic blood pressure to a greater degree than normotensives when salt intakes are reduced.

Mechanisms for salt induced high blood pressure

Many investigators have attributed the blood pressure raising effects of increased salt intake to an increase in the volume of circulating blood. The inherent argument was that the increased plasma sodium would lead to greater thirst, hence increased water intake, as well as a distribution of fluid from the intracellular to the extracellular compartment.[148] However, the data are inconclusive. As reviewed, short-term salt infusions into man or dogs apparently do not raise blood pressure.[149] Possible explanations for these observations might be rapid changes in arterial blood vessels leading to compensatory vasodilation and/or increased natriuresis. Other explanations for salt-induced hypertension, by mechanisms not related to blood volume changes, include possible effects of sodium on its renal excretion through direct effects on various transport systems or by mechanisms involving the arterial vessel walls.[150-152] Specific polymorphisms have also been identified for genes coding for transport proteins within the renal excretion systems, suggesting that individuals carrying these polymorphisms might be more prone to raised blood pressure upon increased salt intake.[153]

In summary, there is yet no universally accepted explanation of how increased salt intake increases blood pressure. Several mechanisms, both volume-dependent and volume-independent, are likely to play a role.

Salt-sensitivity – still a concept to consider?

The term "salt-sensitivity" was coined in order to possibly identify individuals susceptible to lowering of blood pressure upon reductions in salt intake. Conceptually, the term appeared fruitful. However, despite numerous attempts to reach a unifying standardisation, there are still wide differences in how salt sensitivity is defined, and it has been hard to obtain reliable results when performing repeated tests on salt sensitivity.[154-156] Given the inability to identify a separate group of hypertensive individuals, other than by taking arbitrary definitions of what constitutes a raised blood pressure, and recognising that multiple genes with potentially different mechanisms are involved in the control of blood pressure, it is currently unwise to consider the identification of specific salt-sensitive sub-groups within society as a practical strategy for tackling the major public health problem of hypertension.

In summary, it has not been possible to agree on a definition of the term salt-sensitivity, and in any case, such a definition is of little practical use and neglects the important benefit that could arise should the whole population reduce its average salt intake.

Does increased salt intake lead to increased mortality or morbidity of blood pressure-related diseases (stroke and other cardiovascular disorders)?

Hypertension, in addition to being classified as a disease itself, is the major risk factor for stroke, and a substantial risk factor for cardiac diseases.[157] Ischaemic heart disease and cerebrovascular disease are the most important risk factors for death and disability in high-income countries.[158] WHO has calculated that a global salt-reduction strategy could prevent about 8.5 million deaths worldwide.[159]

Definitive proof that reducing salt intake selectively reduces the burden of cardiovascular disease is unrealistic, as it would require major long term randomised trials with very high costs and major logistical difficulties.

Notwithstanding this, in the late 1990s and early in this decade a number of short-term studies were performed and in recent years there have been follow-ups of some of them. Complicating many of these studies were heterogeneous study populations, e.g. inclusion of both normotensives and hypertensives as well as people with normal and elevated body mass index, confounding factors that have been more or less adequately statistically handled. Also, the chosen interventions have in many studies varied from a mere reduction in salt intake to a combined change in several lifestyle factors including diet and physical activity.

The intervention PREMIER-trial showed that a combination of the DASH-diet, in addition to modifications of several lifestyle factors, increased the prevalence of optimal blood pressure (< 120 mmHg systolic and < 80 mmHg diastolic) to 35% compared with 19% among those who were only given usual advice about nutrition and physical activity.[160]

The randomised Treatment of Hypertension in Older Persons (TONE) intervention study from 1998 showed that, in a modest-sized and relatively short term study, marked effects were seen in terms of blood pressure and the reduced need for medication. A reduction in salt intake of as little as 1.8 g/day or less among elderly (60-80 years), led to a significant decrease in the main outcome measure (a composite measure of hypertension, use of anti-hypertensive treatment or cardiovascular event) compared with a control group (relative hazard ratio 0.69; 95% CI 0.59-0.81), in particular among obese participants. However, the small reduction in cardiovascular events in the lower salt intake group was insufficient to achieve statistical significance in this small, short term trial.[161]

A Finnish study prospectively followed two cohorts from 1982 and 1987, respectively, until 1995. For each 100 mmol greater sodium excretion/24-hour (equivalent to a 6 g/day difference in salt intake) the hazard ratios for coronary heart disease and other cardiovascular disease were 1.51 (95% CI 1.14-2.00) and 1.45 (1.14-1.84), respectively. Interestingly, the frequency of acute stroke was not significantly associated with increasing sodium excretion.[162]

The Trials of Hypertension Prevention (TOHP) study

prospectively followed adults with prehypertension recruited from 1987 to 1988 and from 1990 to 1992. This was a randomised trial lasting 1-3 years testing the effects of various lifestyle interventions including reductions in salt intake. Data collected 10-15 years after enrolment were presented in 2007 and showed a 25% reduction (relative risk 0.75; 95% CI 0.57-0.99) among those who had reduced their salt intake between 33 and 44 mmol/24-hr (equivalent to a 2.5-3g reduction in salt intake) compared to the controls.[163] The authors also found that a higher potassium intake was associated with a reduction in the frequency of cardiovascular disease and this is in keeping with the well-recognised effect of dietary potassium in countering the hypertensive impact of sodium.[164] Numerous studies have shown that increasing potassium intake lowers blood pressure, so it is not surprising that in the DASH study, where there was a selective increase in potassium-rich fruit and vegetables, there was a clear reduction in blood pressure in the normotensives and a greater fall in the hypertensives among those allocated to the intervention diet.[165,166] The DASH study also suggested the usefulness of urinary potassium excretion as an index of fruit and vegetable consumption and blood pressure reduction.

Attempts to consider this issue at a national level have come from analyses of the US National Health and Nutrition Examination Survey (NHANES) involving nearly 9,000 participants that were followed between 1971-75 and 1988-1994. This prospective, observational study attempted to link self-reported estimates of salt intake to the incidence of heart disease in three different surveys. All three surveys[166-168] tended to show an inverse relationship between the presumed intake of sodium and the individuals' subsequent mortality but all relied on a single 24-hr recall of the diet which is notoriously inaccurate before one considers the uncertain composition of salt in the foods reportedly consumed. More robust studies from Finland have shown marked secular reductions in salt intake and these changes, accompanied by a substantial reduction in the dietary fat content, were accompanied by a marked fall in the average blood pressure, stroke and myocardial infarction rates of the Finnish population.[170]

So far, probably the most convincing support for a decrease in salt intake in terms of reducing blood pressure-related diseases, comes from a recent meta-analysis of 13 studies involving about 177,000 participants with follow-up periods lasting between 3.5 and 19 years. After adjustments for confounding factors, it was concluded that an increased salt intake (equivalent to a change of 6 g/day) was associated with a relative risk for stroke of 1.24 (95% CI 1.06-1.43), whereas the relative risk for other cardiovascular disease was 1.14 (95% CI 0.99-1.32).[171] Similarly long term, i.e. 10-15 years, prospective analyses of the randomised hypertension prevention trials show that the combination of advice to reduce salt intake and alter diet to improve potassium intakes was associated with a significant reduction in subsequent cardiovascular disease, i.e. myocardial infarction stroke, coronary, revascularization, or mortality from cardiovascular diseases.[175,176]

In summary, almost all large scale studies with longer term follow up and better measures of salt intake now report that reducing salt intake will contribute to a fall in average population blood pressures which will then induce a reduction in the incidence of hypertensive disorders. Individuals with prehypertension, established hypertension and those who are overweight or obese, seem to benefit the most from reducing their daily salt intake.

1.3.2.3 Recommendations

Many studies report that about 10 g of salt is consumed per day among adults in most western European nations.[172] From a physiological point of view, as little as 1-2 g of salt per day is adequate. Most international recommendations are for 5-6 g of salt per day. WHO recommends a daily salt intake of no more than 5 g.[52] The American Heart Association and the American Dietetic Association recommend an upper intake level of 5.75 g or less per day.[173,174] The Nordic recommendations are 6 g/day for women and 7 g/day for men.[101] Some more recent recommendations have been more ambitious—in 2010 the UK's National Institute for Health and Clinical Excellence (NICE) proposed 3 g/day by 2025[25] and the US Dietary Guidelines for America 2010 propose a goal of 3.75 g/day (1.5 g/day sodium).[66]

This requires a co-ordinated government-led approach as shown by the differences in national response to efforts to reduce salt intake. Thus a recent systematic review of 38 studies with about 26,000 subjects' salt intakes in the US[175] showed that salt intakes, of approximately 9 g/day, have been virtually unchanged from 1953 to 2003, and with no particular trend for gender or ethnicity. Within that period there have been several attempts to lower salt intake in the US by using educational approaches to the population with seemingly dismal results. This contrasts, however, with national data from European studies where governments have been implemented policies involving a specific and systematic reduction in salt intakes with the food industry re-formulating its products. The reduction in salt intakes following a systematic multi-sectoral approach can be substantial[176] and is partly responsible for the 80% reduction in cardiovascular disease in Finland over the last 35 years.[170] Salt is used a vehicle for iodine fortification to eliminate iodine deficiency, and concerted action is, therefore, important to ensure that salt reduction strategies are compatible with action to improve iodine intakes.

In summary, given the available evidence it seems justified to propose an interim recommendation of maximally 5 g salt intake per day for adults, with a more ambitious longer-term goal of less than 4 g/day. International experience suggests that a reduction of salt intake should be implemented gradually and requires an integrated approach, both from public authorities and from the industry.[177]

1.3.2.4 Conclusions

- The precise amount of daily salt intake among adults in Europe is not known, but probably lies around 10 g.
- Falls in salt intake reduce blood pressure as do increases in the intake of potassium-rich foods such as fruits and vegetables. Recent follow-up data of randomised trials, with modest salt reductions, show clear evidence of a reduced blood pressure and a fall in cardiovascular morbidity and mortality.
- The mechanisms of how salt raises blood pressure are still being explored.
- A maximal daily consumption of 5 g/day seems reasonable for adults in the interim. In the longer term, a population goal of less than 4 g/day is proposed. In order to achieve this goal on a population-based level, policies for a progressive decrease are needed.

1.3.3 Sugars

This review has been contributed by Marleen A. van Baak of Maastricht University Medical Centre+, Maastricht, The Netherlands.

1.3.3.1 Background

Over the past years a role of dietary sugar intake in the pathogenesis of cardiovascular disease has been suggested.[178-183] This relates to the consumption of dietary sugars in general,[179,181] the intake of sucrose,[178] the consumption of sugar-sweetened beverages,[180] or to a specific role for fructose consumption.[182,183] Effects of dietary sugars on endothelial function, overweight, insulin resistance, dyslipidemia, blood pressure, uric acid and inflammation have been implied as mechanisms underlying a potential association between sugar consumption and cardiovascular disease. In its report on Food, Nutrition and Cardiovascular Disease Prevention in the European Region, published in 2002, the European Heart Network has set a population goal for sugary foods of four or fewer occasions per day for the prevention of cardiovascular disease in Europe[1] based on the available evidence at that time. This section reviews the evidence for a role of dietary sugars in cardiovascular disease and its potential mechanism(s) with emphasis on the new evidence that has emerged since 2002. This comes from large observational studies and from controlled studies in humans. These new data give further support to a potential negative effect of high dietary sugars—and especially dietary fructose—consumption on cardiovascular disease risk, although the evidence remains inconsistent.

1.3.3.2 Definition of dietary sugars

Dietary sugars are glycaemic carbohydrates.[5] The main dietary sugars are the monosaccharides glucose and fructose and the disaccharides sucrose and lactose. Sucrose consists of a fructose and a glucose monomer, lactose of a glucose and galactose monomer. In this paper the term total sugars is used for the combination of all mono- and disaccharides. Added sugars refers to any form of sugars added during food preparation, manufacturing or consumption. When the singular sugar is used, sucrose is meant. High-fructose corn syrup is not addressed as a separate entity, because its composition is very similar to that of sucrose. Sugar-sweetened beverages contain caloric sweeteners such as sucrose or high-fructose corn syrup.

1.3.3.3 Dietary sugar consumption in Europe

The European Food safety Authority (EFSA) has recently published an overview of total sugars and sucrose intake (as % of energy intake (E%)) of children and adults in European countries. In children and adolescents between five and 18 years of age the average total sugars intake varied between 22 and 36 E% and sucrose intake varied between 12 and 19 E%, in adults total sugars varied between 17 and 36 E% and sucrose between 8 and 18 E% among European countries.[184] Reported standard deviations ranged between 5 and 7E% for both total sugars and sucrose intake.[184] No data on added sugars consumption in Europe have been found. However, most of the dietary sucrose consumption comes from added sucrose and EFSA noted that the average intake of (added) sugars in some EU member states exceeds 10 E%, especially in children.[184] In the US average daily added sugars consumption was estimated at around 350 kcal or 90 g/d in 2001-2004, one third came from soft drink consumption.[181]

1.3.3.4 Dietary sugar and risk of cardiovascular disease

Since the early observation by Yudkin and Roddy[185] that patients with recent myocardial infarction (MI) and peripheral artery disease (PAD) had higher consumption of total dietary sucrose than control subjects (mean intake in MI, PAD and control 132, 141 and 77 g/day respectively), the role of dietary sugar in cardiovascular disease has been much debated.[186] Yudkin even argued that the positive association between dietary intake of fat and cardiovascular disease may well be explained by the effect of sugar intake on cardiovascular disease because of the strong correlation between dietary fat and sugar intake.[178] Because dietary sucrose, if it plays a role, is unlikely to be the only dietary factor that influences cardiovascular risk, cross-sectional and observational epidemiological studies cannot be expected to give clear answers to the question whether dietary sugars consumption affects cardiovascular risk. Moreover, only a limited number of observational studies have been published on this topic since the initial reports by Yudkin.[187,188] Bolton-Smith and colleagues[187] found some evidence for a U-shaped relationship between added sugars consumption and the risk for coronary heart disease in Scottish males (not in females), but concluded that in the absence of a clear dose-response relationship this could not been seen as evidence for a role of dietary sugars in cardiovascular disease. Liu and colleagues failed to find a significant association between coronary heart disease risk and total carbohydrate, starch, sucrose, lactose or fructose intake in the Nurses' Health Study.[188]

5 The term "glycaemic carbohydrates" refers to those carbohydrates which can be digested in the small intestine and then absorbed as simple monosaccharides for metabolism in the body whereas "non glycaemic carbohydrates" pass into the lower intestine for potential fermentation to short chain fatty acids which also provide energy to the intestine itself and then the rest of the body.

More studies report on the associations between sugar-sweetened beverage (SSB) consumption and cardiovascular risk factors. In the Nurses Health Study consumption of SSBs was associated with a higher risk of coronary heart disease, even after adjustment for other dietary and lifestyle risk factors in subjects consuming ≥ 2 SSBs per day (relative risk (95% confidence interval (95% CI)) 1.35 (1.07, 1.69)) compared to those consuming < 1 SSB per month).[189] In middle-aged participants of the Framingham Heart Study SSB consumption (≥ 1 SSB per day compared to < 1 SSB per day) was associated with a higher prevalence (odds ratio (OR) (95%CI) 1.81 (1.28, 2.56)) and incidence over four years of follow-up (OR 1.62 (0.96, 2.75)) of metabolic syndrome.[190] The prospective ARIC study showed that SSB consumption (> 1 SSB per day vs < 1 SSB per day) at baseline tended to be associated with the prevalence of chronic kidney disease in a multivariate model (OR (95% CI) 1.46 (0.96, 2.22)), but not with the incidence over the nine year follow-up in middle-aged US adults (OR 0.82 (0.59, 1.16)).[191] A higher sugar-sweetened beverage consumption in a large nationally representative sample of the US adolescents studied between 1999 and 2004 was associated with higher serum uric acid and systolic blood pressure[192] and with higher insulin resistance-associated metabolic parameters and anthropometrics.[193] Higher dietary fructose consumption was associated with a more atherogenic lipid profile (smaller LDL particle size and lower HDL cholesterol) in a relatively small study in 6-14 year-old Swiss children, independent of adiposity.[194] A meta-analysis, based on 11 prospective cohort studies involving 310,819 participants and 15,043 cases of diabetes, reported that higher intakes of sugar-sweetened drinks are associated with type 2 diabetes.[195] Individuals with the highest intake of sugar-sweetened beverages (highest quartile; usually 1-2 servings/day) had a 26% greater risk of developing type-2 diabetes than those in the lowest quartile. Three studies examining metabolic syndrome (19,431 participants) found a 20% greater risk among those in the highest quartile of soft drink intakes compared to individuals in the lowest quartile.

1.3.3.5 Dietary sugars and cardiovascular risk factors: controlled studies in humans

Several papers have recently reviewed the evidence derived from controlled studies in humans on the effect of dietary sugars on cardiovascular risk.[179,181,184]

With respect to glucose tolerance and insulin sensitivity, the European Food Safety Authority (EFSA) concluded that most studies do not find adverse effects of predominantly added sugars (mostly sucrose) up to 20-25% of energy, provided that body weight does not increase.[184] Ruxton and colleagues concluded in 2010 that relevant studies all report similar or better insulin sensitivity on diets high in sugars, with most of these studies including subjects with obesity, type-2 diabetes or at risk from heart disease.[179]

With respect to blood lipids, EFSA indicates that high intake of sugars (> 20% of energy), mainly as added sucrose or fructose, potentially increases triglycerides and LDL-cholesterol, especially in hyperinsulinaemic individuals.[184] The American Heart Association (AHA) concludes, based on their review of studies, that high sugar (glucose, sucrose and fructose) diets (> 20% of energy) increase plasma triglycerides, but that the effects on LDL cholesterol are inconsistent. The review by Ruxton and colleagues is supportive of this view.[179] The negative effect on blood lipids is most clearly seen in the studies with high levels of added fructose.

With respect to the effect of consumption of sugars on body weight, EFSA concluded that the evidence was inconsistent, but that high intakes of sugars in the form of sugar-sweetened beverages might contribute to weight gain.[184] This conclusion is agreement with that of other recent reviews on this topic.[179,181,196,197]

Very few studies have looked at the blood pressure effects of dietary sugars. EFSA does not comment on a potential effect of sugars on blood pressure,[184] the AHA states that the data are inconsistent,[181] and blood pressure is not mentioned in the review by Ruxton and colleagues.[179] In the literature, a number of studies on this topic were found. Israel and colleagues reported in 1983 on an increase in diastolic blood pressure of 2-3 mm Hg in 24 subjects on a high sucrose diet (33% of energy vs 5 or 18% of energy).[198] Van der Schaaf and colleagues, on the other hand, found no change in ambulatory blood pressure in 13 hypertensive subjects on an isocaloric high-sucrose diet (~40% of energy).[199] The composition of the control diet was not reported. Sørensen and colleagues compared sucrose (125-175 g/d depending on total energy intake, ~25% of total energy) with artificial sweetener on top of an *ad libitum* diet.[200] The diets were not isocaloric, which resulted in significant differences in weight change between the groups. Systolic and diastolic blood pressures were significantly higher on the high sucrose diet (8 and 4.5 mm Hg respectively), although adjustment for body weight changes attenuated the difference (6 and 5mm Hg respectively).[200] Further studies are clearly needed to assess whether dietary sugars have an effect on blood pressure.

Inflammation is regarded as a risk factor for cardiovascular disease. Few studies so far have addressed a potential effect of sugars on inflammation. The AHA remarks that there is a clear lack of controlled studies that have assessed the effect of long-term sugar consumption on inflammation and markers of oxidative stress.[181]

1.3.3.6 Role of fructose in cardiovascular disease

It has been suggested that it is not the overall intake of sugars (mono- and disaccharides) that leads to cardiovascular disease, but rather the intake of sucrose or fructose.[183]

Near equal amounts of fructose and glucose are found in all common nutritive sweeteners, including high fructose corn syrup, except for those based solely on glucose, such as pure glucose or regular corn syrup.[201] It is estimated that >95% of Americans aged >19 y consume

<100 g/d of fructose from all sources.[202] Dolan and colleagues recently estimated, based on data from the NHANES study between 1999-2004, that the 95th percentile of fructose consumption in US adults is 136 g/d or 18.8 % of energy intake.[203] Livesey argues on the basis of similar data that intervention studies using >100 g/d of fructose for women and 150 g/d for men are of minor relevance for public health.[202]

In Europe the production of HFCS is restricted to protect the sugar market and is set at approximately 5% of total sugar production.[204] Therefore, in contrast to the US, foods and beverages with added sugars on the European market are predominantly sweetened with sucrose. However, this does not make a difference for the fructose consumption, because sucrose and high fructose corn syrup contain similar amounts of fructose per gram.

Fructose metabolism
The metabolism of fructose is different from that of the other monosaccharides. Fructose is solely metabolised in the liver, in contrast to glucose and other monosaccharides. Dietary fructose activates the fructokinase pathway in hepatocytes. It is rapidly phosphorylated, bypassing 6-phosphofructokinase, one of the rate-limiting enzymes in glycolysis.[205] The post-meal oxidation of fructose is therefore much more rapid and extensive than the oxidation of glucose with equal availability. Studies suggest that fructose may be less satiating than glucose and that sweetness of sucrose increases palatability.[183] Both factors may contribute to over consumption. Moreover, fructose has a lipogenic effect in the liver and could potentially modify the fatty acid composition of VLDL and induce harmful secondary effects like hypertriglyceridemia or insulin resistance.[183,205,206] Chong and colleagues concluded that the contribution of *de novo* lipogenesis to elevated postprandial triglyceride concentrations in humans is probably small, and that they are mainly due to lower activation of LPL and thereby reduced uptake of triglycerides in adipose tissue.[207] The phosphorylation of fructose in the liver requires ATP, which may result in ATP depletion and, as a consequence, production of uric acid from the breakdown of ADP. Uric acid inhibits NO production, increases ROS production and inflammation, and impairs endothelial and kidney function.[182,183]

Fructose and uric acid
Elevated uric acid concentrations are usually found in adults with metabolic syndrome, cardiovascular and renal disease.[183] Few controlled studies in humans have compared the effect of fructose consumption with consumption of other sugars or starch on uric acid concentrations.[183,208] A study by Akhavan and Anderson[209] investigated the effect of acute isocaloric ingestion of glucose/fructose mixtures, with glucose and fructose in different ratios, on the area under the curve (AUC) of uric acid over 75 min after ingestion. The AUC showed a dose-dependent increase with increasing fructose content of the mixture. Data on more long-term effects of high fructose diets on uric acid in humans from controlled trials seem to be lacking.[209]

Fructose and lipids, glucose metabolism and body mass
Several recent reviews have addressed the association between dietary fructose and lipids, glucose metabolism and body mass.[202,203,210] Schaefer concluded that diets containing ≥20% of energy from fructose are more likely to cause lipid abnormalities (hypertriglyceridaemia in those with hyperinsulinaemia and LDL-cholesterol increases in normo-insulinaemic individuals).[210] Diets containing 6-12 % of energy as fructose had very little effect on lipids and may reduce glucose levels modestly.[210] Dolan and colleagues conclude, based on an extensive review of the literature, that there is no evidence for increased plasma triglycerides after long-term ingestion of fructose of up to ~135 g/d, when it is not consumed in calorie excess, in healthy individuals.[203] These authors also report that there is no evidence that fructose consumption up to approximately 100 g/d instead of glucose or sucrose is associated with an increase in body weight.[203]

A neutral or even beneficial effect of moderate fructose diets (0-50 g/d) and a potentially unfavourable effect of diets very high in fructose (>100 g/d) agrees with the outcome of a meta-regression analysis by Livesey on the dose-response relationships between fructose consumption and various health parameters (insulin sensitivity, blood lipids, uric acid, BMI).[202]

Fructose and blood pressure
Hardly any studies have specifically studied the effect of fructose consumption on blood pressure. One study showed that consumption of a fructose drink (500 ml with 60 g of fructose) increased mean blood pressure acutely, over at least two hours after consumption, by ~4 mm Hg in comparison with an isocaloric glucose drink, due to a more pronounced increase in cardiac output in the absence of less change in peripheral resistance, in young healthy volunteers.[180]

1.3.3.7 Current recommendations for dietary sugars with respect to cardiovascular disease prevention

The general population guidelines on added sugars consumption differ among European countries. Most countries have qualitative guidelines for consumption of sugars (e.g. use in moderation, limit intake, little portions, consume occasionally), other countries use quantitative recommendations (e.g. maximum 10 or 15% of energy, <15 g/d).[179]

In its 2002 report, the European Heart Network set a population goal for sugary foods of four or fewer occasions per day for the prevention of cardiovascular disease in Europe.[1] This recommendation was not based on a direct relationship between dietary sugars and cardiovascular disease, but was rather given in the context of preventing overweight and obesity, which are associated with increased risk for cardiovascular disease. The recommendation was derived from the prevention of caries where the frequency of sugar consumption is more relevant than the total amount.[3,179]

Based on a review of the available evidence, the European Food Safety Authority has concluded recently that the data are insufficient to set an upper limit for sugars based on their effects on lipids, body weight or dental caries.[184]

However in the United States, recommendations on intake of sugars for cardiovascular risk reduction have been issued by the American Heart Association (AHA). In its 2002 scientific statement on sugars and cardiovascular disease, it is concluded that since sugars have no nutritional value other than to provide calories, high intake of sugars should be avoided because this will help to reduce the nutrient density of the diet and the intake of excess calories.[211] In line with this conclusion, the 2006 Diet and Lifestyle Recommendations for Cardiovascular Risk Reduction by the AHA specifically mention dietary sugars:[212] the AHA advises to minimise the intake of beverages and foods with added sugars. In its most recent statement on this issue, the AHA recommends a prudent upper limit of added sugars intake of half of the discretionary calorie allowance, which is for most American women no more than 100 kcal per day and for most American men not more than 150 kcal per day, from added sugars (approximately equivalent to 35 g/d of added sugars, i.e. sugar content of 0.33 ml can of soft drink).[181]

1.3.3.8 Conclusion

Based on this review of the recent literature on the association between dietary sugars and existing dietary recommendations it is concluded that:

1. there is limited and inconsistent evidence for an effect of consumption of sugars on cardiovascular disease risk;
2. most consistent evidence is found for an association between high intake of sugar sweetened beverages and risks of cardiovascular disease;
3. most evidence suggests that unfavourable effects of consumption of sugars on blood lipids is related to high intakes of fructose (>100-150 g/d or >15-25 E%);
4. high intakes of sugar-sweetened beverages may increase the risk of overweight;
5. a prudent recommendation would be to avoid high intakes of fructose, especially in the form of sugar-sweetened beverages, even though unequivocal evidence for such recommendation is lacking.

1.3.4 Dietary fibre and complex carbohydrates

This review was contributed by Professor Philip James, of the International Association for the Study of Obesity and a member of EHN's nutrition expert group.

1.3.4.1 Dietary fibre: not a simple defined category

Dietary fibre is a term which has several definitions, but the latest FAO/WHO analysis suggests that a chemical, physiological or botanical definition is preferred.[213] It is clear that FAO, WHO and many academics consider the specification of fibre should best be considered as the non-starch polysaccharides (NSPs) which are predominantly of plant cell wall origin. They favour this as an explicit chemical category within the carbohydrate group.[214] Other investigators have emphasised the specification of fibre as the residual components of plant cell wall which evade digestion in the small intestine.[215] This assessment, linked to the traditional focus on fibre's gastrointestinal effects, therefore includes the non-carbohydrate component lignin which is structurally intimately integrated with the NSPs in the plant cell wall. However, when these different definitions were being considered it was not recognised originally that some starches could be relatively resistant to digestion and indeed the process of cooking is important in determining how digestible the starches are. The granular structure of the starches, their processing and normal cooking all affect the digestibility. Any cooling of cooked food also alters the tertiary structure of starch by a process called retrogradation such that cooked starch after cooling, even when reheated, contains far more "resistant" starch i.e. starch which is likely to be poorly digested and pass into the colon where it is probably fermented. Often the impact of differential cooking, and whether or not the food has previously cooled before reheating, is not considered by those concerned with specifying the indigestible part of fibre. Yet the EFSA board concluded that the term dietary fibre should include all non-digestible carbohydrates.[216]

Thus, in general, two figures for fibre intake are now usually provided one specifying the non-starch component measured by the Englyst methods[217] which provides a lower number than the other "fibre" fraction which includes indigestible material measured by an in vitro assay originally developed by Asp and which attempts to simulate normal digestion.[218] This definition was accepted as the appropriate method for measuring dietary fibre by the Association of Official Analytical Chemists (AOAC) and is strongly supported by the food industry. It is also generally favoured in the US and has recently also been favoured by EFSA[219] but unfortunately it usually gives a higher value for fibre than that derived from measuring the NSP because it also includes, for example, coloured products involving the Maillard reactions of sugar-protein interactions induced by cooking. These products amplify the supposed fibre values of some processed foods, such as corn flakes, but there is no evidence that these products have discrete physiological effects on either the intestine or metabolism. In addition, there are modified starches which are used by the food industry to alter the physical properties of food and most of these modified starches, present in small amounts, are also not digestible so will be measured in standard in vitro tests.

Neither the Englyst nor the AOAC method measures the resistant oligosaccharides and inulin often used as supplements—so separate measurements needs to be made for these components. In Europe there has been a substantial emphasis by some companies on the potential probiotic properties of oligosaccharides and inulin has also been added to food to boost the claim for fibre-enriched products. Given all these uncertainties, and how best to define the different fibre fractions and their

physiological roles, FAO and WHO have focused on the non-starch polysaccharides as a more robust definition and recommended its use to Codex for the purpose of international regulations.

1.3.4.2 The different measures for the fibre content of the diet

To complicate matters further there have been, historically, four main assays for the fibre content of foods. The first was Van Soest's assay of what one might term "crude" fibre based on measures then considered valuable by ruminant nutritionists.[220] These values were often used in USA reports dealing with fibre studies. In the US, food composition tables traditionally have no direct measure of carbohydrates in the foods listed in tables; carbohydrates are calculated as the weight difference once the fat, protein and ash in a dried sample of food had been measured. This, in practice, leads to substantially higher values for the energy content of food than those obtained by direct measurements.[221] Thus US and European measures of carbohydrate energy and fibre intake may differ markedly simply because of the methods used in their estimation.

Then the Southgate analysis was developed and involved an acetone extraction before a measure of enzymatic digestibility.[222] These values were those used traditionally in UK food composition tables before the Englyst assay superseded it.

Now only the Englyst and the AOAC assays are usually quoted when presenting results of fibre studies but care needs to be taken to assess which values were being chosen when particular studies are published dealing with the metabolic or other benefits of fibre.

1.3.4.3 Potential physiological effects and health benefits of fibre

As noted by Cummings and Stephen "the established epidemiological support for the health benefits of dietary fibre is based on diets that contain fruits, vegetables and whole grain cereals for which the intrinsic plant cell wall polysaccharides are a good marker." [214] What this means in practice is that some of the supposed effects of NSP do not relate to NSP as such but to frequently associated components in the diets which have a high NSP value. Thus fruit and vegetables contain NSP and some of these components have a distinct physiological and metabolic effects but fruits and vegetables are also rich in potassium (as are unrefined cereals in general) and the potassium may be one of the useful components which helps, for example, to lower blood pressure. Fruits and vegetables also contain varying amounts of complex molecules which have antioxidant and other properties which could impact on the blood vessel walls and reduce the inflammatory and endothelial thickening and thereby help to protect against cardiovascular diseases. (See section 1.3.5 for an outline of the potential mechanisms for the protective effect of fruit and vegetables.) However, when, for example, the Mediterranean-type diet is highlighted as beneficial for the prevention of cardiovascular disease there are multiple components involved and the higher NSP values may then be more of a marker of the dietary pattern than intrinsically responsible for the diet's preventive role.

Cummings and Stephen have summarised the physiological effects as set out in Table 2 for all the components of dietary carbohydrates. This approach, of course, differs from an approach which attempts to assess the health benefits of NSPs or the dietary fractions conventionally included in the term dietary fibre. If the term fibre or NSP is considered in epidemiological or intervention studies then the potential benefits (and hazards) of these physiological effects need to be considered.

Table 2 The principal physiological effects of carbohydrates including the non-starch polysaccharides as set out by Cummings and Stephen[214]

	Provide energy	Increase Satiety	Glycaemic[a]	Cholesterol lowering	Increase calcium absorption	Source of SCFA [b]	Alter balance of microflora (prebiotic)	Increase stool output	Immuno-modulatory
Monosaccharides	✓		✓						
Disaccharides	✓		✓		✓				
Polyols	✓					✓[c]		✓	
Maltodextrins	✓		✓						
Oligosaccharides (non-α-glucan)	✓				✓	✓	✓		✓
Starch	✓		✓			✓[d]		✓[d]	
NSP	✓	✓		✓[e]		✓		✓	

a Provides carbohydrate for metabolism (FAO, 1998).
b Short chain fatty acids.
c Except erythritol.
d Resistant starch.
e Some forms of non-starch polysaccharide (NSP) only.

1.3.4.3.1 Gastrointestinal effects of fibre

Perhaps the greatest series of meticulous studies on fibre's effects relate to the effect of NSP on total daily faecal output which conventionally is not considered a major disease, albeit that in adults and children on "western" processed or refined diets constipation is a major problem for an appreciable proportion of the general population. It is clear that in deriving goals for fibre often this laxation effect of NSPs has been taken as a simple index, or marker, which is expected to correlate with other effects. This approach was adopted in WHO's 1990 report on diet and the prevention of chronic diseases[223] and has been used again by EFSA in its 2010 specification of Dietary Reference values.[219] This report estimated that an average goal of 16g NSP (for adults) would limit constipation and took account of the observed variation in faecal output between volunteers on defined diets and fibre intakes and the volume needed for volunteers to consider that they did not have constipation or difficulties associated with defaecation. An upper limit of 24 g/d was also set, based on the concern that as whole grain cereal intakes rose then the potentially greater intakes of phytates associated with the whole grain might inhibit the absorption of important minerals such as iron and zinc, and these mineral deficiencies were known to be globally important.

Two further features were already evident 20 years ago. First, that, given the substantial differences between the energy needs of different populations based on their different levels of physical activity and their different average body weights, the NSP values could be expressed as 2.2–3.2 g NSP/MJ dietary energy. This actually also had the implicit value of allowing for the different energy requirements of women and men and of different groups within the population. The second issue related to the broader definition of dietary fibre. On this basis the 16 g and 24 g NSP values were estimated to be equivalent to 27 g and 40 g dietary fibre as measured at that time by a combination of studies using the Southgate and Asp methods.

1.3.4.3.2 Non-gastroenterological effects of fibre

In the WHO 1990 report it was also recognised that the use of faecal bulking was just a simple marker and that there could well be parallel benefits on other metabolic systems and in disease prevention which were far less easy to define in a precise way. Thus it was estimated that the 16 g NSP would be a reasonable figure capable of normally including the 400 g/day of fruit and vegetables which was also being set out as a suitable goal. It was known that the faecal bulking effect of whole grain cereal derived NSPs was greater than the NSPs derived from fruit and vegetables but the whole grain cereal component of the improved diet could also be accommodated within the 16 g/d figure.

At that time it was also known that the whole grain cereals included in the NSP estimation would contribute substantially to lowering the speed of digestibility and would limit the sudden rise in glucose and insulin levels. It was also recognised that there was a poorly quantified potential effect of dietary bulking on satiety. The impact of pectins and guar gums in the NSPs of whole grain cereals, fruit and vegetables in lowering blood cholesterol levels was known but was difficult to quantify. Therefore, no detailed examination of data in relation to blood sugar and insulin fluctuations and blood cholesterol lowering or the prevention of obesity, type 2 diabetes or cardiovascular disease was used in deriving the preliminary WHO NSP goal in 1990.

The updating of the original WHO report was undertaken a decade later and this joint FAO/WHO Consultation found that high intakes of dietary fibre was convincingly important in preventing obesity and probably important in preventing type 2 diabetes and cardiovascular disease.[52] Only a possible preventive role was assigned to fibre for cancer prevention but the more recent WCRF/AICR report,[224] which undertook an enormously exhaustive analysis of epidemiological data relating diet to the development of cancer, concluded that dietary fibre was probably protective against colorectal cancer. In the later WCRF/AICR policy report[225] it was proposed on the basis not only of cancer but to engage other preventive principles that NSP intakes should be at least 25 g/d as a population average.

The new analysis by EFSA sets out their basis for recommending diets with a reasonable content of dietary fibre. They suggest 25 g/d dietary fibre or 2 g fibre/MJ energy intake as the Dietary Reference Value on the basis of analyses relating to gastrointestinal function. On this basis this might be considered using their current methods of analysis as about equivalent to 18.5 g/d NSP.

The WCRF/AICR report, however, considered that the desirable long-term goal should be >600 g/d of vegetables and fruit which would then be accommodated by a mixed Mediterranean diet containing >25 g/d NSP. This implies an energy adjusted value of >2.25 g NSP/MJ or an AOAC fibre value of say >35 g/d or >3 g fibre/MJ.

1.3.4.4 Dietary fibre intake and cardiovascular disease

The analysis of appropriate fruit and vegetable intakes set out in Section 1.3.5 presents a case for considering fruits and vegetables as important in cardiovascular prevention. If one then takes the WHO/FAO 916 report where it specified the NSP goal as >20 g/d with total fibre intakes of >25 g/d fibre this was accepted as accommodating the ≥ 400 g/d of vegetables and fruits which was retained as a reasonable goal.

The FAO/WHO 916 report had a series of background papers on different aspects of diet and prevention of cardiovascular disease and noted, not only the cholesterol-lowering effects of some of the NSPs, but also quoted three cohort studies which had shown the benefits of intakes of whole grain consumption in preventing coronary heart disease

The Population Health Research Institute in Canada[226] undertook a detailed systematic review of the evidence from cohort and randomised intervention studies to assess the potential role of different dietary factors in relation to CHD. They used the Bradford Hill guidelines to generate a causation score based on the four criteria of strength, consistency, temporality, and coherence for each dietary exposure in cohort studies and then examined the results for consistency with the findings of randomised trials. Strong evidence with all four criteria satisfied suggested protective factors including intakes of vegetables, nuts, and "Mediterranean" and high-quality dietary patterns in preventing coronary heart disease. Harmful associations included intakes of trans fatty acids and foods with a high glycaemic index or load. Moderate evidence with three of the Bradford Hill criteria satisfied included, apart from fish, marine omega-3 fatty acids, alcohol and folate, whole grains, dietary sources of vitamins E and C, beta carotene, fruit, and fibre. Among the dietary exposures with strong evidence of causation from cohort studies, only a Mediterranean dietary pattern related to CHD in randomised trials.

In this analysis there were 15 cohort studies trials dealing with dietary fibre and the pooled analysis of these cohorts showed a relative risk of 0.78 (0.72-0.85) for coronary events and secondary outcomes associated with a high fibre diet. There was, however, only one randomised trial that selectively dealt with fibre. Nevertheless, the Mediterranean diet was specified as one which contained a higher intake of vegetables, legumes, fruits, nuts, whole grains, cheese or yogurt, fish, and monounsaturated relative to saturated fatty acids.

Given this perspective the issue then is the sources and amount of fibre which could be considered reasonable from a cardiovascular, as distinct from a gastrointestinal, point of view. Recently the EPIC consortium relating diet to disease has evaluated the carbohydrates and fibre intakes across Europe.[227] Unfortunately, the basis for the assessment was the AOAC figures for fibre and on this basis the fully adjusted high intakes of fibre for health conscious, moderately active, normal weight UK men seems to be about >35 g/d and for UK women > 27 g/d. Figures are not yet available to characterise the fibre or NSP content of a Mediterranean diet from this data set but recent Spanish analyses[228] of fibre intakes in relation to carotid intimal thickening—taken as an important biological index of important atherosclerosis—showed an inverse relationship between fibre intakes and intimal thickening with the higher fibre intakes set at >35 g/d in adults. In other words, the levels are similar to that inferred for men from the EPIC data set.

1.3.4.5 Conclusions

The different suggested goals are set out in Table 3.

Table 3 Suggested population goals for fibre intakes

Study	Intermediate target g/d NSP; (g/d fibre)	Intermediate target should be g/d NSP/MJ; (g/d fibre /MJ)	Longer term goal g/d NSP; (g/d fibre)	Longer term goal g/d NSP/MJ; (g/d fibre /MJ)
WHO 797 1990	16 g NSP			
WHO 916 2003	20 g NSP			
WCRF/AICR 2009			>25 g NSP	
EFSA 2010	(>25 g fibre)	(2 g fibre/MJ)		
This report: EHN 2010	**>20 g NSP (>27 g fibre)**	**>1.6 g NSP/MJ (>2.2 g fibre/MJ)**	**>25 g NSP (>35 g fibre)**	**>2 g NSP/MJ (>2.8 g fibre/MJ)**

On the basis of current evidence in relation to cardiovascular disease it seems reasonable to conclude that fibre intakes should be consumed as whole foods with a mix of whole grain cereals, legumes, vegetables and fruit and that the intermediate target for average population intakes should be >20 g NSP (> 1.6 g/d NSP/MJ) or 27 g AOAC fibre with an optimum goal of a population average intake of >25 g NSP (>2 g NSP/MJ) or >35 g AOAC fibre (2.8 g fibre/MJ).

1.3.5 Fruit and vegetables

This review has been contributed by Karen Lock and Lydia Collingridge of the London School of Hygiene and Tropical Medicine, UK.

1.3.5.1 Introduction

This paper is a summary of the published evidence on the relationship between fruit and vegetables and cardiovascular disease since 2002.

1.3.5.2 Methods

1.3.5.2.1 Literature search

A search was conducted to identify systematic reviews and meta-analyses published on the relationship between fruit and vegetables and cardiovascular disease (CVD) since 2002. We searched Medline, Embase and Cochrane electronic databases for papers published from 2001 to December 2009.

Both free text and MeSH terms were used with limits set as; humans, 2001 and being published in the English language. MeSH terms used were cardiovascular disease AND (fruit OR vegetables). A free text search was performed using "fruit* OR vegetable* OR legume* AND (cardiovascular disease* OR stroke OR CVA OR heart disease OR coronary disease OR peripheral vascular disease OR coronary artery disease OR myocardial infarction OR MI OR heart attack)". They were then refined by either selecting only meta-analysis or by adding "systematic review" into the search criteria.

An additional search of literature reviews on "Mediterranean diets" was conducted for completeness. This was carried out in a similar way to the original search using the same search terms for CVD endpoints as before. Papers would be included if the study focus was on fruit and vegetable intake and a CVD outcome.

To ensure that no important recent papers were missed, a final search was included to look specifically for interventions impacting on CVD outcomes. The same search terms were used as in the original search except "meta-analysis" OR "systematic review" was replaced with "intervention" OR "trial". Limits were set as humans, English, publication date from 2002/01/01 to 2010/02/15 with the search field set as Title/Abstract. This search produced 69 papers. None of the studies found looked at CVD (heart disease or stroke) as an endpoint. They all looked only at intermediate risk factors for cardiovascular disease e.g. BP, obesity or specific plasma serum levels.

1.3.5.2.2 Inclusion criteria

Papers were included if they were reviews of either fruit and/or vegetable consumption and had an aspect of CVD as an endpoint. Studies of the Mediterranean diet were only considered if the focus was on fruit and vegetable intake and the analysis permitted the impact of fruit and vegetable intake on cardiovascular disease to be examined separately taking into account the obvious confounding factors of other dietary influences.

Fruit and vegetables had to be eaten as part of the diet, in vitro studies were not included. We excluded those papers that looked only at dietary supplements or selected plant components, e.g. Vitamin C supplements or garlic supplements. Papers also had to include some measure of cardiovascular disease as an end point, or an intermediate outcome which is a well recognised as a risk factor for CVD, such as blood pressure or serum cholesterol.

The search strategy in Medline, Embase and Cochrane identified 26 potentially relevant unduplicated studies, of which only seven met the inclusion criteria.

1.3.5.2.3 Definition of fruit and vegetables used in this review

As mentioned in the inclusion criteria we used a broad definition of fruit and vegetables in our search terms. MeSH terms were used in our original search as well as free text. We did not include meta-analysis on specific fruits or vegetables (e.g. garlic alone) or studies that looked at supplements based on fruit or vegetable components (e.g. vitamin C).

The seven papers that have been included in this paper used similarly broad definitions of fruit and vegetables. Table 4 illustrates the definitions used by the authors for the different systematic reviews, if this was stated.

A number of the papers included in this study have used cohorts that incorporated potatoes in their definition of vegetables. Although potatoes are not included in the UK's '5-a-day' campaign[229] we did not exclude these papers. This is because without them there would not be enough evidence to draw on. Also, if potatoes have been included in the studies then it is likely that the effect of fruit and vegetables on CVD will be underestimated, not overestimated.

Table 4 Definitions of fruit and vegetables used in the papers

Dauchet et al	He et al	Mente et al	Huxley et al	Pereira et al	Dauchet et al	He et al
Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies[230]	Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies[231]	A Systematic Review of the Evidence Supporting a Causal Link Between Dietary Factors and Coronary Heart Disease[226]	The relation between dietary flavonol intake and coronary heart disease mortality: a meta-analysis of prospective cohort studies[232]	Dietary fibre and risk of coronary heart disease: a pooled analysis of cohort studies[233]	Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies[234]	Fruit and vegetable consumption and stroke: meta-analysis of cohort studies[235]
'...included in the analysis only..."vegetables," "all vegetables," "vegetables rich in carotenoids," "fruit," or "all fruit." Individual fruit or vegetables... were excluded... fruit juices were included with fruit in some studies and potatoes with vegetables in others'	No specific definition stated. Literature search used MeSH and text words for fruits and vegetables. Authors excluded studies that only looked at 'surrogate nutrients of fruits or vegetables'.	No mention of definitions used for fruit and vegetables	Looked at flavonol intake (search terms included 'flavonols' and 'flavonoids'). Mentions the main source of flavonol intake for each of the studies; tea (+/- milk), onions, apples, broccoli, vegetables	Looked at the fibre content of the fruit and vegetables. Mentions that potatoes were included in the vegetable group for two of the cohorts	'...the exposure variable was always fruits and vegetables, the definition of this variable could vary between studies. For instance, fruit juices were included with fruit in some studies and potatoes with vegetables in others'.	No specific definition stated (only mentions 'fruit' and 'vegetable'). Authors excluded studies that only looked at 'surrogate nutrients of fruits or vegetables'

1.3.5.3 Results

There were seven reviews relevant to this paper. Five were about fruit and vegetable intake, one focused on dietary fibre and one looked at dietary flavonol intake. All the reviews included both men and women, although not all were analysed or presented in subgroup analyses. All the papers used a mixture of single and mixed sex cohorts so the overall results are felt to reflect both male and females. The reviews included a combination of studies from Europe and USA, with one cohort from Japan, although the majority of reviews were more heavily weighted by USA cohorts.

Six out of the seven papers state in the title that they are an analysis of cohort studies. The only paper that looked at both cohort studies and intervention studies was by Mente and colleagues.[226] However, this paper did not actually include any evidence from RCTs in the section on fruit and vegetables and used cohort data only like the other six papers. Of interest this paper looked at many different dietary components including the 'Mediterranean diet'. This was one of the few dietary factors that had findings that were also supported by RCT evidence.

Of the reviews that met the inclusion criteria, the majority studied coronary heart disease (CHD) as the cardiovascular outcome. However there were two reviews that focused on stroke as an outcome. There were insufficient published studies to look at the effects of fruit and vegetables on any other disease end points.

The key parts of the papers looking at coronary heart disease as the outcome are summarised in Table 5. Those that used stroke as the endpoint are presented in Table 6.

TABLE 5 SUMMARY OF PAPERS INCLUDED IN THIS REVIEW WITH CHD AS OUTCOME OF INTEREST

Name and year of study	Author	Type of study and inclusion criteria	Data sets used	Key results
Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies 2006[230]	Dauchet, L. Amouyel, P. Hercberg, S. Dallongeville, J.	Meta–analysis of published prospective cohort studies. Studies were included if they reported RR and 95% CI for CHD or mortality and if they presented a quantitative assessment of fruit and vegetable intake. Studies had to look at fruit and vegetable intake per se and not just the nutrient content.	Nine cohorts- 91,279 men, 129,701 women and 5007 CHD events. Seven cohorts from USA, two from Finland. Range of follow up was 5–19 years. Subjects ranged in age from 25–84.	CHD risk decreased by 4% [RR (95%CI): 0.96 (0.93-0.99), P 0.0027] for each additional portion per day of fruit and vegetables intake and by 7% [0.93 (0.89 – 0.96), p< 0.0001] for fruit intake. The association between vegetable intake and CHD risk was heterogeneous.
Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies 2007[231]	He, F. J. Nowson, C. A. Lucas, M. MacGregor, G. A.	Meta–analysis of published prospective cohort studies. Studies were included if they reported RR and 95%CI of CHD with respect to frequency of fruit and vegetable intake.	Twelve studies of 13 cohorts. 278,459 individuals with 9,143 CHD events. Nine cohorts from USA and four from Europe. Range of follow-up was 5-26 years. Subject age ranged 25-84.	Compared with individuals who had <3 servings/day of fruit and vegetables, the pooled RR of CHD was 0.93 (95% CI: 0.86-1.00, P=0.06) for those with 3-5 servings/day and 0.83 (0.77-0.89, P<0.0001) for those with 5+ servings/day.
A Systematic Review of the Evidence Supporting a Causal Link Between Dietary Factors and Coronary Heart Disease 2009[226]	Mente, A. de Koning, L. Shannon, H. S. Anand, S. S	Systematic review. Search of MEDLINE for prospective cohort studies or RCTs investigating dietary exposures in relation to CHD. The Bradford Hill guidelines were used to evaluate systematically whether a causal link between the exposure of interest and CHD exists.	Vegetables – 220,564 subjects, nine cohorts. Men, women, Europe, USA, primary prevention. Fruits – 222,706 subjects, 10 cohorts. Men, women, Europe, USA, primary prevention. Fruit and vegetables – 199,514 subjects, seven cohorts. Men, women, Europe, USA, primary prevention	Vegetables – 0.77 RR (0.68-0.87) 95%CI 4/4 Bradford criteria met. Fruits – 0.81 RR (0.68-0.94) 95%CI 3/4 Bradford criteria met. Fruit and vegetables – 0.79 RR (0.72-0.87) 95%CI 3/4 Bradford criteria met.
The relation between dietary flavonol intake and coronary heart disease mortality: a meta-analysis of prospective cohort studies 2003.[232]	Huxley, R. R. Neil, H. A.	Meta-analysis of prospective cohort studies published before September 2001. Studies were included if they reported data on flavonol intake and CHD mortality.	Seven prospective cohorts of men and women were identified, approximately 105,000 people with a total of 2087 fatal CHD events. Five European and two USA cohorts. Follow up 6-25 years. Age range 30-84.	Comparison of individuals in the top third with those in the bottom third of dietary flavonol intake yielded a combined risk ratio of 0.80 (95% CI 0.69-0.93) after adjustment for known CHD risk factors and other dietary components.
Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies 2004[233]	Pereira, M. A. O'Reilly, E. Augustsson, K. Fraser, G. E. Goldbourt, U. Heitmann, B. L. Et al	Pooled analysis of published prospective cohort studies. Studies had to have at least 150 incident coronary cases, include assessment of usual dietary intake, and used a validation study of the diet assessment method or a closely related tool.	Eleven cohorts totalling 336,244 individuals and 5249 CHD events. Men and women included. Cohorts from USA and Europe. Follow up range 6–10 years. Age range 35-99.	RR of CHD was 0.84 (95%CI: 0.70-0.99, p=0.04) for fruit fibre. RR was 0.9 (95%CI: 0.77–1.07, p=0.23) for cereal fibre. RR for vegetable fibre was 1.00 (95%CI: 0.88–1. 13, p=0.97). A regression model suggested that the effects of fruit and cereal fibre were independent of one another.

Table 6 Summary of papers included in this review with stroke as outcome of interest

Name and year of study	Author	Type of study and inclusion criteria	Data sets used	Key results
Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies 2005[234]	Dauchet, L. Amouyel, P. Dallongeville, J.	Meta-analysis of published prospective studies. Studies were selected if they had RR and 95% CI for any type of stroke and used a validated questionnaire for food intake assessment.	Seven studies were eligible for the meta-analysis, including 90,513 men, 141,536 women, and 2,955 stroke events. Five cohorts from USA, one from Europe and one from Japan. Follow up ranged from 3-20 years. Age range was 25-103.	The risk of stroke was decreased by 11% (RR 95% CI: 0.89 [0.85 to 0.93]) for each additional portion per day of fruit, by 5% (RR: 0.95 [0.92 to 0.97]) for fruit and vegetables, and by 3% (RR: 0.97 [0.92 to 1.02]; NS) for vegetables.
Fruit and vegetable consumption and stroke: meta-analysis of cohort studies 2006[235]	He, F. J. Nowson, C. A. MacGregor, G. A.	Meta-analysis of published prospective cohort studies. Studies were included if they reported RR or hazard ratios and 95% CI and gave details on the frequency of fruit and vegetable consumption.	Eight studies consisting of nine cohorts – 257551 individuals and 4917 stroke events. Five USA cohorts, three from Europe and one from Japan. Men and women included. Follow up 3-20 years. Age range 25-103.	RR of stroke was 0.89 (0.69-0.79) for those with between three and five servings a day compared with individuals who had less than three servings a day. RR was 0.74 (0.69- 0.79) for those with more than five servings day.

1.3.5.4 Fruit and Vegetables and CHD

To start off with we shall look at the results published about the combined effects of fruit and vegetable consumption. Dauchet and colleagues published a meta-analysis of nine cohorts looking at the risk of coronary heart disease and its relationship to fruit and vegetable intake.[230] The paper extracted relative risk data from each paper for each additional portion of fruit and vegetables eaten per day and the effect of CHD. The risk of CHD decreased by 4% [RR=0.96 95%CI (0.93-0.99), p=0.0027] for each additional portion of fruit and vegetables eaten per day. The paper showed a linear trend suggesting the risk reduction improved with higher fruit and vegetable consumption. There was no evidence of heterogeneity between studies.

He and colleagues also published a meta-analysis looking at fruit and vegetable consumption and the effect on CHD.[235] This paper looked at data from eight studies, split into nine cohorts. The paper presented the results by dividing daily intake into three categories; less than three portions of fruit and vegetables per day, three to five portions per day and more than five portions per day. The relative risk was then worked out in comparison to the risk of the group who ate less than three portions per day (i.e. RR in that category = 1). Higher fruit and vegetable intake resulted in a reduced risk in CHD. Those individuals in the three to five portions a day category had a risk reduction of 7% [RR=0.93 95% CI (0.86-1.00), P=0.06] and those who ate more than five portions per day reduced their risk by 17% [RR=0.8395%CI (0.77-0.89, P<0.0001]. This paper again illustrated that fruit and vegetable consumption is not only protective against CHD, but the more fruit and vegetables eaten, the greater the protection afforded.

The paper by Mente and colleagues[226] had a different premise, in so much as they wished to find a causal link between dietary factors and CHD. It looked at different dietary exposures and their influence on coronary heart disease. This paper used both prospective cohort studies and randomised controlled trials. Odds ratios or relative risks were calculated from the cohort studies by comparing the highest intake quantile with the lowest intake quantile (usually quartiles or quintiles). Each exposure was then analysed using the Bradford Hill criteria[236] to find if a causal link existed. This paper showed that fruit and vegetable intake resulted in a 21% reduction in the risk of coronary heart disease [RR=0.79 95%CI (0.72-0.87)]. Data from seven cohorts was used to calculate this figure. However, as the paper did not specify the exact type of dietary exposure, so the amount of fruit and vegetables consumed to produce this risk reduction is not clear. Fruit and vegetable intake was found to have moderate evidence to support the causal link with coronary heart disease according to their modified Bradford Hill criteria.

The evidence suggests that eating more portions of fruit and vegetables will significantly reduce the risk of CHD. The more consumed the greater the risk reduction.

1.3.5.5 Fruit and CHD

Four of the included studies had data on fruit alone and its influence on CHD. Increased fruit intake was shown to protect against coronary heart disease with Dauchet and colleagues[230] quoting a risk reduction of 7% [RR=0.93 95%CI(0.89-0.96), p<0.0001] for each additional portion of fruit eaten per day. Pereira and colleagues[233] carried out a pooled analysis of 10 prospective cohort studies. The paper analysed the links between dietary fibre and CHD, producing data on cereal fibre, fruit fibre and vegetable fibre. The paper attributed a 16% reduction in the risk of CHD [RR=0.84 95%CI (0.70-0.99), p=0.04] to fruit fibre. Further analysis of the data showed that the beneficial effect of fruit fibre was independent to that of cereal fibre. The Mente paper[226] showed a risk reduction of 19% [RR=0.81 95%CI(0.68-0.94)]. As mentioned previously with this paper, there was no information on the amount of fruit consumed that related to this benefit. There was moderate evidence that this was a causal link, with three out of the four modified Bradford Hill criteria being met. The meta-analysis carried out by He and colleagues[231] showed that by eating three to five portions of fruit per day one could reduce the risk of CHD by 11% [RR=0.89 95%CI(0.82-0.98) and by eating more than five portions this reduction in risk grew to 28% [RR=0.72 95%CI(0.66-0.79)] compared to those individuals who ate less than three portions of fruit per day. This was a greater reduction in risk than the data for combined fruit and vegetable consumption.

The evidence suggests that eating more portions of fruit will significantly reduce the risk of CHD. Eating five portions per day has a greater effect than eating three portions.

1.3.5.6 Vegetables and CHD

Mente and colleagues[226] found that vegetable intake decreased the risk of CHD by 23% [RR=0.77 95%CI (0.68-0.87)]. This was one of the few dietary influences that scored four out of four in the modified Bradford Hill criteria, corresponding to strong evidence of a causal link between vegetable consumption and CHD. The Pereira paper[233] found that vegetable fibre had no influence of CHD outcomes [RR=1.00 95%CI (0.88-1.13), p=0.97]. However the Dauchet paper,[230] which looked at the vegetable consumption as a whole entity, found that eating an additional portion of vegetables reduced the risk of CHD by 11% [RR=0.89 95%CI (0.83-0.95), p=0.0023]. He and colleagues[231] also found a benefit to vegetable consumption and reduction in CHD, although this was less profound compared to the effects of fruit consumption. Individuals who ate three to five portions per day had a 7% reduction in risk [RR=0.93 95%CI (0.82-1.06)]. Those who ate greater than five portions per day extended this reduction in risk to 19% [RR=0.81 (%%CI (0.72-0.90)].

The evidence suggests that eating more portions of vegetables will reduce the risk of CHD. The evidence does not suggest that it is the fibre content that results in the benefit. There is strong evidence of a causal link between vegetable consumption and CHD.

1.3.5.7 Fruit and Vegetables and Stroke

Dauchet and colleagues also published a study looking at the relationship between fruit and vegetables intake and stroke.[234] This was a meta-analysis of seven cohorts from the USA, Europe and Japan. The risk of stroke was shown to decrease by 5% [RR=0.95 95%CI (0.92 to 0.97)] for each additional portion of fruit and vegetables eaten. This paper also showed a linear trend suggesting the risk reduction with regards to stroke improved with higher fruit and vegetable consumption, as it also did for CHD.

He and colleagues also published a study looking at the relationship between fruit and vegetables and stroke.[235] This was a meta-analysis of eight studies consisting of nine cohorts. Again it included data from the USA, Europe and Japan. The paper showed the risk of stroke reduced by 11% [RR=0.89 95%CI (0.83-0.97)] for those who ate between three and five portions of fruit and vegetables per day, compared to those who ate less than three portions of fruit and vegetables per day. Those who ate more than five portions per day had a 26% reduction in stroke [RR= 0.74 95%CI (0.69- 0.79)].

The evidence suggests that eating more portions of fruit and vegetables per day significantly reduces the risk of stroke. The more consumed, the greater the risk reduction.

1.3.5.8 Fruit and Stroke

Both the Dauchet and He papers analysed the data in separate groups for fruit and vegetable intake. In the paper by Dauchet and colleagues increased fruit intake was shown to decrease the risk of stroke by 11% [RR=0.89 95%CI (0.85-0.93)] for each additional portion eaten per day.[234] In the study by He and colleagues[235] those who ate three to five portions of fruit per day reduced their risk of stroke by 10% [RR=0.90 95%CI(0.83-0.98)] compared to those who only ate three portions or less of fruit per day. Those who ate more than five portions per day reduced their risk by 13% [RR=0.87 95%CI (0.80-0.95)]. So both these studies illustrated a beneficial effect of fruit intake on the risk of stroke, which was greater the more fruit that was consumed.

The evidence suggests that eating more portions of fruit per day significantly reduces the risk of stroke. Eating three portions per day results in a benefit, but eating more than five portions per day increases this benefit further.

1.3.5.9 Vegetables and Stroke

Vegetable intake also seems to influence the incidence of stroke. Dauchet and colleagues[234] found a 3% reduction in the risk of stroke [RR=0.97 95%CI (0.92-1.02)] with each additional portion of vegetables eaten per day. Although the 95% confidence interval suggests that this may not be statistically significant. The He paper[235] showed a reduction in stroke risk of 8% [RR=0.92 95%CI(0.87-0.97)] for those who ate three to five portions of vegetables

per day, and a reduction of 16% [RR=0.84 95%CI(0.76-0.92)] for those who ate more than five portions per day. These risks are in comparison to individuals who ate less than three portions per day.

The evidence suggests that eating more portions of vegetables per day reduces the risk of stroke. Eating three portions per day results in a benefit, but eating more than five portions per day increases this benefit further.

1.3.5.10 Dietary Components

A further paper has been included in this review; Huxley and colleagues.[232] This paper looked at the relationship between dietary flavonol and coronary heart disease. Flavonol is found in high quantities in tea however it is also found in fruits and vegetables such as onions, apples and broccoli. Those in the top third group of dietary flavonol intake had a 20% reduction in the risk of CHD [RR=0.80 95%CI (0.69-0.93)] compared to those in the bottom third for intake. The main source of flavonoid intake was noted for each group. For many the main source was tea, but there were two studies from Finland where 64% of flavanoid intake came from apples and onions. These two studies produced a greater risk reduction [RR=0.73 95%CI (0.41-1.32) for women and RR=0.67 95%CI (0.44-1.00) for men] than the combined data. However, as these subset groups are much smaller the confidence interval is much wider, making the results less significant.

The evidence suggests that dietary flavonol may result in a reduced risk in CHD. However, these results come from small amounts of data and are not statistically significant.

1.3.5.11 Discussion

There appears to be a growing body of evidence linking diet, specifically fruit and vegetable intake to cardiovascular outcomes. The degree of protection eating fruit and vegetables gives you varies between the studies, as illustrated in Table 7 (CHD as outcome) and Table 8 (stroke as outcome). In these tables we have extrapolated some of the data to try and make comparison between the studies easier, by trying to convert risk reductions into those gained from eating five portions of fruit or vegetables per day.

There does not seem to be a clear picture as to whether fruit or vegetables offer more protection. Of the six studies that analysed the results by fruit and vegetable groups, three studies showed greater protection from vegetables, and three showed greater protection from fruits. In the review only considering fibre, fruit fibre afforded a 16% reduction in CHD risk, compared to no risk reduction from vegetable fibre.[233] However, studies do seem to agree that the more portions of fruit and vegetables you eat, the greater the risk reduction, i.e. there is no upper limit for fruit and vegetable consumption with regards to benefit on CVD risk.

Table 7 CHD and results split by group; fruit and vegetables, fruit alone and vegetables alone

Study	Fruit and vegetables	Fruit alone	Vegetables alone
Dauchet et al	RR - 4% reduction for each additional portion (106g) eaten per day. Linear response, so for five portions 20% reduction.	RR - 7% reduction for each additional portion eaten per day. Five portions results in 35% reduction.	RR - 11% reduction for each additional portion eaten per day. Five portions results in 55% reduction.
He et al	RR - 17% reduction for 5+ portions eaten per day.	RR - 28% reduction for 5+ portions eaten per day.	RR - 19% reduction for 5+ portions eaten per day.
Mente et al	RR - 21% reduction comparing highest and lowest eating groups –highest group likely to consume 5+ portions per day.	RR - 19% reduction comparing highest and lowest eating groups – highest group likely to consume 5+ portions per day.	RR - 23% reduction comparing highest and lowest eating groups – highest group likely to consume 5+ portions per day.
Range of results for 5 portions per day	**17-21% reduction in CHD risk by eating ~5 portions per day.**	**19-35% reduction in CHD risk by eating ~5 portions per day.**	**11-55% reduction in CHD risk by eating ~5 portions per day.**

Table 8 Stroke and results split by group; fruit and vegetables, fruit alone and vegetables alone

Study	Fruit and vegetables	Fruit alone	Vegetables alone
Dauchet et al	RR 5% reduction for each additional portion (106g) eaten per day. Linear response, so for five portions 25% reduction.	RR 11% reduction for each additional portion eaten per day. Five portions results in 55% reduction.	RR 3% reduction for each additional portion eaten per day. Five portions results in 15% reduction.
He et al	RR - 26% reduction for 5+ portions eaten per day.	RR - 13% reduction for 5+ portions eaten per day.	RR - 16% reduction for 5+ portions eaten per day.
Range of results for 5 portions per day	**25-26% reduction in stroke risk by eating ~5 portions per day**	**13-55% reduction in stroke risk by eating ~5 portions per day.**	**15-16% reduction in stroke risk by eating ~5 portions per day.**

The reviews do not reveal any difference in the relationship of fruit or vegetable intake and different CVD outcomes. Two studies produced results where vegetables offered more benefit with regards to CHD, and two showed fruit to be the category with a larger risk reduction. Of the two studies that focused on stroke as an endpoint, He and colleagues[235] showed that vegetables offered more protection, whereas Dauchet and colleagues[234] produced results showing fruit consumption was more protective. However, all papers agreed that fruit and vegetables, in some combination would reduce the risk of both coronary heart disease and stroke.

Due to the nature of the study designs we are unable to say from these reviews that there is definite causal link between fruit and vegetable intake and cardiovascular risk, there is only evidence of a correlation. However other studies including Mente and colleagues[226] have attempted to address this by looking at the Bradford Hill criteria[236] and also by trying to find intervention studies to help show causation. As demonstrated by the additional literature search, there is a lack of published trials looking at dietary interventions (specifically fruit and vegetable intake) and CVD outcomes. Intervention studies would need to be undertaken to show a causal link between dietary factors and CVD. However, despite this lack of trial data the current body of scientific literature provides a strong and consistent evidence base for a relationship between increased intake of fruits and vegetables being protective for CVD.

There are various potential mechanisms by which fruit and vegetable intake could influence the risk of cardiovascular disease, and a summary of these is presented in Figure 10. Knekt and colleagues[237] wrote a paper that looked at antioxidant vitamins and coronary heart disease by carrying out a pooled analysis of nine cohorts. The paper showed that the antioxidant vitamins contained within dietary fruit and vegetables did not appear to offer much protection against the incidence of CHD. Those in the highest quintile of vitamin C intake had a relative risk of 1.23 [95%CI (1.04-1.45), p=0.17] compared to those in the lowest quintile. However supplemental vitamin C did significantly reduce the risk. Those who took >700mg of supplemental vitamin C per day reduced their risk by 25% [RR=0.75 95%CI (0.63-0.93), p<0.001. Flavonoids, which are found in fruit and vegetables, are known to have antioxidant properties. Huxley and colleagues[232] showed that eating foods that contained flavonoids, such as apples, onions and tea did translate into a reduction in the risk of mortality from coronary heart disease. However, it is still not clear if it is the flavonoids within these foodstuffs that give the benefit or some other aspect of the foods studied.

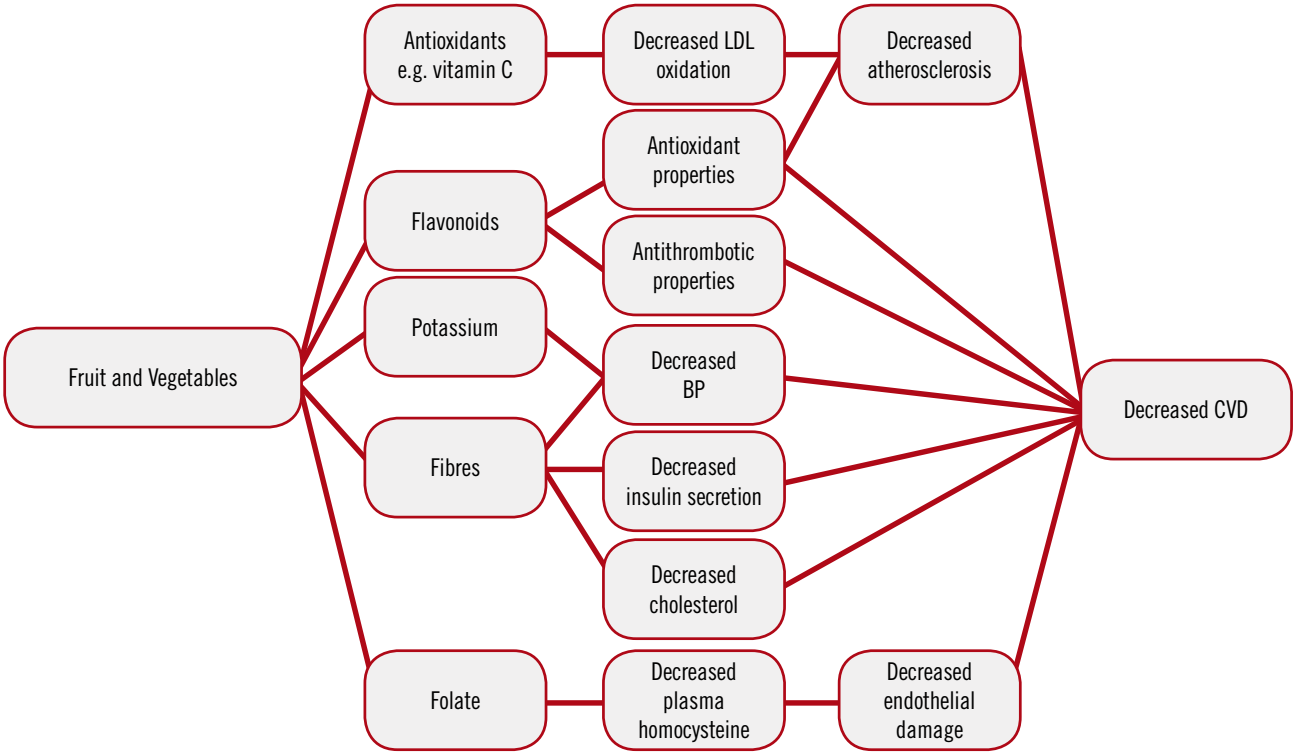
Pereira and colleagues[233] suggest that it is the fibre content of fruit that is the protective factor against CHD, although no such protection was offered from vegetable fibre. It is thought that dietary fibre alters the metabolism of bile acid, which lowers cholesterol.[238] There is also evidence that dietary fibre reduces blood pressure[239] and decreases insulin secretion.[240] It has also been hypothesised that it is the potassium in the fruit and vegetables that afford the risk reduction,[241] although this as a theory was not tested by any of the studies in this review.

As the mechanisms of action regarding fruit and vegetable consumption and their protection against CHD are still not well understood it is difficult to propose what the essential protective ingredients of a diet rich in fruits and vegetables might be.

During the research for this paper there were a number of papers found that focused on the Mediterranean diet and its influence on cardiovascular disease. Mediterranean diets are considered to be those that are based on plant foods, such as fruits, vegetables, cereals, nuts, beans and seeds.[242] As fruit and vegetables take up a high content of the diet we looked at some of the data available. Sofi and colleagues[243] looked at how following a Mediterranean diet influenced the incidence of various diseases. When looking at cardiovascular mortality they found such a diet reduced the risk by 9% [RR=0.91 95%CI (0.87-0.95), p<0.0001]. Panagiotakos and colleagues[244] looked at six studies which related to following a Mediterranean diet and its influence on cardiovascular disease. All the

studies showed a reduction in risk, and this varied from 8% to 45% depending on the study. Despite the obvious confounding factors of other dietary influences that make up the Mediterranean diet, such as using largely olive oil and only eating small amounts of meat, specifically rarely consuming red meats, it could be hypothesized that part of this protection against cardiovascular disease comes from the high fruit and vegetable intake. However, none of the reviews analysed the impact of high fruit and vegetable intake separately and so it is not possible to comment further on these.

Figure 10 Possible mechanisms of fruit and vegetable consumption and CVD



1.3.5.12 Limitations

With all these studies there will be many confounding factors. There are many influences on dietary habits which makes it difficult to dissect the evidence. It is likely that those who eat more fruit and vegetables live a more 'healthy lifestyle' and so do not smoke and also exercise more. Both these factors are known to reduce risk with regards to cardiovascular disease. In this sense there may well have been some overestimation in the effects of fruit and vegetable consumption as a variable. However some of the papers did try and adjust for these potential confounding factors. The paper by Dauchet and colleagues on coronary heart disease mentioned that there was likely to be an overestimation of the risk reduction due to publication bias. All the papers highlighted the problem of actually getting accurate data on the classification and exact amount of fruit and vegetables eaten, which is a problem inherent in these sorts of studies.

1.3.5.13 Conclusions

Evidence from systematic reviews and meta-analyses since 2002 show that eating more fruit and vegetables will decrease the risk of cardiovascular disease, with particular reference to coronary heart disease and stroke. The benefit of fruit and vegetable consumption has been found to be linear, with no upper limit as yet found. The exact risk reduction varies between papers, and the way it is presented also varies. However there seems to be a 17-21% reduction in the risk of CHD if an individual eats five portions of fruit or vegetables per day. Similarly this quantity of fruit and vegetables is also reflected in a 25-26% reduction in the risk of stroke. When divided into categories of either fruit or vegetables the range becomes much larger, and the data less reliable due to the smaller size of data available for analysis.

At present it is not clear what the underlying mechanisms behind these findings are; whether it is the antioxidant properties, the high potassium content, the fibre contained within the fruits and vegetables, or some other process or factor entirely. Nor is it clear if fruits or vegetables, or specific fruits or vegetables, confer greater benefits. More information needs to be gathered as to the mechanisms of action, and perhaps into individual fruit or vegetable benefits. However, it is not necessary to wait to discover the underlying mechanism, before recommending a diet rich in fruit and vegetables to help reduce the burden of cardiovascular disease.

1.3.6 Obesity and overweight

This review is contributed by Professor Philip James, President of the International Association for the Study of Obesity (IASO) and Honorary Professor of Nutrition at the London School of Hygiene and Tropical Medicine, Rachel Leach, member of IASO, and Caroline Small, Executive Director of IASO until March 2010 and honorary Senior Lecturer at Imperial College London.

This section will evaluate the contribution of increased weight and diet to the disease burden of CVD, including both morbidity and mortality. As discussed in section 1.1.1, morbidity leads to an additional burden of disability with enormous implications for society.

This section differs in approach from the other scientific reviews in this paper because obesity is both a condition in itself and a risk factor for cardiovascular disease. This chapter, therefore, examines the latest evidence on the complex inter-relationships between obesity, cardiovascular disease, cardiovascular risk factors, dietary factors and physical activity.

1.3.6.1 Obesity and overweight in Europe

1.3.6.1.1 Measures of obesity and CVD predictors

Body mass index (BMI), waist-hip ratio (WHR) and waist circumference (WC) are all used as predictors of cardiovascular diseases (CVD). However, which measure more accurately reflects CVD risk has been the focus of many investigations. One issue that emerges seems to be that different interpretations can be made depending on whether the study is conducted in a single population with predominantly similar population or across many societies with people of very different build.

The multinational WHO-sponsored MONICA study of 32,000 men and women aged 25-64 yrs from 19 populations participating in the second MONItoring trends and determinants in CArdiovascular disease (MONICA) survey from 1987-1992 found considerable variations in waist circumferences and WHR between different populations. Waist circumference and WHR, were both used as indicators of abdominal obesity but the waist circumference seemed to reflect well the degree of overweight whereas the WHR did not.

In the other major international studies—the INTERHEART[32] and INTERSTROKE analyses of 52 and 22 different countries respectively and spanning, as in the MONICA study, Asians as well as Caucasians—the question was what produced the most consistent prediction of cardiovascular disease. It rapidly became evident that BMI was only a crude measure of risk and that measuring the waist circumference as an index of abdominal fat distribution was more predictive of heart disease but the best measure was the WHR.[245] The proportion of the variance explained by taking the ratio of the waist to hip was not great so the simplicity of using just the waist measure is appealing although the body build of an individual from different ethnic groups can vary substantially so when comparing across ethnic groups the ratio can be useful. Similar results were obtained in the Asia-Oceania Obesity Collaboration.[246]

The waist measure in these international studies was more closely associated than BMI with cardiovascular disease, presumably because it reflected the greater hazards of abdominal obesity and took account of the varying propensity to abdominal obesity in different societies. The WHR, however, allowed for very different proportions of skeletal size in different societies.

Some single national studies give a different perspective. A Canadian study concluded that waist circumference may be the best single indicator of an individual's cardiovascular risk factors in a cross sectional study[247] whereas an Australian study[248] found that WHR had the strongest correlations with CVD risk factors before adjustment for age. All three obesity measures performed similarly after adjustment for age.

These studies are all cross-sectional and therefore assess the best correlations between indices. A comprehensive study by van Dis and colleagues however, compared the absolute risk, hazard ratio and population attributable risk of non-fatal and fatal CVD for BMI and WC in a large prospective cohort of over 20,000 men and women aged 20-65 years and with an average follow-up of 10 years in the Netherlands.[249] Overall the study concluded that the associations of BMI and WC with CVD risk were equally strong. Overweight and obesity had a stronger impact on fatal CVD than on non-fatal CVD. This then limits the criticism involved in the use of the BMI as a predictor of risk, but it does not necessarily apply to all 53 European countries in the European region (as defined by WHO). Indeed there seems to be evidence that populations emerging from poverty have a particular propensity to selective abdominal obesity where the use of waist circumference or WHR may be particularly valuable.[250]

In light of all these studies it is recommended that health professionals, in addition to measuring BMI, should incorporate the use of waist circumference as a simple additional measurement in their routine clinical examination of adult patients.

1.3.6.1.2 The prevalence of overweight and obesity in Europe

New analysis by the International Obesity Task Force (IOTF) of the prevalence of overweight and obesity in Europe in comparison with other regions of the world is shown in Table 9. This reveals that in Europe, the Americas and Middle East more than half of all adults are either overweight or obese and in some parts of Europe more than three quarters of older adults are affected, with eastern Europe having the greatest problems. The usual pattern is

for more women to be obese than men but the prevalence of overweight in men is greater.

The data on children are also alarming as over a quarter of them are overweight with about 5% of the entire European child population being obese with all its long-term adverse health consequences. There is emerging evidence of a potential levelling off in the prevalence of obesity in children in recent years in some European countries. Rokholm and colleagues[251] reviewed 52 studies in over 25 countries and found evidence of stability[6] or a levelling off of the epidemic in children and adolescents. Of the 21 studies in children in 11 European countries reviewed, 20 reported a levelling off, stability or a decrease in prevalence of obesity. Of seven studies in adolescents in seven European countries, all reported a decrease, stability or levelling off in adolescent females, and five for adolescent males. While these results give cause for hope, the authors point out that, in general, the prevalence of obesity still remains higher than ever before and previous stable phases in the epidemic have been followed by increases.

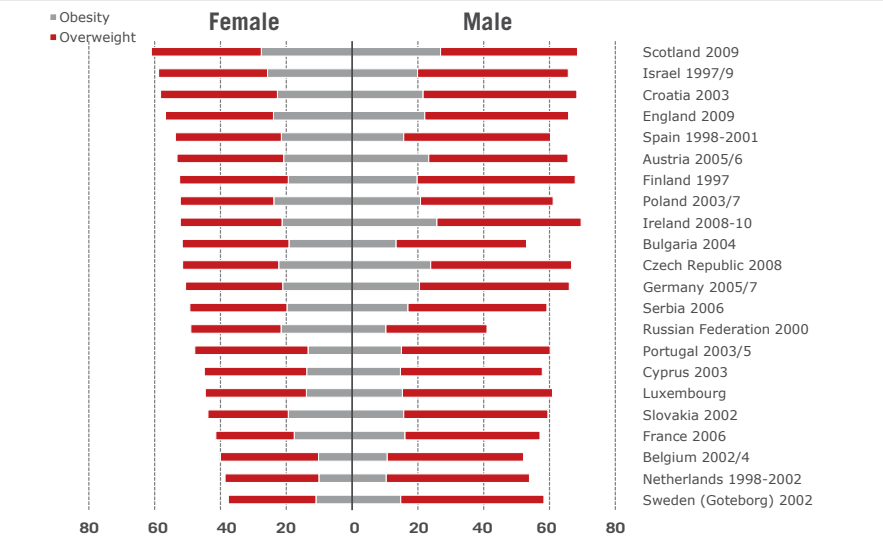
6 Stability refers to no statistically significant changes, while a levelling off refers to a clear change in the trend from an increase towards stability or a clear slowing down in the increase.

TABLE 9 PREVALENCE OF OVERWEIGHT AND OBESITY BY AGE GROUP FOR MEN AND WOMEN IN DIFFERENT WHO REGIONS

Sub-region	Sex	5–14 yrs			15–29 yrs			30–44 yrs			45–59 yrs			60–69 yrs			70–79 yrs			≥80 yrs		
		Ow	Ob	OW+Ob	Ow	Ob	OW+Ob	Ow	Ob	OW+Ob	Ow	Ob	OW+Ob	Ow	Ob	OW+Ob	Ow	Ob	OW+Ob	Ow	Ob	OW+Ob
Afr D	Male	1.8	0.7	2.6	14.2	3.3	17.4	22.4	7.0	29.4	29.3	10.6	39.9	27.8	9.7	37.5	11.8	1.1	12.9	0.0	0.0	0.0
	Female	1.9	0.3	2.2	13.4	4.4	17.8	21.0	11.4	32.5	23.2	14.0	37.1	30.0	19.9	50.0	12.4	6.6	19.0	0.0	0.0	0.0
Afr E	Male	2.6	0.8	3.4	14.8	3.7	18.5	24.8	8.5	33.3	29.6	11.6	41.2	27.8	11.6	39.4	32.0	14.0	46.0	0.0	0.0	0.0
	Female	3.3	1.2	4.5	10.9	4.1	14.9	16.6	10.1	26.7	18.2	14.9	33.1	29.9	27.9	57.8	33.9	31.8	65.7	0.0	0.0	0.0
Amr A	Male	18.5	9.6	28.1	31.3	22.5	53.8	38.8	29.0	67.9	43.5	33.2	76.7	41.6	36.3	77.9	42.1	36.0	78.1	42.3	35.6	77.9
	Female	15.2	11.1	26.3	22.7	27.4	50.1	26.4	33.7	60.1	28.3	36.8	65.1	34.8	33.2	68.0	34.9	33.1	68.0	35.2	32.8	68.0
Amr B	Male	18.2	8.8	27.0	25.8	9.9	35.8	38.7	17.7	56.4	38.8	20.5	59.3	38.5	17.7	56.3	35.2	11.6	46.8	30.9	7.6	38.6
	Female	19.0	7.4	26.4	21.6	10.3	31.9	30.0	21.5	51.5	34.6	27.8	62.4	34.9	27.1	62.0	34.7	21.1	55.9	30.4	14.9	45.2
Amr D	Male	17.5	7.8	25.4	24.1	8.9	33.0	38.7	17.7	56.4	38.8	20.5	59.3	38.5	17.7	56.3	35.2	11.6	46.8	30.9	7.6	38.6
	Female	18.1	7.1	25.2	21.6	15.8	37.4	26.6	23.4	50.1	26.2	18.4	44.6	34.9	27.1	62.0	34.7	21.1	55.9	30.4	14.9	45.2
Emr B	Male	11.1	6.4	17.5	20.5	6.4	26.9	38.0	13.5	51.6	40.6	15.9	56.5	39.2	15.6	54.7	42.5	11.7	54.2	39.3	12.2	51.5
	Female	12.1	6.7	18.8	23.0	10.9	34.0	35.4	28.0	63.4	35.9	36.8	72.7	35.5	35.1	70.6	39.8	27.4	67.2	31.0	16.5	47.4
Emr D	Male	8.4	6.4	14.8	12.9	5.0	17.8	21.8	8.4	30.2	23.1	11.1	34.3	13.8	3.1	16.9	12.5	4.1	16.5	13.6	4.5	18.1
	Female	9.3	6.7	16.0	16.0	7.9	23.9	21.1	22.6	43.8	20.8	29.3	50.0	17.0	9.1	26.2	13.9	6.5	20.4	27.3	9.1	36.4
Eur A	Male	16.6	5.5	22.1	26.9	9.0	35.9	44.8	18.1	62.9	48.6	26.3	74.9	50.2	30.7	80.9	51.7	25.6	77.3	51.5	26.7	78.2
	Female	17.4	5.5	22.8	17.3	8.7	26.0	25.3	17.1	42.4	34.1	27.6	61.7	39.3	35.1	74.4	39.0	31.4	70.4	40.0	32.3	72.3
Eur B	Male	14.6	4.1	18.6	20.7	4.1	24.9	40.9	15.0	55.9	42.7	23.4	66.1	45.8	25.0	70.8	44.3	22.6	66.9	34.7	16.5	51.1
	Female	12.6	2.6	15.1	12.5	3.8	16.3	27.7	15.9	43.6	35.1	30.2	65.3	37.8	38.2	76.0	36.7	37.7	74.4	36.5	28.0	64.5
Eur C	Male	20.2	6.0	26.2	20.2	1.6	21.8	36.7	8.5	45.2	41.1	13.5	54.6	39.1	13.4	52.6	40.5	9.8	50.3	33.8	8.5	42.3
	Female	17.4	4.2	21.5	16.7	5.6	22.3	33.4	22.1	55.5	37.0	35.1	72.1	38.0	36.1	74.1	36.8	27.0	63.8	31.8	16.6	48.4
Sear B	Male	11.7	2.5	14.3	5.4	1.6	7.0	12.6	2.0	14.6	14.2	2.8	17.0	8.2	1.7	9.9	14.8	1.7	16.5	17.5	7.5	25.0
	Female	7.1	0.0	7.1	9.3	2.7	12.0	36.3	6.9	43.2	24.1	6.3	30.5	14.2	4.2	18.4	20.4	5.5	25.9	21.9	9.4	31.3
Sear D	Male	11.7	2.5	14.3	4.9	0.9	5.8	11.6	1.9	13.5	13.4	2.3	15.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Female	7.1	0.0	7.1	6.0	1.3	7.3	14.5	4.4	18.8	16.6	5.9	22.5	2.4	0.0	2.4	9.1	0.0	9.1	0.0	0.0	0.0
Wpr A	Male	13.2	4.5	17.7	17.7	7.8	25.5	26.7	5.8	32.5	28.9	6.8	35.7	23.6	5.5	29.1	20.5	4.0	24.5	15.9	2.9	18.7
	Female	13.2	4.0	17.2	10.4	5.2	15.6	16.3	5.6	21.8	25.4	7.5	32.9	28.6	7.6	36.2	25.9	5.6	31.5	21.3	4.2	25.5
Wpr B	Male	6.5	3.4	9.9	14.0	1.2	15.2	24.2	4.7	28.9	27.0	6.4	33.4	27.9	7.4	35.3	9.7	0.0	9.7	15.9	2.9	18.7
	Female	5.3	1.7	7.0	10.4	1.8	12.2	18.4	6.4	24.9	22.2	4.7	26.8	22.1	9.8	31.9	28.9	3.2	32.1	21.3	4.2	25.5

Within each region there are obviously clear national differences as illustrated in Figure 11 where it becomes clear that the southern Mediterranean countries have a surprisingly high prevalence of adult overweight and obesity rate with women again showing a propensity to obesity.

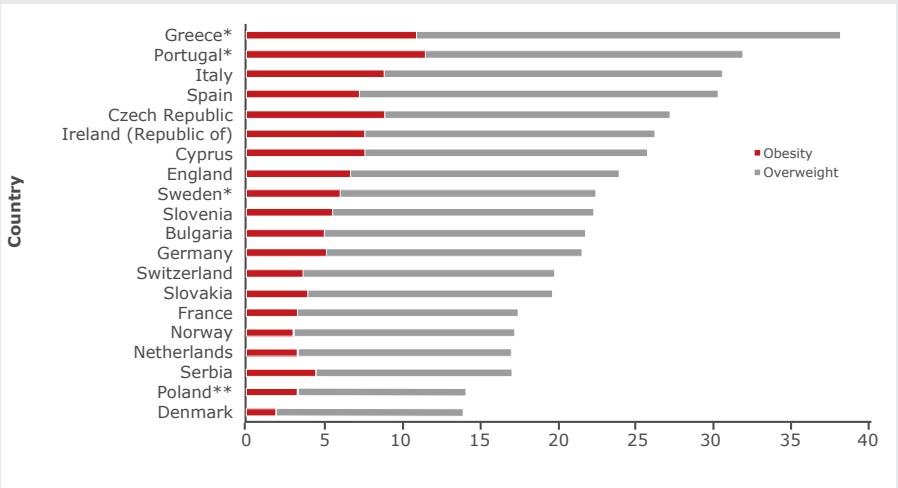
Figure 11 Prevalence of overweight and obesity in some European countries



Note: Men and women shown separately. Prevalences are from non-age-standardised surveys with measurements (not self-reported data) and relate to studies made since 2000. Some of the prevalences are based on sub-national surveys. Sources and references are available from IASO.

This geographical difference is also evident in children as shown in Figure 12.

Figure 12 European prevalence rates of overweight and obesity in children aged 7-11 years



*6-11 yrs ** 7-8 yrs

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It is recognised that there are significant differences and inequalities in the risk factors for cardiovascular health within and between EU member states (see Section 1.1.2), and that differing obesity levels play a role in these differences. The challenge, therefore, is how best to assess the contribution of obesity to the overall differences between countries and the different rates of change in cardiovascular mortality.

1.3.6.2 Diet, physical activity and excess body weight

Given that there is strong evidence that excess body weight is associated with adverse levels of blood pressure, blood cholesterol and glucose levels,[252] the issue is whether it is the obesity itself or the way in which excess weight amplifies the cardiovascular risk factors which is really responsible for cardiovascular disease in Europe. Alternatively, perhaps it is the dietary factors promoting obesity which also independently cause increases in blood pressure, blood cholesterol, higher blood glucose and lower HDL cholesterol levels. If there are independent mechanisms then altering the diet may prevent and deal with both cardiovascular disease and obesity at the same time. Delineating the exact contribution of weight gain *per se* from its accompanying features—namely, low physical activity and a poor diet—is, however, difficult because all these factors are so interdependent that cause and effect is hard to establish. Nevertheless some evidence is emerging to clarify the field.

1.3.6.2.1 Dietary fat and obesity

Dietary fat does not appear to have a selective metabolic effect on total energy balance. This is illustrated by the fact that an additional reduction in bodyweight does not occur when obese adults reduce selectively the proportion of calories from fat in their diet, if their precise energy intake is controlled.

Dietary fat can have an impact on energy balance, however, by increasing the energy density of the diet which when dense readily allows individuals to unconsciously overeat and put on weight. In other words, if people tend to eat the same amount of food, as the fat content of the food increases their energy intake will increase. Research by Stubbs showed that when individuals eat a high fat diet, rather than a modest or low fat diet of identical appearance and taste, an individual unconsciously consumed their usual amount (mass) of food. However, individuals were unaware that the higher the content of the fat in the diet the more energy they accumulated over the subsequent days. When sedentary they only maintained energy balance on a 20% fat diet and were in positive energy balance on the common European intake of 40% fat. If the adults were more physically active then their spontaneous intake of diets with different fat contents was the same in the short term. Thus when they took more exercise from walking and cycling they just achieved energy balance on 40% fat. However, individuals readily went into positive energy balance and gained weight when fat intakes increased further. Thus there is interplay between the amount of fat in the diet and the degree of exercise: the less exercise is undertaken the lower the fat content of the diet must be.[253,254] Stubbs also showed that increasing the energy density of the diet by increasing the refined carbohydrate content has the same effect as fat induced energy dense diets. Thus it is not necessarily a specific factor in fat that induces overeating—it seems to be simply a question of dietary energy density as concluded by WHO.[52]

1.3.6.2.2 Dietary fat, weight gain and weight loss

The short-term intervention physiological studies described above chime with the observations of the impact of the extraordinary nutrition transition which has affected most poor societies in the world over the last 25 years where the fat content has escalated in association with economic improvements from about 5-15% up to 30-35%.[255] This marked increase in dietary fat content has been accompanied by an explosion in the development of obesity,[256] so that now 80% of noncommunicable diseases occur in the low and middle income countries of the world rather than the affluent west.

Studies that attempt to specify the effect of fat on the development of obesity need to be distinguished from studies based on the assessment of slimming diets. It is now well recognised that as adults gain weight there seems to be a progressive adaptation in the brain—at the post leptin receptor level—which adjusts the normal brain responses to changes in energy balance and comes to resists the impact of attempts to lose weight. Thus the current obesity epidemic has occurred despite so many people attempting to reduce their weight. There is strong evidence of a hypothalamic adaptation whereby obese individuals when reducing their weight by only 10% have metabolic responses which favour weight gain and are also associated with a major drive to eat. These effects can be reversed to some extent by returning the blood levels of the hormone leptin to its high level seen in the obese adults.[257] When obese individuals have lost weight, their reduced body weight maintenance needs are accompanied by a normal drive to increase energy intake above that required to maintain their reduced weight. The failure to reduce energy intake in response to the reduced energy expenditure which normally occurs on weight loss reflects both decreased satiation and the perception of how much food is eaten. Multiple changes in neuronal signalling in response to food conspire together with the decline in energy output to keep body energy stores (body fat) above a brain defined minimum. Much of this biological opposition to sustained weight loss is mediated by the fat cell derived hormone leptin.[258]

Furthermore, detailed studies of those obese adults who have forced themselves to lose at least 15kg and maintained this weight loss for at least a year find that they are constantly needing to monitor their intake, ensure that they have a lower fat intake of 20-25% and engage in marked physical activity to combat this biological drive

to return to their previous weight.[259] This emphasises the importance of preventive measures to limit weight gain in children and adults, rather than relying on community-wide slimming programmes.

1.3.6.2.3 Potential selective effects of different fatty acids on weight gain

There are several studies which suggest that different fatty acids may play different roles in promoting weight gain. Astrup and colleagues have suggested that saturated fatty acids and particularly trans fats induce greater weight gain[260] even though short-term studies on satiety fail to show differences between saturated, monounsaturated and polyunsaturated fatty acids.[261]

1.3.6.2.4 Fat intakes and dietary energy density

On the basis of the detailed observations on fat intake it would appear sensible to consider fat intake to be a key to the development of obesity. However, Stubbs proceeded to show in his studies that if the fat content of the food was maintained but the energy density of the diet was adjusted then a high energy density diet induced by using refined carbohydrates with little unrefined fibre-rich foods had equivalent effects on energy intake as high fat diets.[253] This observation is in keeping with the nature of the global nutritional transition of the last 20-30 years. This is characterised by an increase in fat intake, a reduction in physical activity and a marked increase in sugar intake with a switch from unrefined foods to fibre-poor refined carbohydrates, increasing the overall energy density of the diet.

Although some studies examining modern societies, e.g. in the US, do not find a relationship between the fat content and the subsequent weight gain of adults[262] these diets are rich in sugars and refined foods so their energy density overall is high. When analysed as a body of research the WHO, in the 916 technical report,[52] specified that high energy dense diets and a low fibre intake, in addition to physical inactivity, were extremely conducive to excess weight gain. Thus high fat diets do promote the development of obesity but they seem to operate through a mechanism that relates to their impact on energy density.

In addition, there are suggestions that fat intake is not as satiating as dietary protein or carbohydrates.[263] Many observations also suggest that the mouth feel induced by fat, especially in combination with sugar, is very attractive and a substantial stimulant of excess consumption.

1.3.6.2.5 Selective taste receptors for fat

Evidence of a selective taste receptor for the essential fatty acids and n-3 fatty acids[264] has now been found and more recently for longer chain fatty acids.[265] This implies that—as with sugar, salt and the umami taste receptors—there is a primeval system of taste which includes a drive for at least some specific fatty acids. This presumably originally developed in response to the fundamental need for some fat and, in particular, the essential fats, especially n-3 fats required for human brain development. These gustatory responses may then amplify the mouth feel effects of fat to promote the consumption of fat rich diets.

1.3.6.2.6 Other dietary effects linked to the development of both obesity and hypertension

International comparisons show that weight gain and the development of high blood cholesterol track each other as societies become economically more affluent, whereas hypertension occurs in poor countries before the problem of obesity emerges.[266] Studies also show that dietary changes can lower blood pressure independent of weight change. These observations reflect the fact that other dietary factors contribute to hypertension in addition to weight gain and its associated high-energy diets.

One extremely important factor is the salt content of the diet (as explored in detail in section 1.3.2). In addition to the benefits of reduced blood pressure associated with reducing salt intakes, new evidence now suggests that if the salt content of current European diets can be reduced then children will tend to drink less. With current dietary patterns, this means they will consume fewer sugar containing drinks with their weight enhancing properties.[267]

Other important factors that can affect blood pressure include fruit and vegetable consumption, fibre intakes and dietary energy density. These issues are covered separately in sections 1.3.2, 1.3.4 and 1.3.5.

1.3.6.2.7 Physical activity: effects on energy balance, obesity and cardiovascular disease

A number of mechanisms for the beneficial effects of exercise on cardiovascular health have been put forward (see Section 1.3.7). These mechanisms—which include an improved lipid profile, increased insulin sensitivity, improved vascular function and reduced inflammation—are interlinked with the effects of weight loss and changes in regional fat distribution.

Extensive analyses have shown that even short periods of moderately intense exercise improve physical fitness—and thereby insulin sensitivity—with very beneficial effects on cardiovascular disease. The major cardiovascular preventive effect of physical activity (PA) can be attained by activity such as, for example, brisk walking of 150 minutes per week. However, longer periods of physical activity (e.g. an extra 60-90 minutes of brisk walking or cycling daily) may be needed to improve the chances of maintaining body weight, given the prevailing dietary patterns in Europe.

This is particularly important because, while physical activity has been shown to be of modest benefit in weight loss, it plays a crucial part in maintaining weight loss. When there is substantial weight loss physical activity becomes

much more important—as already set out in the studies by Wing and Hill on the post-obese US adults.[259] The contribution of physical activity to the degree of weight loss following bariatric surgery has also been investigated where physical activity appears to be associated with a greater weight loss of over 4% BMI in post-surgical patients.[268]

1.3.6.3 Obesity and the risk of CVD

The multi-factorial nature of cardiovascular disease is well recognised. Recent reanalyses[269] of data from the 52-country INTERHEART study[32] and the 22-country INTERSTROKE study[270] confirmed the importance of additional diet-related factors, alongside the "classic" risk factors of tobacco use, high-blood pressure and raised blood cholesterol. Abdominal obesity is identified as an important risk factor in both studies—accounting for 36% and nearly 20% of the incidence of heart attacks from which the women and men recovered respectively and 26% of strokes among male and female stroke survivors

The two recent major international studies mentioned above have an unparalleled range and detail in their analysis of risk factors, but they rely on retrospective analyses of risk factors in those who have survived their cardiovascular episode. Two integrated analyses involving multiple cohort studies (and which do not, therefore, depend on retrospective analyses) also confirm the strength of the principal factors observed in case-control studies. The two studies in question—the Prospective Studies Collaboration[271] of a million adults in 61 prospective studies and the Asia Pacific Cohort[272] involving over 350,000 adults in 29 cohorts—reaffirmed the need to consider these classic risk factors in Europe, where many of the studies were conducted.

It is well known that the main causes of premature death among obese people are heart disease, coronary thrombosis and congestive heart failure and all are significantly more frequent among obese people than normal weight individuals.[273] In the integrated data from the Prospective Studies Collaborative analyses of 61 studies involving nearly a million adults it is clear that there is an almost linear relation between BMI and systolic/diastolic blood pressure.[271] High blood pressure, raised concentrations of plasma low-density cholesterol (LDL) and low concentrations of high density cholesterol (HDL) fractions are all important risk factors in cardiovascular disease and all these risk factors are amplified by weight gain. Total cholesterol has been positively associated with ischaemic heart disease mortality at all blood pressure levels.[274] However, in older age groups higher blood pressures rather than greater total blood cholesterol levels are more important in determining cardiovascular deaths as shown in an extensive meta-analysis of over 55,000 vascular deaths.[275] Nevertheless, both the earlier and later meta-analysis of the role of obesity showed that weight gain amplified blood pressure and cholesterol levels, as well as increased the likelihood of increased fasting blood glucose levels and diabetes. So it is not surprising that there was a persistent effect of excess weight on cardiovascular events even in the 70-89 year-old group.[271]

Among 14 larger studies in a meta-analysis undertaken eight years ago, there was an average increase in coronary heart disease risk of 14% for each 2 kg/m^2 higher body mass[276] and this was later confirmed in the much bigger Prospective Studies Collaboration where a five unit BMI increase was accompanied by a 30% increase in total death rates and a 40% increase in cardiovascular mortality.[271] There was only limited evidence of effect modification by age, sex, ethnicity, or other variables. The authors concluded that the evidence from many studies, including randomised controlled trials, implied that the association between coronary heart disease risk and body mass index is in part or substantially mediated by the effect of weight gain on high blood pressure, dyslipidaemia, and impaired glucose tolerance. The effects of smoking and BMI were, in this predominantly European and US based meta-analysis, additive whereas in the Asia-Pacific region there was evidence that increased BMIs in smokers conferred an even greater risk than equivalent weight gain in non-smokers.[277] These studies amplify the need to quit smoking in those who are overweight.

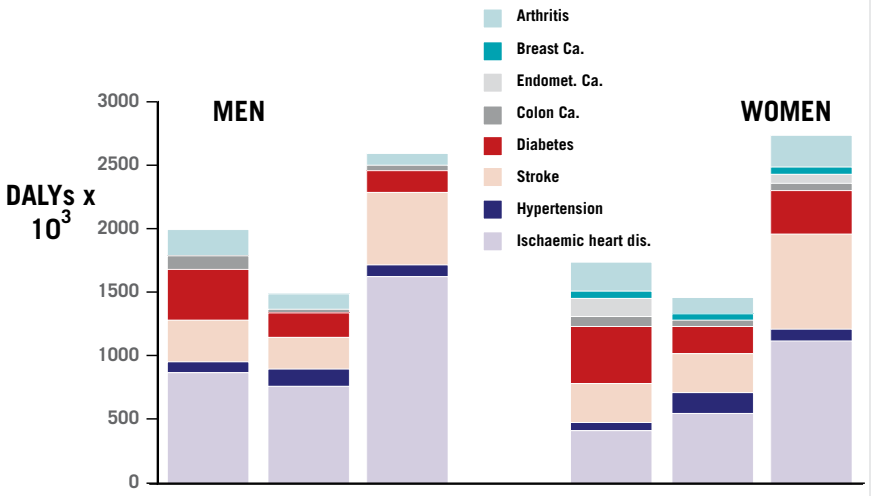
Although it is well accepted that excess body weight during midlife is associated with an increased risk of death[278] it is also known from long standing evidence from insurance statistics that the earlier the obesity occurs in adult life the greater the risk of premature death.[279] New data from Denmark demonstrates that even modestly overweight children between the ages of seven and 13 years have an increased risk of premature death from cardiovascular disease.[280]

1.3.6.3.1 Impact of excess weight gain on cardiovascular disability in Europe

It is important to consider the impact of "excess weight gain" as important in public health not just "obesity" which, using the standard WHO classification, occurs when adults have a BMI of ≥30.

The relative impact of weight in excess of the ideal average BMI of 21 on disability and premature deaths in Europe, calculated as part of the analyses of risk factors is shown in Figure 13. The analyses show that cardiovascular diseases are the biggest contributor to the overall burden of ill-health and this is, in part, attributable to weight gain.

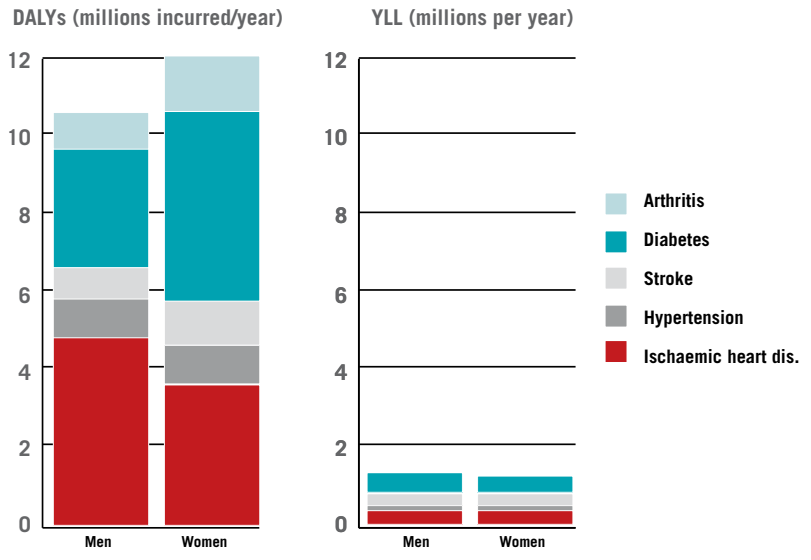
Figure 13 Disease Burdens in Europe linked to increasing body weights



Euro A: EU countries in 2003 + Croatia, Czech, Norway, Switzerland
Euro B: Bulgaria, Poland, Romania, Turkey, Balkans, some Asians Republics
Euro C: Baltic countries, Hungary, Russia, Ukraine, Belarus, Kazakhstan, Moldavia.

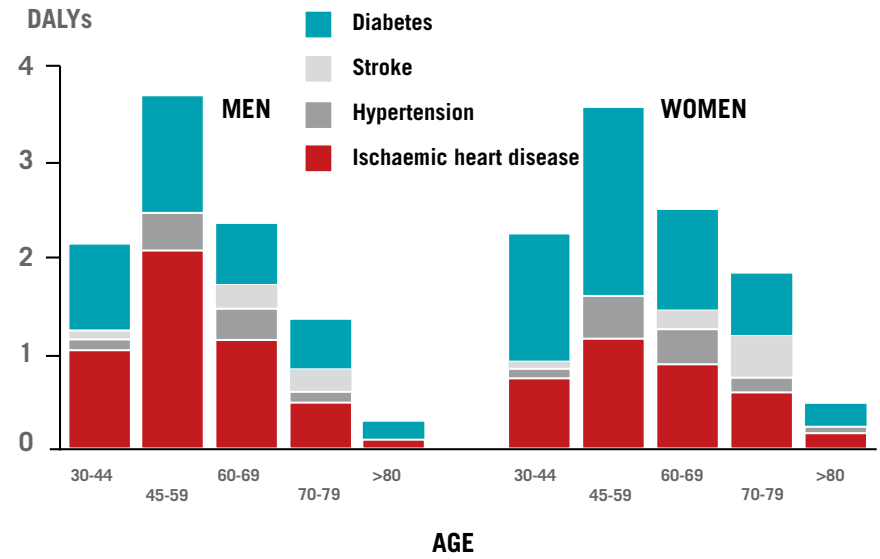
In early 2004 the greatest burden was evident in eastern Europe. Figure 14 emphasises that the burden is dominated not by premature death (i.e. deaths<75yrs), but by the disabilities encountered by people when they have high blood pressure, diabetes, heart disease or by the handicaps of having had a stroke; this burden of disability is nearly 10 times that incurred by premature deaths.

Figure 14 The relative effects of excess weight gain on disability and premature mortality from cardiovascular disease and diabetes



When considering how this burden affects adults of different ages one should recognise, as shown in Figure 15, that the burden of disease incurred by excess weight occurs to a substantial extent before retirement age. Therefore not only does this present a burden to society as a whole but the economic impact of obesity is likely to be very substantial in working men and women.

Figure 15 The age relationships of disability and premature deaths as expressed in DALYs for men and women separately in Europe in 2004



1.3.6.3.2 Metabolic changes induced by weight gain

In man the biggest reservoir of fatty acids for supplying the body's long-term energy needs is in the adipose tissue. In European countries, where the amount of fat in diets is generally high, human adipose tissue normally stores fatty acids derived from the diet rather than from fat synthesised in the body from dietary carbohydrates.

The absorbed dietary fat is stored in the liver as well as adipose tissue and as weight gain develops increasing amounts of fat are stored in muscle both within the muscle fibres themselves and in the cells surrounding the contractile fibres. It is now increasingly recognised that the storage of fat outside adipose tissue itself is particularly hazardous and affects metabolism in general as well as the function of both liver and muscle.

As fat cells expand with weight gain they respond by increasing their release of fatty acids into the plasma.[281] Hormones, in particular insulin, are a key regulator in this process of lypolysis and insulin functions under normal conditions to restrain the hormone sensitive lipase activity in the fat cells. Fat deposited in the visceral fat area is recognised to be particularly hazardous. This is because large quantities of fatty acids are released directly into the portal blood stream where they have an immediate impact on the liver. The liver normally removes the fatty acids from the blood stream and recycles them as triglycerides which are carried by low density lipoproteins. Visceral obesity is therefore a potent source for increasing liver fat stores where they interfere with the action of insulin.[282] In addition, as the fat cells expand, there is an increase in their production of several inflammatory cytokines including tumour necrosis factor alpha (TNFα) and this induces a fall in the secretion of a crucial metabolic hormone adiponectin which not only enhances insulin action and reduces the likelihood of diabetes but adiponectin also protects the endothelium of the blood vessels from a range of inflammatory and early atherosclerosis responses. This therefore suggests a direct interplay between the expanding fat cells in obesity and the impairment of arterial vessel function as well as explaining the fundamental link between weight gain and the development of diabetes with its cardiovascular damaging effects.

There are many other complex interactions of the metabolic pathways by which visceral and intramuscular fats induce insulin resistance as a forerunner to the development of glucose intolerance and type 2 diabetes. Obesity is now well accepted as having deleterious effects on hyperglycaemia, hyperinsulinaemia and hypertriglyceridaemia. These all appear to contribute to increased arterial stiffness

which interacts with insulin resistance, hypertension and endothelial function in a highly complex manner.[283] In patients with hyperinsulinaemia there are changes in the arterial wall, in particular characteristic decreases in the elasticity of the arterial wall and there is some evidence suggesting that insulin resistance precedes the onset of hypertension in high-risk patients. Improvements in insulin sensitivity improve hypertension and vessel wall function.

During the early phases of obesity, primary sodium retention occurs as a result of increase in renal tubular reabsorption. Extracellular-fluid volume is expanded and the kidney-fluid apparatus is reset to a hypertensive level, consistent with the concept of hypertension from volume overload. The plasma renin activity, angiotensinogen, angiotensin II and aldosterone values show significant increases during the development of obesity thereby explaining the retention of sodium and water.[284] As obesity develops fat cells expand and secrete more leptin. This increased circulating leptin signals to the hypothalamus and induces an increase in sympathetic nervous system activity which increases blood pressure. Obese adults have a marked increase in the spontaneous sympathetic nervous activity which therefore links to their greater propensity to hypertension.

There are many different ways of reducing weight but most if not all induce beneficial changes in insulin resistance and in hypertension. Many studies have demonstrated a positive improvement in a variety of co-morbidities on weight reduction whether this is undertaken by exercise,[285] lifestyle modifications,[273] diet,[286] drug therapy,[287] or bariatric surgery.[288] Fat loss through dieting or exercising produces comparable and favourable changes in plasma lipoprotein concentrations.[285] Thus weight loss, particularly when there is a decrease in visceral adiposity, leads to improved plasma lipids, enhances insulin sensitivity, improves endothelial function and lowers blood pressure.

1.3.6.3.3 Obesity as an inflammatory disease promoting cardiovascular disease

Obesity has now been found to involve a chronic, sub-acute state of inflammation which accompanies the accumulation of excess lipid in adipose tissue and liver (hepatic steatosis). Evidence suggests that changes in both inflammatory cells and biochemical markers of inflammation are associated with obesity.[289] Circulating mediators of inflammation participate in the mechanisms of vascular insult and atheromatous change, and many of these inflammatory proteins are secreted directly from fat cells and adipose tissue–derived macrophages. Two of these inflammatory molecules are tumour necrosis factor (TNF) alpha and interleukin 6 (IL-6). TNF-alpha inhibits the release from fat cells of the beneficial protein adiponectin and TNF-alpha and IL-6 both induce insulin resistance and counteract the beneficial effects of adiponectin. The increased release of fatty acids from the expanded fat cells in obesity and changes in the levels of retinol-binding protein 4 (RBP-4) may also induce oxidative stress and subsequent endothelial dysfunction with changes in thrombosis and endothelial processing of lipids through the PAI-1 and ICAM-1 molecules. Thus, the combination and interactions of fat accumulation, insulin resistance, liver-induced inflammation and dyslipidaemia may all lead to the premature atherosclerotic process.

The adipocyte-specific secretory protein adiponectin has been of particular significance in this field of research. Its levels are decreased in obesity and its secretion is markedly suppressed by the inflammatory peptide, tumour necrosis factor alpha which is secreted by fat cells as they expand.[290] Adiponectin may mediate some of its demonstrated cardio-protective effects through its anti-inflammatory properties. So the fall in adiponectin levels as well as the secretion of inflammatory factors from the expanded fat cells, particularly in the abdominal area may contribute to the increased cardiovascular risk associated with obesity.

1.3.6.3.4 Obesity and hypertension

The risk of hypertension is up to five times higher among obese people than among those of normal weight[291] and it has been estimated that up to 85% of the cases of hypertension arise in individuals with BMI values above 25kg/m^2.[292] Overall the mechanistic relationship between hypertension and obesity is complex and represents an interaction of ethnic, gender, demographic, genetic, neurohormonal, and biochemical, as well as dietary factors and physical inactivity. In addition, upper body (android) obesity, especially in the presence of increased visceral fat, is more strongly associated with hypertension than lower body (gynoid) obesity.[293]

As discussed in Section 1.3.1, studies have shown that differences in total fat intakes alter blood pressure levels in people with normal blood pressure and those with hypertension.[165]

It has long been recognised that the type of fat, rather than the total amount of fat *per se* in the diet, is important in the development of the increased levels of total blood cholesterol and low density lipoprotein (LDL) cholesterol levels, as outlined in section 1.3.1. Increases in LDL cholesterol induced by saturated fats can occur independently of any weight change, so reductions in cardiovascular risk can be induced even if a lower body weight is not achieved.

Nevertheless, the marked amplification of LDL cholesterol levels in association with higher body weights is remarkable and this effect is most marked in those with higher BMIs in the so-called "normal" range.[271] Thus the weight increase itself appears to be the factor which amplifies LDL cholesterol levels as there is little evidence that the percent of dietary saturated fatty acid in the diet are appreciably different in adults with BMIs of 20 compared with BMIs of 25. This suggests that obesity *per se* increases LDL cholesterol levels. Katan, in his analysis of the differences between the responses of lean and obese adults to changes in saturated fatty acid intake, notes that the obese (with their higher rates of cholesterol synthesis and higher prevailing levels of LDL cholesterol) are less responsive to lowering the saturated fatty acid intake.[294] This he

ascribes to the very much bigger inflow of cholesterol to the clearance mechanism of hepatic LDL receptors. This inflow suppresses LDL cholesterol clearance so the overall LDL levels remain high and the additional benefit of lowering saturated fatty acid intake is then modest. The implication of these studies is that weight loss is also important as well as reducing the saturated fatty acid intake but achieving sustained weight loss is much more difficult than reducing saturated fatty acid intake.

The type of fat with the exception of *trans* fats—probably of both industrial and ruminant origin[295]—does not seem to impact on the levels of high density lipoprotein (HDL) cholesterol levels which are recognised to be a further indicator of the risk of cardiovascular disease. The lower the HDL cholesterol level the greater the risk of cardiovascular disease. As weight gain occurs there is a progressive fall in HDL levels and this is evident as weight increases from a BMI as low as 20.[271] The fall in HDL cholesterol levels is progressive as the BMI increases into the obesity range. Losing weight can reverse this trend in HDL levels once the weight has re-stabilised at the lower level.

There seem to be, therefore, real advantages in not only modifying the type of fat in European diets but also lowering the intake of total fat to reduce the likelihood of weight gain and to promote some weight loss which will then induce favourable increases in HDL cholesterol levels.

1.3.6.4 Weight loss and its potential benefits

In light of the evidence that obesity is harmful to cardiovascular health, weight loss is often recommended as a therapeutic goal to patients. The data suggest that modest weight loss in hypertensive patients reduces systolic blood pressure.[296,297] In addition, non-surgical weight loss for extreme obesity results in significant improvement in many metabolic parameters and blood pressure.[298] It also has a well-recognised beneficial effect in markedly reducing the likelihood of those with glucose intolerance developing diabetes. This was first suggested in Swedish studies[299] and confirmed in a meticulous Finnish intervention trial[300] as well as in other studies in the US, [301] China[302] and India.[303] Thus one would predict a marked effect on cardiovascular morbidity and mortality with weight loss in obese subjects.

Studies examining weight loss in the general population have found an equivocal association between weight loss and mortality.[304-306] However, this approach requires further examination of the underlying causes of the weight loss. The primary limitation of the observational literature on weight change and mortality is the lack of information about an individual's intention to lose weight.[307,308] The weight losing population includes a mixture of individuals losing weight on purpose and those who lose weight unintentionally. Unintentional weight loss is frequently associated with poor health. Thus, it is difficult to draw strong conclusions on the impact of weight loss on mortality from such studies.

<u>Intentional weight loss</u>

In a prospective study to assess intentional—as distinct from unintentional—weight loss among individuals with diabetes, those who intentionally lost up to ~20lb had a 25% lower all causes and cardiovascular disease mortality.[309] The modest intentional weight loss is associated with improved blood pressure, lipid concentrations, insulin sensitivity and glycaemic control[310] so reducing these risk factors. Weight loss may also reduce the high risk of vascular complications and death among individuals with diabetes.[311]

Even the intention to lose weight is associated with reduced mortality, regardless of whether weight loss has occurred.[312] Individuals who reported trying to lose weight had a 23% lower mortality rate than those not trying to lose weight.[312] These results suggest that trying to lose weight may be a marker of healthy behaviours, such as being more physically active or eating healthier foods, and these lifestyle behaviours may be more important determinants of health status than weight loss *per se*, particularly given the biological resistance to weight loss in those who are already obese.

Bariatric surgery has beneficial effects on diabetes, other cardiovascular risk factors, cardiovascular symptoms, sleep apnea, joint pain and health related quality of life.[288,313-316] The prospective Swedish Obesity Subjects (SOS) was the first study to clearly demonstrate that surgery for obesity was associated with a reduction in mortality.[317] These findings are in agreement with other surgical retrospective cohort studies[318-320] and with observational prospective studies that attempted to separate intentional from unintentional weight loss occurring before the baseline examination.[309,310,321]

In the SOS study it took many years for the favourable treatment effect on mortality to be shown[317] and it also requires long follow up periods for the negative effect of obesity on mortality to become evident.[311] In the Framingham[322] and Manitoba[323] studies obesity became a significant predictor of mortality only after 26 years. Yet in the observational studies on bariatric surgery, with more marked weight loss than that seen in the SOS study, the 50% reduction in total mortality and the even greater falls in cardiovascular mortality occur within five to seven years.

<u>Unintentional weight loss</u>
<u>in those with cardiovascular disease</u>

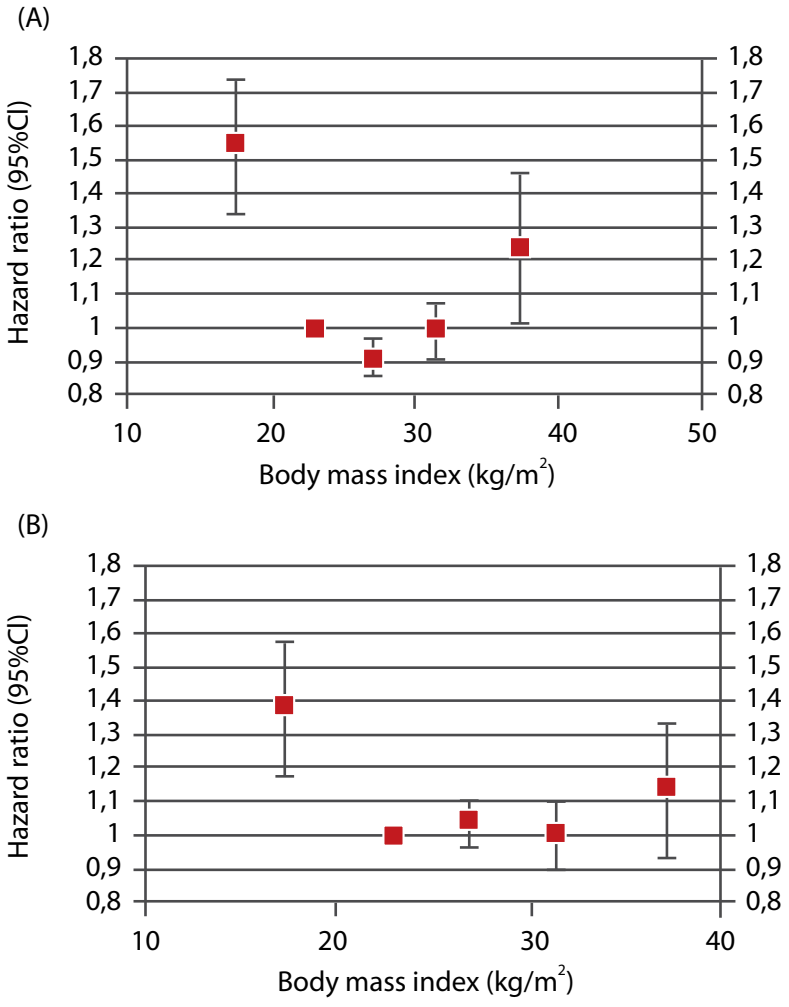
Unintentional weight loss is associated with *increased* mortality in those whose cardiovascular system is already compromised. Obesity may confer a more favourable prognosis, and an elevated BMI is an independent predictor of improved survival at one and two years in those with heart failure (HF).[324] However, this may reflect the absence of cachexia and aggressive disease in those who live longer, rather than an effect of obesity in conferring a positive health benefit.

The survival of patients with HF but without cachexia has also been reported.[325] The study concluded that BMI was a predictor of one year survival independent of exercise and left ventricular function: HF patients with a higher BMI had improved survival. However, it was also found that the survival of the lowest quintile BMI was similar to that of the cachexia patients over the first two years. These results suggest that it is difficult to find an appropriate "non cachectic" control group as BMI is directly proportional to disease status. It can be concluded that in HF patients with unintentional and involuntary weight loss mortality is high, but the converse simply indicates a less advanced or aggressive disease. Thus the so called "obesity paradox" in cardiovascular-compromised patients needs to be treated with great caution. Many inappropriate extrapolations from these studies are made to the non-diseased "general" population that obesity is "protective".[326]

This cautionary approach is supported by a study of a large, high-risk population over a prolonged period of time (10.4 years). This analysis concluded that obesity needs to be considered an important risk factor in patients with myocardial infarction (MI) or HF with systolic dysfunction. This study also concluded that there was no evidence of any protective effect of obesity.[326]

In conclusion, there is a strong tendency towards a U shaped relation between BMI groups and hazard of death as illustrated in Figure 16 from Abdulla and colleagues.[326] A higher hazard ratio is observed in patients with a low BMI. Importantly, the previously described paradox that obese patients with HF have a lower mortality is not substantiated in this more extensive analysis.

Figure 16 The prospective analyses of all adults who were admitted initially to Danish hospitals with either myocardial infarction or heart failure



The results of the adjusted multivariant all-cause mortality showed U-shaped form of curve in myocardial infarction (A) but not in heart failure patients (B)

1.3.6.5 Secular changes in obesity and other risk factors associated with changes in cardiovascular disease in Europe

Despite positive improvements in coronary heart disease mortality over the past decade it could be argued that this fall would have been more dramatic if the prevalence of obesity, a major risk factor for CVD, had not tripled in the European region over the same time frame.[327]

There is evidence to suggest that the decline in CVD mortality is slowing down, particularly in the younger age groups, in a number of European industrialised countries and the continuing prevalence of obesity is a significant factor in this slow down (see Section 1.1). In England, for example, reports of improvements in hypertension management are encouraging.[328] However, in a EU comparison, women in England have been reported to have amongst the highest systolic blood pressures (systolic blood 127)[329] and the highest prevalence of obesity[7] (2007: 24.4% females BMI >30). In contrast, some EU countries where the obesity rates are lower, such as Denmark (2005: 11% females BMI >30), have amongst the lowest reported systolic blood pressure (systolic blood 115). With a wave of childhood obesity imminent (in England, for example, 17.0% of boys and 19.6% of girls are overweight according to the 2007 Health Survey England,[330] the risk factors underlying CVD in younger generations give significant cause for concern.

1.3.6.6 A prudent diet and its impact

Long-term prospective studies have shown that dietary patterns have an important impact on cardiovascular morbidity and mortality. Thus if the elderly follow a Mediterranean diet then they have lower mortality rates and lower cardiovascular disease irrespective of their obesity levels.[331] A study by Brunner and colleagues[332] also demonstrated that a healthy eating pattern significantly reduced the risks of diabetes and major coronary events. Furthermore dietary change, as a result of health promotion advice, is effective among motivated high-risk individuals but tends to be modest among others.[334] These studies and others show the importance of dietary quality for population health and the reduction of cardiovascular mortality.

An analysis of Finnish data reinforces the importance of diet in CVD. When vegetable consumption trebled, and there was a substantial fall in the intake of total fat and saturated fat with a 15% decrease in total serum cholesterol concentrations[335] along with a significant decline in salt intake, there was a very marked fall in the population's blood pressure and cardiovascular disease. However, there was an increase in the average BMI of the Finnish population. Therefore, given the epidemic of obesity in Europe, with its associated increased risk of hypertension and greater blood cholesterol levels, it is still possible to reduce markedly the major risk factors for cardiovascular disease, the incidence of myocardial infarcts and strokes as well as cardiovascular mortality. This emphasises the importance of focusing on the dietary changes which are associated with obesity. Taken together, the studies suggest that dietary changes are important even if weight loss is limited. Nevertheless, preventive measures involving marked dietary improvements and increased physical activity should impact favourably on both the development of obesity and the prevention of cardiovascular disease.

WHO in its obesity report[336] suggested that the total fat intake should be as low as 20-25% and added sugar—through its effects in increasing energy density and, particularly, when consumed in drinks—is also conducive to weight gain. However, there are no goals as yet set out for the optimum dietary density of the diet. Therefore, it is clearly important to alter the diet by setting interim and longer term targets for the average fat, sugar, carbohydrate and fibre content of European diets as well as suitable targets for vegetable and fruit intake.

1.3.6.7 Population goals for body mass index

In the analyses of the impact of different risk factors on premature death and disabilities the WHO has emphasised that one should identify the ideal level of a risk factor as the optimum value for a population's average value for that risk factor in terms of its prediction of health and then consider deviations from this level as increasing risk. Therefore, WHO has emphasised that an "ideal level" of a risk factor should be set where it induces minimum health problems in a country.

For example, an individual is normally considered to have high blood pressure when the systolic blood pressure is 140+mmHg. However, the optimum average systolic blood pressure for the whole adult population has been shown to be 115mm Hg (i.e. far lower than the conventional level for diagnosing people as having the clinical disorder of hypertension). Similarly, when body weight is considered, then the average optimum body mass index (BMI) for adults was found to be 21 (i.e. far lower than the standard cut-off of BMI 25+ for specifying adults as "overweight"). Thus, as outlined earlier, it is important to consider the public health impact of excess weight gain, not just of obesity (BMI>30).

This report proposes a BMI of 23 as an intermediate target and a longer-term goal for a BMI of 21. These recommendations are in line with WHO recommendations for a population median BMI range BMI of 21-23.[52] Several Asian and African communities (with little or no adult underweight) had average BMIs in the 21-23 range in the 1980s before the onset of the nutrition transition and the westernisation of their diets. In the longer term European countries should be aiming towards the lower end of the WHO recommended range. WHO's report on obesity concluded that "*adults in affluent societies with a more sedentary lifestyle are likely to gain greater benefit from a median BMI of 21.*"[336]

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1.3.6.8 Conclusions

Avenell and colleagues in their Cochrane analysis could not distinguish between the effects of weight loss *per se* and the accompanying changes in diet leading to the weight loss.[337] This reinforces the important preventive health strategy of focusing on the provision of a high quality diet, high in fruit and vegetables and low in fats, sugars and salt for the prevention of cardiovascular disease in Europe.

1.3.7 Physical activity

The following review has been contributed by Professor Emeritus Ilkka Vuori, Finland.

1.3.7.1 Introduction

This paper summarises the evidence on the relationship between physical activity (PA) and cardiovascular disease that has been published mainly during the past 10 years. The primary focus is on aspects related to primary prevention of the clinical events of coronary heart disease and stroke.

1.3.7.2 Methods

The paper is mainly a review of reviews. The material was collected by conducting a search of the Medline, Embase and Cochrane electronic databases to identify systematic reviews and meta-analyses published on the relationship between PA and CVD since 2002. The terms used for the exposure were physical activity or exercise, and for the outcome the following terms: cardiovascular disease, coronary heart disease, coronary artery disease, coronary disease, myocardial infarction, stroke, peripheral vascular disease or peripheral arterial disease. In addition, committee reports on the topic were identified through scientific meetings and by personal communication. In section 1.3.7.5 *Biological plausibility* selected reviews published in the leading journals of the topic have been used.

The reviews on the relationship between PA and CVD were included if they fulfilled the criteria of systematic review and included meta-analysis and were written in English. A large majority of the studies included in the reviews were prospective cohort studies of at-risk populations, and the quality of the studies was assessed by established procedures in all included reviews.

1.3.7.3 Results

1.3.7.3.1 Coronary heart disease

The US Physical Activity Guidelines Advisory Committee Report (PAGAC 2008)[338] includes a comprehensive, rigorously conducted systematic review of 16 cohort and four case-control studies on men, and 13 cohort and six case-cohort studies on women published between 1996 and 2007. In addition, five cohort and four case-control studies reported the results on men and women combined. The studies include approximately 124,000 men aged 15 to 96 years and more than 200,000 women aged 20 to 85 years at baseline. All included studies provide self-report information on the PA of the subjects, a standardised assessment of cardiovascular clinical events, and a comparison of event rates in subjects assigned to two or more categories of PA. In all the cited studies, the multivariate adjusted relative risks were recorded and used in all analyses. The adjustments included, as a minimum, age, body mass index (BMI), cigarette smoking, blood pressure, and blood lipid concentrations. Inclusion of, for example, BMI, blood pressure, and blood lipids may in some cases inappropriately decrease the magnitude of the relation between the PA exposure and the clinical outcome because some of the benefit of PA may be mediated through these "mediating" or "intermediate" variables. In studies where RRs are presented using both limited and multivariate adjustments that accounted for the "intermediate" variables, the RRs for the limited adjustments show about 10% greater effects.

The review revealed that for men the median relative risk (RR) was 0.81 for moderate intensity or amount of activity versus no or light activity, and 0.68 for vigorous intensity or high amounts of PA versus no or light activity. The lower CHD event rate for more active men was reported for both non-fatal and fatal CHD with no systematic difference in CHD incidence versus CHD mortality.

In the 13 prospective cohort studies of women the median RR of having clinical CHD event was 0.78 for women reporting participation in moderate intensity or amount of PA compared to women reporting no or light intensity activity. The corresponding RR was 0.62 for women reporting performing vigorous or high amounts of activity as compared to women reporting no or light intensity activity.

The analyses showed no evidence that the effects of PA on CHD are different in men, pre-menopausal and post-menopausal women. The magnitude of the inverse associations between PA and CHD events for men and women older than 65 years were at least as strong as for younger adults.

A few of the prospective studies have reported PA two or more times during the follow-up. The results show that those men or women who reported increased activity during the follow-up had lower relative risk of CVD events compared to subjects who remained sedentary or at the previous activity level. In these studies the change in activity preceded the clinical events. These findings increase the evidence that links higher levels of physical activity with lower risk of CVD.

A meta-analysis by Nocon and colleagues[339] including 676,605 subjects in 24 cohort studies with at least 5,000 participants provided results that correspond to the findings presented above. Overall, in fully adjusted model the cardiovascular mortality was 35% (RR 0.65, 95%CI 0.60, 0.70) lower in the most active as compared with least active subjects. Adjusting only for age, the risk reduction

was increased to 47% (RR 0.53 (95% CI 0.46, 0.61) in all 13 studies that reported both fully adjusted and age-adjusted results. Thus, the reported risk reductions are conservative as in the 2008 PAGAC report.[338] The risk reduction was larger (RR 0.43, 95% CI 0.33, 0.57) in the eight studies (analyses of 10 groups) in which PA was assessed by fitness test as compared with those 16 studies (analyses of 20 groups) that assessed PA by questionnaire (RR 0.70, 95% CI 0.66, 0.74). Significant risk reduction was found in 26 of the analyses, and in most studies it was 30–50%. The size of the risk reduction was similar in men and women, and the three studies that also included older participants reported similar decreases in mortality as in the rest of the studies.

Another recent European meta-analysis by Sofi *et al*[340] included 513,472 individuals in 26 prospective cohort studies with 4-25 years of follow-up. In the included studies the intensity of LTPA (leisure time physical activity) had been assessed in at least three categories. High intensity level of LTPA was associated with 27% lower risk of CHD incidence or mortality (RR 0.73, 95 % CI 0.66, 0.80) as compared with the low intensity level. The high intensity level of LTPA was determined so that it is attainable by ordinary people. The corresponding decrease of risk associated with moderate intensity level of LTPA was 12% (RR 0.88, 95% CI 0.83, 0.93).

1.3.7.3.2 Dose-response relationship

The two meta-analyses referred to above[338,340] showed strong evidence for dose-response effect between PA and CHD. One meta-analysis included studies only on women. In five studies out of 30 the relative PA level (amount or volume) was reported for four levels. When these studies were combined, a significant dose-response relationship was found between the relative level of PA and risk of CHD: RR 1 (= reference), 0.78, 0.53, 0.61, p for trend <0.0001.

In populations where the reference group is very sedentary even very modest amounts of moderate intensity PA such as one to two hours walking per week are associated with reduced rates of CHD.[338,341] The risk is decreased with increasing amounts or intensity of PA, and much of the benefit is derived when the subjects perform 150 or more minutes per week of moderate-intensity PA. Greater amounts or higher intensity of activity provide increasing benefit. However, the shapes of any dose-response relations have not been well defined, potentially because of the inaccuracy involved in assessing physical activity. The relations are all most closely related to volume, with less information about intensity and none for frequency and duration of sessions. There is very limited and mixed data related to the effects of short bouts (~10 min or less) of PA (accumulation), but they seem to be effective in increasing cardio-respiratory fitness.[338]

1.3.7.3.3 Stroke

Several systematic reviews provide data of the relationship between PA and risk of stroke. Lee and colleagues[342] included data on 285,509 men and women from 18 cohort and five case-control studies in their meta-analysis. For the cohort studies RR for stroke incidence or mortality for the most active versus the least active individuals was 0.75 (95% CI 0.69, 0.82) and for the moderately active versus least active individuals 0.83 (95% CI 0.76, 0.89). For case-control studies, highly active individuals had 64% lower risk of stroke incidence (RR 0.36, 95% CI 0.25, 0.52) and moderately active individuals 48% lower risk (RR 0.52, 95% CI 0.40, 0.69) than their least active counterparts. The authors conclude that moderate and high levels of PA are associated with reduced risk of total, ischaemic, and haemorrhagic strokes.

The meta-analysis by Wendel-Vos and colleagues[343] included data from 31 studies published in English before 2001, including 24 prospective cohort studies and seven case-control studies. Persons categorised as most active compared to the least active during their leisure time were at significantly lower risk for all strokes (RR 0.78, 95% CI 0.71, 0.85), ischaemic stroke (0.79, 95% CI 0.69, 0.91), and haemorrhagic stroke (0.74, 95% CI 0.57, 0.96). When persons categorised as moderately active during their leisure time were compared with those categorised as least active, the RR of the active persons for all strokes was 0.85 (95% CI 0.78, 0.93), for ischaemic stroke 0.83 (95% CI 0.64, 1.09), and for haemorrhagic stroke 0.76 (95% CI 0.55, 1.05). Only six studies examined the association of occupational physical activity and risk of stroke. Persons categorised as most active in their occupation had RR 0.74 (95% CI 0.49, 1.12) for total stroke, RR 0.57 (95% CI 0.43, 0.77) for ischaemic stroke, and RR 0.31 (95% CI 0.13, 0.76) for haemorrhagic stroke when compared to the least active. Based on four studies, also moderate amount of occupational activity seemed to protect against total stroke, RR 0.64 (95% CI 0.48, 0.87).

The meta-analysis of Oguma and Shinoda-Tagawa[341] included 30 articles on women only. The aim of the study was to quantify the dose-response relationship of PA on CVD outcomes including stroke. When the seven studies reporting three relative levels of PA were combined, the RRs showed a dose-response relationship for stroke: RR = 1 (reference), 0.73, 0.68, p for trend <0.0001.

The PAGAC report includes data from studies published between 1996 and 2007, eight studies on women, 11 studies on men, and six studies on men and women combined. For all strokes in men, the median RR was 0.65 for moderate-intensity versus no or light activity and 0.72 for high-intensity or amount versus no or light activity. In women, the median RR was 0.82 for all strokes combined for moderate-intensity activity versus no or light activity, and 0.72 for high-intensity or amount versus no or light activity.

Reimers and colleagues[344] analysed 33 prospective cohort studies and 10 case-control studies published until December 2008. This meta-analysis included the studies of the earlier investigations cited above. LTPA and occupational PA were not differentiated. Two groups were compared: lowest level of PA as the reference, and the

group with highest level of PA or the one with the greatest risk reduction (in some studies the intermediately active group). The RR for ischaemic stroke in 12 cohort studies for the higher activity level as compared with the low level was 0.75 (95% CI 0.67, 0.84). The corresponding RR for cerebral haemorrhage in seven studies was 0.67 (95% CI 0.52, 0.86), and for undifferentiated type in 23 studies 0.71 (95% CI 0.64, 0.80). In most studies in which PA was graded on a scale with three or more levels, the risk of stroke declined with increasing PA. However, a clear dose-response relationship has not yet been demonstrated. In eight studies the risk was lowest in the intermediate PA category, but in five studies the risk was highest in this category. The risk of ischaemic stroke was 24% and 27% lower in the more active women and men, respectively, as compared with the least active subjects. The corresponding risk reductions for cerebral haemorrhage were 8% for women and 40% for men, and for undifferentiated type of stroke 29% and 28%, respectively. The risk reductions were statistically significant only for men—this was due, at least in part, to the smaller number of studies on women.

On the basis of the limited amount of data analysed in the cited studies, the inverse association between PA level and stroke risk appears very similar for men and women, and there seems to be no systematic difference in the relationship of LTPA amount to either total or non-haemorrhagic stroke in men or women aged 45 to 64 years versus 65 to 74 years at baseline.

A recent analysis of the follow-up data of the Health Professionals Follow-up Study (43,685 men) and of the Nurses´ Health Study cohorts showed among men daily PA (assessed several times during the follow-up) decreased the risk of total stroke by 22% and risk of ischaemic stroke by 25%. The corresponding risk reductions for women were 28% and 31%. Of the other risk factors included in the analyses (that were also repeatedly assessed during the follow-up) only “not smoking” decreased the risk more, 40–50% than “daily exercise”, while the effect of “optimal weight” on the risk was of the same order as that of “daily exercise.”[345]

1.3.7.3.4 Peripheral arterial disease (PAD)

The relationship between PA and PAD has been reported in few studies only, and the evidence suggesting protective effect of PA on PAD is weak.[338] However, PA particularly as structured supervised exercise training is a powerful and cost-effective secondary preventive measure for those with established PAD with or without claudication symptoms.[338,346-349]

1.3.7.4 Domain and type of physical activity

Most of the analysed data are related to LTPA, and most forms of LTPA consist mainly of endurance or aerobic activity—or at least aerobic activity has been the main component that has been assessed. However, also PA performed in occupational work, domestic chores, or while commuting appears to provide benefit.[338] A meta-analysis including a total of 173,146 subjects in seven prospective cohort studies and one case-control study analysed the relationship between commuting to work by walking or cycling and several cardiovascular endpoints (mortality, incident coronary heart disease, stroke, hypertension and diabetes).[350] The overall meta-analysis revealed a robust protective effect of active commuting on cardiovascular outcomes, integrated RR 0.89 (95% CI 0.81, 0.98). The relationship was stronger among women (RR 0.87(95% CI 0.77, 0.98)) than among men (RR 0.91 (95% CI 0.80,1.04)).

Walking is the most common and most feasible type of physical activity. Several recent systematic reviews and meta-analyses have provided consistent evidence of protective effect of walking on CHD[351] or CVD.[341,350,352] The meta-analysis of Oguma and Shinoda-Tagawa[341] referred to above included also an analysis of the relationship of walking and risk of CVD. When the included studies were combined by absolute walking amount, even one hour/week walk was associated with reduced risk of CVD outcome. The meta-analysis of Hamer and Chida[353] included 18 prospective studies and 459,833 subjects. The pooled hazard ratio of CVD mortality in the highest walking category compared with the lowest was 0.69 (95% CI 0.61, 0.77). The effect was robust among men and women. There was evidence of dose-response relationship across the categories of walking volume, but walking pace was a stronger predictor of risk compared with walking volume. The systematic review of Boone-Heinonen and colleagues[352] including 21 peer-reviewed publications revealed in general dose-dependent reductions in the risk of fatal or non-fatal CVD with higher duration, distance, energy expenditure and pace of walking. Adjustment for clinical CVD risk factors generally attenuated but did not eliminate the associations. Zheng and co-workers[351] found in their meta-analysis of 11 prospective cohort studies and one controlled randomised trial including 295,177 subjects that an increment of approximately 30 minutes of normal walking a day for five days a week was associated with 19% (95% CI 14 – 23%) CHD risk reduction, and the risk decreased as the walking dose increased. The results were similar in men and women and in different age groups. The findings of all three analyses were consistent with the current physical activity guidelines that recommend at least 150 minutes of moderate-intensity aerobic activity per week to attain significant health benefits.

Cycling is a popular leisure time activity and in some countries a significant mode of commuting. The amount of studies exploring the health-related effects of cycling is rather limited, however. A recent systematic review of the published data[354] revealed the following main findings concerning cardiovascular health. Strong evidence from randomised controlled trials indicates that the intensity of the spontaneously chosen speed of commuting cycling is higher than the intensity of walking, and sufficient to lead to significant increase of aerobic capacity. Moderate evidence supports modest favourable changes in selected cardiovascular risk factors. Results regarding the association of cycling and cardiovascular mortality and morbidity are

mixed. A recently published prospective study on a large Dutch population sample and with detailed data of cycling and other physical activities found significantly smaller risk of fatal and non-fatal cardiovascular disease incidence among cyclists compared with non-cyclists (fully adjusted hazard ratio 0.82 (0.71-0.95))[355] Two other prospective cohort studies[356,357] did not find significant association between cycling and cardiovascular disease morbidity or mortality, possibly due to narrow classification of cycling. In a Chinese study cardiovascular disease mortality was 25–37% lower among cycling compared to non-cycling women, but due to the small number of deaths in the cycling categories the difference was not significant.[358] Considering the available scientific evidence as a whole it may be concluded that regular cycling, for example as commuting, provides cardiovascular health benefits in terms of aerobic fitness and some risk factors, and may decrease the risk of cardiovascular diseases.

Stair use is one option to integrate PA into everyday life. It is vigorous activity with oxygen uptake reaching approximately 80% of maximal level in young healthy adults. A short review that summarises the effects of studies on the biological effects of stair use concludes that climbing stairs is effective in improving aerobic capacity and some CVD risk factors.[359] For 100–150 climbed floors each week corresponding to 8–12 minutes of daily exercise, the improvement in aerobic capacity may reach, in previously untrained persons, more than 10%, corresponding to an increase of approximately 1 MET. This magnitude of improvement has been associated with a 15% reduction of CHD/CVD mortality in epidemiological studies.[360] Thus, habitual use of stairs seems a promising mode of PA for CVD prevention.

Resistance exercise. Interest in the health-related potential of resistance exercise has gradually increased. The main part of commonly practiced resistance exercise is dynamic as contrasted to static components of the activity, and consequently the effects of resistance exercise refer mainly to those of the dynamic activity. Participation in resistance exercise as a major part of PA practiced in fitness studios has increased during the past decades. However, the proportion of resistance exercise of the PA volume at population level remains small. Relationship between resistance exercise and CVD has not been a subject of any major epidemiological study. The eventual role of resistance exercise in CVD prevention is most likely mediated through its effects on some biological risk factors of CVD.[361,362]

1.3.7.5 Biological plausibility

Physical activity fulfils well all criteria of a causal risk factor of CHD and stroke. The temporal sequence between the exposure and outcome is correct in a large number of prospective studies, in which PA is measured before the CVD outcomes occur. Furthermore, change of PA during the observation period as revealed by repeated assessments is logically reflected in the outcomes, and is seen as strengthening of the associations between PA and the outcomes.[363,364] In well-conducted epidemiological studies the consistency of the relationship between PA and CVD outcomes is good, and the findings are similar in men and women of different ages as well in populations from a large number of countries. The strength of the associations is significant both statistically and from the public health point of view. The strength of the relationship of PA and CVD is well comparable to that of other cardiovascular risk factors.[345,365-369] The same applies also for secondary prevention of patients with CHD.[370]

The strength and consistency of the relationship between PA and health outcomes is decreased by the inaccuracy of PA assessment by questionnaires leading to misclassification of subjects. There is a great need to begin to assess PA also in large epidemiological studies by objective methods such as accelerometers, step counters, heart rate measurements, and various combinations of the new techniques.

The decreased risk of CVD related to PA is partly explained by the effects of PA on other risk factors, and partly it is independent.

Blood pressure. Recent meta-analyses[338,371] show that aerobic PA decreases resting blood pressure in healthy subjects, mean reduction of systolic pressure 2.4 mmHg, and diastolic pressure 1.6 mmHG (2%/2%). In pre-hypertensive subjects the corresponding decrease is 3.1/1.7 mmHG (1%/2%), and in hypertensive subjects 6.9/4.9 mmHG (5%/5%). Changes are independent of changes in body weight. No consistent dose-response relationships between the changes in blood pressure and characteristics of aerobic PA have been observed. Limited data suggests that resistance training can also have a blood pressure lowering effect.

Blood lipids. Aerobic PA influences blood lipids favourably. High amounts of PA modestly increase high-density lipoprotein (HDL) cholesterol and decreases serum triglycerides, most in subjects with largest baseline abnormalities. The effects of PA on LDL cholesterol are inconsistently demonstrated. The same applies to the effects of resistance exercise on blood lipids.[338,361,372,373]

Impaired glucose tolerance and Type 2 diabetes (T2D). A large number of prospective cohort and cross-sectional studies and several randomised controlled trials show convincingly that increased levels of PA are associated with significantly improved glucose tolerance and decreased risk of T2D.[338] A key mechanism is the favourable effect of PA on the sensitivity of muscle and other tissues to the action of insulin. Any amount of PA appears to be better than none, but higher intensity and more frequent PA increase the preventive effect. Approximately 30 minutes of moderate-intensity activity at least five days per week decreases the risk of T2D by 25 to 36%, and the findings apply to men and women. Resistance training has shown promise as a mode of PA in the treatment of T2D, but the eventual role of resistance PA in the prevention of T2D has not been explored in any large prospective study. PA has also a secondary preventive role for CVD regarding T2D,

because strong evidence indicates that PA decreases the risk of CVD in subjects with T2D or impaired glucose tolerance. Furthermore, several studies have found a steeper response of CVD risk to PA in diabetic subjects than in those with normal glucose tolerance.

Metabolic syndrome. Regular PA is associated with substantially decreased risk of metabolic syndrome, and several cross-sectional and one prospective study strongly suggest dose-response between the amount of PA and metabolic syndrome in men and women.[338]

Overweight and obesity. There is favourable and consistent effect of aerobic PA on achieving weight maintenance (less than 3% change in body weight). There is large inter-individual variability with weight stability and PA, and many individuals need more than 150 minutes per week of moderate PA to maintain stable weight. The currently recommended amount of PA needed to prevent weight gain is between 150 and 250 minutes per week of moderate-intensity aerobic activity.[338,374] A recent analysis of weight gain of approximately 34,000 healthy US women consuming a usual diet revealed that initially normal weight women (BMI of less than 25) needed to perform about one hour moderate-intensity activity daily in order to maintain their weight or to prevent weight gain more than 2.3 kg during 13 years.[375] This amount of activity corresponds to one current US recommendation for the prevention of unhealthy weight gain.[376] Among heavier women no relationship was found between PA and weight gain suggesting that PA alone, at least in the amounts the study subjects performed, was not sufficient to maintain energy balance.

In fact, evidence suggests that physical activity is of modest benefit when attempts to reduce weight are made. Greater amounts of aerobic PA than 150–250 min per week are needed to achieve clinically significant weight loss (at least 5% of body weight), but moderate-intensity PA will improve weight loss in connection with moderate dietary restriction.[338,374,377-379]

There is clear evidence, however, that physical activity plays a crucial part in maintaining weight loss. Cross-sectional and prospective studies indicate that after weight loss, weight maintenance is improved with moderate-intensity aerobic PA more than 250 minutes per week, but there is lack of well-designed randomised controlled trials. There is a dose effect of PA, with greater weight loss and enhanced prevention of weight regain with doses of PA that approximate 250 to 300 minutes per week. The evidence on the effects of resistance training in prevention of weight gain or in weight reduction is less consistent.[338,374] The contribution of physical activity to the degree of weight loss following bariatric surgery has also been investigated where physical activity appears to be associated with a greater weight loss of over 4% BMI in post-surgical patients.[380]

Aerobic PA decreases also total abdominal adiposity and intra-abdominal adiposity. Moderate-intensity aerobic activity approximately 150 to 300 minutes per week leads to reduction of abdominal obesity that is consistent with improved metabolic function. Limited evidence suggests that resistance training has a small and less consistent effect on abdominal obesity.[338,361,374]

Regarding the risk of CVD, it is important to note that prevention of weight gain is an effective way to prevent the development of undesirable changes in the metabolic CVD risk factors, and even small (less than 3%) or no decrease in body weight by PA leads to significant beneficial changes in those risk factors.[374] Furthermore, PA also counterbalances the risk caused by overweight (BMI 25.0 to 29.9) on CVD mortality or events.[381]

Other mechanisms. Several studies have reported that the reduced risk of CVD in the active subjects is not fully explained by the traditional risk factors such as hypertension, high cholesterol, high BMI and diabetes.[382-384] A recent study in a large cohort of women[385] found that 59% of the PA-induced reduction in CVD events was explained by differences in a large number of traditional and novel cardio-metabolic risk factors, particularly by inflammatory/ haemostatic factors and blood pressure. However, the remaining 41% of the risk reduction due to PA was independent of these effects. In another study the 52% higher risk of CVD in physically inactive as compared with physically active (at least 150 min/wk) men and women was not mediated by the measured cardio-metabolic risk factors (blood pressure, triglycerides, low-density lipoprotein cholesterol, high density lipoprotein cholesterol, glucose, and waist circumference).[386] The findings of these studies strongly suggest that, even in the absence of positive changes in cardio-metabolic risk factors, increasing PA level would decrease the risk of CVD.

The reduced risk of CVD related to PA beyond the traditional and even novel risk factors may be explained by several mechanisms. PA has been shown to decrease chronic low-grade inflammation that is an important factor in the pathogenesis of atherosclerosis as well as insulin resistance.[387,388] PA also exerts several direct effects on the vascular wall. Habitual PA favourably modulates several expressions of arterial ageing and preserves vascular function. Thus, adults who regularly perform aerobic PA demonstrate smaller or no age-associated increases in large elastic artery stiffness, reductions in vascular wall endothelial function, and increases in carotid artery intimal medial thickness. Moderate-intensity aerobic exercise training improves carotid artery compliance and can improve vascular endothelial function in previously sedentary middle-aged and older individuals by several mechanisms.[338,389-392] The effects of exercise on the vascular wall may be induced via the impact of repetitive increases in shear stress on the endothelium, which transduce structural and functional adaptations that decrease arteriosclerotic risk. The beneficial effects of PA on the vascular wall may be enhanced by decreased sympathetic and increased parasympathetic outflow caused by PA.[393,394]

1.3.7.6 Health-enhancing potential of PA

In addition to the substantial benefits to cardiovascular health, physical activity is associated with lower risk of several of the most common chronic diseases and manifestations of ill health as follows:[338]

- type 2 diabetes and metabolic syndrome: 30% to 40% lower risk (strong evidence),
- hip fracture: 36% to 68% lower risk (moderate evidence),
- osteoarthritis: 22% to 83% lower risk (weak evidence),
- functional or role limitations: about 30% lower risk (moderate to strong evidence),
- falls: 30% lower risk (strong evidence),
- colon cancer: 30% lower risk (strong evidence),
- breast cancer: 20% to 40% lower risk (strong evidence),
- depression: 20% to 30% lower risk (strong evidence),
- dementia: 20% to 30% lower risk (strong evidence).

The risk reduction of various diseases is reflected in the reduced risk of all-cause mortality of 30% (strong evidence).

If a reversed expression is used, burden to health of physical inactivity (PIA), the estimated number of deaths in Europe in 2004 caused by PIA is 992,000 annually, 10.4% of all deaths. About half of these deaths are caused by CVD. The loss of disability adjusted life years (DALYs) in Europe caused by PIA is estimated as 5.5% of all DALYs. Among the factors influencing the burden to health, PIA ranks fourth as a risk factor for all-cause mortality and sixth among the risk factors for loss of DALYs.[31]

It is also worth mentioning that PA not only decreases risk of diseases but also improves cardiovascular and muscular fitness (strong evidence), cognitive function in the elderly (strong evidence), functional health in the elderly (moderate evidence), and sleep quality (moderate evidence).[338]

All data presented above indicates that PA is one of the major factors that have very large potential to maintain and improve health and decrease burden of ill health in Europe. A substantial part of all of the risk reducing and health-enhancing effects of PA is attained by moderate amount and intensity of aerobic PA, e.g. by brisk walking (four to six km/h depending on the fitness of the subject) for 150 minutes per week. This dose and type of physical activity strongly supports the view that it is a feasible population-level means to enhance health.

However, for most of the major health benefits of PA, including the reduction of the risk of the most common cardiovascular diseases, there is a dose-response relationship between the risk and PA, especially the amount of PA.[338] Thus, by increasing the amount of daily or weekly PA above the current recommendation, the risk of most of the most common non-communicable chronic diseases would further decrease, although with diminishing gain. One target level for a large number of the European people employed in sedentary occupations could be about one hour moderate-intensity PA daily, based on the evidence and recommendation to prevent weight gain (see above). Adoption of this level of PA would lead to massive avoidance of overweight and obesity and the associated health losses.

All data on the associations of PA and risk of chronic non-communicable diseases, and a major part of the data as the basis for the current PA recommendations, are derived from studies assessing PA by self-report using various questionnaires. This leads to underestimation of the preventive potential of PA due to misclassifications and over-reporting of the activity, may increase the relative weight of LTPA to the cost of the other domains of PA in attempts to assess the total activity, and hinders reliable quantitative assessment of PA in research and surveillance. In the light of the great potential of PA for health, and consequently the need to promote it using valid quantitative recommendations as well as to monitor and to survey it reliably at individual, group and population levels, objective methods to assess PA should be taken into much wider use. Appropriate technologies are available.[395] Funds should be allocated for their use in sufficiently large scale at national and international levels in order, for example, to begin to collect data for a new generation of epidemiological studies that would increase the reliability and accuracy of the use of PA for health. Objective data are needed also to survey PA levels and trends in populations in order to evaluate the effectiveness and cost-effectiveness of the attempts to increase PA.

An example of the value of objective, accurate methods in assessing PA are the recent studies revealing the health risks of sedentariness, especially sitting e.g. in driving a car and viewing TV. Large amount of especially uninterrupted sitting has been shown convincingly to be a risk factor of e.g. overweight and obesity[396-398] as well as of metabolic diseases[399-401] and all-cause and CVD mortality[402] even independently of PA. Increased emphasis on sedentariness is important not only for human, but also for environmental health. Substantial increase of PA and decrease of sitting in large numbers of people, and increasing and improving the environmental conditions to enhance this development would contribute substantially to efforts to reach important societal goals such as saving energy, decreasing environmental hazards due to, for example, motorised transport, and preventing climate change.

1.3.7.7 Conclusions

The quantity and quality of scientific data on the cardiovascular and other health-related effects of physical activity has increased greatly during the past decade. The evidence now shows convincingly that insufficient physical activity is one of the key causal risk factors of CVD, particularly of the most prevalent of them, CHD and stroke. Because of the high prevalence of insufficient physical activity, the CVD burden caused by it is great. The effects of insufficient physical activity are mediated partly through the traditional major risk factors, and partly they are independent. This rather recently shown fact emphasises the essential, irreplaceable role of PA for cardiovascular health.

Strong epidemiological evidence indicates that a major part of the preventive effect of PA can be attained by activity that is applicable in large scale in all European populations: moderate-intensity endurance or aerobic activity such as brisk walking on several days during the week, in total approximately 150 minutes per week. Thus, a population goal of 150 minutes moderate-intensity physical activity per week is proposed as an interim target for cardiovascular health.

Higher, but still attainable, amounts of moderate-intensity PA, however, would further increase the CV and other health benefits. Furthermore, there is increasing evidence that overall levels of physical activity, from general activity such as brisk walking or cycling, need to be higher—an extra 60-90 minutes daily, for example—to improve the chances of maintaining body weight given the prevailing dietary patterns in Europe. Thus more modest exercise patterns may be compatible with lower energy density diets, but in current circumstances there will be a need in Europe to promote higher levels of physical activity if the benefits from the decline in cardiovascular risk factors are not to be wiped out by rising levels of obesity. Thus, a further goal of one hour of moderate-intensity activity on most days (around 300 minutes/week) is appropriate in sedentary populations to avoid overweight and obesity, and the associated health consequences. In the longer term, moderate activity for an hour a day (60 minutes daily) should be the target.

PA in all domains, during leisure time, in domestic chores, in transport, and in occupational work has been shown to be effective.

Prevention of CVD through increased PA brings also several other health benefits by decreasing substantially the risk of several of the most common chronic diseases and by improving and maintaining physical, mental, and cognitive functions. PA also decreases— and in overweight (BMI 25-29.9) persons may even totally counterbalance—the risk of CVD as well as several other health hazards of overweight and obesity, probably because a large part of overweight and obese individuals are insufficiently physically active. It is clear that sufficient physical activity is an essential factor in attaining and maintaining health and functional capacity at all ages, and without sufficient physical activity all attempts to improve the health of the Europeans remain deficient.

Rapidly increased scientific evidence indicates that complete sedentariness, especially sitting, increases the risk of overweight and obesity, and of metabolic and cardiovascular diseases even independently of PA. Thus, sedentariness is a domain of its own that has to be tackled by efficient policies and actions.

Prevention of CVD through PA by increasing opportunities and motivation to participate in it and by decreasing sitting during leisure time, in transport, and in occupational life gives strong support also to other important societal goals such as fighting against obesity, traffic congestion and accidents, air pollution, excess use of energy, and climate change.

1.3.8 Alcohol

This review is contributed by Simona Costanzo, Augusto Di Castelnuovo, Maria Benedetta Donati, Licia Iacoviello and Giovanni de Gaetano of Università Cattolica del Sacro Cuore, Campobasso, Italy.

1.3.8.1 Summary

A large number of epidemiological data—both on apparently healthy people and on patients with a history of cardiovascular events—indicate a consistent reduction in the risk for cardiovascular events or for all-cause mortality among moderate alcohol drinkers, but, on the other hand, the harm of excess and irregular drinking.

A J-shaped relationship between cardiovascular and total mortality and increasing amounts of alcohol consumed was consistently observed in several meta-analyses, showing that low to moderate consumption of alcohol (no more than one drink daily in women and two drinks in men) significantly reduces total mortality, while higher doses increase it. Definitions of standard drinks vary between countries, but in this paper a standard drink is considered to contain 10 g of alcohol (ethanol), which is equivalent to 250 ml (half a pint) of beer, a small glass of wine or a 40 ml measure of spirits.

There is no reason to discourage adults who are already regular light-moderate alcohol consumers from continuing. Although low-moderate, non-binging alcohol consumption—in the absence of contraindications and in the context of healthy eating and a healthy lifestyle—reduces the risk of coronary heart disease, it is not recommended that adult abstainers begin drinking. This is because even moderate alcohol intake is also associated with increased risk of other harm and there is a risk that some of those who start to drink will consume more than the low-risk drinking limits. For these reasons it is not recommended that anyone be advised to drink more frequently on health grounds.

The hazards of excess or binge drinking should always be highlighted and heavy or binge drinkers urged to cut their consumption.

Regarding cardiovascular patients, if not contraindicated, regular alcohol consumers should not exceed one drink/day for women (10 g) or up to two drinks/day (20 g) for men as a component of a balanced cardio-protective dietary pattern, with appropriate energy intake levels. At present, a cardiovascular patient who is teetotal should neither be recommended, nor “prescribed” to start drinking for health gain.

The protective effect of regular and moderate alcohol consumption in coronary heart disease and ischaemic stroke has been consistently shown in many epidemiological studies.[403-406] The association between alcohol consumption and CHD mortality is described through a J-shape relationship, where teetotallers and heavy drinkers are at the highest risk whereas light-moderate drinkers are at the lowest risk. However, if low alcohol intake is

inversely related to CHD, the other side of the coin shows an increased risk for certain cancers, cirrhosis and death from accidents mainly associated with increasing alcohol consumption.[407-409] Total mortality was also reduced in moderate drinkers; however, excess drinking is definitely harmful.[408]

The absence and the unfeasibility for ethical considerations of randomised controlled trials leave room for residual uncertainty about cardiovascular protection by alcohol consumption and consequently there has been a point-counterpoint debate on this topic in scientific literature.[409,410,411]

In the last decade, several open questions have been addressed: the confounding factors and the weakness of study design (choice of reference category), the role of different alcoholic beverages, the effects of alcohol in women and men or in unhealthy individuals (i.e. cardiovascular patients), the harm of irregular (binge) drinking.

1.3.8.2 The role of confounding and study design

A weakness of several studies on alcohol consumption is the heterogeneity of the reference groups, which sometimes have included lifelong teetotallers, former drinkers and/or occasional drinkers.[410] However, the lower risk associated with moderate alcohol consumption does not appear substantially related to the inclusion of the above mentioned categories in the reference groups.[408,410]

It has been suspected that the protective effect of moderate alcohol consumption on cardiovascular disease might be due to a favourable risk profile in moderate drinkers, but a higher prevalence of cardiovascular risk factors in abstainers.[408,410-413] Unmeasured factors that might differ between drinkers and abstainers might contribute to the apparently lower risk among drinkers (the so-called uncontrolled or residual confounding). It has been argued that drinkers have many healthier characteristics than non-drinkers and thus show lower ischaemic heart disease risk. However, when all the possible confounding factors are taken into consideration to assess whether other factors might account for these mortality differences, a large meta-analysis found there was still more than a 10% lower total mortality rate in the light to moderate drinkers.[408]

1.3.8.3 Is wine better than beer?

Many epidemiologic studies have explored the hypothesis that consuming alcohol in the form of wine (or beer) might confer a protection against cardiovascular disease above that expected from its alcohol content.[404] Wine and beer might indeed conceivably show additional non-ethanol related beneficial effects. In fact, while the potential mechanisms underlying the effects of alcohol have essentially been limited to lipid metabolism and the haemostatic system, those related to wine or beer consumption have been extended to specific antioxidant and vaso-relaxant properties of their different polyphenolic and phenolic constituents.[404,410,414]

In spite of a large number of experimental studies suggesting a protective role of wine-derived polyphenols on ischaemic CVD risk, epidemiological evidence of a greater effect of wine versus beer has not been established (see also Section 1.3.10.2 on polyphenols). A meta-analysis conducted on studies that investigated the relation between fatal or not fatal vascular events and specific alcoholic beverages (wine and beer), whatever the amount consumed, showed an overall relative risk (RR) of wine drinkers in respect to non-drinkers of 0.68 (95% CI: 0.59-0.77), whereas the protection associated with beer drinking showed an overall RR of 0.78 (95% CI: 0.70-0.86).[404] However, after the exclusion of the studies that did not simultaneously adjust for different types of alcoholic beverages (the most unbiased method to control for confounding), no meaningful difference in the RRs of vascular risk between wine and beer drinkers as compared with abstainers were apparent (0.75 vs 0.77).[404] The difference between wine and beer consumption was also investigated considering the amount of alcohol intake.[404] In contrast with wine, for which a clear inverse dose-effect curve was found, the fitted models failed to show any significant relationship between different amounts of beer intake and vascular risk.

Using ecological studies, which are not well controlled for confounding, international comparisons showed lower coronary artery disease mortality in wine-drinking countries (e.g. France) than in countries where beer or distilled spirits are the preponderant alcoholic beverages.[405] Several studies suggest that alcohol consumption at mealtimes is more favourable for CHD, and wine is more often drunk with meals than beer or liquor.[405]

Whether wine is better than beer or spirits remains therefore to be elucidated, but it seems likely that the ethanol component of different beverages is a major factor that reduces the cardiovascular risk.[411]

1.3.8.4 Is the effect of alcohol different in men and women?

There is controversial evidence that alcohol consumption may have a different protective role in men and women.

In a meta-analysis of studies on alcohol consumption and stroke,[403] a greater protection in women than in men was apparent. In another meta-analysis of studies reporting trend (dose–response) effect of alcohol intake on vascular risk separately for men and women, the predicted dose–response models were very similar for both sexes.[415] At 12 grams of alcohol per day the relative risk was 0.83 (95% CI: 0.69–0.99) in men and 0.72 (95%3CI: 0.54–0.96) in women. A maximum reduction (RR = 0.80; 95% CI: 0.57–1.12 in men and RR = 0.66; 95% CI: 0.39–1.12 in women) was predicted in both groups at 18–24 grams of alcohol per day, but statistical significance was only reached up to the amount of 12 grams of alcohol per day.

On the contrary, an earlier meta-analysis[416] had found a lower protective effect of alcohol consumption against coronary artery disease risk in women.

Thus, a possible sex difference in the protective effect of alcohol on vascular risk might exist and explain some seemingly controversial results in different epidemiological studies.[417]

Finally, in a meta-analysis on alcohol and total mortality[408] the dose-response curves are similar for both sexes at low alcohol intake (up to 10 grams per day), but they differ with increasing intake; in fact, the protection in women disappears at a dose consistently lower than in men (18 vs 38 grams of alcohol per day, in men and women, respectively).

Additional studies are needed to test whether women benefit more from alcohol than men or whether they, in practice, drink lower amounts on average, thereby maximising the benefit.

1.3.8.5 Alcohol consumption and cardiovascular secondary prevention

Promoting the secondary prevention of cardiovascular events in survivors of primary vascular events, as well as in hypertensive or diabetic patients, is important.[418,419] Among factors implicated in secondary prevention, a substantial benefit can come from more physical activity and a better diet.

A possible association between alcohol consumption and secondary events (cardiovascular and all-cause mortality) in patients with cardiovascular disease has been recently investigated.[412,419] A meta-analysis relating to cardiovascular mortality showed a J-shaped pooled curve with a significant maximal protection (average 22%) by alcohol at about 26 grams/day. Similarly, a meta-analysis on mortality from any cause, also showed a J-shaped pooled curve (average maximal protection 18% at 7 grams/day).[410] So in patients with cardiovascular disease, light to moderate alcohol consumption (5-25 grams/day) was significantly associated with a lower incidence of both recurrent cardiovascular events and all-cause mortality.

Guidelines for the management of hypertension[420] recommend avoiding binge drinking, and suggest regular alcohol consumption limited to no more than 2-3 drinks/day for men and 1-2 drinks/day for women, if not total abstention. A recent analysis investigating whether reducing alcohol consumption lowered blood pressure without losing the cardiovascular benefits of drinking in moderation,[421] concluded that "the hypertensive patient over the age of 60 who drinks over 16 drinks per week should be advised to reduce his or her alcohol intake, but a daily drink may be advisable and the patient should not stop drinking entirely; it is not suggested that the non-drinker should start drinking".

Diabetic patients have a CHD risk two to four times higher than non-diabetics. A meta-analysis of 15 prospective cohort studies showed a J-shaped relation between alcohol consumption and risk of developing diabetes, with a 30% lower risk in moderate alcohol consumers (1-2 drinks/day).[422] Moreover, moderate alcohol consumption was associated with a lower incidence of heart disease or total mortality in patients with type 2 diabetes.[423,424] Obviously, the decrease in cardiovascular disease risk associated with moderate alcohol consumption in hypertensive or diabetic subjects does not reduce the importance of controlling blood pressure or blood glucose, regardless of drinking habits.

1.3.8.6 Detrimental effects of alcohol consumption: abuse and binge drinking

Abuse of alcohol, binge drinking, and drinking outside meals have all been associated with detrimental effects including foetal alcohol syndrome, liver cirrhosis, pancreatitis, certain cancers, cardiomyopathy, hypertriglyceridaemia, hypertension, haemorrhagic stroke, overweight, alcohol intoxication and addiction.[404,425]

Excess alcohol intake and binge drinking must be avoided by everybody, but in patients with CVD, it can have more serious unhealthy consequences, such as exacerbation of existing pathological conditions. Alcohol abuse or binge drinking are major causes of hyperlipidaemia, vasoconstriction, increased clotting activity and a lower threshold for ventricular fibrillation.[405,411]

A recent meta-analysis found that both binge and heavy drinking are associated with excess CHD risk.[426] Compared with regular drinking, binge drinking (defined in this meta-analysis as the consumption of three or more drinks within one to two hours) doubles total and cardiovascular mortality risk in cardiovascular patients.[419]

Episodic heavy alcohol drinking is reportedly associated with risk of atrial fibrillation (AF). However, recent studies failed to obtain evidence that chronic alcohol intake (especially moderate consumption) was an important factor in the development of AF; moreover, no association of alcohol consumption with risk of death was found among subjects with AF.[404,427]

Few studies have investigated whether drinking with or without meals modifies the negative association between moderate alcohol consumption and CVD or total mortality. Drinking out of mealtimes is considered to be associated with increased CHD and hypertension risk, independently of the amounts drunk.[419,428]

Moderate regular drinking, possibly during meals, appears as the ideal behavior to get the optimal cardiovascular benefit of alcohol, while excess or binge drinking is to be absolutely avoided.

1.3.8.7 The influence of age

A small number of prospective studies explored the influence of age on the relationship between alcohol consumption and mortality. In the majority of these studies, the J-shaped curve was confirmed in elderly cohorts, while a positive relation was found for women up to 44 years and for men up to 35 years. In young men the curve was steeper than in young women, and the reduction in mortality in the lightest drinkers was larger and was maintained up to higher levels of consumption than for women.[429] As causes of death have different incidence throughout the age categories, the effect of alcohol on health and disease may differ in different life periods.[430] The reduced mortality risk in light/ moderate drinkers is substantially due to reduced coronary heart disease. However, in the young cohorts cardiovascular events or deaths are relatively low while car accidents or violence are prevalent, so a possible alcohol protection is quite difficult to detect in the general population under 40.

1.3.8.8 What is the optimal amount of alcohol intake?

A J-shaped relationship between total mortality and increasing amounts of alcohol consumed was observed in several meta-analyses, including altogether more than one million people and hundred of thousands fatal events, showing that low to moderate consumption of alcohol (no more than one drink/day in women and two drinks/day in men) significantly reduces total mortality, while higher doses increase it.

Official definitions of standard "drinks" or "units" vary between countries (see Table 10, below). The most commonly favoured definition—also used by WHO—is 10 g of alcohol (ethanol) per drink, but definitions vary from 8 to 14 grams of pure ethanol in a standard drink.[431] A 250 ml glass (half a pint) of beer (5% alcohol by volume), a 100 ml glass of wine (12% ABV) or a 30 ml measure of spirits (40% ABV) contains 10 g of alcohol. Larger measures or stronger drinks clearly contain more alcohol—a large glass (175 ml) of 14% wine, for example, contains almost 20 g of alcohol.

Table 10 Official definitions of standard drinks or units

Standard drink (g ethanol)	Country
8 (expressed as "units" rather than drinks)	United Kingdom
9.9	Netherlands
10	Australia, France, Hungary, Ireland, New Zealand, Poland, Spain
11	Finland
12	Denmark, Italy, South Africa
14	United States

Source: International Center for Alcohol Policies. ICAP http://www.icap.org/PolicyIssues/DrinkingGuidelines/StandardDrinks/tabid/126/Default.aspx

1.3.8.9 Conclusions

The rates of fatal and non-fatal cardiovascular events are significantly lower for people who drink low to moderate amounts of alcohol than for people who do not drink at all or drink heavily.

Available epidemiologic data—derived at present from a large number of prospective observational studies—confirm the hazards of excess drinking, but also indicate the existence of potential windows of alcohol intake which may confer a net beneficial effect of drinking, in terms of survival, both in men and in women. Moderate alcohol consumption is a traditional component of the Mediterranean diet, and largely contributes to the "adherence to Mediterranean diet scores" frequently used as predictors of lower mortality.

The main message for an adult general population can be summarised as follows: Heavy or binge drinkers should be urged to cut and modify their consumption. There is no reason to discourage people who already regularly consume small to moderate amounts of alcohol—no more than one drink (10 g) per day for women or two drinks per day (20 g of alcohol) for men—from continuing. A similar message can be addressed to patients who already suffered a cardiovascular event.

Although low-moderate, non-binging alcohol consumption—in the absence of contraindications and in the context of healthy eating and a healthy lifestyle—reduces the risk of coronary heart disease, it is not recommended that adult abstainers begin drinking. This is because even moderate alcohol intake is also associated with increased risk of other harm (including breast cancer, violence, accidental death or injury and road traffic accidents). Furthermore, there is a risk that some of those who start to drink will be drawn into consuming more than the very limited amounts of alcohol associated

with decreased cardiovascular risk. For these reasons it is not recommended that anyone be advised to drink more frequently on health grounds.

The overall evidence also suggests a reduced risk of ischaemic stroke in light-moderate drinkers, but little or no protection against haemorrhagic stroke. Cerebrovascular events substantially increase in heavy alcohol and binge consumers: moreover, heavy alcohol consumption is a risk factor for both haemorrhagic and ischaemic stroke in young adults.[432,433]

The overall message is that, if alcohol is consumed at all, it should be limited to not more than two drinks a day (20 g) in men and not more than one drink (10 g) per day in women.

Randomised controlled trials are considered to offer a more solid answer than observational studies to many questions in medicine, but intervention trials on diet in general and on alcohol in particular, are difficult, mainly because of selection, blinding or compliance problems and ethically questionable to perform.[434] Ideally, a large randomised controlled trial on alcohol, comparing a reference group of long-term abstainers, excluding former or occasional drinkers and a comparable "intervention" group of light to moderate no-binging drinkers, with appropriate follow-up, collecting not only vascular but total mortality data too, would help in establishing the effect of alcohol on health. Randomised controlled trials on intermediate or surrogate endpoints could represent a more feasible and likely alternative.

1.3.9 Folate

This review is contributed by Professor Helene McNulty RD and Dr Mary Ward RD from the University of Ulster, Northern Ireland.

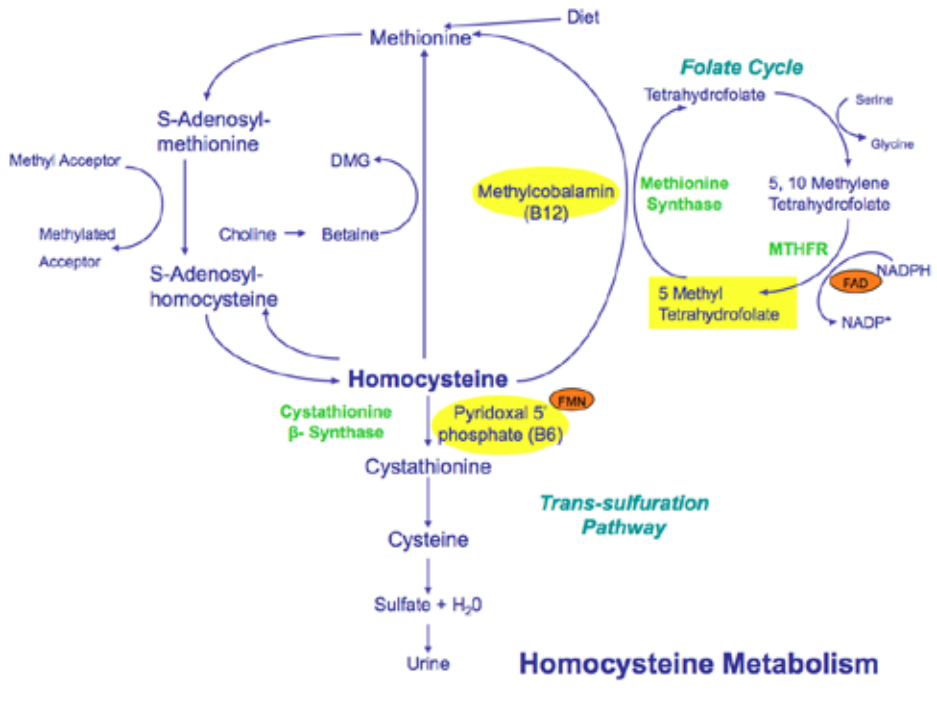
1.3.9.1 Summary

In recent years there has been much interest in the potential protective effect of the B-vitamin folate in cardiovascular disease. Such an effect may or may not be mediated via the role of folate (and the metabolically related vitamins B12, B6 and B2) in maintaining healthy plasma homocysteine concentrations. A number of recent secondary prevention trials in at-risk patients have, however, failed to show a benefit of homocysteine-lowering therapy on CVD events generally. Given that all of these trials were performed in patients with well established pathology, current evidence appears to suggest that the administration of high dose folic acid (with or without related B-vitamins) to CVD patients is of no benefit in preventing another event, at least in the case of heart disease. The evidence supporting a beneficial role for those B-vitamins at this time is however somewhat stronger for stroke, with one meta-analysis of randomised trials showing that homocysteine-lowering with folic acid reduced the risk of stroke by 18% and by an overall 25% in patients with no history of stroke (but with pre-existing cardiovascular or renal diseases). The decline in stroke-related mortality in the North American population that relates to the timing of introduction of mandatory folic acid fortification, adds further support to the potential benefit of enhancing folate status and/or lowering homocysteine in the prevention of stroke. Evidence supporting a causal relationship between elevated homocysteine (or sub-optimal folate) and CVD also comes from genetic studies. The most important genetic determinant of homocysteine in the general population is the common 677C→T variant in the gene encoding for the folate-metabolising enzyme methylenetetrahydrofolate reductase (MTHFR). People homozygous for this polymorphism (TT genotype)—about 10% of populations worldwide—typically have higher plasma homocysteine levels and a 14 to 21% higher risk of CVD. Apart from folate, riboflavin (vitamin B2) is required as a co-factor for MTHFR, and has a major modulating effect on the expected (high homocysteine) phenotype in people with the TT genotype. New evidence shows that riboflavin intervention results in marked lowering of blood pressure specifically in patients with the TT genotype, an effect that may be independent of the homocysteine-lowering effect of riboflavin also seen only in individuals with the TT genotype. Of note, the fact that these effects of riboflavin are achievable with intervention at dietary levels, suggests that there may be important implications for dietary riboflavin requirements specifically in individuals with this common genetic variant in folate metabolism. The latest evidence linking folate and related B-vitamins to CVD, the relevant gene-nutrient interactions and the practical considerations in relation to achieving optimal B-vitamin status aimed at preventing CVD will be considered.

1.3.9.2 Introduction

This section considers the latest evidence in relation to the potential roles played by folate and related B-vitamins in preventing cardiovascular disease and relevant gene-nutrient interactions that appear to be implicated. These effects may be mediated via homocysteine, the metabolism of which requires an adequate status of up to four B-vitamins (Figure 17). Plasma homocysteine is very responsive to intervention with the relevant B-vitamins, in particular folate[435] but also (albeit to a lesser extent) vitamins B12,[436] B6[437] and B2 (riboflavin).[438] Although the literature in this area tends to focus on plasma homocysteine as the cardiovascular risk factor, it is also possible that folate and related B-vitamins have roles in CVD that are independent of their effects on homocysteine and that elevated plasma homocysteine may merely be a marker of sub-optimal B-vitamin status.

FIGURE 17 HOMOCYSTEINE METABOLISM SHOWING ROLE OF FOLATE AND RELATED B-VITAMINS



1.3.9.3 Suboptimal folate status

Folate is required for one-carbon metabolism, involving the transfer of one-carbon units for DNA and RNA biosynthesis, methylation reactions and amino acid metabolism. Classically folate deficiency results in megaloblastic anaemia. Although this is relatively rare in the absence of an underlying clinical cause, many otherwise healthy populations in Europe and elsewhere have inadequate folate intakes when considered from the perspective of achieving an optimal folate status (a status associated with lowest risk of folate-related disease), rather than merely preventing overt folate deficiency (megaloblastic anaemia).[439] On the basis that normal homocysteine metabolism requires an adequate supply of folate, the measurement of plasma homocysteine concentration provides a reliable functional marker of folate status. When folate status is sub-optimal, plasma homocysteine concentration is invariably found to be elevated and supplementation with folic acid can lower homocysteine levels in healthy and diseased populations by about 25%.[435]

1.3.9.4 Latest evidence linking low folate/elevated homocysteine with CVD

1.3.9.4.1 Evidence from population studies and randomised trials

There is much evidence over the past 15 years to link low folate status and/or elevated plasma homocysteine with an increased risk of CVD. In 2002, two major meta-analyses of numerous retrospective and prospective studies predicted that lowering homocysteine by 3µmol/l (or by about 25% based on typical homocysteine concentrations in Western populations of 12µmol/l) would reduce the risk of heart disease by 11-16% and stroke by 19-24%.[440,441] However, since 2004, a number of secondary prevention trials in at-risk patients published in high profile journals failed to demonstrate a benefit of homocysteine-lowering therapy on CVD events generally.[442-446] The most recent of these, the SEARCH trial, again found no significant effects of combined folic acid and vitamin B12 on major coronary events (RR 1.05 95%, CI 0.97-1.13) despite the large sample size (n=12064).[446] Thus the balance of evidence suggests that the administration of high dose B-vitamins to CVD patients with established disease is of little/no benefit in preventing another event, particularly in the case of heart disease. One of the aforementioned negative trials, however, did show a clear benefit of B-vitamin intervention in reducing the risk of stroke but for some reason the authors largely overlooked this result in their original report.[443] Instead, the beneficial effect of B-vitamins in relation to stroke risk were separately reported three years after the original report.[448] Likewise, a recent meta-analysis of 13 randomised trials by Lee and colleagues, predominantly concerning secondary prevention, found a mild beneficial effect of folic acid in reducing the overall risk of stroke but this did not reach statistical significance when all 13 trials were considered together (RR 0.93, 95% CI 0.85-1.03; P=0.16).[449] However, another meta-analysis of randomised trials considered stroke events separately in patients with a

history of stroke and in patients without a history of stroke but with pre-existing cardiovascular or renal diseases. This meta-analysis showed that homocysteine-lowering by folic acid reduced the risk of stroke by an overall 18%, but to a much greater extent (by 25%) in patients with no history of stroke (RR 0.75, 95% 0.62-0.90; P=0.002).[450] Consistent with the idea that folic acid may have a beneficial effect in preventing stroke (though by no means conclusive evidence) are population data from North America (1990-2002), showing an improvement in stroke mortality that relates to the timing of introduction of mandatory folic acid fortification, but no corresponding improvement over the same time period in the UK where no such fortification policy was put in place.[451] Thus current evidence to support the case for folic acid/homocysteine-lowering in CVD is stronger for stroke than for heart disease.

1.3.9.4.2 Evidence from genetic studies

Other evidence for a causal relationship between low folate status/elevated homocysteine and CVD comes from genetic studies. The most important genetic determinant of homocysteine in the general population is the common 677C→T variant in the gene encoding the folate-metabolising enzyme methylenetetrahydrofolate reductase (MTHFR) required for the formation of 5-methyltetrahydrofolate, which in turn is required to convert homocysteine to methionine. People who are homozygous for this polymorphism (TT genotype), the reported frequency of which varies from 3% to 32% in populations worldwide,[452] have reduced MTHFR activity (resulting in impaired folate metabolism) and higher homocysteine concentrations *in vivo*;[453] an effect which is found to be particularly marked in people with low folate intake.[454] Importantly, evidence from various meta-analyses involving over 25,000 cases shows that individuals with the MTHFR 677 TT genotype have a significantly higher risk of CVD (by 14 to 21%) compared to those without this genetic factor.[441,455,456] There does, however, appear to be a large geographical variation in the extent of excess cardiovascular disease risk associated with this polymorphism. Studies that have examined this variation show that the excess risk of coronary heart disease is not significant in North American populations, while the polymorphism carries a significantly increased (but variable) risk of coronary heart disease elsewhere in the world including in European populations.[455,456]

Although largely overlooked, riboflavin (vitamin B2) is required as a co-factor for the MTHFR enzyme and therefore is needed to generate 5-methyltetrahydrofolate. Evidence shows that enhancing riboflavin status (through intervention with low-dose riboflavin) results in a marked lowering in homocysteine specifically in people with the MTHFR 677 TT genotype.[438] Presumably this effect of riboflavin works by neutralising the variant form of the enzyme which is reported to become inactive as a result of having an increased propensity to dissociate from its FAD (riboflavin) cofactor.[457] Thus ensuring optimal riboflavin intake and status may be important in modulating the risk of CVD specifically in people who have the TT genotype, i.e. 10% of Europeans generally, and much higher in certain sub-populations such as southern Italy where the reported frequency is as high as 26%.[452]

1.3.9.4.3 Folate related B-vitamins and blood pressure

Hypertension (i.e a blood pressure of 140/90mmHg or greater) is estimated to carry an almost three-fold greater risk of developing CVD,[458] while treating hypertension significantly reduces cardiovascular events, and stroke in particular.[459] Despite the significant associations that are observed between homocysteine and blood pressure in observational studies, intervention studies to lower homocysteine have shown little or no corresponding blood pressure response, suggesting that there is no causative relationship between elevated homocysteine concentrations *per se* and high blood pressure.[460] However, most published studies in this area have not considered the role of the MTHFR 677C→T polymorphism, but evidence is now emerging showing an important association between this common genetic factor and hypertension.[460] Moreover, evidence just published shows for the first time that riboflavin is an important determinant of blood pressure specifically in individuals with the TT genotype.[461] Of greater note, riboflavin intervention (at dietary levels, 1.6 mg/d) was shown to result in marked lowering of blood pressure in CVD patients with the TT genotype (from 144/87 to 131/80mmHg), with no response observed in the other MTHFR genotype groups.[461] Thus riboflavin is effective in reducing blood pressure specifically in patients with the MTHFR 677 TT genotype. There may be important implications of these findings for the prevention and treatment of hypertension among the 4-26% of Europeans[452] who carry this genotype.

The precise mechanism linking the MTHFR 677C→T polymorphism, riboflavin and blood pressure is as yet unclear, although it is possible that nitric oxide (NO), a potent vasodilator, may be implicated. Vascular concentrations of 5-methyltetrahydrofolate were recently shown to be important in regulating NO and endothelial function in patients with the MTHFR 677-TT genotype.[462] By stabilising the variant MTHFR enzyme, riboflavin supplementation may restore 5-methyltetrahydrofolate levels in vascular cells, improve NO bioavailability and in turn lower blood pressure specifically in patients with the MTHFR 677 TT genotype.

This novel genotype-specific effect of riboflavin on blood pressure may also partly explain the inconsistencies in the evidence as to the role of this common polymorphism in cardiovascular disease generally.[455,456] The policy of population-wide riboflavin fortification of food that has existed for over 50 years in North America would ensure higher and less variable intakes of riboflavin (compared with European populations), which could in turn be predicted to neutralise any phenotypic effect of the variant MTHFR enzyme. Thus differences in prevailing nutrient intakes in different regions could result in this polymorphism carrying an increased risk of CVD in Europe, but not in North America.[455,456]

1.3.9.5 Public health considerations in relation to folate and related B-vitamins

In practice achieving an optimal status of the B-vitamins folate, vitamin B12 and riboflavin can present particular challenges.

1.3.9.5.1 Achieving optimal folate status

Achieving optimal folate status in a population is a major challenge because of the poor stability and poor bioavailability of folate from natural food sources (e.g. green leafy vegetables, green beans, liver) compared with folic acid (the synthetic form of the vitamin found in fortified foods and supplements).[437] Thus there is a particular challenge for certain European populations with limited or no access to fortified foods where consumers are dependent on natural food folate sources as a means to optimise status. The decline in stroke-related mortality in the US and Canada relating to the introduction of mandatory fortification of food with folic acid[431] suggests that, although primarily aimed at preventing neural tube defects, folic acid fortification may also play a role in the primary prevention of stroke. However, there are also safety concerns regarding long-term exposure to high dose folic acid. Traditionally these involved the potential risk that high folic acid intake might mask the anaemia of vitamin B12 deficiency while allowing the associated irreversible neurological symptoms to progress,[463] but more recently the new concern was raised that it may be associated with an increased risk of cognitive impairment in older people with low vitamin B12 status.[464] Furthermore, despite considerable evidence that folate within the dietary range plays a protective role against various cancers,[439] recent evidence has raised the concern that high dose folic acid (1 mg/d) may promote colorectal tumorigenesis in patients with pre-existing lesions[465] or significantly increase the risk of prostate cancer in men.[466] Because of these concerns, many governments worldwide have delayed decisions to implement population-based folic acid fortification policies as in the United States and Canada.

Thus mandatory (population-wide) folic acid fortification remains very controversial. However alternative strategies to increase folate status through health promotion and educational campaigns have generally been found to be ineffective, even in the case of preventing neural tube defects where the evidence for a beneficial effect is beyond any doubt.[437] Optimising folate should be a priority for public health, but will only be achieved with levels of intake of the vitamin greater than those currently provided by a typical diet as eaten in most European countries.

1.3.9.5.2 Achieving optimal vitamin B12 status

Dietary B12 is provided by animal foods (e.g. meat, fish, eggs, milk, cheese) and B12 intakes are generally found to be good in most healthy populations (often greatly exceeding recommended values). However, the achievement of an optimal status may present a particular difficulty for many older people because of the common problem of age-related food-bound B12 malabsorption estimated to effect up to 30% of otherwise healthy older people.[437,467] This arises mainly from atrophic gastritis, a chronic inflammatory condition resulting in decreased gastric acid production (hypochlorhydria), which diminishes B12 absorption because of the essential role of gastric acid in the release of protein-bound B12 from food. Unlike the treatment required for the classical (but rare) B12 deficiency symptom of pernicious anaemia involving B12 injections for life, older adults with low B12 status due to food-bound malabsorption should be able to absorb free (crystalline) vitamin B12 because it is not bound to protein. In fact, on the basis of this assumption, the Institute of Medicine in the United States recommends that people aged 50 years and over consume most of their vitamin B12 from crystalline B12 found in fortified foods and supplements. No such recommendation exists at this time in any European country.

Vitamin B12 is metabolically closely related to folate and, like folate, is also required for the remethylation of homocysteine to methionine. Thus achieving optimal folate status only may not lower homocysteine to desirable levels (<10μmol/l),[468] and optimising vitamin B12 may have benefits over and above the effect of folate. A further 7% reduction in homocysteine can be achieved with vitamin B12 additional to that with folic acid alone.[435] Any small additional decrease in homocysteine could be predicted to confer a further benefit in terms of cardiovascular risk given that the relationship with homocysteine is a graded one. Of note, low biomarker status and functional deficiency of vitamin B12 (including B12-related elevated homocysteine) was found to be particularly prevalent in Asian Indians (even among non-vegetarians).[469] Thus Asian immigrants may be a vulnerable sub-group within Europe in relation to vitamin B12 status.

1.3.9.5.3 Achieving optimal riboflavin status

Riboflavin is provided in the diet predominantly through consuming milk and dairy foods. In the UK, the Scientific Advisory Committee on Nutrition has expressed particular concern about the high proportion of the British adult population (and particularly younger women) with apparently poor riboflavin status as determined from national survey data using the gold standard biomarker of status, erythrocyte glutathione activation coefficient (EGRac).[470] In general, dietary intakes of participants were found to compare favourably with recommended values with the exception of young women where high proportions were found to have low intakes.[470] Whether there is a general problem of poor riboflavin status in the UK and other European populations, as indicated by the large proportion with abnormal EGRac values, is unclear at this time, and requires further investigation. However, the new findings discussed above, that the higher blood pressure in

patients with a common genetic variant in folate metabolism is responsive to low-dose riboflavin,[461] raises the possibility that improving riboflavin status may have an important role in preventing hypertension among these genetically at-risk people.

1.3.9.6 Conclusions

There is little evidence to support a beneficial role for supplemental folic acid in preventing further disease in patients with existing CVD. There remains interest in the question of whether optimal folate status and/or maintenance of lower homocysteine will have a role in the primary prevention of stroke, but further research is needed to establish this. Preliminary evidence indicates that optimising riboflavin intake may have a role in preventing hypertension specifically in people with a common genetic variant in folate metabolism, but this needs to be confirmed in much larger trials.

An adequate B-vitamin status can be achieved with a heart healthy diet that includes whole grain foods, leafy green vegetables, whole grain foods, lean meat and low fat dairy produce. In practice, however, achieving an optimal status of the B-vitamins folate, vitamin B12 and riboflavin, can be problematic for some groups. In the case of folate, poor stability and poor bioavailability of the natural vitamin from food sources means the typical diet of most Europeans is sub-optimal in folate. In the case of vitamin B12, although dietary intakes are generally more than adequate, the achievement of an optimal status presents a particular difficulty for many older people because of the common problem of age-related food-bound B12 malabsorption. People of Asian origin, particularly those on a vegetarian diet, may have low vitamin B12 intakes and may also be at risk of low riboflavin status because of the avoidance of milk. In order to offer maximal protection against elevated homocysteine in all individuals, including those genetically predisposed to impaired folate metabolism, an optimal status of all four relevant B-vitamins should be ensured but this remains challenging in practice.

1.3.10 Antioxidants (including polyphenols)

The review on polyphenols and cardiovascular disease is contributed by Dr Riitta Törrönen, University of Eastern Finland, Finland. The introduction to this section and the short summary on antioxidant vitamins contains some material contributed by Dr Vardis Dilis of the Hellenic Health Foundation in Athens.

Although essential for aerobic life, oxygen is also thought to be implicated in the process of ageing and to contribute towards the development of various degenerative diseases. The latter role is attributed to numerous oxidation reactions occurring in the body, resulting in a chronic accumulation of oxidative damage.

"Reactive species" include "free radicals" as well as other oxidizing agents that may easily be transformed into free radicals. Free radicals include oxygen, nitrogen or chlorine compounds and may be formed as natural products of metabolism, or can derive from exogenous sources. Although very important in neural transmission and cell signaling, free radicals are very reactive by possessing uncoupled electrons and may therefore damage other molecules. On the contrary, "antioxidants" are compounds that prevent the oxidation of susceptible molecules and can be produced endogenously (e.g. superoxide dismutase, catalase, uric acid) or can be provided through the diet (e.g. vitamins C and E, flavonoids). Disruption of the balance between free radical formation and their clearance by antioxidants leads to oxidative stress, a state that brings about excessive damage to biological molecules (proteins, lipids and DNA). There is evidence that the oxidation of low-density lipoprotein (LDL) is a key process implicated in the development of atherosclerosis, while the accumulation of DNA damage is thought to be a crucial step in carcinogenesis.

Dietary antioxidants fall into several groups, two of the main being antioxidant vitamins and polyphenols (including flavonoids). The following section on antioxidant vitamins has not been based on a specially commissioned scientific review. The section on polyphenols (Section 1.3.10.2) is based on a specially commissioned scientific review.

1.3.10.1 Antioxidant vitamins

There has been a longstanding theory that the antioxidant vitamins—E, C and beta carotene (a form of vitamin A)—may be protective against cardiovascular disease and may contribute to the protective effects of fruits, vegetables and the Mediterranean diet. While observational data comparing the health of people on antioxidant-rich diets with those with lower antioxidant intakes give support to this hypothesis, the results of clinical studies of high doses of antioxidant supplements to see if it works in practice have been contradictory. A 2009 review article reported that a total of nine primary and 11 secondary prevention trials (involving a total of 150,000 and 60,000 participants respectively) have been disappointing.[471]

Further research is needed to achieve a consensus on the associations between oxidation and chronic disease, and the extent to which antioxidants may reduce oxidative stress and protect health.

On the basis of the theoretical health benefits of antioxidants, antioxidant-rich foods and dietary supplements are today promoted by the industry and some health professionals as carriers of beneficial nutritional properties that may protect against premature aging. The European Food Safety Agency (EFSA) has recently reviewed a number of health claims in relation to vitamins A, C and E. The only one of the submitted claims directly related to cardiovascular health ("vitamin E can neutralise free radicals and help maintain a healthy heart") was rejected.[472] In contrast, claims that vitamin C and vitamin E can "protect DNA, proteins and lipids from oxidative damage" were approved.[470,473] EFSA rejected more general claims for "antioxidant activity", "antioxidant content", and "antioxidant properties".[474]

1.3.10.2 Polyphenols

1.3.10.2.1 Introduction

Polyphenols are phenolic phytochemicals widely distributed in plants, and are also present in significant amounts in a wide range of plant-derived foods and beverages. They constitute a very diverse group of secondary metabolites synthesised by plants and are ubiquitous throughout the plant kingdom. Several thousand molecules having a polyphenolic structure (i.e. several hydroxyl groups on aromatic rings) have been identified in higher plants, and several hundred are found in edible plants.[475] They are divided into several classes and subclasses according to their chemical structure. Flavonoids, phenolic acid, tannins, lignans and stilbenes are the most common polyphenolic classes in plant foods. Flavonoids are further classified as flavonols, flavones, flavanols (flavan-3-ols, catechins), flavanones, anthocyanidins and isoflavones (Table 11). Proanthocyanidins (also called condensed tannins) are oligomers and polymers of flavanols.

Table 11 Dietary flavonoids and examples of food sources

Flavonoid subclass	Food sources
Flavonols	apple, beans, berries, broccoli, kale, onion, red wine, tea, tomato
Flavones	celery, parsley
Flavanones	citrus fruit
Flavanols (catechins)	apple, berries, cocoa or dark chocolate, grape, red wine, tea
Proanthocyanidins	apple, berries, cocoa or dark chocolate, grape, red wine
Anthocyanidins	berries, cherry, plum, red cabbage, red onion, red grape, red wine
Isoflavones	soybean, soy foods, soy protein isolate

Polyphenols are an integral part of the human diet, and have been linked to improved human health through reduced risk of chronic diseases, especially cardiovascular disease. Unlike vitamins or other essential nutrients, polyphenols are not required for growth and development and for maintaining vital body functions throughout life, and do not cause classical deficiencies. However, they may be essential for maintaining body functions and health through the adult and later phases of life. Recently, they have been discussed as 'lifespan essentials' because they may increase the chance of reaching the full genetically determined lifespan and the quality of life during aging by reducing the incidence of chronic, age-related diseases.[476,477]

Polyphenols are the most abundant dietary antioxidants, with total intake of nearly 1 g/day, which is much higher than that of all other classes of phytochemicals and known dietary antioxidants. The intake of polyphenols is approximately 10 times higher than that of vitamin C and 100 times higher than that of vitamin E and carotenoids.[478]

Research on dietary flavonoids and other polyphenols, their bioactive properties, and their role in the prevention of chronic diseases truly began after 1995.[478] The diversity and complexity of their chemical structures as well as differences in bioavailability have delayed and complicated the research on their health benefits. The antioxidant properties of polyphenols have been extensively studied. However, the mechanisms of action go beyond radical scavenging or antioxidant functions. Polyphenols affect cell metabolism, physiology and health also through regulation of gene expression and interactions with receptors, enzymes and other proteins.

Today, there is an enormous amount of scientific literature on the potential cardioprotective properties of polyphenols, including data from *in vitro* studies, animal experiments and human intervention trials as well as prospective epidemiological studies. The data available supports the concept that certain polyphenols in the diet, especially flavonoids, may be associated with benefits on cardiovascular health.[479-481] Proposed mechanisms are presented in Table 12. However, it is important to understand that, in this relatively new area of investigation, the research that has been done to-date has limitations. Many of the studies on polyphenols are relatively small in size and short-term in nature. In addition, they have often measured intermediate end-points rather than health outcomes.

Table 12 Proposed mechanisms by which polyphenols may reduce the risk of CVD

Oxidative stress
• scavenge reactive oxygen and nitrogen species
• chelate redox-active transition metal ions
• spare and interact with other antioxidants
• inhibit redox-sensitive transcription factors
• inhibit pro-oxidant enzymes
• induce antioxidant enzymes
Growth of atherosclerotic plaque
• reduce adhesion molecule expression
• reduce inflammation
• reduce the capacity of macrophages to oxidatively modify LDL
Platelet function and haemostasis
• inhibit platelet aggregation
Blood pressure and vascular reactivity
• promote nitric oxide-induced endothelial relaxation
Plasma lipids and lipoproteins
• reduce plasma cholesterol and triglycerides

From: Manach et al. 2005[479]

This review provides a summary of the current scientific evidence on the effects of dietary polyphenols on CVD, based on recent critical reviews and meta-analyses of epidemiological studies and randomised controlled human trials (RCTs). In some cases, also original studies are cited. To date, the evidence is largely limited to certain groups of flavonoids and to foods or beverages rich in flavonoids, such as cocoa products, black and green tea, red wine, and soy products. Less evidence is currently available on other important dietary sources of polyphenols, such as fruits and berries.

1.3.10.2.2 Flavonoids and CVD

Prospective studies

Arts and Hollman[482] reviewed 12 prospective cohort studies on flavonoid intake and the risk of coronary heart disease (CHD), focusing on the flavonoid subclasses of flavonols, flavones and flavanols (catechins). Seven of the studies found protective effects of flavonols and flavones or of flavanols with respect to fatal and nonfatal CHD, and reductions of mortality were up to 65%. A similar result was obtained also in a 2007 systematic review conducted by Erdman and colleagues.[480]

A meta-analysis of prospective cohort studies indicated that intake of flavonols was inversely associated with non-fatal and fatal stroke.[483] Data from six cohorts involved 111,067 subjects with at least 2,155 non-fatal and fatal cases, and the persons were followed for from six to 28 years. This meta-analysis showed that a high intake of flavonols compared with a low intake was associated with a 20% lower risk of stroke incidence. It was concluded that flavonols may reduce stroke risk.

A large prospective study of 34,489 postmenopausal women with 16 years of follow-up investigated the associations between intakes of total flavonoids or seven subclasses of flavonoids and cardiovascular mortality.[484] Significant inverse associations were observed between anthocyanidin intake and CHD, CVD, and total mortality, between flavanone intake and CHD mortality, and between flavone intake and total mortality. No association was found between flavonoid intake and stroke mortality.

Randomised Controlled Trials (RCTs)

Erdman and colleaguesd[480] reviewed eight human intervention studies examining the effects of flavonols on markers for CHD. These studies used pure quercetin aglycone or glycosides, onions, or a variety of flavonol-rich foods. Duration of the interventions varied from one to 42 days, and the number of subjects from six to 19. The daily dose of quercetin equivalents varied from 20 to 250 mg. Favourable effects were found on antioxidant biomarkers. Plasma lipid profiles were not affected by flavonol supplements. The authors also reviewed studies on other flavonoids and concluded that the clinical evidence is encouraging but not sufficient to support a role of dietary flavonoids in reducing the risk of CVD.

The effect of oral isoflavone supplementation on endothelial function in postmenopausal women has been investigated in many studies. A meta-analysis of nine RCTs (including 525 subjects) showed that oral isoflavone supplementation did not improve endothelial function (measured as flow-mediated dilation, FMD) in postmenopausal women with high baseline FMD levels but led to significant improvement in women with low baseline FMD levels.[485] In these trials, the source of isoflavone was soy protein (six trials) or tablets

(three trials), the doses of isoflavone ranged from 50 to 99 mg/day, and the duration of supplementation ranged from two weeks to 12 months. The result of this meta-analysis suggests that oral isoflavone supplementation should be applied to targeted subjects (with baseline FMD levels <5.2%), but not to all postmenopausal women.

1.3.10.2.3 Flavonoid-rich foods and CVD

1.3.10.2.3.1 Cocoa products

Chocolate and other cocoa products are made from beans of *Theobroma cacao*. Cocoa beans contain approximately 6-8% polyphenols by dry weight.[486] Cocoa is particularly rich in the flavanols epicatechin, catechin, and proanthocyanidins (oligomers of epicatechin and catechin).

Prospective studies

There are no meta-analyses available on epidemiological evidence relating to consumption of cocoa products and cardiovascular health. Therefore, four recent studies examining the association of cocoa consumption with blood pressure (BP) and CVD risk are cited here.

The association of cocoa intake with BP and cardiovascular mortality was assessed in a Dutch cohort of 470 elderly men.[487] Men with habitual intakes of 4.2 g of cocoa per day (equal to 10 g of dark chocolate) had a 3.7 mmHg lower systolic BP and a 2.1 mmHg lower diastolic BP than non-consumers of cocoa. During 15 years of follow-up, cocoa intake was associated with 50% lower risk of cardiovascular and all-cause mortality.

The association of chocolate consumption with BP and the incidence of CVD (myocardial infarction and stroke) was investigated in a German cohort of 19 357 middle-aged men and women.[488] Systolic BP was 1.0 mmHg and diastolic BP 0.9 mmHg lower in the top quartile compared with the bottom quartile of chocolate consumption. After a mean follow-up of eight years, an increase in chocolate consumption of 6 g per day was associated with a 39% lower risk of the combined outcome of myocardial infarction (MI) and stroke. The inverse relation was stronger for stroke than for MI. Baseline BP explained 12% of the lower risk. Chocolate consumption had a strong inverse association with cardiac mortality also in a Swedish cohort of 1,169 patients surviving their first acute myocardial infarction.[489]

In addition to these European cohorts, the association between chocolate consumption and CVD has been evaluated in a large U.S. cohort of 34,489 postmenopausal women with a 16 year follow-up.[484] This study found a borderline significant inverse association between chocolate intake and CVD mortality.

RCTs

There are several recent reviews and meta-analyses on the effects of cocoa products on cardiovascular health[481,490-492] and particularly on blood pressure.[493-495] In addition to BP, cardiovascular risk factors such as endothelial function, blood lipids, and platelet function have been investigated within these RCTs.

In their meta-analysis of the effectiveness of different flavonoid subclasses and flavonoid-rich foods on cardiovascular risk, Hooper and colleagues[481] reviewed 133 RCTs examining the effects on endothelial function (measured as flow-mediated dilatation, FMD), BP, and blood lipids. They found that chocolate or cocoa was the only group to show significant improvement of FMD. Daily consumption of 50 g dark chocolate increased FMD after acute (3.99%, six studies) and chronic (1.45%, two studies) intake. The time course suggested a peak effect at approximately two hours.

Chronic intake of chocolate or cocoa also had beneficial effects on blood pressure. Both systolic (5.88 mmHg) and diastolic BP (3.30 mmHg) was reduced.[481] This effect is similar to that identified earlier in a review by Taubert and colleagues.[493] These authors commented that the magnitude of the hypotensive effects of cocoa (dark chocolate 100 g/day) is clinically noteworthy; it is in the range that is usually achieved with monotherapy of β-blockers or angiotensin-converting enzyme inhibitors.[493]

Recently, Desch *et al* published a meta-analysis of RCTs assessing the antihypertensive effects of flavanol-rich cocoa products (dark chocolate or cocoa-containing beverages).[494] Ten RCTs comprising 297 individuals were included in the analysis. The populations studied were either normotensive adults or patients with prehypertension/stage 1 hypertension. Treatment duration ranged from two to 18 weeks. The mean BP reduction was 4.5 mmHg for systolic BP and 2.5 mmHg for diastolic BP. The meta-analysis confirms the BP-lowering capacity of flavanol-rich cocoa products in a larger set of trials than previously reported.

Also the meta-analysis by Ried and colleagues, including 15 trial arms, demonstrated a small but significant BP-reducing effect of dark chocolate and flavanol-rich cocoa products.[495] Compared with the previous meta-analyses with fewer trials, the size of the effects was smaller. The mean reduction was 3.2 mmHg for systolic and 2.0 mmHg for diastolic BP. However, subgroup meta-analysis revealed a significant reduction only for hypertensive or prehypertensive subgroups (5.0 mmHg for systolic and 2.7 mmHg for diastolic BP). Flavanol-rich cocoa products did not significantly reduce BP below 140/80 mmHg.

Hooper and colleagues concluded that there is sufficient evidence to suggest that chronic intake of chocolate or cocoa has no overall effect on blood LDL or HDL cholesterol.[481] In a more recent meta-analysis of the short-term effects of cocoa product consumption on lipid profile, Jia and colleagues found that cocoa consumption marginally lowered total cholesterol and significantly lowered LDL cholesterol (by 0.15 mmol/l).[496] However, no significant change was observed in LDL cholesterol in high-quality studies. There was no dose response and no effect in healthy subjects.

Ostertag and colleagues critically reviewed 25 well-controlled human intervention studies examining the effect of polyphenol-rich diets on platelet function.[497] One consistent finding was that cocoa-related products have platelet-inhibiting effects when consumed in moderate amounts. Significant inhibition of platelet aggregation and activation upon acute of chronic intake of flavanols from cocoa was observed. It was calculated that intake of 100 mg flavanols led to 3-11% inhibition of platelet function, and that 100 mg flavanols may be obtained from 11 g dark chocolate (cocoa content 70%), from 52 g milk chocolate, or from 50-100 ml cocoa drink (containing 8% pure cocoa). The authors concluded that the physiological relevance of the beneficial effects of cocoa consumption on platelet function is comparable to standard doses of aspirin: consumption of 100 g dark chocolate with 70% cocoa solids could result in similar effects to 81 mg of aspirin in an acute setting. The effect can be obtained in healthy subjects as well as in people at risk for CVD, and appears to be largest upon acute consumption.

1.3.10.2.3.2 Tea

Both green and black tea is made from leaves of the plant *Camellia sinensis*. Green tea is produced by steaming fresh leaves to inactivate polyphenol oxidase, followed by drying. It is a rich source of flavonoids, especially catechins such as epicatechin, epicatechin gallate, epigallocatechin, and epigallocatechin gallate. These catechins comprise 30-50% of the solids of green tea and 90% of total flavonoids.[498] Black tea undergoes a fermentation process in which the leaves are kept at room temperature for 16-24 hours and are then cut and dried. In the fermentation process polyphenol oxidase converts catechins to theaflavins and thearubigin polymers. The major fraction of black tea polyphenols is composed of thearubigins, which account for >20% of the solids and 47% of total flavonoids.[498] Black tea represents one of the main contributors to intake of flavonoids in European countries, while green tea is an important source of flavonoids in the Asian countries.

Prospective studies

The meta-analysis by Peters and colleagues reported that the incidence rate of MI was estimated to decrease by 11% with an increase in black tea consumption of three cups per day.[499] The analysis was based on 10 cohort and seven case-control studies. Gardner and colleagues reviewed 21 studies and found evidence that an intake of three or more cups of black tea per day was associated with reduction of CHD risk.[500] Grassi and colleagues reviewed 15 epidemiological studies and concluded that an inverse association between tea (black and green tea) intake and CVD has been demonstrated.[498]

Four prospective cohort studies reviewed by Kuriyama have shown that green tea consumption is inversely associated with mortality from CVD.[501] One of the studies reviewed included 40,530 individuals from Japan.[501] In that study the inverse association with CVD mortality was more remarkable than that with all-cause mortality, and was also more pronounced in women. Women who consumed five or more cups (500 ml or more) per day had 31% lower risk for CVD death compared to those who consumed less than one cup of green tea per day. There was no statistically significant association between green tea consumption and MI mortality. In women, green tea consumption was significantly associated with reduced mortality from stroke, especially cerebral infarction (62% lower risk in women who consumed five or more cups compared with those who consumed less than one cup/day).

A meta-analysis including nine studies involving 4,378 stroke incidents among 194,965 individuals demonstrated that drinking tea (black or green tea) daily significantly reduced risk of stroke.[502] In subjects drinking three cups or more of tea per day the risk of a fatal or non-fatal stroke was reduced by approximately 21% as compared with nondrinkers of tea. The effect does not appear to be specific to green or black tea or to Asian or non-Asian populations. The authors concluded that tea drinking might be one of the most actionable lifestyle changes to significantly reduce the risk of stroke.

RCTs

Taubert and colleagues reviewed five RCTs on tea consumption involving a total of 343 subjects with a median duration of four weeks and showed that tea intake had no effects of blood pressure.[493] The data from black and green tea interventions were pooled. In the meta-analysis of Hooper and colleagues, ingestion of black tea resulted in an acute rise in systolic (5.69 mmHg) and diastolic BP (2.56 mmHg), independent of caffeine content.[481] However, chronic consumption of black tea did not show significant effects on BP.[481]

Chronic consumption of black tea also increased FMD by 3.40%, whereas the acute effect was modest (1.70%) and not significant.[481] Black tea had no effect on LDL or HDL cholesterol.

A meta-analysis of four interventional studies showed that green tea significantly reduced LDL cholesterol (–0.23 mmol/l) but had no overall effect on HDL cholesterol.[481] The authors estimated that two to five mugs of green tea per day (up to one-half of the usual fluid intake) would be required to achieve a clinically relevant reduction in LDL cholesterol concentration.

Kuriyama reviewed 30 RCTs and found that 17 studies indicated statistically significant beneficial effects of green tea on CVD risk profile, and 11 studies showed no such results.[501] Two studies on blood pressure or aortic stiffness showed acute harmful effects.

Ostertag and colleagues reviewed three studies examining black tea for its potential effect on platelet function. Consumption of one litre black tea per day inhibited platelet activation by 4-10%.[497] Two other studies did not find significant effects of tea consumption on platelet function.

1.3.10.2.3.3 Red wine

Red wine is produced by fermenting grape juice in the presence of grape solids. It is composed of more than 500 compounds, of which only a few are present at concentrations >100 mg/l.[503] Red wine contains a wide variety of polyphenols, most of which are derived from the skin and seeds of the grapes. Typical polyphenols include proanthocyanidins, anthocyanins, flavanols, flavonols, cinnamic derivatives, and resveratrol. White wine, on the other hand, is made by separating the juice from the solids by pressing and then allowing it to ferment. Therefore, white wine has a lower content of polyphenols than red wine. A typical commercial bottle of red wine contains approximately 1.8 g/l of total polyphenols, while a typical bottle of white wine has only 0.2-0.3 g/l of polyphenols.[503] One glass of red wine provides about 200 mg polyphenols, in comparison to 30 mg in a glass of white wine.

Prospective studies

Di Castelnuovo and colleagues performed a meta-analysis of 13 studies involving 209,418 subjects on the relationship between wine consumption (including red and white wine) and vascular risk.[404] A significant inverse association between light to moderate wine consumption and vascular risk was observed, with 32% reduction of overall vascular risk associated with drinking wine. Both non-fatal vascular endpoints and cardiovascular mortality were significantly reduced in wine drinkers. There was strong evidence from 10 studies involving 176,042 persons to support a J-shaped relationship between different amounts of wine intake and vascular risk. A statistically significant inverse association was found up to a daily intake of 150 ml of wine. The authors of this meta-analysis emphasised that the hazards of excess drinking should always be highlighted, and heavy drinkers should be pushed to cut their consumption.

In a large U.S. study of 34,489 postmenopausal women with 16 years of follow-up, consumption of red wine was inversely associated with CHD and CVD mortality.[484]

Opie and Lecour reviewed evidence for and against the "red wine hypothesis", which proposes that red wine is more likely to confer cardiovascular benefits than white wine.[504] There is strong epidemiological and mechanistic evidence for J-shaped relation between moderate alcohol consumption and total mortality. However, epidemiological data favouring a specific benefit of red over white wine is not strong and the "French paradox" (the inverse relation between CHD mortality and red wine consumption, with France having the lowest mortality despite its high-fat diet) could at least in part be explained by confounding factors. (See also Section 1.3.8 on alcohol and cardiovascular disease and Section 1.3.11 for this paper's recommended population goals in relation to alcohol consumption).

RCTs

Human studies have shown that there is incomplete and only indirect evidence that red wine might potentially be more cardioprotective than white wine.[504] The specific components of red wine that are active on cardiovascular endpoints, are the polyphenols, especially resveratrol and proanthocyanidins (which are high in pinot noir). In their comprehensive review Opie and Lecour concluded that red wine potentially has beneficial effects "beyond alcohol" and may be cardioprotective when consumed in moderate amounts and preferably with meals.[504]

The studies on the chronic effects of red wine do not suggest significant effects on endothelial function measured as FMD.[481] In acute studies red wine showed a modest (1.25%) but not significant benefit on FMD. Also another review found that data about the acute effects of red wine constituents on endothelial function is inconclusive.[505] The authors concluded that one should be very careful in suggesting red wine consumption in high-risk populations, such as patients with CHD, as the acute postprandial effect is not yet clear.

In the meta-analysis by Hooper and colleagues chronic consumption of red wine did not show significant effects on blood pressure, or LDL or HDL cholesterol.[481] Consumption of wine rich in polyphenols has produced inconsistent effects also on platelet function.[497]

1.3.10.2.3.4 Soy products

Soy foods are rich sources of isoflavones. Soybeans contain 35-40% protein on a dry-weight basis, and isoflavones are closely associated with the proteins. Soy foods contain 1-4mg isoflavones/g, whereas soy isoflavone supplements contain up to 500 mg/g.[506]

The U.S. FDA approved a food-labelling health claim for soy proteins in the prevention of CHD in 1999 but clearly indicated that "the evidence did not support a significant role for soy isoflavones in cholesterol-lowering effects of soy protein".[506] Since then, similar health petitions for soy proteins have been approved also in the United Kingdom and some other countries. This has led to an enormous increase in the number of soy products, and to extensive research on the health effects of soy intake.

RCTs

The Nutrition Committee of the American Heart Association has assessed 22 RCTs published since 1999 and found that isolated soy protein with isoflavones significantly decreased LDL cholesterol but had no effect on HDL cholesterol, triglycerides, lipoprotein(a), or blood pressure.[507] The consumption of soy protein ranged from 25 to 135 g/day, and that of isoflavones from 40 to 318 mg. Isolated soy protein with isoflavones compared with milk or other proteins decreased LDL cholesterol concentrations by approximately 3%.

In the meta-analysis conducted by Hooper and colleagues soy protein isolate—but not other soy products or components—significantly reduced LDL cholesterol (–0.19 mmol/l) and diastolic BP (–1.99 mmHg).[481] Soy

protein isolate or isoflavone extracts showed no statistically significant effects on HDL cholesterol, systolic BP or FMD.

It can be concluded that the beneficial effects observed in cardiovascular risk factors are very small compared with the large amount of soy protein required (studies all done on intakes above 25g per day). The evidence favours soy protein rather than soy isoflavones as the responsible nutrient. For this reason, use of isoflavone supplements in food or pills for cardiovascular health is not recommended.[507,508] In contrast, many soy products may be beneficial to cardiovascular and overall health because of their high content of polyunsaturated fats, fibre, vitamins, and minerals and low content of saturated fat.[507]

1.3.10.2.3.5 Fruits and berries

In a large U.S. study of 34,489 postmenopausal women with 16 years of follow-up, intakes of foods rich in flavonoids associated with reduction of mortality.[484] Consumption of apples and pears, oranges, grapefruit, blueberries, red wine, celery, strawberries, brussels sprouts, bran, chocolate, and fruit juices decreased the risk of CHD and CVD mortality. However, after multivariate adjustment, only apples and pears, grapefruit, and red wine remained significantly inversely associated with CHD mortality, and apples and pears, red wine, strawberries, bran, and chocolate for CVD mortality. Stroke mortality was reduced by consumption of apples and pears, red wine, bran and chocolate; only intake of bran remained statistically significant after multivariate adjustment.

Chong and colleagues reviewed RCTs investigating the effects of fruit polyphenols on four risk factors of CVD: platelet function, BP, vascular function and blood lipids.[509] They concluded that there is some evidence to suggest that fruits containing relatively high concentrations of flavonols, anthocyanins and proanthocyanidins, such as pomegranate, purple grapes and berries, are more effective than other fruits investigated in reducing CVD risk factors—particularly with respect to reducing BP, inhibition of platelet aggregation and increasing endothelial-dependent vasodilatation. Flavanone-rich fruits, such as oranges and grapefruits, may have some hypocholesterolaemic effects but little impact on other risk factors. However, to date the scientific evidence of beneficial effects of fruits and berries on CVD risk factors is limited, inconsistent and inconclusive. It should also be noted that besides being rich sources of various polyphenols, fruits and berries contain a wide range of other potentially cardioprotective components including fibre, folate, antioxidant vitamins and carotenoids.

1.3.10.2.4 Conclusions

There has been a lot of scientific interest in the potential cardioprotective properties of dietary polyphenols. However, due to the chemical diversity and complexity of these phytochemicals, the task is challenging. To date, certain groups of flavonoids as well as foods and beverages rich in flavonoids have been the main focus of attention. Although an extensive amount of information has been published, the overall evidence is still insufficient—not conclusive but promising. It must be stressed that the studies that have been done to date remain relatively small and short-term and do not always measure final health outcomes. The data presented in this review suggest the following conclusions:

- Reviews and meta-analyses of epidemiological studies suggest that dietary intake of flavonols may be associated with a reduced risk of CHD and stroke; for other flavonoids the evidence is more limited. Flavonols are common constituents of our diet (Table 11), and increasing the consumption of fruits, berries, vegetables and tea would increase the intake of flavonols (and also the intakes of flavones, flavanols, flavanones, anthocyanidins, and proanthocyanidins).

- Human intervention studies have shown that several biomarkers of cardiovascular risk are influenced by consumption of flavonoid-rich foods. Relatively consistent and clinically relevant effects have been observed on endothelial function, blood pressure, and platelet function.

- Epidemiological studies have demonstrated that both black and green tea consumption may be associated with reduced risk of CVD, especially stroke. Beneficial effects from RCTs are less evident. Habitual consumption of green tea may have a small favourable effect on LDL cholesterol.

- According to the recommendations of The American College of Cardiology Foundation Task Force, moderate consumption (one to two cups/day) of tea is possibly useful for cardiovascular risk reduction.[508]

- Recent meta-analyses of RCTs have provided evidence that regular consumption of flavanol-rich dark chocolate (10-100 g/day) or other cocoa products may improve endothelial function, reduce blood pressure, and inhibit platelet function. The blood pressure lowering effects as well as reduction of CVD risk are supported by epidemiological evidence but one needs to remember there are other constituents in chocolate already known to reduce blood pressure including the minerals potassium, calcium and magnesium.

- It is also important to remember that chocolate and other cocoa products are usually high in sugar, fat, and energy—100g of plain chocolate provides over 500kcal, 28g of fat (predominantly saturated fat) and a substantial amount of sugar (62.6g).[510] Therefore, questions such as the optimal dose and long-term side effects (such as weight gain and adverse metabolic changes) warrant further investigation before cocoa products can be recommended for reduction of hypertension or other cardiovascular risk factors.

- There is epidemiological evidence on the inverse association between wine consumption and cardiovascular risk, but the evidence is weaker from randomised controlled trials. Thus the association may

be nothing to do with polyphenols and may reflect the impact of other factors, such as, alcohol. (See also Section 1.3.8 on alcohol and cardiovascular disease and the recommended population goals for alcohol consumption).

- It should be noted that the polyphenols proposed to be responsible for the cardioprotective effects of dark chocolate (flavanols, proanthocyanidins) or red wine (proanthocyanidins, anthocyanins, flavanols, flavonols, and cinnamic derivatives) can be obtained also from many commonly consumed fruits, berries, and vegetables (Table 11) with no adverse effects on nutrition.

- Despite the interesting evidence that is emerging in relation to polyphenols, there is not currently sufficient conclusive evidence to justify a specific public health recommendation on polyphenols in relation to cardiovascular disease prevention.

1.3.10.3 Conclusions on antioxidants

The EHN's Nutrition Expert Group concluded that there is not currently sufficient conclusive evidence to justify making any public health recommendation in relation to antioxidants. This maintains the earlier conclusion of our 2002 report.

1.3.11 Proposed population goals

The population goals represent a recommended average intake or level for the population as a whole. They are not dietary guidelines for individuals. We recognise that there are different needs depending on factors such as age and gender. Subgroups of the population, such as infants and children, pregnant women, older people and people who are overweight or obese may have different requirements. Equally, people's needs may vary depending on their occupation—manual workers and people who work night shifts, for example, may have different requirements.

These population goals are intended primarily for use in informing and monitoring policy. They allow policy makers to make direct comparisons between the current dietary intakes and physical activity levels of a population and to identify the gap between actual and recommended dietary/activity patterns. When developing national population goals, policy makers will need to consider the proposed population goals in Table 27 in conjunction with current national dietary patterns.

Because the goals represent population averages, some of the population will have intakes (or physical activity levels, or BMI) above the goal and others will be below. The process of determining the population goals takes this fact into account—by setting the population mean at a level which results in the majority of individuals achieving the desired intakes, BMI or physical activity levels.

Examination of existing population goals across Europe reveals that there are considerable differences between the various goals and recommendations.[511] While these disparities can be partially explained by physiological and environmental variations across Europe, they also reflect differences in the philosophical approach to setting goals and in the assumptions made in the process.[511]

Intermediate targets and ambitious longer-term goals

Following review of the up-to-date scientific evidence on the relationships between diet and cardiovascular disease (as summarised in the series of reviews commissioned), the EHN Nutrition Expert Group has proposed two different sets of population goals:

- Intermediate targets are based on an assessment of current dietary and physical activity patterns in Europe and include pragmatic considerations of what might realistically be aimed for in the next five to 10 years;

- Ambitious longer-term goals highlight the levels we should ultimately be aiming for, if the pragmatic constraints that feed into the intermediate targets can be overcome.

It is important to set out both sets of goals for several reasons. First, experience has shown that where pragmatic population goals have been proposed on the basis that they reflect what can realistically be achieved in the European or North American diet, these goals can be widely interpreted (and reproduced) as being "ideal" goals which reflect the best case scenario. It is important to recognise the intermediate targets for what they are—a compromise between current evidence for optimum heart healthy intakes/levels and judgements about what can realistically be achieved in the European context in the short term.

Secondly, the inclusion of ambitious longer-term goals is important to flag up the fundamental changes that are needed to the European food system, and to the way that European societies are currently organised, to achieve an ideal diet and optimal physical activity levels. By highlighting in this way the longer-term goals that Europe should be working towards, we aim to help policy makers—as well as the food and agriculture sectors—to understand the challenges ahead. In this way, we show policy makers the scale and scope of changes that are needed, so that ambitious policy thinking can develop appropriate solutions. Ambitious longer-term goals are needed to really drive forward innovative and far-reaching policy solutions.

1.3.11.1 The process of setting goals

In the European Heart Network's previous policy paper, *Food Nutrition and Cardiovascular Disease Prevention in the European Region*,[1] the population goals were based on the Eurodiet Core Report.[3] Since the report was published in 2002, a number of authoritative bodies have published population goals relating to physical activity and nutrition.

In order to set these interim and ambitious population goals, the EHN Nutrition Expert Group took a range of factors into account:

- The latest scientific evidence as summarised by our expert reviews in the previous sections;
- Existing global or European population goals for each nutrient or food—namely the goals issued by the World Health Organization (WHO), the Food and Agriculture Organization (FAO), the European Union's European Food Safety Authority (EFSA) and the earlier Eurodiet report. The population goals set in the 2009 World Cancer Research Fund/American Institute for Cancer Research report (*Food, Nutrition, and Physical Activity: a global Perspective*[225]) are also considered—since these are based on a very recent review of the evidence and the scope of the report is global;
- Where such goals do not exist, or where they may be considered to be outdated in relation to the current scientific thinking or dietary patterns, more recent population goals issued by national bodies or international organisations are taken into account;
- When developing the more ambitious long-term goals, the Expert Group also took into consideration whether diets complying with the proposed population goals can be shown to be feasible in practice.

The following sections set out previous and existing population goals for each of the items under consideration, and demonstrate the EHN Nutrition Expert Group's decision-making process for each goal.

1.3.11.2 Total fat

The European Heart Network's earlier target from the 2002 report, based on the 2001 Eurodiet report,[3] was for less than 30% of energy from fat. Previous recommendations for total fat intake for adult populations are shown in Table 13.

Table 13 Population goals for total fat

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: <30% of dietary energy (E%)** **Longer-term goal: 20-25 E%**	**The interim target maintains EHN earlier recommendation and is in line with WHO guidance.** **The more ambitious goal in the longer term is aimed at promoting a healthy energy balance given current physical activity levels and is also in line with WHO guidance for sedentary individuals and societies.**
FAO (2010)	Fats and fatty acids in human nutrition. Report of an expert consultation[71]	Maximum of 30-35 E%	Full agreement among the experts was not reached, thus maintaining the previous recommendation was considered prudent.
EFSA (2010)	Scientific Opinion on DRVs for fats[512]	20-35 E%	This is based on "practical considerations (eg current levels of intake, achievable dietary patterns)."
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	15-30 E%	This was formulated to accommodate countries where the fat intake is typically above 30% and those where fat intakes are very low.
European Heart Network (2002)	Food, Nutrition and Cardiovascular Disease Prevention in the European Region[1]	<30 E%	
Eurodiet (2001)	Core Report [3]	<30 E%	"Sedentary societies will probably need to be on a lower fat intake, eg 20-25%, to avoid excessive weight gain."
WHO (2000)	Obesity Report (Technical Series 894).[336]	30-40 E% for "people who sustain moderately high levels of physical activity throughout life"	20-25 E% for "sedentary individuals and societies"

The scientific review by Professor Pedersen (see Section 1.3.1) concluded that the previous goal remains appropriate as an interim target. The Nutrition Expert Group, therefore, proposes to maintain the previous 2002 goal (<30 E%) as an interim goal for total fat (which includes all dietary lipids, both invisible fat from animal and vegetable sources and visible fat from vegetable oils, butter and margarine). We know that it is possible, in 21st century Europe, to achieve this goal: EFSA reported in 2010 that total fat intakes European countries range from less than 30% up to 47% of energy.[512] In fact, average total fat intakes for both men and women in Portugal are already below 30% and levels in Norway have dropped to 31% for both men and women.[512]

In formulating a more ambitious longer-term goal, the Expert Group was conscious of the fact that previous recommendations for the total fat content of European diets were formulated on the basis of judgements about what is pragmatic and realistic, given that average total fat intakes in US and northern European countries were over 40% in the 1970s.

Such recommendations have often been accompanied by caveats that total fat intakes should be lower in more sedentary societies. The Eurodiet report, for example, concluded that sedentary societies probably need lower fat intakes, e.g. 20-25%, to avoid excessive weight gain. For this reason, WHO's 2000 report on obesity suggested that fat intakes of 20-25% of energy are needed "to minimise energy imbalance and weight gain in sedentary individuals and societies."

Since then, many of those countries that had very high fat intakes have successfully reduced the fat content of diets and EFSA found that only 15% of average fat intakes in European countries are now above 40% of energy.[512] There has, however, been an explosion in the prevalence of obesity and diabetes—hence the need for an ambitious population goal for total fat which places emphasis on promoting diets which prevent unhealthy energy imbalance.

The longer-term goal (20-25 E%), therefore, is proposed to prevent unhealthy energy imbalance. It is also intended to reduce the saturated fat intake—because current dietary patterns mean that, in many countries, higher total fat intakes translate to consumption of more saturated fats. This applies particularly to northern and central Europe. The Greek version of the Mediterranean diet is unusual in that it manages to combine a high total fat intake with high consumption of vegetables and fruit, as well as a low saturated fat intake—since the fat is mostly consumed as olive oil.

We know that it is possible to achieve diets with total fat contents as low as 20% on a population average level. Populations have been reported with average fat intakes as low as 15%, and both WHO and EFSA have concluded that total fat energy of at least 20% is consistent with good health.[50,52,512]

Both the interim and longer-term goals are compatible with a healthy diet overall. The aim is to reduce the energy-density of the diet and, given the current patterns of fat consumption in many European countries, bring about a corresponding reduction in saturated fat intakes. It is important that unrefined carbohydrate from foods naturally rich in non-starch polysaccharides (fibre) replace the fat as a source of energy.

1.3.11.3 Saturated fatty acids

The European Heart Network's 2002 report proposed a population goal of less than 10% of dietary energy from saturated fat. This is in line with WHO's recommendations in the 2003 report, *Diet, Nutrition and the Prevention of Chronic Diseases* (the 916 report).[52]

Table 14 Population goals for saturated fats

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: Less than 10 E% Longer-term goal: <7 E%**
FAO (2010)	Fats and fatty acids in human nutrition. Report of an expert consultation[71]	Not more than 10 E%
EFSA (2010)	Scientific Opinion on DRVs for fats[512]	"as low as possible"
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	<10 E%
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	<10 E%
Eurodiet (2001)	Core Report [3]	<10 E%

On the basis of the review of the latest evidence (see Section 1.3.1), EHN has decided to maintain the goal of less than 10% of energy as the interim target.

There is a strong case for setting a more ambitious goal in the longer term. It is interesting to consider more recent guidelines on saturated fat intakes. EFSA's 2010 scientific opinion, for example, recommends that saturated fatty acid intakes should be "as low as possible within the context of a nutritionally adequate diet."[512] Another example of a very recent recommendation is from the 2010 version of the Dietary Guidelines for Americans, which advocates a long-term goals of 7% energy from saturated fats.[66]

As with targets for total fat, goals for saturated fat have taken pragmatic considerations into account—judgements about what is realistic in a modern European context. We do, however, know that diets with saturated fat intakes below 7% of dietary energy are possible—as shown by data on Japanese, Greek and Chinese populations.[48,63] EHN has, therefore, decided that an ambitious longer-term goal of <7 E% is advisable.

1.3.11.4 Trans fats

In the previous version of this report, EHN's population goal for trans fatty acids was <2 E%. Since then, WHO and others have recommended even lower average intakes.

Table 15 Population goals for trans fatty acids

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: <1 E%** **Longer-term goal: <0.5 E%**	
FAO (2010)	Fats and fatty acids in human nutrition. Report of an expert consultation[71]	<1 E%	This may need to be revised to take account of the distribution of intakes and thus the need to protect substantial subgroups from having dangerously high intakes.
EFSA (2010)	Scientific Opinion on DRVs for fats[512]	"as low as possible"	
WHO (2009)	Scientific update on trans fats[115]	There is a need to review the <1 E% goal	The report makes the case for reducing the target population mean so that the great majority of the population have intakes of <1 E% and "to protect large subgroups from having high intakes."
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	<1 E%	
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	<2 E%	
Eurodiet (2001)	Core Report [3]	<2 E%	

To reflect the growing evidence that the intake of trans fats should be reduced as far as possible, the new interim target for trans fats is <1 E%. This is in line with WHO's recommendation in the 916 report.[52]

In a 2009 scientific update from WHO, experts made the case for shifting the goal for the population mean in order to ensure that the vast majority of the population has intakes of less than 1% of energy. They argued that there is "*sufficient epidemiological and experimental evidence*" to support revision of WHO's previous recommendation of not more than 1% of energy.[115] This is important because data on the Danish experience, for example, have shown that even when the population mean for trans fat intakes is reduced to 1%, there are still subgroups of the population with high intakes.[513]

EFSA has recommended that trans fats intakes should be "as low as possible". It is now well recognised that it is possible to virtually eliminate industrially produced trans fatty acids, leaving only trans fats from natural sources in the diet. EHN proposes, therefore, a more ambitious longer-term goal of less than 0.5% energy.

1.3.11.5 Polyunsaturated fatty acids

It is important that the intake of polyunsaturated fatty acids comprises a combination on n-6 and n-3 fatty acids. Both help to reduce blood lipids when substituted for saturated fats. Total PUFA intake comprises linoleic acid (LA), alpha-linolenic acid (ALA) and the very long chain fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

In the 2002 report, EHN recommended that n-6 polyunsaturated fatty acids (PUFAs) should account for between 4% and 8% of dietary energy. In relation to n-3 PUFAs, the population goal was for 2 g/day linolenic acid plus 200 mg/day very long chain fatty acids. Since then, WHO and EFSA have published new guidelines and an FAO/WHO Expert Consultation has reviewed the evidence.

TABLE 16 POPULATION GOALS FOR POLYUNSATURATED FATTY ACIDS

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target:** **- 6-11E% from total PUFA;** **- Alpha-linolenic acid 1-2E%;** **- Very long chain PUFA (EPA and DHA) 250 mg-500 mg/day.** **Longer-term goal:** **- 5-8 E% for total PUFA;** **- Alpha-linolenic ≥2 E%;** **- Very long chain fatty acids 250-500 mg/day.**
FAO (2010)	Fats and fatty acids in human nutrition. Report of an expert consultation[71]	- 6-11 E% from total PUFA; - Total n-3 PUFA <2 E% - N-6 PUFA 2.5-9 E%
EFSA (2010)	Scientific Opinion on DRVs for fats[512]	- EFSA did not set a goal for total PUFAs - n-6 PUFAs: >4 E% for linoleic acid - n-3 PUFAs: >0.5 E% for alpha-linolenic acid (ALA), 250 mg/day for eicosapentaenoic acid (EPA) plus docosahexaenoic acid (DHA)
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	- n-6 PUFAs: 5-8 E% - n-3 PUFAs: 1-2 E%
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	- n-6 PUFA: 4-8 E% - n-3 PUFA: 2 g/day linolenic + 200 mg/day very long chain fatty acids
Eurodiet (2001)	Core Report [3]	- n-6 PUFA: 4-8 E% - n-3 PUFA: 2 g linolenic + 200 mg very long chain

Having reviewed the evidence—and in line with the most recent WHO/FAO Expert Consultation—the EHN Expert Group proposes a range of 6% to 11% of energy from total polyunsaturated fatty acids (PUFAs) as an interim target. More specifically, alpha-linolenic acid should make up between 1% and 2% of energy, while average intakes of very long chain n-3 polyunsaturated fatty acids—such as EPA and DHA—should be between 250 mg and 500 mg per day.

Average PUFA intakes in Europe are commonly between 5% and 7% of energy.

The longer-term goal for polyunsaturated fatty acid intakes is for between 5% and 8% of dietary energy. Alpha-linolenic should constitute at least 2% of energy and intakes of very long chain fatty acids should be 250-500 mg. This lower percentage of energy from polyunsaturated fatty acids is proposed in proportion to the planned drop in the total fat content of the diet to comply with the longer-term goal for total fat.

1.3.11.6 Monounsaturated fatty acids

EHN's 2002 report did not set a specific goal for monounsaturated fatty acids. In this version, EHN proposes an intermediate target of 8% to 13% of energy. This is based on calculating the difference between total fat and a subtotal of saturated, polyunsaturated and trans fatty acids. This reflects the approach adopted by WHO in setting a population goals for monounsaturated fats based on the difference between other types of fatty acids.

Table 17 Population goals for monounsaturated fatty acids

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: 8-13 E%; Longer-term goal: 7.5-9.5 E%.**
FAO (2010)	Fats and fatty acids in human nutrition. Report of an expert consultation[71]	By difference. i.e. total fat - (saturated fat + PUFAs + trans)
EFSA (2010)	Scientific Opinion on DRVs for fats[512]	EFSA decided not to set a dietary reference value for monounsaturated fatty acids
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	By difference. i.e. total fat – (saturated fatty acids + PUFAs + trans fatty acids)
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	No goal specified
Eurodiet (2001)	Core Report [3]	No goal specified

In the longer term, the population goal is for 7.5% and 9.5% of energy to be from monounsaturated fatty acids. This is coherent with the proposed changes in dietary fat intakes proposed by the longer-term ambitious goals for total fat and other types of fatty acids.

1.3.11.7 Fruit and vegetables

The EHN's 2002 report included a population goal for an average intake of 400 g/day of fruits and vegetables. This has since been adopted as a goal by WHO and has formed the basis for the widely adopted message promoting five portions of fruits and vegetables a day.

Table 18 Population goals for fruit and vegetables

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: >400 g/day** **Longer-term goal: >600 g/day**	
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	> 600 g/day	Applies to non-starchy vegetables and fruits
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	≥ 400 g/day	
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	> 400 g/day	
Eurodiet (2001)	Core Report [3]	> 400 g/day	

This report maintains the 400 g/day goal for fruits and vegetables as the interim target. This should relate to the amount actually consumed—that is, peeled and prepared fruits and vegetables.

Yet the evidence suggests that the relationship between fruit and vegetables consumption and cardiovascular protection is linear—the more the better, and no upper limit has been found. In the absence of any recent WHO or EFSA population goal for fruits and vegetables, the Expert Group looked for more recent proposals for population goals based on up-to-date reviews of the evidence. In 2009, the World Cancer Research Fund/American Institute for Cancer Research proposed a population goal of 600 g/day of fruit and vegetables to ensure that most people in the population consume at least 400 g/day, and in order to prevent cancer and other chronic diseases.[225]

We know (from food availability data) that most countries in Europe still struggle to meet the target of 400 g/day population goal. Only five countries have a per capita supply of fruit and vegetables above 700 g/day—the figure estimated to enable consumption of 400 g/day after waste is accounted for (see Section 1.4.2.1). We do know, however, that higher fruit and vegetable intakes are possible—Greece, for example, supplies over 1,100 g/person/day—and many European countries have seen increasing fruit and vegetable consumption levels (see Figures 27 and 28, Section 1.4.2.1).

The EHN, therefore, proposes this higher target of 600 g/day of fruits and vegetables as a longer-term population goal.

1.3.11.8 Salt

In 2002, EHN proposed an adult population goal of less than 6 g per day for salt. Since then, other bodies have generally recommended an average of between 5 g and 6 g per day.

Table 19 Population goals for salt

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: <5 g/day. Longer-term goal: <4 g/day.**
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	<5 g/day Proportion of the population consuming more than 6 g of salt/day to be halved every 10 years.
EFSA (2005)	Scientific opinion on the tolerable upper intake level of sodium[172]	Data not sufficient to establish an upper level for sodium from dietary sources.
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	<5 g/day
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	<6 g/day
Eurodiet (2001)	Core Report [3]	<6 g/day

EHN proposes a goal of less than 5 g/day as an interim target, which is pragmatic for the next 3-5 years, given that average salt intakes in Europe are currently often around 10 g/day. This is consistent with the WHO and WCRF recommendations outlined above.

It is widely recognised that, in the longer term, a further reduction is desirable—and recent experience shows that gradual reductions in population salt intakes are possible.

At least two sets of national guidelines issued in 2010 have proposed more ambitious long-term goals: the UK's National Institute for Health and Clinical Excellence (NICE) proposed 3 g/day by 2025,[25] and the US Dietary Guidelines for America 2010 propose a goal of 3.75 g/day (1,500 mg/day sodium),[66] lower than the 5.75 g/day (2,300 mg/day sodium) in the 2005 version of the guidelines.[51] With this perspective, EHN proposes a longer-term goal of less than 4 g/day of salt.

1.3.11.9 Physical activity

The population goal for physical activity in EHN's 2002 report was for a Physical Activity Level (PAL) of 1.75, equivalent to about 60 minutes per day of moderate activity such as walking. This was based on the Eurodiet report's goal to prevent weight gain in populations on high fat diets. The specific goal for preventing cardiovascular disease was for 30 minutes per day of moderate activity.

Table 20 Population goals for physical activity

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: 150 minutes of at least moderate intensity endurance or aerobic activity per week (such as brisk walking at a pace of 4 to 6 km/h) for cardiovascular health.** **In addition, a goal of one hour of at least moderate activity on most days (around 300 minutes per week) is recommended for additional health benefits and to prevent overweight and obesity.** **Longer term: Higher, but still attainable, amounts of at least moderate intensity physical activity, say 60 minutes a day, will bring additional health benefits.**	**A large part of the protective effect of physical activity for cardiovascular health could be obtained with 150 minutes of at least moderate intensity activity weekly. A further goal of 300 minutes per week is appropriate to help prevent overweight and obesity, in light of the sedentariness of European populations and current dietary patterns.** **To attain higher levels of PA in the long term, it is important to increase active travel and to build physical activity into daily routines, such as commuting and other chores.**
WHO (2010)	Global recommendations on physical activity for health[514]	At least 150 minutes of moderate-intensity aerobic physical activity or 75 minutes of vigorous physical activity per week; For additional health benefits: 300 minutes of moderate physical activity or 150 minutes of vigorous activity per week.	
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	Average physical activity levels (PALs) to be above 1.6. The proportion of the population that is sedentary to be halved every 10 years.	
EU (2008)	EU Physical Activity Guidelines[515]	A minimum of 30 minutes of daily moderate-intensity physical activity for adults	
FAO/WHO/ UNU (2004)	Human energy requirements. Report of a Joint FAO/WHO/ UNU Expert Consultation.[516]	Moderate-intensity activity, such as brisk walking, for an hour per day on most days of the week.	
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	One hour per day on most days of the week of moderate-intensity activity	Needed to maintain a healthy body weight. This recommendation for physical activity is not relevant for populations which already have high energy expenditure levels.
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	Physical Activity Level (PAL) of 1.75	Equivalent to about 60 minutes per day of moderate activity such as walking.
Eurodiet (2001)	Core Report [3]	Physical Activity Level (PAL) of >1.75.	Equivalent to 60-80 minutes of walking daily to avoid weight gain on high fat intakes; this includes the 30 minute goal for preventing cardiovascular diseases and diabetes.

Having reviewed the latest scientific evidence (see Section 1.3.7), this report recommends an interim target of 150 minutes of moderate-intensity endurance or aerobic physical activity (such as brisk walking at a pace of 4 to 6 km/h) per week for cardiovascular health. In addition, a goal of one hour of moderate activity on most days (around 300 minutes per week) is proposed as a supplementary interim target for additional health benefits and in order to help prevent significant weight gain.

This is consistent with the latest recommendations from WHO (issued in November 2010). This target is recommended on the basis that such levels of physical activity are achievable on a large scale in European populations.

In the longer-term, higher, but still attainable, amounts of moderate-intensity physical activity are recommended to bring additional health benefits: 60 minutes a day is recommended, in line with WHO's recommendations from the 2003 report on diet, nutrition and the prevention of chronic diseases. To attain these higher levels of physical activity (such as one hour daily) at the population level in the long-term, it is important to increase active travel and to build physical activity into daily routines, such as commuting and other chores.

1.3.11.10 BMI

EHN's 2002 population goal in relation to overweight and obesity was for a body mass index (BMI) of 23 kg/m^2, based on analysis of levels of overweight across the European Union at the time. This differed slightly from the Eurodiet recommendation for a BMI of 21-22 as the optimum population mean BMI which both limits the likelihood of underweight and of obesity.

TABLE 21 POPULATION GOALS FOR BMI

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: BMI <23 Longer-term goal: BMI of 21**
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	Median adult BMI for different populations to be between 21 and 23, depending on the normal range
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	Median BMI of 21-23 kg/m^2
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	BMI 23 kg/m^2
Eurodiet (2001)	Core Report [3]	BMI 21-22 kg/m^2
WHO (2000)	Obesity report (894)[336]	Population median BMI range of 21-23

This report proposes a BMI of 23 as an intermediate target and a longer-term goal for a BMI of 21. These recommendations are in line with WHO recommendations for a population mean BMI range BMI of 21-23.[52] In the longer term European countries should be aiming towards the lower end of the WHO recommended range. WHO's report on obesity concluded that '*adults in affluent societies with a more sedentary lifestyle are likely to gain greater benefit from a median BMI of 21.*'[336]

1.3.11.11 Total carbohydrates

In the previous version of this report, EHN proposed a population goal of more than 55% of energy from total carbohydrates.

Table 22 Population goals for total carbohydrates

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: > 55E% Longer-term goal: 60-70 E%**	**The carbohydrate should be derived principally from whole-grain cereals, fruit, berries, vegetables and legumes.**
EFSA (2010)	Scientific Opinion on DRVs for carbohydrate and dietary fibre[184]	45-60 E%	
FAO/WHO (2007)	Scientific update on carbohydrates[517]	50-75 E%	The scientific update suggested a possible revision of the 2003 WHO recommendation (below) to drop the lower end of the range to 50%.
EU (2008)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	55-75 E%	"The percentage of total energy available after taking into account that consumed as protein and fat, hence the wide range."
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	>55 E%	
Eurodiet (2001)	Core Report [3]	>55 E%	

In this report, EHN maintains this figure of >55 E% as an interim target, but also proposes a longer-term goal of between 60% and 70% of energy. The more ambitious longer-term goal is calculated by difference on the basis of the other changes in the diet that are recommended in these population goals. As the fat content of the diet decreases (with an ambitious longer-term goal of 20-25 E% from total fat) and protein remains at 10-15% of energy, the proportion of energy from total carbohydrate will increase.

These targets are broadly in line with WHO's recommended range of 55-75% of energy from carbohydrate from the 2003 916 report.[52] More recently, EFSA has proposed 45 to 60% as the Reference Intake for carbohydrates.[184]

As the proportion of energy from carbohydrate increases, the nature of the carbohydrate becomes even more important—with the emphasis on carbohydrates from whole-grain cereals, fruits, vegetables and legumes.

Average adult carbohydrate intakes in European Union countries currently range from 38% to 54% of energy.[184]

1.3.11.12 Added sugars

The previous EHN report set a population goal for sugary foods consumption of four or fewer occasions per day. Since then, WHO has set a population goal of less than 10% of energy from free sugars. This goal (also proposed by different national bodies) was reiterated by the more recent WHO/FAO Scientific Update on carbohydrates.[517]

TABLE 23 POPULATION GOALS FOR ADDED SUGARS

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: <10 E%** **Longer-term goal: tentatively 5 E%**	**The rationale for these goals is focused on reducing the energy density of the diet, with a particular emphasis on the importance of limiting the intake of sugar-sweetened drinks.** **The tentative long-term goals is proposed to highlight the changes needed in the diet to ensure maintenance of a healthy energy balance with current physical activity levels.**
EFSA (2010)	Scientific Opinion on DRVs for carbohydrate and dietary fibre[518]	No DRV set	
FAO/WHO (2007)	Scientific update on carbohydrates[517]	<10 E% (free sugars)	
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	Free sugars <10 E%	The term "free sugars" refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices.
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	Sugary foods on four or fewer occasions/ day	"This accords well with goals of less than 10% of energy used in other reports."
Eurodiet (2001)	Core Report [3]	Sugary food consumption, four or fewer occasions per day	This limited intake is compatible with many member states' limits on total sugar intake and the Nordic concern to limit the intake of children and those adults on low energy intakes to no more than 10%.

In line with WHO, this report proposes a mean population intake of less than 10% of dietary energy from added sugars as an interim target. This target is in line with WHO's 2003 recommendation that less than 10% of energy should be from 'free sugars' (all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices).

In the longer term, EHN considers that a more ambitious goal is advisable, with a view to reducing the energy density of the diet—important for ensuring a healthy energy balance can be maintained in conjunction with improved physical activity patterns.

1.3.11.13 Sugar-sweetened beverages

There has been increasing emphasis on the role of sugary drinks in the diet, given the global increase in sweetened soft drink consumption in recent decades. In response to the growing contribution of soft drinks to energy intakes, various authorities have recently introduced (or attempted to introduce) policy initiatives to reduce sugar-sweetened beverage consumption.

Table 24 Population goals on sugary drinks

Body (year)	Report	Recommendation / Conclusion	Comment
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Intermediate target: to reduce as much as possible, consumption of sugar sweetened beverages.** **Longer-term goal: zero consumption of sugar-sweetened beverages.**	**This is a new population goal for the EHN, reflecting the growing consensus that it is important to limit sugar-sweetened drink intakes and the increasing momentum behind policy initiatives with this objective.** **The term sugar-sweetened beverages includes sweetened fruit juices and dairy based drinks.**
EFSA (2010)	Scientific Opinion on DRVs for carbohydrate and dietary fibre[518]	No DRV set	The scientific update did conclude that "there is justification for the recommendation to restrict the consumption of beverages high in free sugars to reduce the risk of excessive weight gain."
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	Population average consumption of sugary drinks to be halved every 10 years	
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	No specific population goal	
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	No specific population goal	
Eurodiet (2001)	Core Report [3]	No specific population goal	

In the US for example—where sugar-sweetened beverages are the primary source of added sugar in the diet[181] and are calculated to contribute an average of 172 and 175 kcal daily for children and adults respectively[519]—the federal government and several states have looked at introducing economic measures (e.g. a soda tax, a ban on purchasing sugary drinks with food stamps) to reduce intakes. Elsewhere in this report, section 1.4.2.4 describes how soft drinks consumption has increased in Europe. In the UK, for example, sugar-sweetened drinks are estimated to contribute an average of 90-100 kcal per day (see section 1.4).

Although neither WHO nor EFSA has defined a population goal in relation to sugar-sweetened beverages, both have mentioned the role of sugary drinks in relation to their 'free sugars' targets.[52,184] In 2009, the World Cancer Research Fund proposed that population average consumption of sugary drinks should be halved every 10 years.[225] In the absence of European or WHO guidelines on sugary drinks consumption, some national bodies have introduced population goals. Denmark, for example, has introduced a population goal for older children and adults to drink a maximum of half a litre of soda or juice per week.[520] Finland has recently issued population guidance on drinks—advising adults to drink 1.5 litres of liquids in addition to food every day and advising that this should be mainly made up of water, with up to one glass of fruit juice daily and otherwise very infrequent consumption of juice/juice drinks, soft drinks and sweetened or fatty milks.[521]

EHN proposes an interim target to "reduce, as much as possible, consumption of sugar-sweetened beverages (including sweetened dairy-based drinks)". In the longer term, the population should be aiming for zero consumption of sugar-sweetened drinks. Water is to be encouraged as the beverage of choice, although there remains a place for unsweetened, low fat dairy drinks and unsweetened fruit juices in limited amounts. It is important that supportive policies are in place to ensure easy access to drinking water.

1.3.11.14 Fibre

In the 2002 report, the EHN proposed a population goal of more than 25 g per day (or 3 g/MJ) from dietary fibre.

TABLE 25 POPULATION GOALS FOR DIETARY FIBRE

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**Interim target: >20 g non-starch polysaccharide (NSP) per day (>27 g fibre/day) equivalent to 1.6 g NSP/MJ (>2.2 g fibre/MJ) Longer-term goal: >25 g NSP (>35 g fibre) equivalent to >2 g NSP/MJ (>2.8 g fibre/MJ) Fibre should be consumed in the diet as whole grain cereals, legumes, vegetables, fruits and berries.**
EFSA (2010)	Scientific Opinion on DRVs for carbohydrate and dietary fibre[184]	>25 g fibre (2 g fibre/MJ)
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	>25 g fibre (2 g fibre/MJ)
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	>25 g NSP
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	20 g non-starch polysaccharides/day
Eurodiet (2001)	Core Report [3]	> 25 g/day (or 3 g/MJ)

The proposed goals include figures for fibre according to two different definitions in widespread use. Non-starch polysaccharides (NSP) refer to fibre of plant cell wall origin. The Association of Official Analytical Chemists (AOAC) method of measuring dietary fibre includes indigestible material and tends to give a higher value for fibre content of foods, hence the higher population goals proposed.

This report proposes an interim target of more than 20 g NSP, in line with WHO's 2003 recommendation from the 916 report.

More recently, the World Cancer Research Fund[225] have recommended a more ambitious goal of more than 25 g of non-starch polysaccharides per day.[518] EFSA proposed that 25 g per day of dietary fibre (non-digestible carbohydrates plus lignin) is adequate for normal laxation, but notes that higher dietary fibre intakes may reduce risk of coronary heart disease, type 2 diabetes and improved weight maintenance.[518] In line with these conclusions, this report recommends a more ambitious longer-term goal of 25 g/NSP/day.

In both cases, the fibre should be consumed as whole grain cereals, legumes, vegetables, fruits and berries.

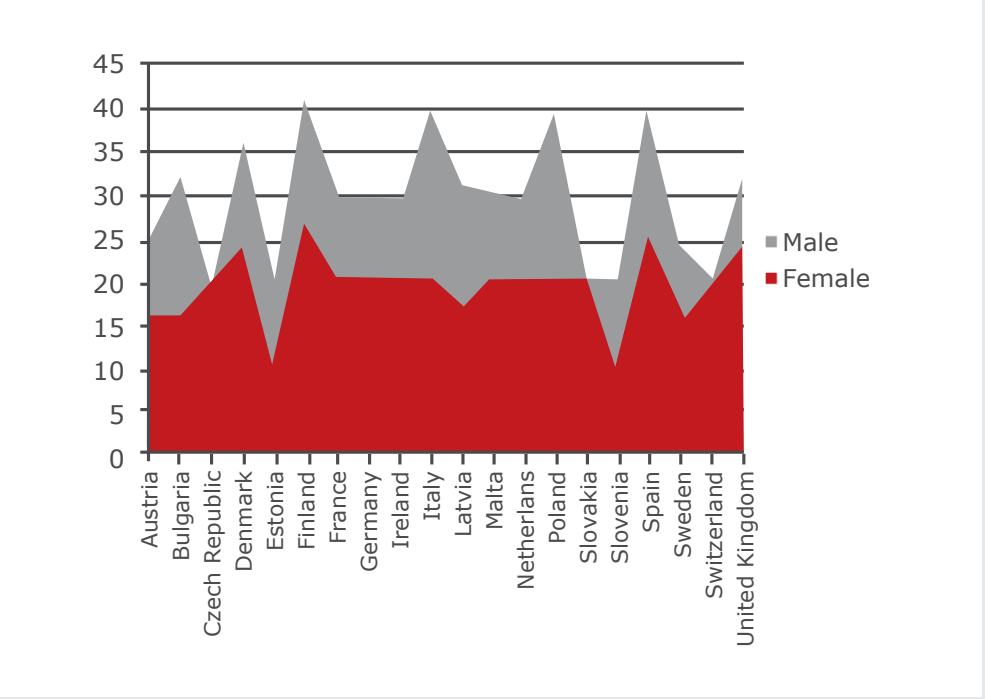
1.3.11.15 Alcohol

Many international and national bodies recognise the cardiovascular protective effect of regular, low to moderate drinking especially in older age groups but there is also limited evidence in younger age groups. The potential benefit, however, has to be considered alongside the vast burden of heavy alcohol-related harm which also exists. For this reason, health bodies have avoided a general recommendation for low to moderate alcohol use to prevent cardiovascular disease.

Official bodies have instead tended to communicate guidelines on low-risk drinking. WHO considers that the setting of region-wide specific drinking guidelines is not advisable, and WHO's key message continues to be "less is better."[522] The global strategy to reduce the harmful use of alcohol adopted by the World Health Assembly in May 2010, outlines global commitments, sets out key objectives and defines a series of guiding principles, but does not propose population goals or guidelines for low-risk drinking.[45] WHO's Regional Office for Europe cautions that, even at the national level, such guidelines can be mis-interpreted and it advises member states to take existing drinking patterns and cultures into account if they do decide to set country-specific population-based drinking guidelines.[522]

The World Cancer Research Fund found that the most common recommendations were for women to drink no more than one alcoholic drink per day and men to drink no more than two drinks daily.[225] A 2009 survey presented to the European Commission's Committee on National Alcohol Policy and Action summarised recommendations about sensible drinking in 20 European countries (see Figure 18 below).[523] The guidelines range from 10 g to over 25 g alcohol per day for women and from 20 g to over 40 g daily for men.

Figure 18 Sensible drinking guidelines for adult men and women in 20 European countries (in g/day of alcohol)



Source: Anderson P and Gordon R. Recommendations about sensible drinking amounts. Results of a survey. Presentation to European Commission's Committee on National Alcohol Policy and Action.

On the basis of the expert review commissioned for this policy paper (see Chapter 1.3.8), the main messages in relation to alcohol are shown in Table 26. This guidance is in line with a number of recent national dietary recommendations in, for example, the Netherlands, Finland and the US.[66,521,524]

TABLE 26 INTERNATIONAL GUIDANCE ON LOW RISK ALCOHOL CONSUMPTION

Body (year)	Report	Recommendation / Conclusion
This report: EHN (2010)	**Diet, Physical Activity and Cardiovascular Disease Prevention in Europe**	**If alcohol is consumed at all, it should be limited to not more than 20 g alcohol (two drinks) per day in men and not more than 10 g alcohol (one drink) per day in women.[8]** **Heavy or binge drinkers should be urged to cut and modify their consumption. Binge drinking is defined as an intake of 60 g or more on one occasion, monthly or more often, during the past 12 months.[9]** **Healthy people who already regularly consume small to moderate amounts of alcohol—no more than one drink (10 g of alcohol) per day for women or two drinks per day (20 g of alcohol) for men—should not be discouraged from continuing. A similar message can be addressed to patients who already suffered a cardiovascular event.** **It is not recommended that adult abstainers begin drinking. This is because even moderate alcohol intake may be associated with increased risk of other harm and there is a risk that some of those who start to drink will be drawn into consuming more than the very limited amounts of alcohol associated with decreased cardiovascular risk. For these reasons it is not recommended that anyone be advised to start drinking on health grounds.**
WCRF (2009)	Food, Nutrition, and Physical Activity: a Global Perspective[225]	If alcoholic drinks are consumed, limit consumption to no more than two drinks a day for men and one drink a day for women (one drink contains 10-15 g of ethanol).
WHO (2003)	Diet, Nutrition and the Prevention of Chronic Diseases (the 916 report).[52]	Consumption of alcohol is not recommended. If it is consumed it should not exceed 20 g alcohol (two "units") per day.
European Heart Network (2002)	Food Nutrition and Cardiovascular Disease Prevention in the European Region[1]	No goal. For those that choose to consume, consumption should be moderate

It is important that these guidelines on low-risk drinking are not seen as population goals. Unlike the other goals proposed in this chapter, these guidelines do not represent a target for mean population intake. The messages here on alcohol are intended to help national health policy makers define messages on low-risk drinking for their populations.

Overall targets for the population have to form part of integrated national alcohol policies which take current patterns of drinking and alcohol-related harm into account. There are huge variations, for example, in the proportion of abstainers in the population between countries—the percentage of adult abstainers in the country with the highest proportion of male non-drinkers in Europe is 27 times higher than in the country with the lowest.[43]

WCRF's 2009 report is unusual in proposing a population goal: to reduce the proportion of the population drinking more than the recommended limits by one third every ten years. The EU has not yet defined any specific goals, but has highlighted three key indicators to monitor the overall performance of its alcohol strategy (volume of consumption, pattern of consumption and alcohol-related health harm).

8 The official definitions of a standard drink varies between countries. The most commonly used definition (also used by WHO) is 10 g of alcohol (ethanol). Alcoholic beverages, however, are not drugs and should not be consumed on a strict gram basis.
9 This has been defined as one of the key indicators for monitoring the progress of the EU's first alcohol strategy.

It is important that these guidelines on low-risk drinking are not seen as population goals. Unlike the other goals proposed in this chapter, these guidelines do not represent a target for mean population intake. The messages here on alcohol are intended to help national health policy makers define messages on low-risk drinking for their populations.

Overall targets for the population have to form part of integrated national alcohol policies which take current patterns of drinking and alcohol-related harm into account. There are huge variations, for example, in the proportion of abstainers in the population between countries—the percentage of adult abstainers in the country with the highest proportion of male non-drinkers in Europe is 27 times higher than in the country with the lowest.[41]

WCRF's 2009 report is unusual in proposing a population goal: to reduce the proportion of the population drinking more than the recommended limits by one third every ten years. The EU has not yet defined any specific goals, but has highlighted three key indicators to monitor the overall performance of its alcohol strategy (volume of consumption, pattern of consumption and alcohol-related health harm).

1.3.11.16 Summary of population goals

The various population goals presented in this paper are summarised in Table 27.

Table 27 Proposed population goals

Component	Intermediate targets[10]	Longer-term goals	Comment
Total fat (dietary lipids)	Intermediate target is less than 30% of energy.[11]	Ambitious longer-term goal is 20–25% of energy. The rationale for this more ambitious goal for total fat is to lower the energy density of the diet, and, given prevailing patterns of fatty acid consumption, to reduce saturated fat intake. Unrefined carbohydrate from foods naturally rich in non-starch poly-saccharides (fibre) should replace the fat as a source of energy.	The interim target (<30 E%) maintains EHN's earlier 2002 target, based on the 2001 Eurodiet report[3] and is in line with WHO's recommendation.[52] We know that this is feasible—average total fat intakes are already <30 E% in Portugal and 31 E% in Norway.[512] The longer-term goal (20-25 E%) is proposed to prevent unhealthy energy imbalance and, given prevailing dietary patterns of fatty acid consumption, to reduce saturated fat intake. This applies particularly to northern and central Europe. Earlier population goals were largely based on pragmatic considerations dating back to a time when total fat intakes in northern European countries were over 40% in the 1970s. These goals were often accompanied by caveats that total fat intakes should be lower in more sedentary societies: - Eurodiet recommended 20-25 E% for sedentary societies; - WHO proposed 20-25 E% for "sedentary individuals and societies" in its 2000 report on obesity. Although many countries have successfully reduced the fat content of diets, there has been an explosion in the prevalence of obesity and diabetes. Thus, there is a need for an ambitious population goal to prevent unhealthy energy imbalance. We know that it is possible to achieve diets with average total fat as low as 20 E%. Total fat energy of at least 20% is consistent with good health.[52, 512]

10 The population goals are for the average (mean) of populations and are not for individuals.
11 These and other goals are given for percentage of dietary energy excluding alcohol.

Component	Intermediate targets	Longer-term goals	Comment
Saturated fat	Intermediate target is less than 10% of energy.	Ambitious longer-term goal is less than 7% of energy.	Population goals for saturated fat range from "as low as possible" to <10 E%. The interim target (<10 E%) maintains the European Heart Network's 2002 target, based on the 2001 Eurodiet report,[3] and is in line with WHO's 2003 goal from the 916 report.[52] There is a strong case for setting a more ambitious goal, and recent guidelines have advocated lower saturated fat intakes (EFSA "as low as possible"; US 2010 DGA proposes 7 E% as long term goal[66]). Data on Greek, Japanese, and Chinese populations show that saturated fat intakes below 7% are possible.[48]
Trans fats	Intermediate target is less than 1% of energy.	Ambitious longer-term goal is less than 0.5% of energy.	The interim target (<1 E%) is in line with WHO's recommendation.[52] This evolution of EHN's 2002 goal (<2%) reflects the growing evidence about the harmful effects of trans fatty acids. The more ambitious longer-term goal (<0.5 E%) reflects the conclusions of a recent WHO scientific update on trans fats that the goal for 1 E% as a population mean for trans fat intakes needs to be revised to ensure that the vast majority of the population has intakes of 1% or lower.[115] This is also in line with EFSA's recommendation that trans fats intakes should be "as low as possible".
Polyunsaturated fatty acids (PUFA) Both n-6 and n-3 fatty acids help to reduce blood lipids when substituted for saturated fats. Total PUFA intake comprises linoleic acid (LA), alpha-linolenic acid (ALA) and the very long chain fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).	Intermediate targets: The intake of polyunsaturated fatty acids should be between 6 and 11% of energy. Alpha-linolenic acid should make up between 1 and 2% of energy. Average intakes of very long chain n-3 polyunsaturated fatty acids—such as EPA and DHA—should be between 250 mg and 500 mg per day.	The longer-term goals for polyunsaturated fatty acid intakes is for between 5 and 8% of energy. Alpha-linolenic should constitute at least 2% of energy and daily intakes of very long chain fatty acids should be 250-500 mg.	Population goals for total polyunsaturated fatty acids range from 4-8 E% to 6-11 E%. The interim target (6-11 E%) presented here is in line with the proposals by the 2008 FAO/WHO Expert Consultation. Average PUFA intakes in Europe are commonly around 5-7 E%.[512] In the longer term a lower percentage of energy from polyunsaturated fatty acids is recommended, in proportion to the planned drop in the total fat content of the diet.
Monounsaturated fatty acids	The intermediate target for monounsaturated fatty acids (MUFA) is between 8% and 13% of energy.	In the longer-term, the % of energy from monounsaturated fatty acids should be between 7.5% and 9.5%.	Few specific population goals have been set for monounsaturated fatty acids—most recommendations are for MUFA to make up the difference between total fat minus saturated, polyunsaturated and trans fatty acids. The targets proposed here, quantify the goals for MUFA based on such calculations. In the longer term, a lower percentage of energy from monounsaturated fatty acids is proposed, in proportion to the planned drop in the total fat content of the diet.

Component	Intermediate targets	Longer-term goals	Comment
Fruit and vegetables	The intermediate target is more than 400 g/day. This should relate to the amount actually consumed—that is, peeled and prepared fruits and vegetables.	The ambitious longer-term goal is more than 600 g/day. This should relate to the amount actually consumed—that is, peeled and prepared fruits and vegetables.	Population goals for fruit and vegetables intakes tend to range from >400 g/day to more than >600 g/day. The interim target proposed here maintains the EHN's recommendation from the 2002 report and is in line with WHO's recommendation. The evidence suggests that the relationship between fruit and vegetables consumption and cardiovascular protection is linear—the more the better, and no upper limit has been found. The higher proposed population goal of 600 g/day is in line with the most recent global population goal proposed by the World Cancer Research Fund in 2009.[225]
Salt	The intermediate target is less than 5 g/day.	The ambitious longer-term goal is less than 4 g/day.	International population goals for salt intake range from <5 g/day to <6 g/day. The interim target proposed here (<5 g/day) is in line with the latest WHO and WCRF recommendations. Given current salt intakes in Europe (probably around 10 g/day), this interim target is pragmatic for the next 3-5 years. In the longer term, a further reduction is desirable and a longer-term goal of <4 g/day is proposed. This reflects the tendency in more recent guidelines (two national guidelines from 2010) to propose more ambitious goals (as low as <3 g/day).
Physical activity	Interim target: 150 minutes of at least moderate intensity endurance or aerobic activity per week (such as brisk walking at a pace of 4 to 6 km/h) for cardiovascular health. In addition, a goal of one hour of moderate activity on most days (around 300 minutes per week) is recommended for additional health benefits and to prevent overweight and obesity.	Longer term: Higher, but still attainable, amounts of at least moderate-intensity physical activity, say 60 minutes a day, will bring additional health benefits.	The interim targets are consistent with the latest recommendations from WHO (issued in November 2010) and are recommended on the basis that such levels of physical activity are achievable on a large scale in European populations. In the longer term, higher, still attainable amounts of at least moderate-intensity physical activity are recommended to bring additional health benefits: 60 minutes a day is recommended, in line with WHO's recommendations from the 2003 report on diet, nutrition and the prevention of chronic diseases. To attain these higher levels of physical activity (such as one hour daily) at the population level in the long term, it is important to increase active travel and to build physical activity into daily routines, such as commuting and other chores.
BMI	The intermediate target is a population average BMI of less than 23 for adults.	The ambitious longer-term goal is for a population average BMI of 21 for adults.	Recommendations for population goals for BMI range from 21 to 23. The intermediate target (<23) maintains EHN's previous recommendation and is also in line with WHO's recommendation for a population median BMI range BMI of 21-23 kg/m^2.[52,336] The longer-term goal is for a population average BMI of 21, reflecting WHO's conclusion that '*adults in affluent societies with a more sedentary lifestyle are likely to gain greater benefit from a median BMI of 21.*'[336]

Component	Intermediate targets	Longer-term goals	Comment
Total carbohydrates	More than 55% of energy. The carbohydrate should be derived principally from whole-grain cereals, fruit, berries, vegetables and legumes.	Up to 60% - 70% of energy. The carbohydrate should be derived principally from whole-grain cereals, fruit, berries, vegetables and legumes.	Population goals for total carbohydrates range from 45 E% to 75 E%. The interim target for >55 E% is in line with the lower end of WHO's recommended range.[52] The longer-term goal reflects the fact that as the fat content of the diet decreases (see ambitious goal for total fat, above) and protein remains at 10-15% of energy, the proportion of energy from total carbohydrate will increase. These goals are in line with WHO's recommended range of 55%-75% (the top of the WHO range is higher because the 2003 guidelines accommodate non-European populations with fat intakes as low as 15 E%).
Added sugars	The intermediate target is less than 10% of energy.	The ambitious longer-term goal is tentatively set at 5 E%.	Recent international guidelines generally propose <10 E% as the population goal for added sugars. The interim target (<10 E%) is in line with WHO's 2003 recommendation. The more recent WHO/FAO Scientific Update on carbohydrates reiterated this population nutrient goal of <10% of energy from free sugars.[517] The tentative longer-term goal of 5 E% reflects the fact that further reductions in added sugars are desirable to reduce the energy density of the diet—increasingly important in light of the overweight and obesity epidemic—with a particular emphasis on the importance of limiting the intake of sugar-sweetened drinks.
Sugar-sweetened beverages	The intermediate target for sugar-sweetened beverages (including dairy-based drinks) is to reduce consumption, as much as possible.	The ambitious longer-term goal is zero consumption of sugar-sweetened beverages.	There has recently been increasing emphasis on the role of sugary drinks in the diet, and a number of countries have introduced policy initiatives to reduce intakes. The only international recommendation relating to sugar-sweetened drinks is from WCRF, to halve population average consumption of sugary drinks every 10 years.[225] Some national bodies have introduced national population goals. Denmark for example has set a target of less than 0.5 litres per week of sugar-sweetened drinks. Finland has issued guidance that sugar-sweetened drinks (including sweetened fruit juice and sweetened dairy-based drinks) should be consumed only very infrequently. The longer-term goal aims for zero consumption of sugar-sweetened drinks (including dairy-based drinks). Water is to be encouraged as the drink of choice, although there remains a place for unsweetened, low fat dairy drinks and unsweetened fruit juice (in limited amounts).

Component	Intermediate targets	Longer-term goals	Comment
Dietary fibre	The intermediate target is >20 g non-starch polysaccharides per day (>1.6 g/ day of non-starch polysaccharides/ MJ) or 27 g AOAC fibre. Fibre should be consumed as whole foods with a mix of whole grain cereals, legumes, vegetables, fruit and berries.	The ambitious long-term goal should be >25 g non-starch polysaccharides (>2 g non-starch polysaccharides/MJ) or >35 g AOAC fibre (2.8 g fibre/MJ). Fibre should be consumed as whole foods with a mix of whole grain cereals, legumes, vegetables, fruit and berries.	The interim target (>20 g NSP/day) is in line with WHO's 2003 recommendation. The longer-term goal (>25 g NSP/day) is coherent with the World Cancer Research Fund[225] target for >25 g NSP/day and reflects EFSA's conclusion that dietary fibre intakes higher than 25 g per day may bring wider health benefits.[184]

Notes:

E%: Many of the goals are expressed as a percentage of dietary energy (E%). The total of dietary energy excludes calories obtained from alcohol.

Protein: Although a goal for protein intakes is not necessary in relation to the prevention of cardiovascular disease, a balance of fat, carbohydrate and protein is important. WHO recommends that between 10% and 15% of dietary energy should come from protein.[52]

Total energy (calories) intake should be adequate to support growth and development and to reach and maintain desirable body weight and micronutrient intakes should be adequate to ensure health, according to existing recommendations.

Breastfeeding: No population goal for breastmilk or breastfeeding is included in the table. It is important to emphasise, however, the important health benefits of breastfeeding. On a population basis, we recommend exclusive breastfeeding for the first six months of life, followed by continued breastfeeding with appropriate complementary foods for up to one year of age.

Water: Although it is not included in the table, water is essential for adequate hydration of the body and an adequate water intake is vital. Recommended adequate intakes for total water range from 2.2 to 3.7 litres per day for adults, including water from foods and beverages (including drinking water). Recommendations for water from drinks are around 1.5 litres per day. The European Food Safety Authority has proposed that adequate total water intakes should be 2 litres for women and 2.5 litres for men.[525] These adequate intakes are based on moderate levels of physical activity and a moderate environmental temperature—requirements will be higher in hotter climates or for people involved in vigorous physical activity. Increasing attention is focusing on the potential contribution of different beverages to energy intake, overweight and obesity. With this in mind, it is important that supportive policies are in place to ensure easy access to drinking water.

Folate: EHN's 2002 policy paper contained a population goal of more than 400 µg/day of folate from food. A new review on folate was commissioned for this report (see Section 1.3.9). On the basis of this review of up-to-date evidence the EHN's Expert Group decided that the evidence is not currently sufficient to justify inclusion of a population dietary goal for folate specifically targeted at preventing CVD.

Antioxidants and polyphenols: EHN's 2002 policy paper concluded that there was insufficient evidence to recommend a population goal for antioxidant vitamins. A new review on one group of anti-oxidants, polyphenols, was commissioned for this report (see Section 1.3.10). The EHN's Nutrition Expert Group concluded that there is not currently sufficient conclusive evidence to justify making any public health recommendation for antioxidants.

Alcohol: If alcohol is consumed at all, it should be limited to not more than 20 g alcohol (two drinks) per day in men and not more than 10 g alcohol (one drink) per day in women.[12] Heavy or binge drinkers should be urged to cut and modify their consumption. Binge drinking is defined as an intake of 60 g or more on one occasion, monthly or more often, during the past 12 months. People who already regularly consume small to moderate amounts of alcohol—no more than one drink (c.10 g of alcohol) per day for women or two drinks per day (20 g of alcohol) for men—should not be discouraged from continuing. A similar message can be addressed to patients who already suffered a cardiovascular event.

It is not recommended that adult abstainers begin drinking. This is because even moderate alcohol intake may also be associated with increased risk of other harm and there is a risk that some of those who start to drink will be drawn into consuming more than the very limited amounts of alcohol associated with decreased cardiovascular risk. For these reasons it is not recommended that anyone be advised to begin drinking on health grounds.

Dietary cholesterol: Although dietary cholesterol does impact on blood LDL cholesterol levels, the main dietary determinant of blood LDL cholesterol levels is saturated fat intakes.[512] Dietary

cholesterol is mainly found in foods which are significant sources of saturated fatty acids. For these reasons, no specific recommendation for dietary cholesterol is proposed.

Definitions

Fruit and vegetables: In general, the term "fruit and vegetables" includes fresh, frozen, dried and canned fruits and vegetables. Potatoes are usually excluded because in most European diets these are consumed as starchy staples and are major sources of complex carbohydrates. Beans and pulses may be included, although in some countries these are eaten as a non-animal protein source. Different studies examined in the course of the expert review on fruit and vegetables use a variety of definitions—these are outlined in the detailed scientific review paper.

Added sugar: Added sugar includes sucrose, fructose, maltose, lactose, starch hydrolysate (glucose, high fructose syrup), honey and fruit and berry-concentrates and other isolated sugar preparations, that are used as such or added as component in foods during food preparation.

Dietary fibre: The proposed goals include figures for fibre according to two different definitions in widespread use. Non-starch polysaccharides (NSP) refer to fibre of plant cell wall origin. The Association of Official Analytical Chemists (AOAC) method of measuring dietary fibre includes indigestible material and tends to give a higher value for fibre content of foods, hence the higher population goals proposed.

Dietary implications of the population goals

It is important that population goals are translated into meaningful food-based dietary guidelines at the country level. In this way the dietary advice to the general public is adapted to the context to take account of local eating patterns and cultural factors. There are, however, some broader principles that can be applied.

Nutrient-dense vs energy-dense foods: A diet rich in nutrient-dense foods, rather than one that is rich in foods which are energy-dense and nutrient poor, is important to ensure that the diet can provide essential nutrients and is appropriate for healthy weight maintenance. Foods naturally high in dietary fibre or water—such as vegetables, fruits and cereals prepared without fats and oils—usually have a low energy density. Foods with a high energy density (>225-275 kcal per 100g) include many fatty and sugary processed foods, such as some ready meals, snacks, baked goods, desserts and confectionery. Policy makers need to ensure that people, across the socio-economic spectrum, have access to nutrient dense foods such as fruits, vegetables, legumes, whole grains, lean meats and low-fat dairy products.

Towards a plant-based diet: In many countries, a diet that meets these population goals favours a shift towards a predominantly plant-based diet from an animal-based diet. In other words, a diet that has higher intakes of vegetables, fruits and whole grains and lower meat and dairy intakes. This shift would also be in the right direction in terms of climate change and food security (see Section 2.5).

Fish consumption: The population goal for an intake of 250-500 mg/day of very long chain n-3 polyunsaturated fatty acids is consistent with twice weekly consumption of oily fish. Unfortunately, despite good nutritional reasons to support a recommendation for frequent consumption of fish, the European Heart Network is aware of the potential consequences of such a recommendation on the current state of the world's fish stocks. Robust regulatory policies to ensure the sustainable management of fisheries and other marine resources are clearly essential to ensure that future generations are able to enjoy fish as part of a healthy diet.

1.3.12 The Mediterranean diet

The previous sections have reviewed evidence on individual nutrients, foods or food components. Several investigators, however, have focused on the epidemiological evidence concerning particular types of diet. Since foods and drinks are not consumed in isolation, they have studied different dietary patterns—such as vegetarian, vegan or religious diets—rather than looking for associations between individual nutrients and health. One dietary pattern which has been the subject of intense interest is the traditional Mediterranean diet of southern Europe.

What is the Mediterranean diet?

Traditional diets vary across the Mediterranean region but tend to be largely plant-based—meat being consumed in small amounts and relatively infrequently—with high intakes of vegetables, fruits, fish, nuts, grains, pulses and with olive oil as the principal contributor to fat intakes. Scientific interest in the idea of the Mediterranean diet first centred on the diets of different communities—such as Greece (Crete and Corfu), Croatia, Serbia and Italy. Studies by Ancel Keys and colleagues in the early 1960s were was the starting point for highlighting the benefits of the pattern of eating associated with the "Mediterranean Diet". Different scoring systems have been developed to enable researchers to assess how diets measure up to a traditional Mediterranean diet. The elements making up these scores generally include:

High intakes of beneficial components:
- Vegetables
- Legumes (pulses)
- Fruits and nuts
- Whole grains
- Fish and other seafood

Olive oil (a high ratio of monunsaturated lipids to saturated fats) as the predominant oil

Low intakes of:
- Meat
- Poultry
- Dairy products
- Sugar containing products

Moderate alcohol intakes (defined differently depending on the scoring system, with the upper limits of moderate ranging from 30 g to 50 g of alcohol per day for men and 15 g to 25 g for women)

Since the initial interest in the Mediterranean diet, researchers have gone on to define such a diet[526,527] in general terms (see box), and to compare cardiovascular risk and mortality of people with differing degrees of adherence to the Mediterranean diet. Recent meta-analyses[243,528] found that the Mediterranean diet appears to be protective against cardiovascular disease (all nine studies) and total mortality (all 10 studies).

Trichopoulou and colleagues have gone on to identify the contribution of the different components of the Mediterranean diet to the reduction in mortality in the Greek cohort of the European prospective cancer study (EPIC).[529] Of the nine components of the Mediterranean diet studied, those making the greatest contribution to lower mortality were:

- moderate (rather than excessive or minimal) alcohol consumption (24%);
- low consumption of meat and meat products (17%);
- high consumption of vegetables (16%);
- high consumption of fruits and nuts (11%);
- high monounsaturated to saturated fat ratio (c11%);
- high consumption of legumes (pulses) (10%).

Moderate consumption of alcohol—mostly as wine with meals—is a traditional part of the Mediterranean diet. The authors caution, however, that recall of alcohol intake is likely to be better than that for the other eight components of the diet and this may result in overestimation of the contribution of alcohol to the Mediterranean diet's health benefits. (See chapter 1.3.8 for a review of the evidence on alcohol and cardiovascular disease).

Further research is ongoing to investigate the health effects of the Mediterranean diet and, importantly, to examine whether there are synergistic interactions between the different components of the diet.

This paper has followed the approach taken in our 2002 report by proposing population goals for each nutrient or food component. This reflects, among other things, the fact that there is more research into the health effects of individual foods, nutrients or food components than into diets (which are difficult to define and characterise). Issues relevant to the Mediterranean diet, however, are tackled elsewhere in this chapter (see the sections on fruit and vegetables (1.3.5), fats (1.3.1), alcohol (1.3.8), anti-oxidants (1.3.10) and folate (1.3.9)).

Despite the scientific interest in its potential health benefits, the Mediterranean diet is under threat. The diet observed by Keys and colleagues in the early 1960s is vanishing. Section 1.4 3 describes how traditional foods in southern Europe are being replaced by a more modern diet and lifestyle, particularly among younger generations.

1.3.13 Promoting lifestyle strategies rather than a pharmacological approach

This report promotes a public health approach to preventing cardiovascular disease through diet and physical activity, rather than a pharmacological strategy. Despite recent advances in medicine, including the now widespread use of statins to manage cholesterol levels, lifestyle strategies have the greatest potential to reduce cardiovascular risk in the general population.

In addition, the benefits of lifestyle strategies should be more readily accessible to all (notwithstanding governments' responsibilities to ensure that all socio-economic groups have access to affordable healthy diets and a healthy built environment). The benefits of pharmacological interventions will only reach those who have access to affordable treatment—this may exclude the populations of Europe's poorest countries and the most disadvantaged groups throughout Europe.

1.3.13.1 The polypill

In recent years there has been growing interest in the use of drugs to alter cardiovascular risk factors. Drugs to lower blood pressure, statins to lower blood lipids and aspirin (anti-platelet therapy) are now widely used. In addition, there has been considerable interest in the concept of a "polypill" which combines several medications to prevent cardiovascular disease. A single pill, it was proposed, would be easier for people to take and would, therefore, improve treatment compliance. A polypill combining several generic medications would, furthermore, be relatively inexpensive and could be used widely in low- and middle-income countries.

The original polypill concept, put forward by Wald and Law in the BMJ in 2003,[530] consisted of three blood-pressure lowering drugs at low doses, a statin, aspirin, and folic acid. The authors suggested that, if taken by everyone over the age of 55, the polypill had the potential to reduce cardiovascular events by over 80%. A small number of trials have since been initiated of different polypill formulations—although interest has largely focused on the use of the polypill in people with signs of existing cardiovascular disease (secondary prevention) rather than population-based primary prevention as Wald and Law had suggested.[531-533]

Whether the polypill concept lives up to its promise remains to be seen. What is clear, however, is that any pharmacological approach should be used in conjunction with a lifestyle strategy to prevent cardiovascular disease. Medication is used to target specific risk factors without addressing a poor diet, or other lifestyle factors. A lifestyle strategy, on the other hand, reduces overall risk and can also enhance the improvements that may be obtained by a more medical approach.

1.3.13.2 Supplements and functional foods

The past 20 years have also seen a growth in the availability of dietary supplements and "functional foods" (foods modified with the purpose of having specific disease-preventing or health-promoting effects beyond basic nutrition). Shoppers are faced with a wide array of products on the shelves and in the chill cabinets bearing claims for the possible health benefits of eating particular food products.

In 2006 the EU adopted a regulation on nutrition and health claims with the aim of, among other things, protecting consumers from false or misleading claims, and ensuring that any health claims made on food labels in the EU are backed up by evidence. As a result, the European Food Safety Authority (EFSA) is responsible for checking the evidence behind health claims on food products and the European Commission and member states then decide whether to authorise the claims.

A list of over 4,600 health claims (whittled down from 44,000) on food has been compiled for the Panel on Dietetic Products, Nutrition and Allergies (NDA) to assess. By the end of 2010, the Panel had assessed 1,745 claims. So far, the vast majority of claims have been rejected.

EFSA approved health claims relevant to cardiovascular risk factors

In relation to major cardiovascular risk factors, namely blood cholesterol, triglyceride levels and blood pressure, EFSA's NDA Panel has assessed claims for the following substances positively (until December 2010).

Alpha linolenic acid: an essential n-3 polyunsaturated fatty acid. EFSA approved claims that alpha linolenic acid (ALA) contributes to maintenance of normal blood cholesterol concentrations for foods providing at least 15% of the proposed labelling reference intake of 2 g of ALA per day, which can be obtained as part of a balanced diet. (See Section 1.3.1 for more on alpha-linolenic acid and other polyunsaturated fatty acids.)

Beta glucans: soluble cereal fibres (non-starch polysaccharides) which are found naturally in bran from barley, oats, rye and wheat. EFSA's opinion related to both beta-glucans naturally found in foods and added to foods.[534] The Panel concluded that beta-glucans from non-processed or minimally-processed oat or barley have been shown to significantly reduce LDL-cholesterol levels. Some human intervention studies with relatively high doses of beta-glucan added to processed foods had failed to show an effect—the cholesterol lowering effect of beta-glucans may be weakened by some processing and manufacturing techniques. EFSA proposed, therefore, that foods providing at least 3 g/day of beta-glucans from oats, oat bran, barley, barley bran, or from mixtures of non-processed or minimally-processed beta-glucans should be able to carry the claim "Regular consumption of beta-glucans contributes to maintenance of normal blood cholesterol concentrations."

DHA and EPA: the very long chain n-3 polyunsaturated fatty acids docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) are found in fish oils. EFSA's Panel concluded that DHA in combination with EPA can contribute to the reduction of levels of blood triglycerides and to maintenance of normal blood pressure.[535] Intakes of EPA and DHA of 2 g/day—which can be consumed as part of a balanced diet—are needed to obtain the claimed effect on triglycerides. Around 3 g/day are needed for the claimed effect on blood pressure. DHA alone was assessed as helping to maintain normal levels of blood triglycerides.[536] A claim that DHA and EPA contribute to normal cardiac function was also approved—the panel notes that intakes of EPA and DHA of about 250 mg per day are needed for this effect to be realised. See Section 1.3.1 for more on DHA and EPA.

Guar gum: a water-soluble fibre (a galactomannan) that is not digested in the human gut and is used as a thickener in foods. EFSA concluded that a cause and effect relationship has been established between the consumption of guar gum and the reduction of blood cholesterol concentrations.[537] Foods should provide at least 10 g per day of guar gum per day to be able to bear a claim.

Glucomannan: a water-soluble fibre food additive, also known as Konjac mannan, derived from the Konjac plant. EFSA approved claims that 'regular consumption of glucomannan helps maintain normal blood cholesterol concentrations'.[538]

Hydroxypropyl methylcellulose (HPMC): a food additive used to emulsify or thicken foods and which is not digested in the human gut. EFSA concluded that HPMC can contribute to maintenance of normal blood cholesterol levels if at least 5 g per day of HPMC is consumed in two or more servings.

Linoleic acid: an omega-6 polyunsaturated fatty acid found in some vegetable oils and legumes. EFSA has approved claims that "Linoleic acid may help to maintain normal blood cholesterol concentrations for foods" that contain at least 15% of the proposed labelling reference value of 10 g of linoleic acid per day, which can be obtained as part of a balanced diet.[539] (See section 1.3.1)

Pectins: found in fruit and vegetables, and also used as thickeners by the food industry. They are not available for digestion in the human gut. EFSA considers pectins help to maintain normal blood cholesterol levels.[540] In order to bear the claims, foods should provide at least 6 g per day of pectins.

Plant sterols/plant stanols: found in a range of foods (vegetables, vegetable oils, nuts, grains, seeds and legumes) and added by the food industry to a range of products such as yoghurts, spreads and other dairy products. EFSA found a clinically significant LDL-cholesterol lowering effect from doses between 1.5 and 2.4 g of plant sterols/stanols.[541] The Panel found no difference between sterols and stanols at those doses. Studies in lower doses (0.8-1.0 g) also suggest statistically significant lowering of LDL-cholesterol levels in people with normal or slightly raised levels. To be able to carry the claim "plant sterols/stanols contribute to the maintenance of normal blood cholesterol levels" foods must provide at least 0.8 g per day.

The above list does not include the many claims that were approved for vitamins and minerals. Nor does it include claims that were approved for other effects that might be indirectly beneficial to cardiovascular health (eg maintenance of normal body weight).

In our 2002 report, EHN questioned whether manipulation of food ingredients is a useful approach for cardiovascular disease prevention in the whole population. In the intervening years the European Commission, through EFSA, has made progress towards tackling concerns about the safety and efficacy of supplements and functional foods. Nonetheless, serious questions remain.

There are questions, for example, about how effective this pharmacological type of approach involving foods can be in tackling the broader problem of poor diet: will people just eat functional foods while continuing to consume many foods high in saturated fat, salt and sugars? There are also worries that some people on cholesterol-lowering medication, for example, may think that they can stop taking their medicine if they use functional food products. Furthermore, the healthy eating and physical activity patterns proposed in the previous sections are not only effective in relation to heart disease or stroke—they have also been shown to help prevent a wide range of chronic diseases.

There are also major concerns about equality of access. Given that existing inequalities mean that poorer Europeans suffer most from cardiovascular disease, real solutions need to be readily accessible and affordable for all. The claimed health benefits of supplements and functional foods, however, tend to be more expensive—making the products inaccessible to those most in need.

Access is also an issue affecting participation in physical activity. Active travel offers an affordable and convenient source of physical activity, yet inactive modes of transport have increasingly dominated in recent years. Addressing this is a priority, through developing local transport plans which encourage support walking and cycling, and removing barriers to physical activity, such as subsidised parking. Children must have adequate opportunities throughout the day to be physically active, including access to parks and green spaces for play, as well as the provision of physical activity at school. Policy makers should pay particular attention to the needs of hard to reach and disadvantaged communities when developing infrastructures to promote physical activity.

It is increasingly clear that sustainable long-term solutions require serious changes in the food system and the foods it produces. It is also important to change the broader environment which influences what we eat and how active we are. Too much of a focus on functional foods—generating hopes for a technical “quick fix” to these problems—may divert attention from the real dietary changes needed. Similarly, a focus on schemes to promote physical activity needs to be underpinned by policies that support physical activity across the health, transport, education, and sport/recreation systems and services. Governments must not be distracted from making the strategic changes which are needed.

1.4 CHANGING PATTERNS OF DIET AND PHYSICAL ACTIVITY IN EUROPE

This chapter has been contributed by Dr Tim Lobstein of the International Association for the Study of Obesity, UK.

Despite our relatively clear and consistent understanding of the links between diet, physical activity and cardiovascular disease, the prevention of cardiovascular disease—and other diseases linked to diet and physical activity—suffers from several substantial problems. The first of these is that the practical assessment of people's diets and physical activity levels is a very inaccurate science with a variety of approaches using different methodologies and suffering different degrees of under-reporting, over-reporting and distortion; the second is that detailed and representative dietary intake and behavioural surveys are expensive and hence only undertaken occasionally; and thirdly as a result there is a serious lack of survey material in Europe and a very poor level of comparability between surveys in different countries. Put simply, we cannot say with certainty exactly who is eating what in Europe, how active they are or how things might have changed in recent decades.

However, there are important indicators that can provide some guidance on what is happening. In this section we have used some dietary survey material where the surveys are sufficiently comparable to bear detailed examination. We have also made extensive use of indirect evidence, largely from food production and supply statistics, to illustrate the patterns and trends in food supplies in the European Region. It is important to note that food supply statistics from the Food and Agriculture Organization (FAO) take account of imports, exports, stock levels and waste during production, but do not take account of waste after the food is sold to consumers. Also, the methods for collecting these data are not necessarily identical in each country, and therefore comparisons between countries should be treated cautiously. However, on the assumption that the methods for collecting data within a country do not change significantly year on year, trends over time for a country or group of countries are more reliable.

There are also serious methodological issues in relation to measuring physical activity (see section 1.3.7). Most of the data available have been obtained using measures which depend on subjective assessments of physical activity and cannot give an accurate picture of activity levels. It is, therefore, very difficult to make comparisons between countries and over time.

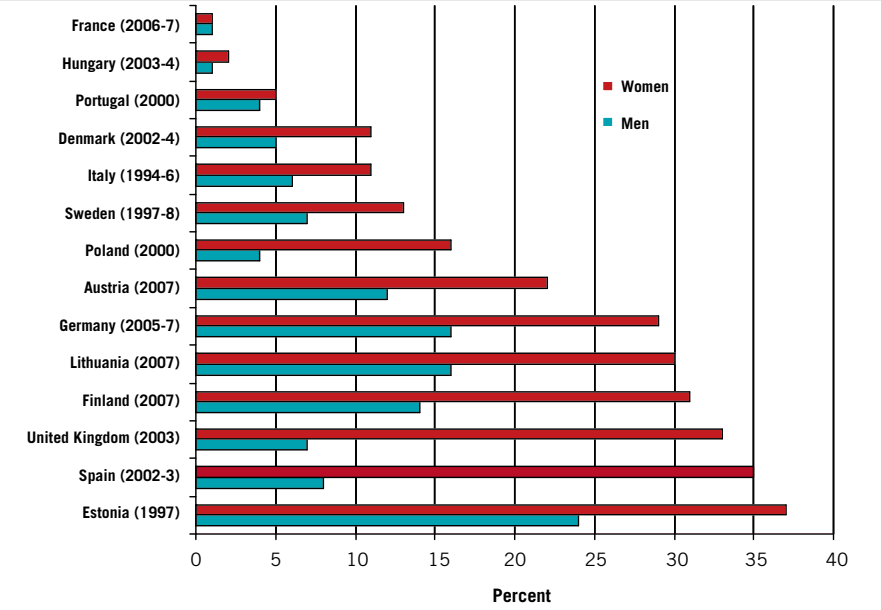
1.4.1 Food components

1.4.1.1 Salt

Salt plays an important role in food manufacturing by making processed, and potentially poorer-flavoured, foods more palatable and enjoyable. The use of salt is likely to be linked to consumption of snack foods, fast foods, and savoury products such as processed meats.

Production statistics are very poor sources of information on salt (sodium chloride) consumption. Estimates of salt intake have been made in several European dietary surveys and these indicate a continuing high level of consumption compared with the level recommended in national and international dietary guidelines. A review of dietary surveys of salt consumption in 14 EU member states by Elmadfa and colleagues[542] found average population intakes varied across countries from 6.5 to 18.3 grams per day for men and 4.3 to 14.0 grams per day for women, compared to WHO's recommended intake of less than 5 grams per day for both population groups.[52] The highest consumers were found in Hungary. An estimate of the proportion of the adult population achieving the recommended consumption levels in each of the surveyed countries is shown in Figure 19. It should be noted that the data are derived from national surveys undertaken at different times, using different dietary intake estimation methods.

Figure 19 Percent of adult population consuming less than 5 g salt per day
Re-calculated from dietary survey data reported in Elmadfa 2009

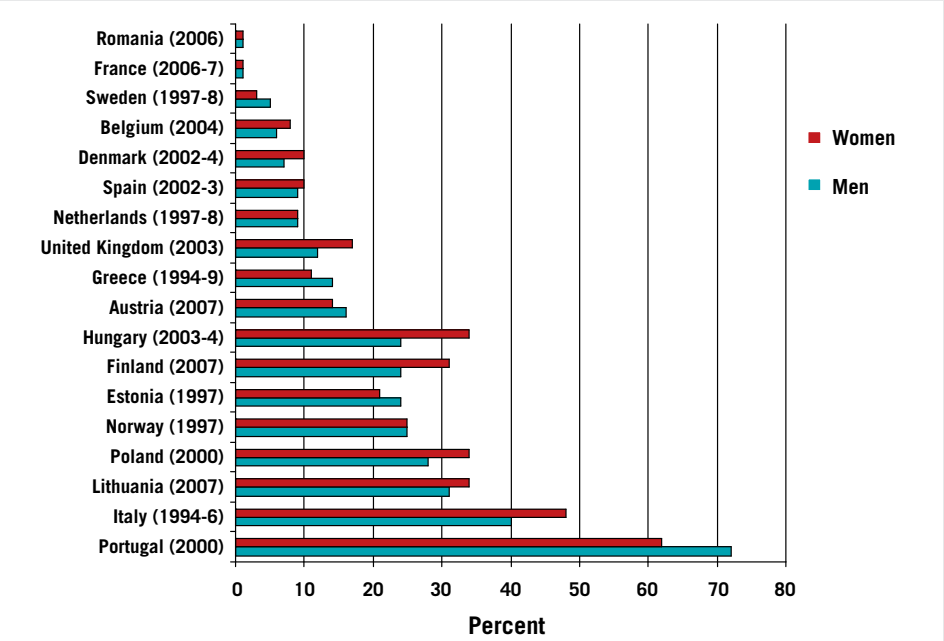


1.4.1.2 Saturated fat

Saturated fatty acids are found in several sources of food: meat and meat products, milk and dairy products, and to a lesser extent in seeds, nuts and vegetable oils. Dietary surveys of saturated fat consumption

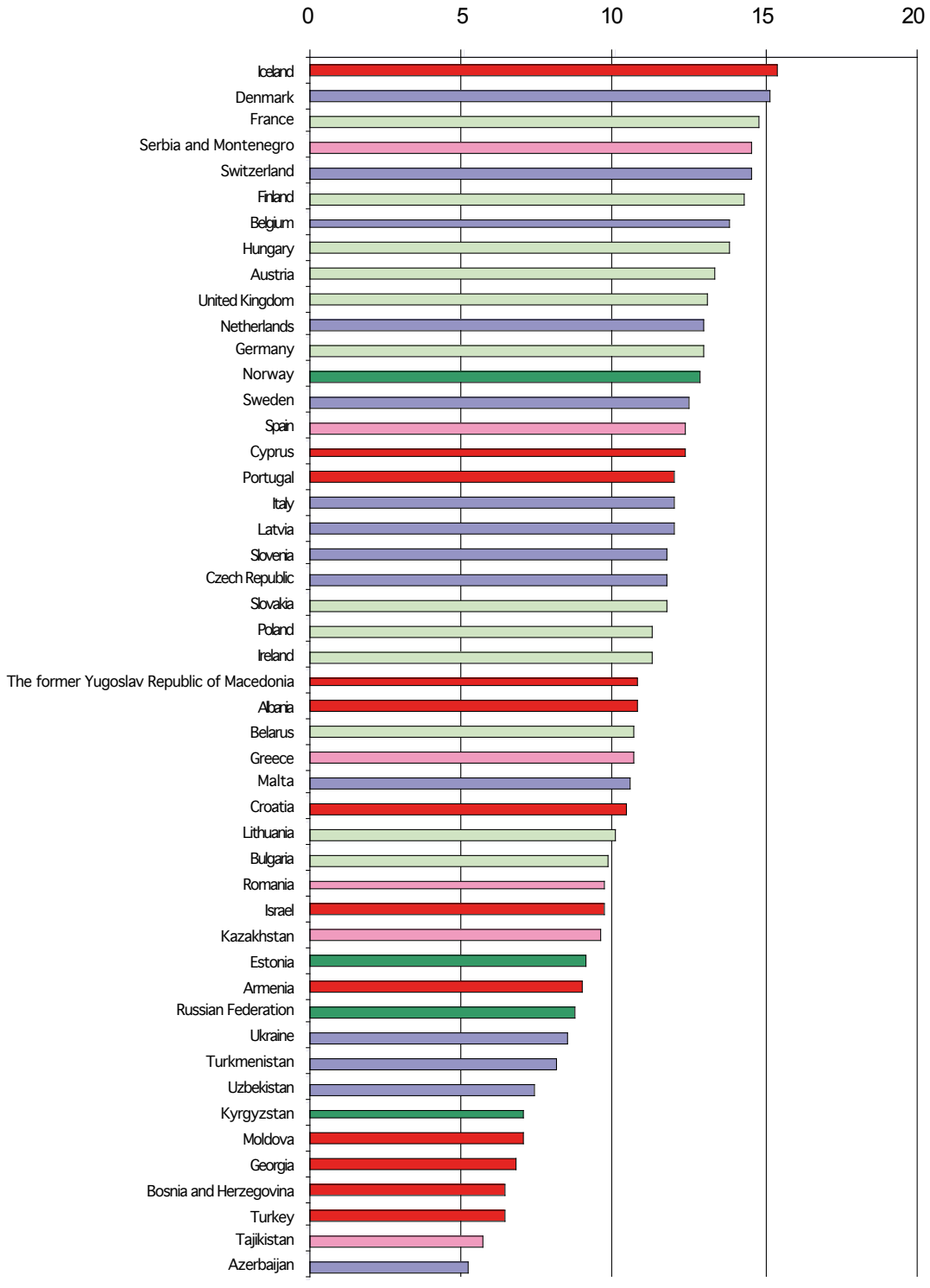
In a review of surveys of adult diets in 18 EU member states, Elmafda[542] found that average population intake ranged from 8.8% to 26.3% of dietary energy for men, and 9.4% to 24.8% of dietary energy for women, compared to WHO's recommended level of intake of less than 10% dietary energy for both population groups.[52] The highest consumers were found in Romania. An estimate of the proportion of the adult population achieving the recommended consumption levels in each of the surveyed countries is shown in Figure 20.

FIGURE 20 PERCENT OF ADULT POPULATION CONSUMING LESS THAN 10% ENERGY FROM SATURATED FAT RE-CALCULATED FROM DIETARY SURVEY DATA REPORTED IN ELMADFA 2009



However, these figures rely on relatively small sample sizes and relatively crude assumptions about the saturated fat content of the foods being eaten. In terms of food supplies, a rough calculation of the saturated fat content as a percentage of the total food energy can be made, based on assumptions on meat, dairy and vegetable oil contents applied uniformly across all countries. The results are shown in Figure 21 overleaf.

FIGURE 21 SATURATED FAT AS A PERCENTAGE OF ENERGY AND CHANGE (ILLUSTRATED BY THE COLOUR CODE) IN SATURATED FAT IN THE FOOD SUPPLY BETWEEN 1992/3 AND 2006/7



Source: calculated from FAO food supply tables (http://faostat.fao.org). The bars illustrate supply levels in 2006/7, the colour coding illustrates the change since 1992/3. Pink = >5% increase. Red = >10% increase. Pale green = > 5% decrease. Dark green = > 10% decrease. Purple = stable. Less than 5% increase or decrease.

1.4.1.3 Trans fats

Trans fatty acids are found naturally in small quantities in dairy foods, and potentially in larger quantities in processed foods, where they have been added from artificially-generated sources (usually from partial hydrogenation of vegetable oils, a process which serves to extend the shelf life of the oil). They have been in use in Europe in margarines and in the fats used for baked goods (e.g. biscuits, pastries, pies) and deep fried foods (e.g. French fries).

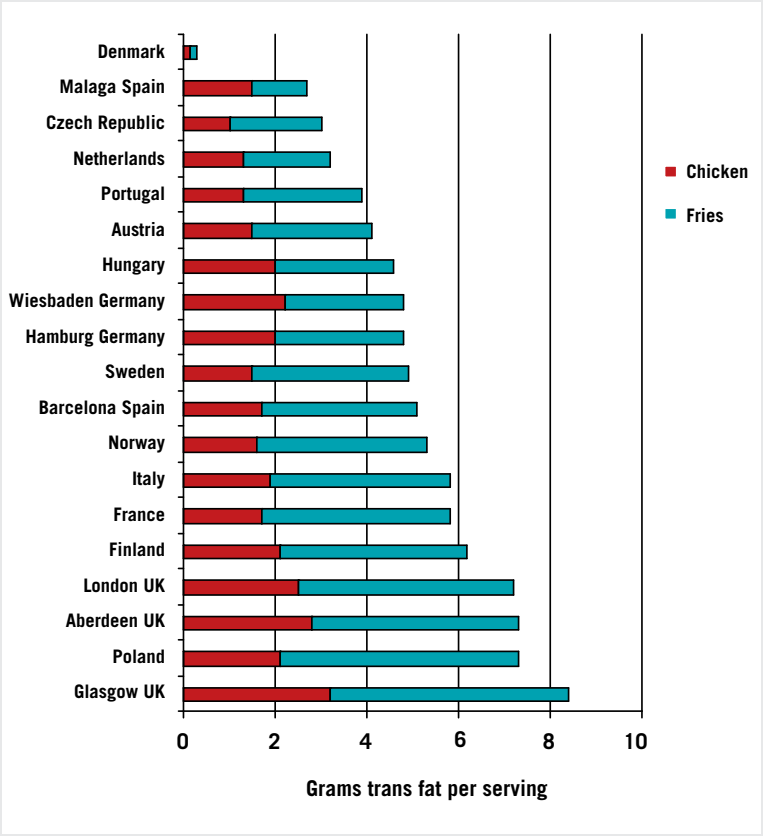
Few surveys of trans fat content of dietary intake have been reported. The trans fat content of some foods has been reported for a sample of products taken in a survey conducted between November 2004 and November 2005 (the composition of these foods is likely to have changed considerably since then). The researchers measured the quantity of trans fat found in a standard single portion of French fries and a standard single portion of chicken in McDonald's and KFC fast food outlets in 13 cities in Europe and the results are shown in Figure 22. Follow-up research on in the same places by Dr Stender in 2009 found that the amount of trans fatty acids had decreased dramatically since 2005, but that popular foods with high amounts of trans fatty acids were still easily available in eastern Europe.[543]

The results shown in Figure 22 relate to major fast food chains and further research is needed into trans fat levels in individual fast food outlets, which may present considerable cause for concern.

There is no known safe level of consumption, and WHO's current the recommendation is for population intakes to average below 1% of total food energy, equivalent to less than two grams of trans fat per day.[52]

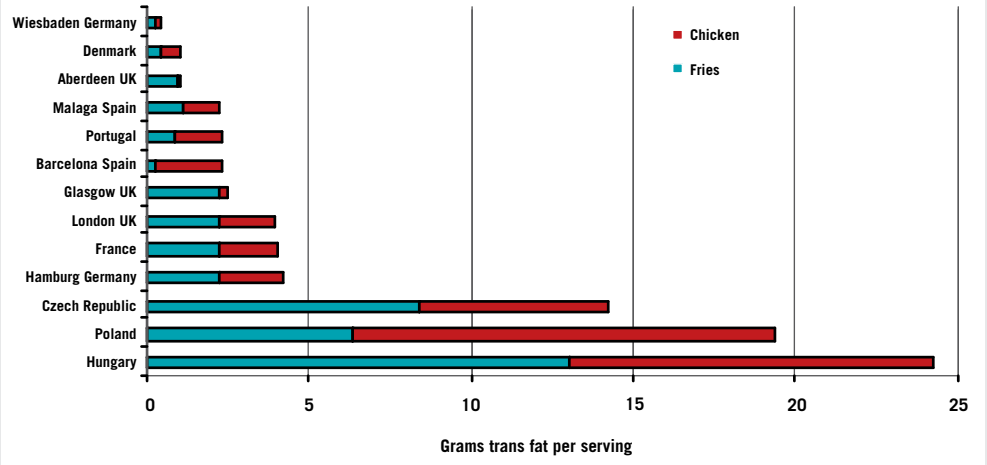
It appears in some national surveys that the consumption of fast food products is likely to show social class differences, with lower income groups, and especially young men in lower income groups, likely to be the highest users of fast food outlets.[544] For this sub-group of the population, a significantly higher than average exposure to a diet with high levels of trans fats may be occurring.

FIGURE 22 GRAMS OF TRANS FAT FOUND IN A SINGLE SERVING OF CHICKEN AND FRENCH FRIES FROM MCDONALD'S (UPPER) AND KFC (LOWER) FAST FOOD OUTLETS IN 2005



Source: Stender et al 2006[513]

TRANS FAT FOUND IN KFC CHICKEN AND FRIES IN 2005



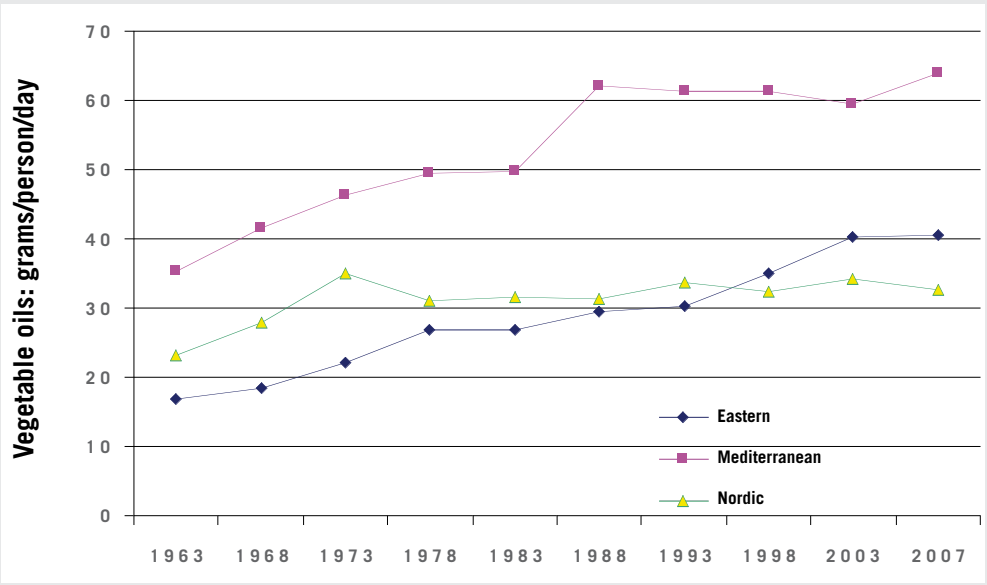
1.4.1.4 Vegetable oils

Although olive oil has been a core part of the Mediterranean diet for centuries, the arrival of mass-produced, cheap vegetable oils from soy, sunflower, rape and other seed and nut sources has been relatively recent. These oils vary in their fatty acid composition but like olive oil they share one feature, namely they are among the most energy-dense forms of food ingredient, typically providing some 9,000 kcals per litre.

Of all the food ingredients which can be mapped in production trends world wide, vegetable oils have risen most significantly in the last half-century. Their use in Europe is no exception. Trends in vegetable oil supplies are shown in Figure 23 below, which indicates trends in three European regions.

In northern Europe, vegetable oils have largely replaced animal fats as a cooking medium in domestic food consumption. The high levels of vegetable oil use in Mediterranean countries indicates that supplies of oils from crops other than olives are being consumed in addition to the traditional consumption of olive oil. In all regions, vegetable oils are used in a wide range of processed foods and fast food cooking processes.

FIGURE 23 REGIONAL TRENDS IN VEGETABLE OIL SUPPLIES IN EUROPE

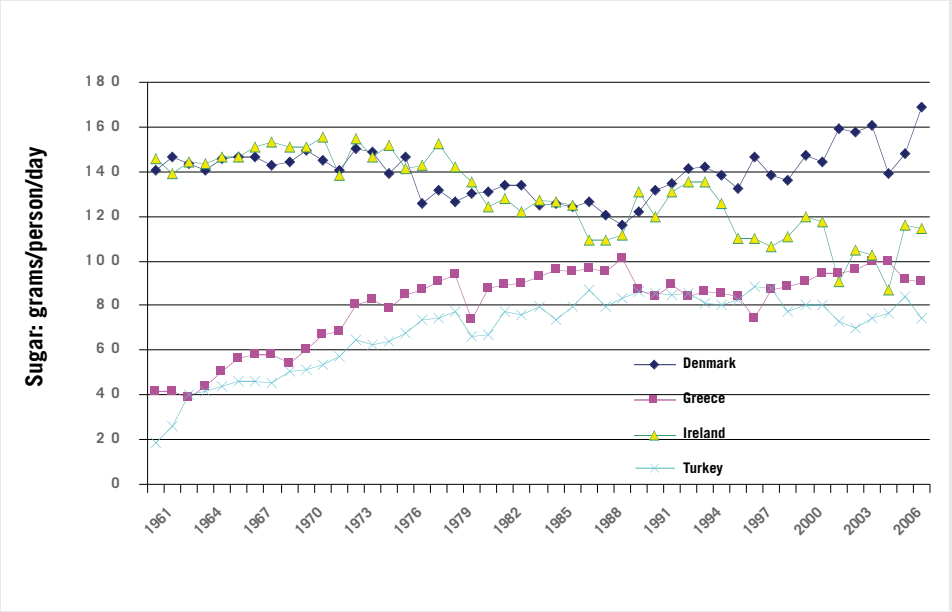


Source: calculated from FAO food supply tables (http://faostat.fao.org).
Eastern = Bulgaria, Hungary, Poland, Romania
Nordic = Denmark, Finland, Norway, Sweden
Mediterranean = Greece, Portugal, Spain, Turkey

1.4.1.5 Sugar

Like vegetable oils, sugar is a relatively dense form of energy, with a kilogram providing some 3,900 kcals. Consumption of sugar has been high in northern Europe for many decades, but has also increased in southern Europe in recent years. The trends can be illustrated using two typical countries from each region, as shown in Figure 24.

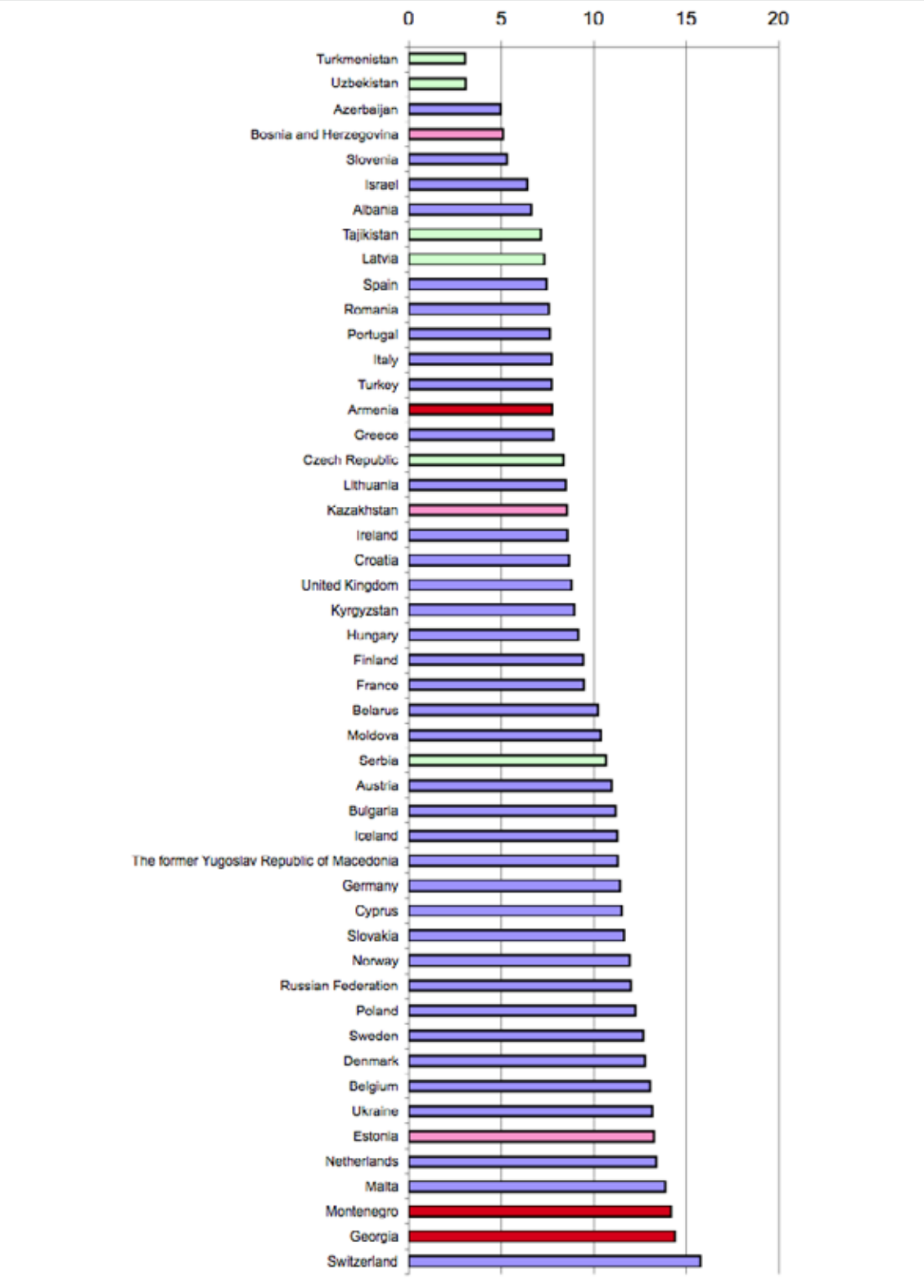
FIGURE 24 FOUR CASES STUDIES: TRENDS IN THE SUPPLY OF SUGAR IN DENMARK, IRELAND, GREECE AND TURKEY



Source: calculated from FAO food supply tables (http://faostat.fao.org).

Overall sugar supplies are generally above the most recent WHO recommended levels of under 10% food energy. Several countries are below this level, although for some of these the reported levels may be temporary as they have recently shown a significant decline in sugar supply.

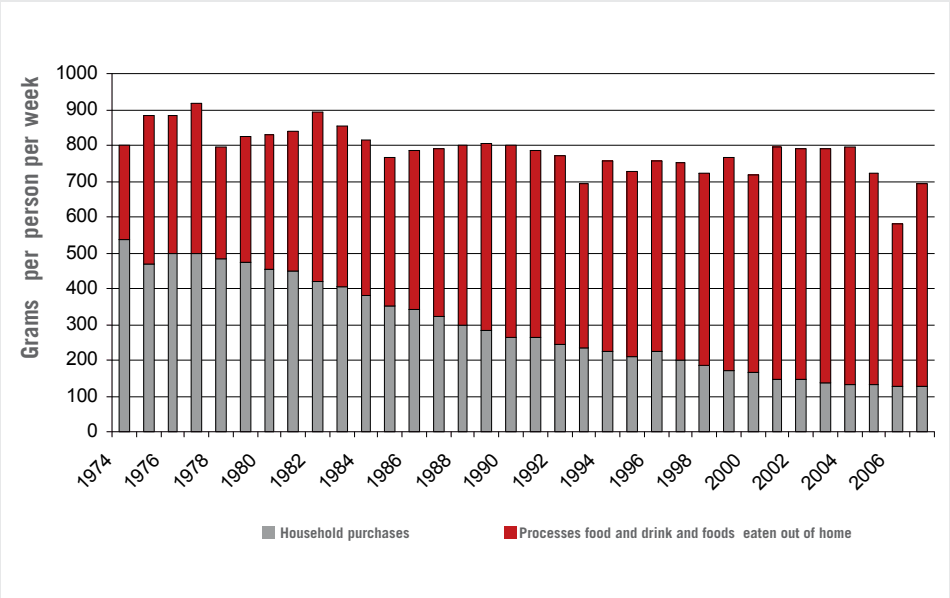
Figure 25 Sugar in the food supply as a percentage of total food supply energy levels, 2006-2007. Colours indicate change since 1992-1993.



Source: calculated from FAO food supply tables (http://faostat.fao.org).
Pink = >30% increase. Red = >100% increase. Pale green = >30% decrease. Blue= stable. Less than 30% increase or decrease.

Sugar consumption is largely in the form of 'hidden' sugars, i.e. sugar in processed food products. Household purchases of sugar have dropped dramatically in recent years, but the amount present in the food supply has continued at high levels—reflecting the sugar present in processed foods and drinks and foods eaten outside the home. Data from the UK, for example, illustrate this trend clearly, as shown in Figure 26.

FIGURE 26 SUGAR IN THE UK FOOD SUPPLY. HOUSEHOLD PURCHASES HAVE BEEN LARGELY REPLACED BY SUGAR IN PROCESSED FOODS AND FOODS EATEN OUTSIDE THE HOME



Source: calculated from FAO food supply tables (http://faostat.fao.org) and UK Household Food Survey and Family Food (https://statistics.defra.gov.uk/)

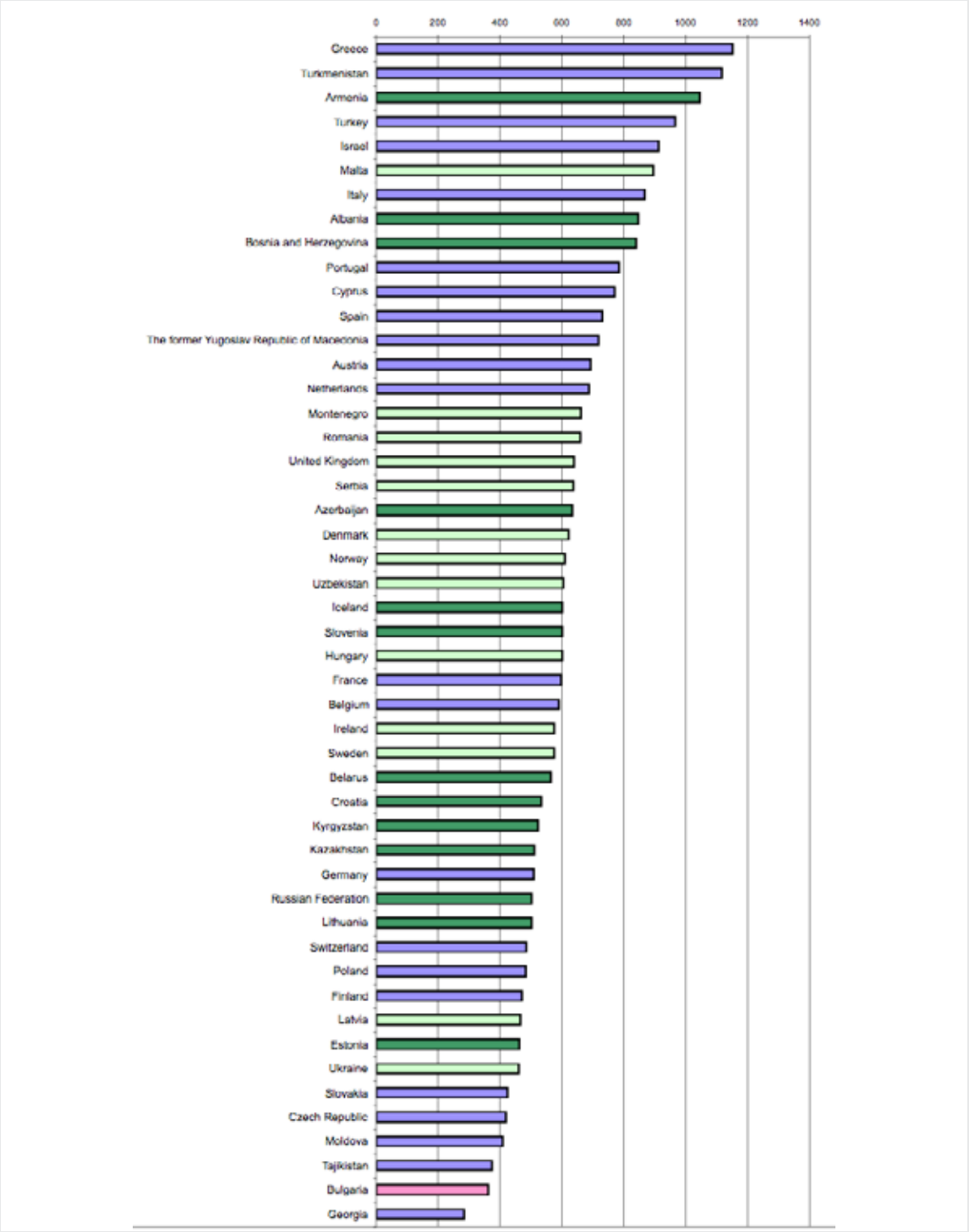
For more information on trends in sugar-sweetened processed foods, see Confectionery and Soft Drinks, below.

1.4.2 FOODS AND DRINKS

1.4.2.1 Fruit and vegetables

Highly recommended for the prevention of many diet-related diseases, fruits, vegetables, nuts and pulses are being provided in greater quantities throughout much of Europe. The supply figures do not distinguish fruits and vegetables eaten in relatively unprocessed form, or as juices, syrups, purees and other processed forms, often in association with additional fats and sugars. Please note that the figures for food supplies do not account for waste after the food has been sold for consumption. In the case of fruit and vegetables the waste may be a significant fraction of the total supplied, with some suggestions that over 700 g may need to be supplied for 400 g to be consumed.[545]

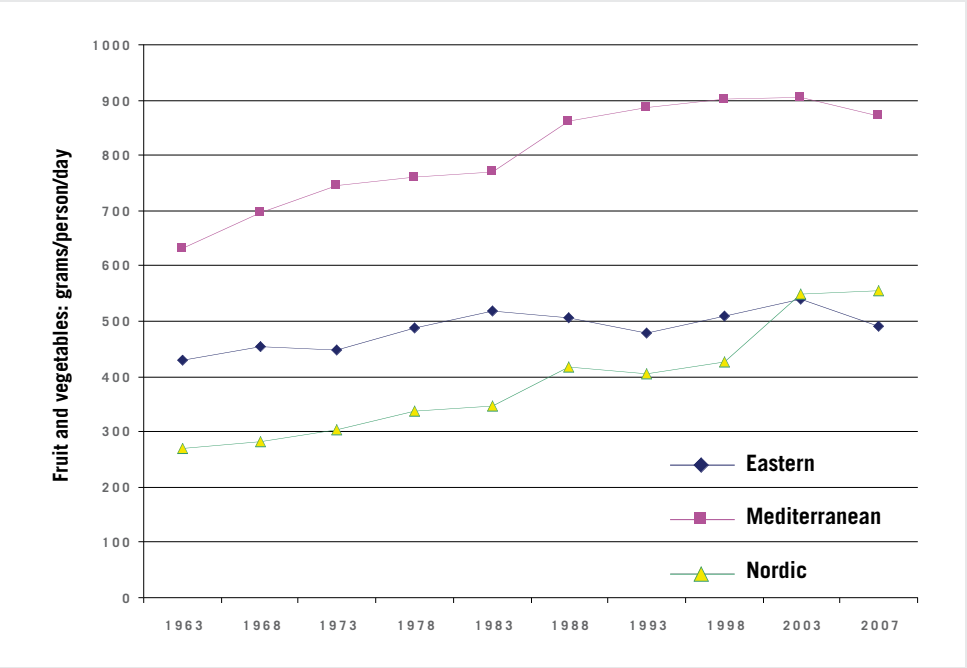
Figure 27 Fruit and vegetables (including pulses and nuts) in the food supply, 2006-2007. Colours indicate change since 1992-1993.



Pale green = > 25% increase. Dark green = >50% increase. Blue = stable. Less than 25% increase.

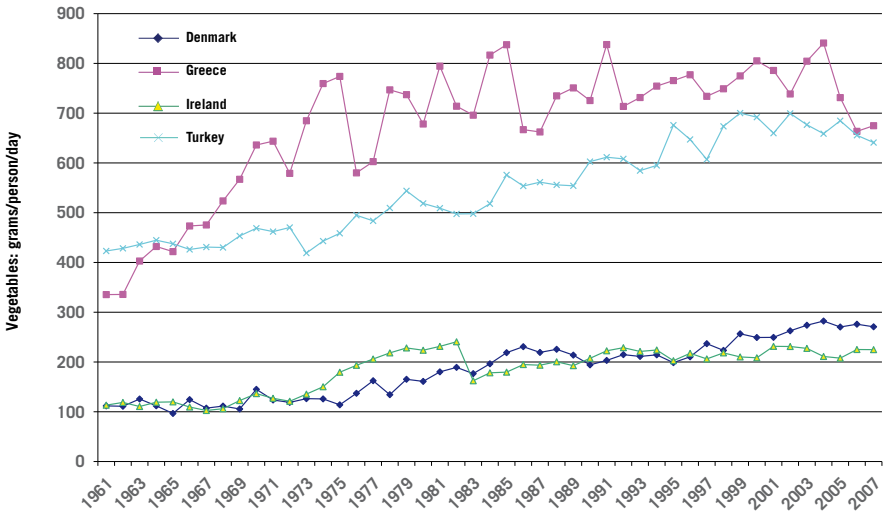
Recent trends indicate a continuing increase in fruit and vegetable supplies in much of Europe, with Mediterranean countries continuing to enjoy the highest consumption levels (Figure 28). However, specific country analyses indicate that this region may be increasing its vegetable consumption (whether in fresh or processed form is not clear), while northern European countries increase their fruit consumption (again the form is not clear) (Figures 29 and 30).

FIGURE 28 REGIONAL TRENDS IN FRUIT AND VEGETABLE SUPPLIES



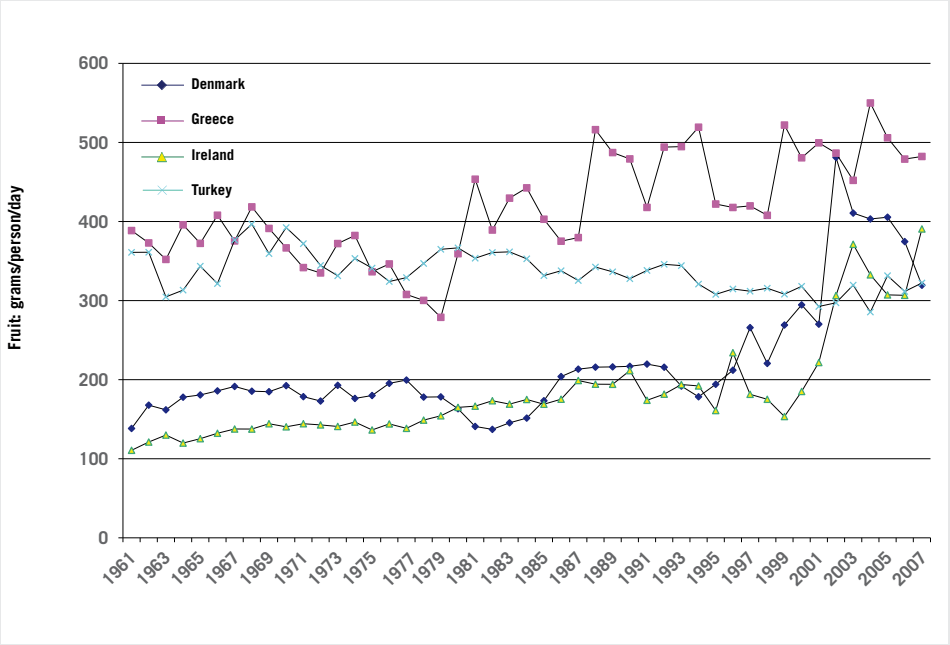
Source: calculated from FAO food supply tables (http://faostat.fao.org).
Eastern = Bulgaria, Hungary, Poland, Romania
Nordic = Denmark, Finland, Norway, Sweden
Mediterranean = Greece, Portugal, Spain, Turkey

FIGURE 29 FOUR CASES STUDIES: TRENDS IN THE SUPPLY OF VEGETABLES (AND PULSES) IN DENMARK, IRELAND, GREECE AND TURKEY



Source: calculated from FAO food supply tables (http://faostat.fao.org).

Figure 30 Four cases studies: trends in the supply of fruit (and nuts) in Denmark, Ireland, Greece and Turkey

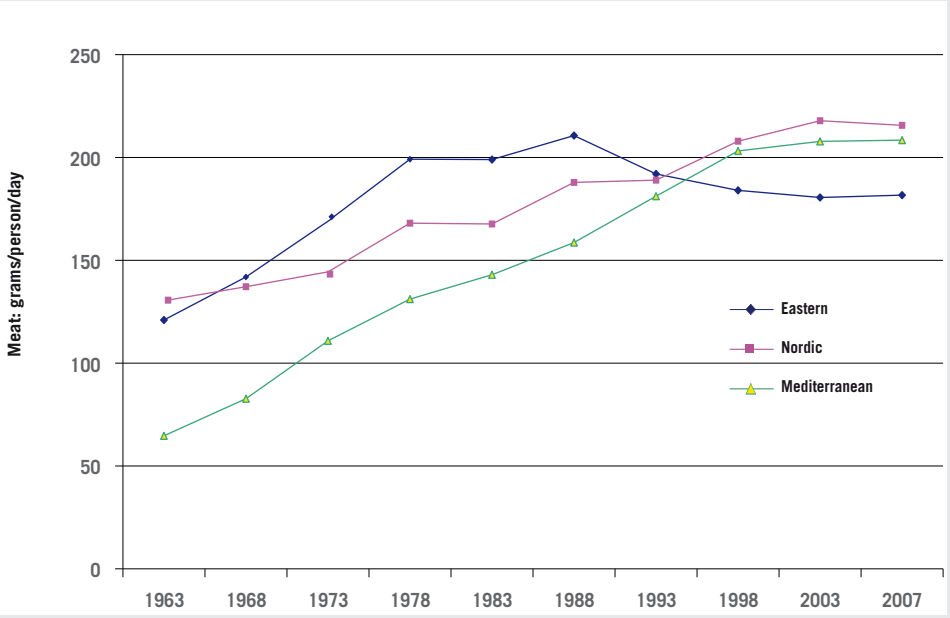


Source: calculated from FAO food supply tables (http://faostat.fao.org).

1.4.2.2 Meat, milk and butter

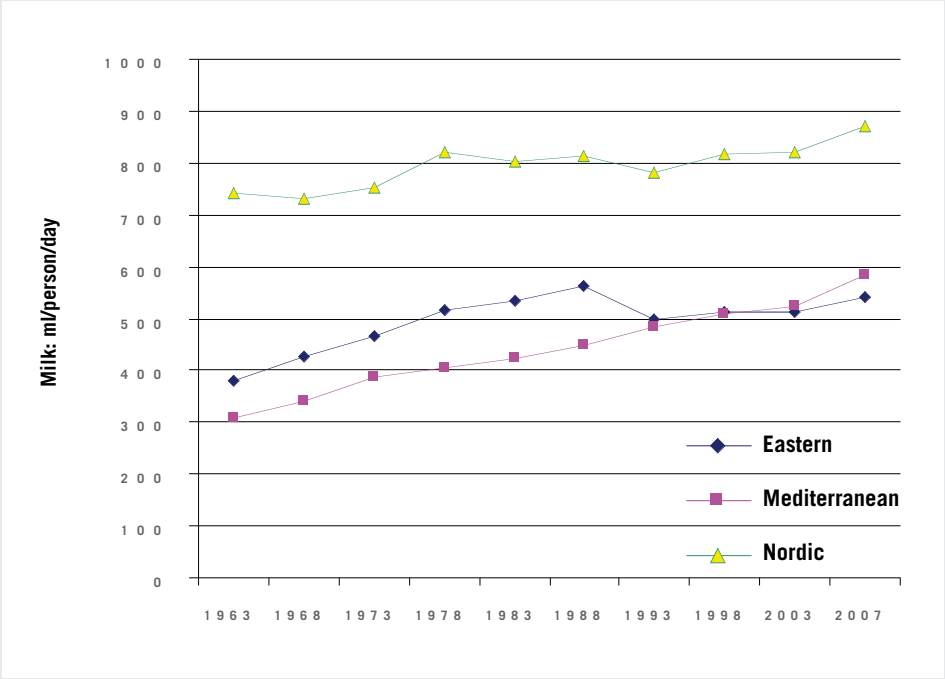
With strong support from the Common Agricultural Policy, EU member states have experienced strong support for their meat and milk producers to the point where surplus production became a problem in the 1980s. The effect on northern European diets was to increase meat supplies and maintain high levels of supplies of milk and dairy foods. In southern Europe, especially the Mediterranean region, meat supplies have increased nearly four-fold in a generation and fresh whole milk has also seen a strong increase (Figures 31 and 32).

Figure 31 Regional trends in meat supplies



Source: calculated from FAO food supply tables (http://faostat.fao.org).
Eastern = Bulgaria, Hungary, Poland, Romania
Nordic = Denmark, Finland, Norway, Sweden
Mediterranean = Greece, Portugal, Spain, Turkey

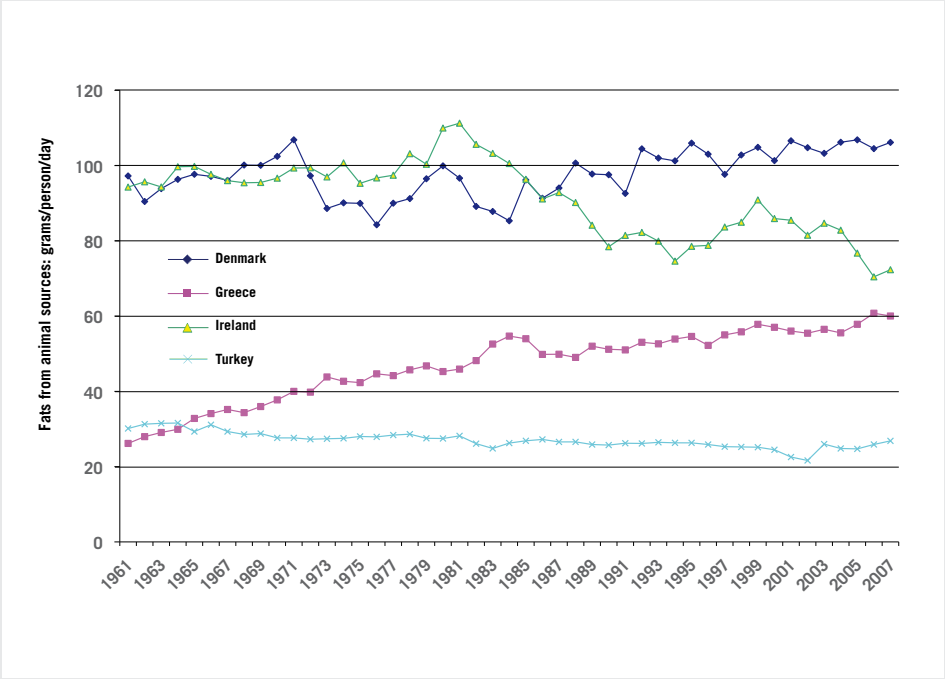
FIGURE 32 REGIONAL TRENDS IN FRESH WHOLE MILK SUPPLIES



Source: calculated from FAO food supply tables (http://faostat.fao.org).
Eastern = Bulgaria, Hungary, Poland, Romania
Nordic = Denmark, Finland, Norway, Sweden
Mediterranean = Greece, Portugal, Spain, Turkey

This picture of increasing meat supplies and maintenance of milk and dairy supplies for northern European countries, while a rapid increase is seen for southern Europe is born out by examination of four countries' data. Figure 33 shows continuing high levels of animal fat in the diets of populations in Denmark and Ireland, and a rapid increase in the diets of the population in Greece. In Turkey, however, animal fat consumption appears to have remained remarkably low, possibly as a result of Turkey remaining outside the Common Agricultural Policy.

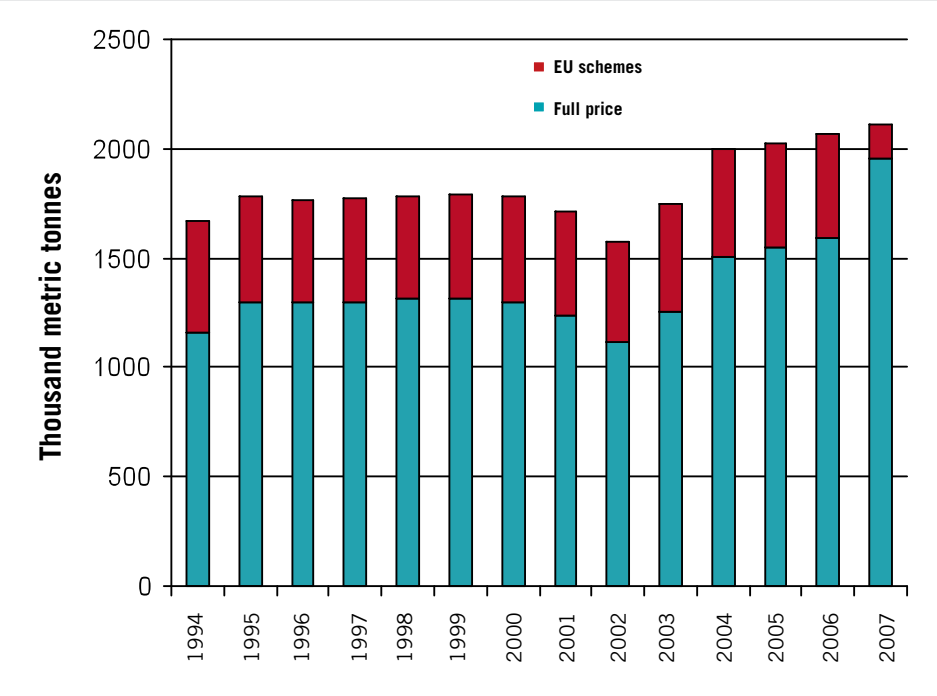
FIGURE 33 FOUR CASES STUDIES: TRENDS IN THE SUPPLY OF ANIMAL FAT IN DENMARK, IRELAND, GREECE AND TURKEY



Source: calculated from FAO food supply tables (http://faostat.fao.org).

Further evidence of the responsibility of the Common Agricultural Policy for introducing high levels of animal fat into the food supply can be shown in the case of butter. Consumer demand for butter has declined significantly in the last three decades, partly as a response to health education campaigns encouraging the switch to vegetable oils as a healthier option for cooking and at table (as vegetable margarines and lower fat spreads). However, specific measures under the Common Agricultural Policy allowed public money to be used to purchase the extra production of butter and re-sell this to the food industry at below-market cost to add to a variety of processed foods, including biscuits, pastries, confectionery and ice cream. At its peak, more than 30% of butter production was being subsidised to be put directly into processed food, and this level has only recently been significantly reduced (Figure 34).

Figure 34 Butter supplies in the EU showing the amount entering the food chain through the regular market and through the CAP 'special schemes'

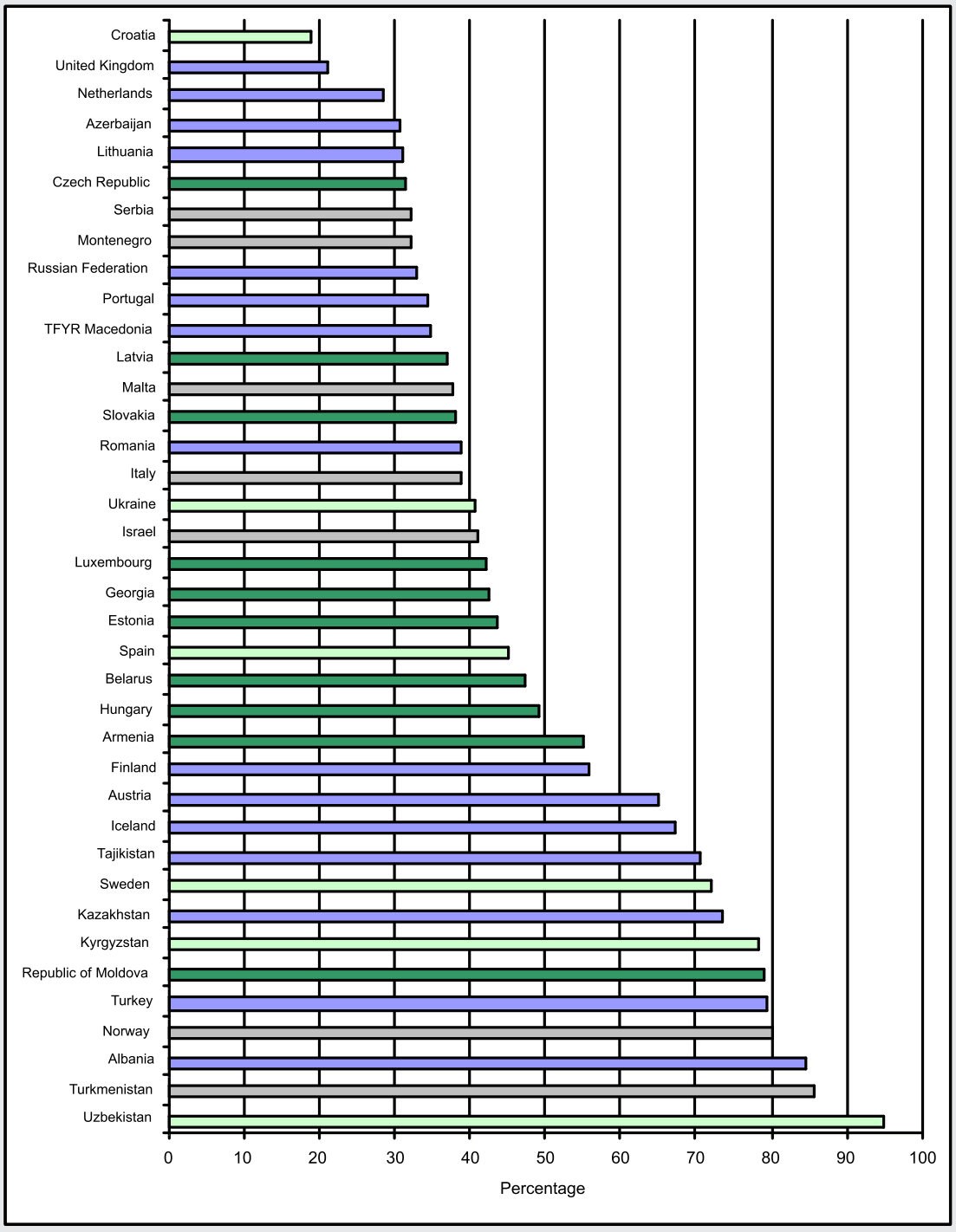


Source: European Commission annual accounts.

1.4.2.3 Breastmilk

Patterns of breastfeeding vary considerably across Europe. In the figure below (Figure 35) the estimates given are of the percentage of infants still receiving some breastmilk at age six months. The survey data is not collected on a regular basis in most countries, and estimates are provided here based on latest available surveys in the period 1998-2007. The comparison for estimating whether there has been a change was based on available data from the period 1986-1997. A bar with no colour coding (i.e. grey) indicates that no comparable survey was available to estimate change. It is encouraging to note that a significant decline in breastfeeding rates was reported in none of the countries listed.

FIGURE 35 PERCENTAGE OF INFANTS RECEIVING BREASTMILK AT AGE 6 MONTHS



Latest available surveys 1998-2007. Colours indicate change compared with surveys conducted in 1986-1997. Source: Health for All Database (www.euro.who.int/HFADB).
Pale green = > 25% increase. Dark green = >50% increase. Blue = stable. No increase more than 25%.
Grey = no previous data for comparison.

1.4.2.4 Snacks, biscuits, confectionery and soft drinks

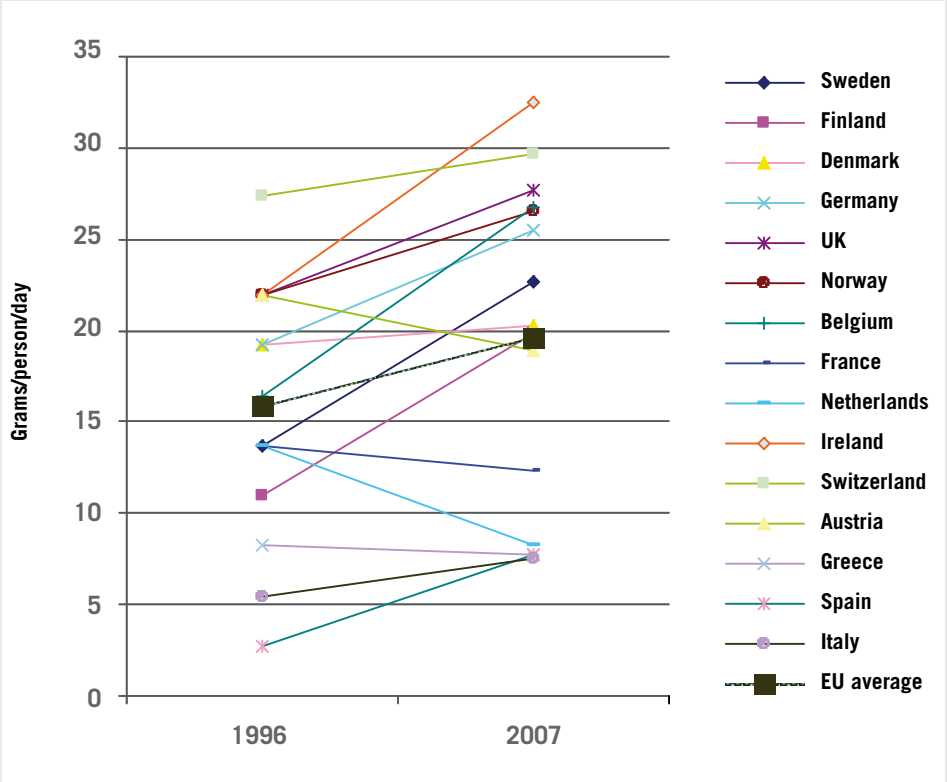
Information on the quantities and trends of consumption of foods such as snacks, biscuits, sweets and soft drinks are hard to obtain. They are not recorded in the food supply statistics from FAO, which focuses on primary, single ingredient products. They often fail to be recorded in shopping basket surveys of household expenditure because they are frequently consumed outside the home, while travelling, shopping or in the workplace. They are notoriously under-reported in dietary intake surveys as they appear to be easily 'forgotten' when diaries are filled in or interviewers ask for recent food recollections. The data presented here have been obtained from manufacturers' statistics and market intelligence reports.

The markets for savoury snacks has shown significant growth in recent years. In the UK, sales of crisps and savoury snacks grew by 5% in the year 2007-2008 to be worth some €2.92bn.[546] The market for savoury snacks in Greece increased by an average of 4.2% every year in the five years to 2008.[547] In Turkey the savoury snacks market grew an astonishing 8.8% every year in the five years to 2008.[548]

A similar extraordinary growth in the snacks market was seen across the whole of Eastern Europe, with an average for the region of 7.4% annual growth every year for the five years to 2008.[549] The region has seen substantial investments in food businesses since the opening of the markets in the early 1990s. During that decade, inward investment by western European and North American food companies totalled US$5bn, of which 60% went to building capacity for the production of confectionery and soft drinks.[550] The results of this investment were to encourage the growth of consumption of particular foods: in Poland, for example, chocolate confectionery sales rose 26% over the period 1999-2004, while sugar confectionery rose 22% and soft drinks consumption rose over 50%.[551,552] In Russia, which has seen similar patterns of inward investment in food businesses, the market for snacks has grown rapidly: sales volume trebled from 66,000 tonnes in 1998 to more than 200,000 tonnes in 2000, and grew a further 85% in 2001.[553]

In western Europe the markets for sweetened snacks are relatively mature, with only small growth in some sectors over the last two decades. Chocolate confectionery has seen significant growth in several countries, but a decline in several others, with an average net increase in the region of some 25% over the decade. (see Figure 36).

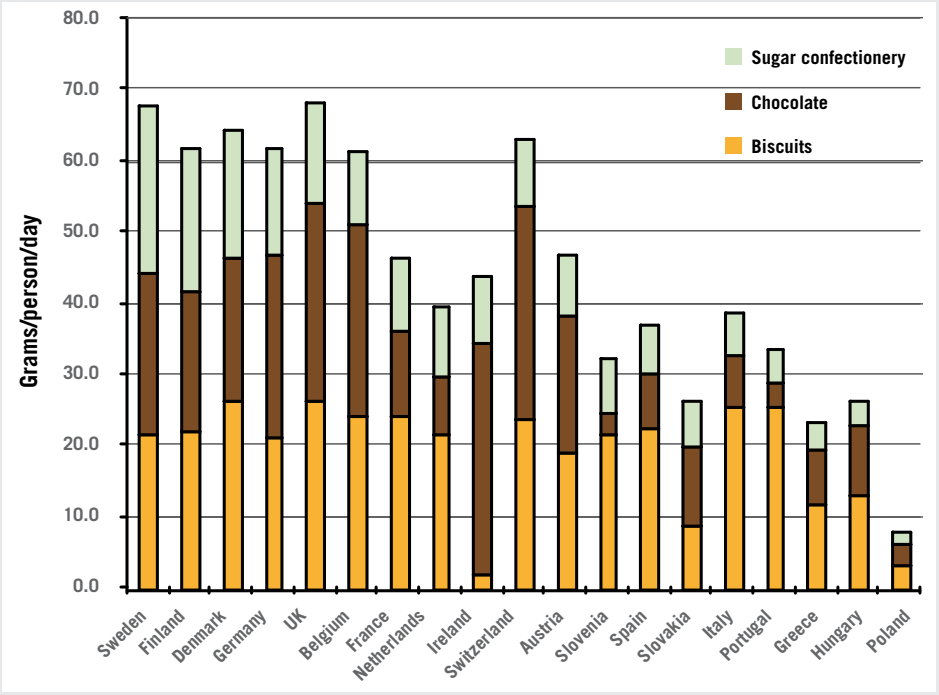
Figure 36 Chocolate consumption per person, 1996-2007



Source: Collated from CAOBISCO (www.caobisco.com)

Cultural differences in preferences for sugared confectionery, chocolate and sweetened biscuits are indicated in the current (2007) consumption figures (see Figure 37) which shows the UK leading the region in the consumption of sweetened snacks, at an average of nearly 70 grams per person per day, equivalent to 300-400 kcals.

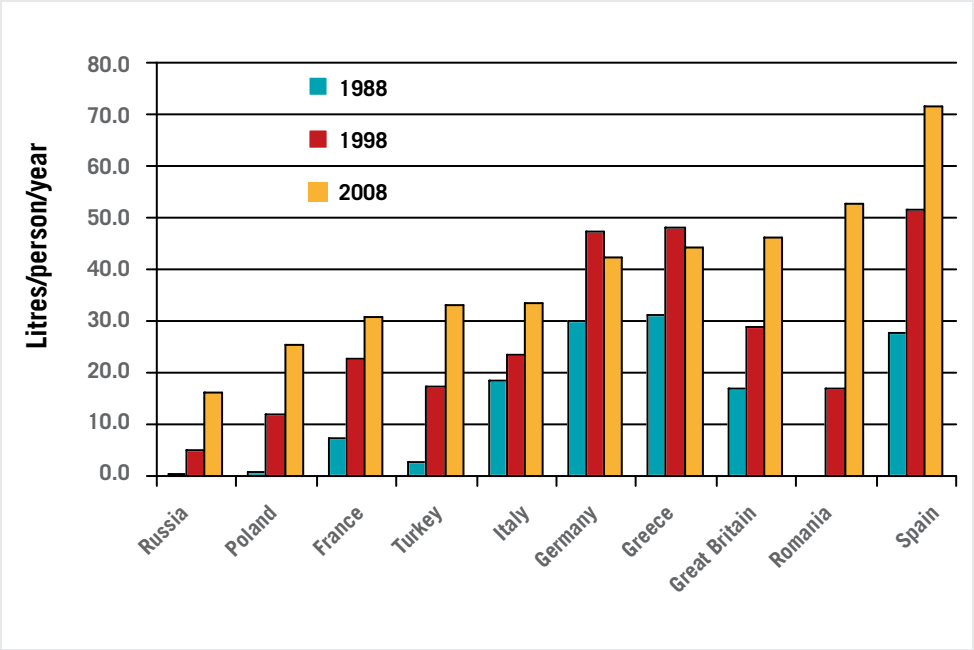
FIGURE 37 CONSUMPTION OF SWEETENED SNACKS, 2007



Source: Collated from CAOBISCO (www.caobisco.com)

Soft drink consumption is also difficult to monitor using supply figures, household purchases and dietary intake surveys. Industry sales figures from the leading company, Coca-Cola indicate a substantial rise in soft drinks consumption over the last few decades (Figure 38), with Spain drinking over 70 litres per person per year, or about 200 ml per day, from this company alone. Not all this consumption will be sweetened soft drinks, however, as the sales figures include other product lines from this company.

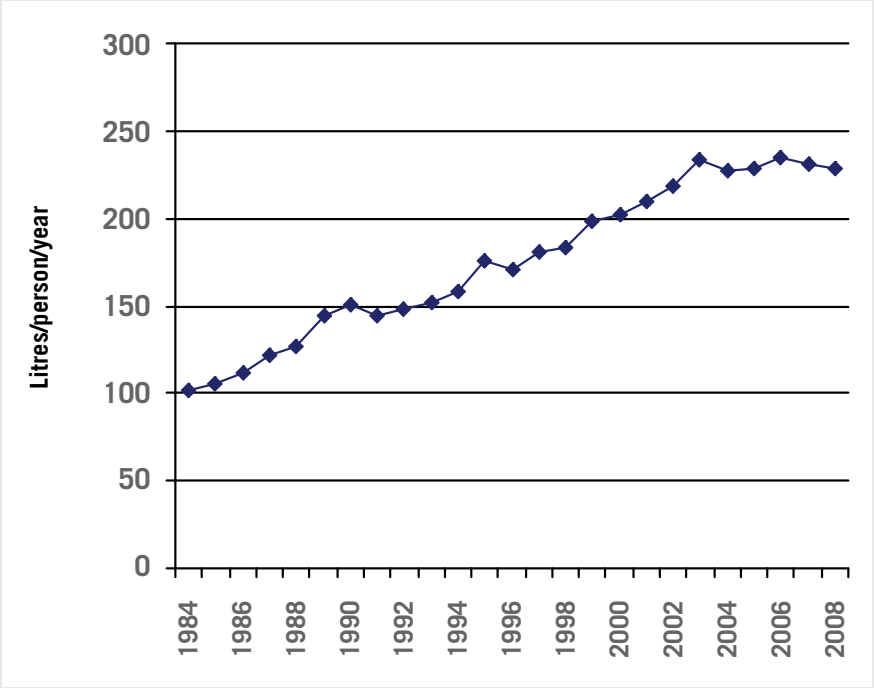
FIGURE 38 AVERAGE SALES OF COCA-COLA PRODUCTS, 1988, 1998, 2008



Source: Company reports.

Overall sales of soft drinks for the UK, for example, show a substantial rise in just 14 years (Figure 39). Again, not all this consumption will be for sweetened soft drinks but on the assumption that half the products are sweetened, at a level of 8% sugar, these products are supplying some 90-100 kcal per day to the average diet.

Figure 39 Total soft drinks sales in the UK, 1984-2008



Source: Collated from British Soft Drinks Association reports (ww.britishsoftdrinks.com)

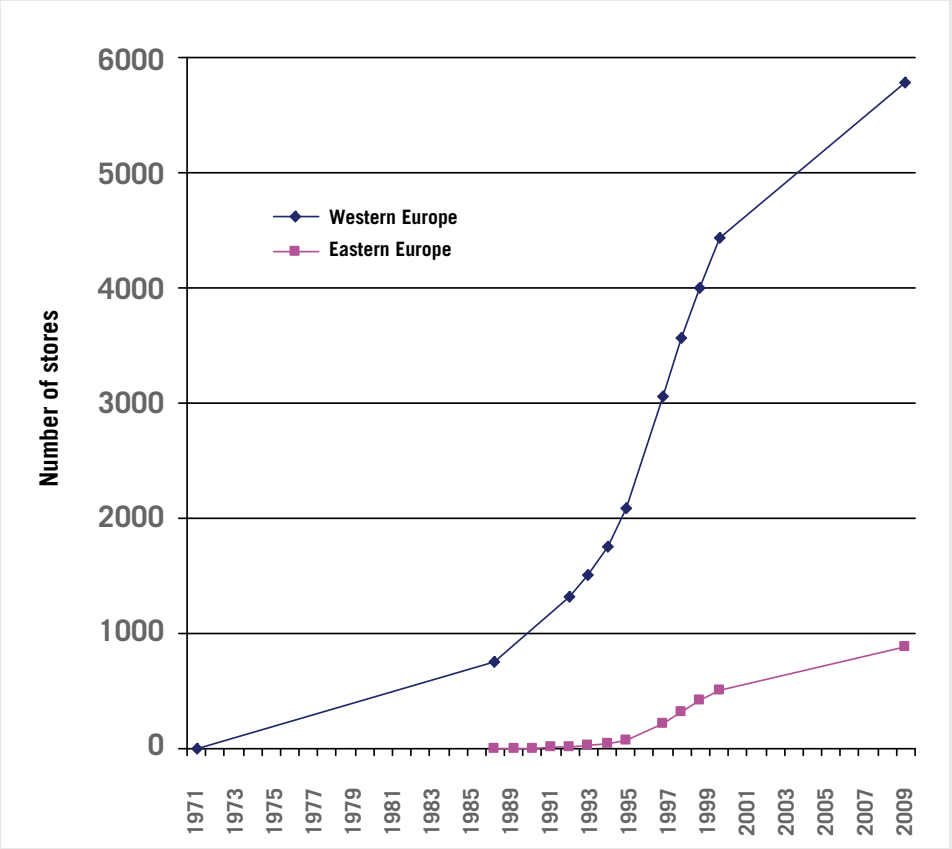
1.4.2.5 Fast food outlets

In many ways the changing diets of a population are reflected in their consumption of fast-service foods. The development of fast food chains in Europe signifies not only changing patterns of eating out, which itself has risen significantly in the last two decades, but also changing expectations of what is being eaten when not at home. Furthermore the rise in fast food chains alters the purchasing patterns from primary producers, raising demand from farmers and suppliers for particular commodities: a certain type of potato (the Idaho Burbank Russett for McDonald's), intensively reared chickens, cheaply produced beef for beef patties, white buns, soft drink ingredients etc. These high levels of demand reduce the wholesale prices, making it easier for others to enter the market and expand the fast food sector, easier for caterers in the public sector to follow the same types of menu, and easier for retailers to supply similar foods to shoppers for home consumption. Thus the large-scale investment in fast food services drives other aspects of the food supply.

Data for the whole sector are not easily found. Company data is found in annual reports and press releases. In this section we show data for the leading investor in fast foods in Europe, the US company McDonald's, which in late 2009 had 6,669 company-owned or franchised stores in the European region, in 39 countries, serving 12 million meals per day, each meal typically providing some 300-500 kcal with ingredients which are criticised for being higher in fats, saturated fats, sugars and salt and lower in fruit, vegetables and dietary fibre, compared with the recommended food-based dietary guidelines.

The rise in this company's activity can be seen in the figure below (Figure 40) showing the number of stores operated in Europe since the company opened its first outlet in the region, in the Netherlands in 1971. Expansion in the region has not been uniform. The company had few operations in eastern Europe until the early 1990s when the market became attractive to foreign investment. Both Poland and Russia have seen major investments, with over 200 stores each by 2009.

FIGURE 40 NUMBER OF MCDONALD'S STORES IN THE EUROPEAN REGION, 1971-2009



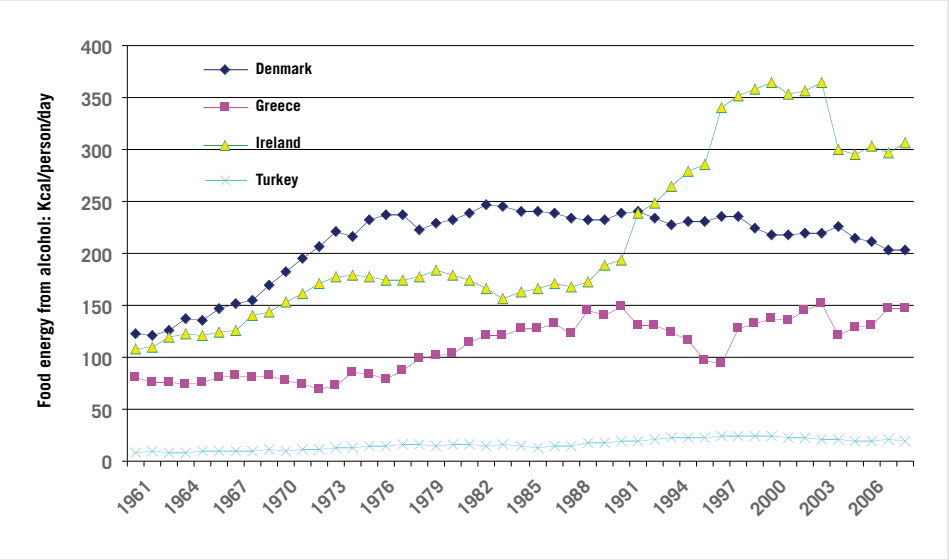
Source: Company reports, website archives.

As noted earlier, the consumption of fast food products is likely to show social class differences, with lower income groups, and especially young men in lower income groups, likely to be the highest users of fast food outlets.

1.4.2.6 Alcoholic drinks

Consumption levels range considerably across Europe, from under 5 litres of alcoholic drinks per year (Albania) to over 200 litres per year (Italy) per person. Taking the case of four countries it is clear that patterns of consumption—as measured by energy (kcal) from alcohol— have been changing dramatically in recent decades (see Figure 41) in some countries and not in others. A steep increase in consumption in Denmark in the 1970s was followed by an even greater increase in Ireland in the 1990s. A lesser increase in Greece nonetheless has led to a virtual doubling of consumption 1972-2002 and very low levels, but also rising, in Turkey, presumably reflecting its cultural traditions but also its increasing market for alcoholic drinks among tourists and visitors, and possibly among younger population groups of Turkish residents.

Figure 41 Four cases studies: trends in the supply of alcoholic drinks in Denmark, Ireland, Greece and Turkey

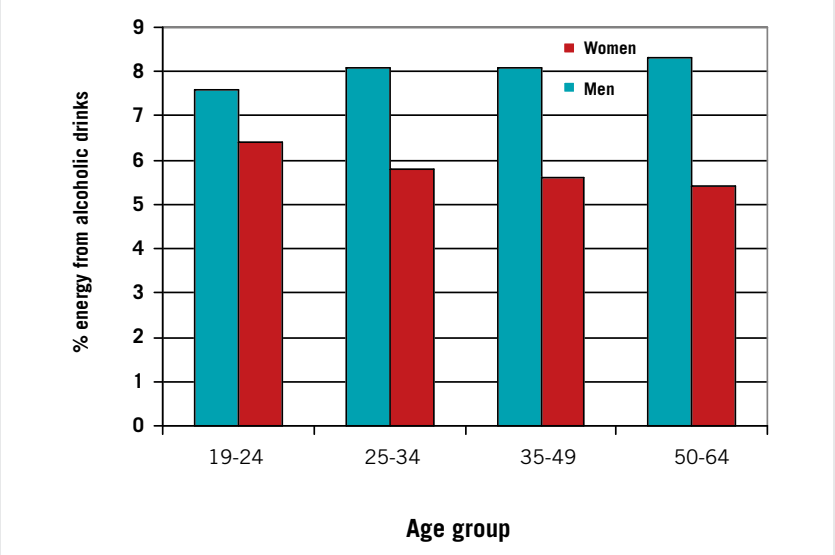


Source: calculated from FAO food supply tables (http://faostat.fao.org).

Section 1.3.8 reviews the evidence on the relationship between alcohol and cardiovascular health. It should be borne in mind that alcoholic drinks provide a source of energy, sometimes more than expected. The alcohol alone provides 7 kcal per gram, and additional energy may be obtained from other constituents of the beverage.

Based on self-reported dietary patterns among UK adult drinkers, alcohol contributes an average 187 kcal for men, and 93 kcal for women, equivalent to 8% and 6% of total energy intake respectively.[554] Not all these calories were from alcohol: in the UK survey, alcoholic drinks were estimated to contribute over 10% of the average sugar intake.

Figure 42 Percentage dietary energy intake from alcoholic drinks in the UK



Excluding non-drinkers. UK data, 2003. Source: NDNS 2003.

There is some evidence that alcoholic beverages serve to increase appetite for food and thereby increase total caloric intake.[555] In one study, healthy volunteers who consumed approximately two drinks during or prior to a meal failed to compensate for the energy by eating less at the meal, and total energy intake thus increased.[555,556] There was also no compensatory reduction in energy intake during a meal eaten later in the day. Evidence for the effects of alcoholic drinks on total energy intake in the longer term is not consistent.

1.4.3 Vanishing traditional diets

The trends shown in the tables above indicate that, especially in southern Europe, there have been some significant changes in the nutritional quality of the diets. Two aspects are of concern: firstly, the possible changes in the quality of high value foods, such as fruits, vegetables, lean meats and fish—for example changes in the types of fruit and vegetables, or changes in the nutrient profiles of meat or fish. Secondly, the larger changes in the patterns of food eaten, and especially the loss of the traditional diets in the Mediterranean region with their relatively high levels of fruit, vegetables, olive oil and fish, and their replacement with soft drinks, confectionery, snack foods and fast food meals.

Several recent studies can throw further light on these trends. In a consideration of the types and quality of fruits and vegetables being consumed, Granado and colleagues[557] analysed the fruit and vegetables being purchased by households in Spain over recent decades and noted that changing patterns—such as a decline in green leafy vegetables and green beans, and an increase in citrus fruits and bananas—might alter the nutritional qualities of the total diet. Table 28 indicates the most recent changes, 1990-2004, and these indicate a decline in the average daily intake of carotenoid levels for all six forms examined. It was assumed that carotenoid levels in each type of fruit remained level, although there is some evidence that modern growing and processing methods (e.g. growing under glass, ripening after harvesting) may reduce the levels of these micronutrients compared with more traditional methods.

Table 28 Average daily intake of carotenoids from fruit and vegetables consumed, Spanish household surveys 1990 and 2004

	1990	2004
Lutein (µg)	558	380
Zeaxanthin (µg)	100	73
Lycopene (µg)	1437	1163
B-Cryptoxanthin (µg)	398	313
A-Carotene (µg)	306	261
B-Carotene (µg)	1303	1065
Total (mg)	4.1	3.3

Source: Granado, 2007[557] Total fruit and vegetable supplies from FAOSTAT.

A similar problem arises with the promotion of high-cropping, easy-storing varieties of fruit and vegetable. Different apple varieties have significantly different vitamin C levels. Some types have three or even five times more of this vitamin than others, although the heaviest cropping varieties (Golden Delicious especially) have amongst the lowest levels (see Table 29).

Table 29 Vitamin C content of different apple varieties

Variety	Vitamin C (per 100g)
Sturmer	20 mg
Discovery	16 mg
Cox's Orange	9 mg
Russet	8 mg
Worcester	5 mg
Golden Delicious	4 mg
Granny Smith	4 mg
Red dessert	3 mg

Source: J Blythman 1996[558]

The implications of this research are that the previous recommended population average intake of a minimum of 400 grams fruit and vegetables per day may need to be raised. (See the longer-term goal proposed for fruit and vegetable consumption in section 1.3.5 of this report.)

Nutritional concern can also be raised about the move from wild-caught fish to farmed fish as a source of long-chain polyunsaturated fatty acids. Generally, the profile of the oils in fish reflects the diets being fed to the fish. An indication of how a switch from marine sources of fish feed to plant oil sources of fish feed may alter the fatty acid profile of the farmed fish is shown in the table below.

Table 30 Fatty acids in the flesh of farmed trout according to their feed (Grams per 100 gram total fatty acids)

	Trout fed with marine oils	Trout fed with a mix of marine and plant oils
Saturates	558	380
Monounsaturates	42	38
Polyunsaturates Omega 3 Omega 6	31 9	16 19

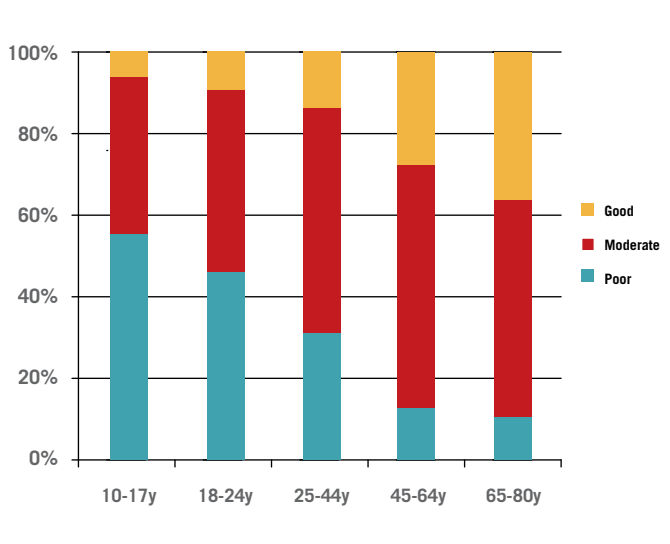
Source: Sargent and Tacon[559]

Similar issues are raised concerning poultry. Whereas wild game birds, such as grouse, have a low fat content, and that fat is comprised of 60% polyunsaturated fatty acids, in farmed poultry the fat content is significantly higher and the polyunsaturated fraction less than 20% of the total.[560]

In terms of wild and commercial varieties of plant crops, a study of edible vegetables eaten in Crete by Trichopoulou and colleagues[561] found that wild green-leafed plants used in traditional cuisine are rich in phytonutrients, such as antioxidant flavonols and flavones. The study found that over 150 varieties of edible wild greens are believed to be consumed in Greece, often in the form of traditional green pies made with virgin olive oil. Analyses of these pies showed the concentrations of antioxidants were considerably higher than in well-recognised sources such as red wine. A second paper demonstrated that a representative weekly menu of Greek traditional foods and dishes fulfilled the recommended macro- and micro-nutrient targets proposed by the European Commission Scientific Committee for Food.[562]

These findings raise a second question, concerning the changes in European diets over time and across the generations. The changes have been very rapid in much of Europe, and especially so in recent years in the Mediterranean region. In a study of changing patterns of diets in Catalonia, Spain, Serra-Majem and colleagues[563] examined the compliance of adults in the region with guidelines proposed by the Spanish Society of Community Nutrition and in particular whether the population was maintaining the traditional aspects of a Mediterranean diet. When asked if they ate a Mediterranean-type diet, 63% or respondents said they did, although using a 15-item index to evaluate actual dietary intake it was found that this figure fell to 52% of the sample having a rating of 'good' or 'moderate' adherence to the Mediterranean diet. Furthermore, adherence was much stricter among older adults (89% among those aged over 65 years) compared with younger groups (falling to just 44% among those aged 10-17 years) (Figure 43).

Figure 43 Adherence to a traditional Mediterranean-type diet in Catalonia, Spain



Source: Serra-Majem et al (2007).[563]

The figures indicate a rapid transition between the generations, with very few younger people adhering to the traditional patterns of diet. The problem may actually be worse in the Mediterranean region than elsewhere: researchers using a formalised dietary scoring system (the computerised MedDietScore, a validated predictor of heart-healthy Mediterranean dietary patterns[564]) found that nutrition students in Holland scored more highly (more like a traditional Mediterranean Diet) than nutrition students in Greece.[565]

Finally, the move from healthier diets to less healthy ones across the generations is also seen in non-Mediterranean regions. A study of older women, also conducted in Holland, found a 30% lower mortality rate among those consuming a 'traditional healthy Dutch diet' (emphasising vegetables, fruit, non-alcoholic drinks, dairy products, and potatoes) compared with either a less healthy Dutch diet (meat, potatoes, vegetables, and alcoholic beverages) or a more Mediterranean-like diet (oils, pasta and rice, sauces, fish, and wine).

1.4.4 Physical activity trends

Broad societal changes that took place in increasingly industrialised and urbanised European societies in the second half of the 20th century mean that it is easier for many Europeans to live largely sedentary lives. A shift away from more physically active occupations, lower energy expenditure levels within various occupations, fundamental changes in travel and transport, along with increasing mechanisation in the home are some of the factors involved.[566]

It is, however, difficult to make any definitive statements about how physical activity patterns have evolved in Europe, because there is very little monitoring and surveillance of physical activity levels. Furthermore, the data which do exist on physical activity have usually been collected using subjective measures of physical activity, such as self-reported questionnaires. The current interest in the use of objective measures, which record the intensity and duration of physical activity, is too recent to enable us to plot how activity levels have changed over time.

A global systematic review by Knuth and Hallal[567] included 25 studies in adults, six of which were conducted in Europe. The authors concluded—while acknowledging the issues around comparability between studies—that current data suggest that leisure-time physical activity (LTPA) has been increasing over time for adults in high income countries, while occupational physical activity has been declining. Many countries have also seen a drop in physically active forms of travel—in Great Britain, for example, trips made on foot, as a proportion of all travel journeys, fell by 24% between 1995/7 and 2008.[568]

Table 31 Physical activity trends in Europe

Adapted from systematic review by Knuth and Hallal[567]

Study first author, year	Country/ data source	Comparative period	Comparative ages	Main outcome measure	Description of measurement technique	Main results
Lahelma et al,1997	Finland/ Monitoring of health behaviors	1978–2003	25–64 y	Leisure-time physical exercise	Postal questionnaire	The prevalence of leisure- time PA increased in both sexes.
Lindahl et al, 2003	Sweden/ Northern Sweden MONICA Project	1990–99	25–64 y	Leisure-time and occupational PA	Face-to-face questionnaire	Occupational PA decreased in males. Leisure-time PA was stable in both sexes.
Lindstrom et al, 2003	Sweden	1986–1994	20–80 y	Leisure-time PA	Postal questionnaire	Physical inactivity in leisure-time increased from 15 to 18% in males and from 19 to 27% in females.
Stamatakis et al, 2007	England/ Health Survey for England (HSfE)	1991–2004	16+ y	Walking, occupational, domestic and leisure-time PA	Face-to-face questionnaire	The prevalence of subjects classified as active in the workplace decreased from 27 to 24% in women and from 43 to 39% in men. Sports participation increased, particularly among middle-aged adults.
Steptoe et al, 2002	European countries/ European Health and Behavioral Study (EHBS)	1990–2000	17–30 y	Physical exercise	Self-administered questionnaire	Physical exercise increased in males in most countries. Among females, physical exercise increased in 4 countries and decreased in 3.
Uitenbroek et al, 1992	Scotland	1987–1991	18–44 y	Leisure-time PA	Telephone questionnaire	There was a substantial decline in the proportion of sedentary individuals, in both cities investigated.

Experimental studies using the doubly labelled water stable isotope technique (recognised to be the most accurate), however, have not found evidence of a decline in physical activity in recent years. One study in the US and one in the Netherlands evaluated physical activity energy expenditure (PAEE) and physical activity level (PAL) at various time points between 1998 and 2005 and found that it did not decrease in either population over the period.[569]

Despite these uncertainties about how physical activity levels have changed in Europe over time—and debate about the degree to which changes in physical activity have contributed to the obesity epidemic—there is widespread agreement that European populations need to be more active (see Section 1.3.7). WHO estimates that at least two thirds of the adult population of the EU countries are insufficiently physically active for optimal health.[570]

It is fundamental, therefore, that policies are implemented to create environments that are conducive and supportive of physical activity. It is also vital that governments introduce measures to enable monitoring and surveillance of their populations' physical activity levels and how they change over time.

1.4.5 Concluding comments on diet and physical activity patterns

This section has highlighted a number of important trends in food supply, food consumption and physical activity patterns in the European region, with profound implications for chronic disease risk. While fruit and vegetable consumption has increased in most countries, the nutritional quality may have deteriorated. Greater opportunities for food purchasing and falling prices for many foods have helped stimulate the demand for food and thereby raised the levels of intake.

A major problem in the analysis of diets and dietary trends is the quality of the data available. As noted at the start of this section, there are no comprehensive surveys across the European region, no uniform methods of assessment, and no single year in which all countries undertake a survey. The trends in actual dietary intakes must be inferred from the available material, and the ability to pursue evidence-based policies will inevitably be limited. The data on physical activity levels and trends are, if anything, even less robust.

The problem is especially acute when dealing with questions about population averages—it is quite plausible that a target is being met by the population on average while actually no person is eating a healthy diet. An analysis of the UK National Diet and Nutrition Survey of Adults, 2000–2001 found that only seven adults per 1000 were likely to be eating a diet that met the recommendations for health in terms of five targets (fat, saturated fat, NME sugars, sodium and fruit and vegetables).[571]

A significant generation shift is also apparent especially in southern Europe. The traditional foods are increasingly being replaced by a more 'modern' diet with a greater proportion of calories from oils and fats, sugars and processed starches, and for some sections of the population, alcohol. The loss of traditional diets may help to explain one of the paradoxical findings on heart disease mortality: namely that higher mortality rates are found among lower income people in much of Europe, but this is reversed in Mediterranean countries specifically among older generations.[572] It appears that older people's adherence to traditional, healthy diets may be protecting them from the cardiovascular ill health that now dominates the disease burden of Europe.

2 Policy Options in a Rapidly Changing World

The European policy landscape relating to diet and physical activity has changed enormously since the European Heart Network published *Food Nutrition and Cardiovascular Disease Prevention in the European Union: Challenges for the New Millennium in 2002*.

2.1 THE POLICY CONTEXT

There has been a series of major food, nutrition and physical activity policy initiatives at the global, European and EU levels (see box). These various initiatives reflect the growing understanding that comprehensive and integrated policies are needed to bring about changes in diet and physical activity patterns. International agencies, the European Commission and national governments increasingly recognise that many different policies—including agriculture, urban planning, trade, taxation, transport and education—can impact on what people eat and how physically active they are.

At the same time some policy makers—at both international and local levels—have pushed forward with progressive and innovative action to prevent diet-related ill-health. Initiatives such as one London borough council's proposal to use planning restrictions to limit fast-food outlets next to schools, the Danish government's groundbreaking ban on trans fatty acids or the European Commission's initiative to reduce the amount of salt in manufactured foods would have been widely dismissed as radical and unthinkable at the start of the millennium.

This recent policy progress inspires belief that governments of all shapes and sizes can take action. It is encouraging that some policy makers are now thinking in much broader terms about how we can prevent diet-related disease, beyond the traditional approach focused on education and information. Moreover, governments have shown that they can come together and cooperate internationally, through WHO and the EU, to tackle major health challenges. National heart foundations and other public health advocates have continued to play an important role in shaping public opinion and creating the appropriate climate for bold public health policies, as well as advocating for broad-reaching action to improve the population's health.

There is still, however, much to be done. The examples of innovative, progress policy often relate to isolated cases (for example, to date few countries have followed Denmark's trans fat ban, even though such a measure was first called for in the 1980s). There are other issues that have been on the policy agenda for years, sometimes decades, which show a frustrating lack of progress. Public health advocates, for example, have been calling for a simplified front-of-pack nutrition labelling scheme to help consumers make healthier choices at a glance for over 20 years. However, discussions on the European Commission's proposal continue to disappoint. Similarly, the proportion of food eaten outside the home has increased dramatically in recent years, yet people ordering a meal in restaurants or cafés very rarely have access to any information to help them choose the healthiest option. Across Europe, the hospitals that treat our sick often serve or sell foods that contribute to the massive burden of diet-related ill-health. Many schools and childcare institutions charged with nurturing Europe's children, sell unhealthy snacks and drinks, and in most countries children are exposed to adverts for foods high in fat, salt or sugar while they watch television. In addition, the agricultural policy that was designed to protect Europe's population from food shortages continues to raise concerns because its policies and mechanisms result in an overabundance of meat that is relatively cheap and of dairy products, thereby increasing supplies of saturated fat.

It is also clear that we can no longer consider food and nutrition policy in isolation, without taking into account the broader global context. Any policy to promote healthier diets or increase physical activity needs to be coherent with strategies to tackle today's major social and environmental challenges, including climate change, water and food prices.

Major developments in food, nutrition and physical activity policy and strategy since 2002

Since EHN's 2002 report *Food Nutrition and Cardiovascular Disease Prevention in the European Union: Challenges for the New Millennium* there have been various developments in terms of global and European strategy to deal with nutrition, physical activity and health:

A new global framework

The World Health Organization (WHO) issued an important report on *Diet, nutrition and the prevention of chronic diseases (WHO Technical Report Series 916)* and in 2004 the World Health Assembly endorsed the *WHO Global Strategy on Diet, Physical Activity and Health*. This was, followed by the *WHO Action Plan for the Prevention of Non-Communicable Diseases* in 2008. Since May 2007—following a resolution by the World Health Assembly—WHO has been in the process of developing a set of recommendations on the marketing of foods and non-alcoholic beverages to children.

European action plans

WHO Europe's Action Plan for Food and Nutrition Policy, 2000 – 2005, successfully pushed the development of policies relating to food and nutrition at the national level. Despite this progress in policy development, however, many countries are still struggling to put policies into action. So a second European action plan for 2007-2012 was endorsed by the WHO Regional Committee in September 2007. Prior to that, health ministers across the continent signed the *WHO Europe Charter on Counteracting Obesity* in November 2006.

An EU strategy

In October 2007, the European Commission adopted a White Paper on *Together for Health: a strategic approach 2008-2013* aimed at establishing a strategic framework for health in the EU. A White Paper on *A Strategy for Europe on Nutrition, Overweight and Obesity-related health issues* was presented in 2007. This strategy was reviewed in 2010 and a final report is due in 2013. The White Paper followed on from the 2005 Green Paper entitled *Promoting healthy diets and physical activity: a European dimension for the prevention of overweight, obesity and chronic diseases* and a European Parliament resolution which reinforced the Green Paper's messages. In relation to physical activity, EU sports ministers endorsed the EU Physical Activity Guidelines in November 2008.

The Lisbon Treaty—recently ratified in all EU member states—reiterates and somewhat strengthens the role of the EU in protecting public health. Article 168 of the new Treaty reiterates existing public health policy objectives and includes provision for incentive measures to "*protect and improve human health and in particular to combat the major cross-border health scourges…*"

The EU Strategy also established a new High Level Group of member states' representatives, and it also emphasised the work of the EU Platform for Action on Diet, Physical Activity and Health. This body, referred to as the Platform, was established in March 2005 to provide a common forum for all interested actors at European level.

These major developments, particularly the EU nutrition White Paper and the creation of the EU Platform and the High Level Group, go some way towards answering EHN's call in our 2002 paper for "*a comprehensive and integrated European food and nutrition policy*" and for new structures involving senior policy makers. Yet further progress is needed in translating these strategic documents into concrete and effective action on the ground.

Support structures are important for the development and implementation of effective nutrition policy. Such structures need to ensure close collaboration and coordination to implement an integrated strategy that spans a wide range of policy areas. EHN welcomed the establishment of the EU High Level Group of member states' representatives. It is important that the Group should be more than a simple exchange mechanism and must be able to provide real leadership in the implementation of EU strategy. In relation to the Platform, EHN supports the formulation of specific operational objectives that would help to provide a focus for commitments and ensure that commitments pull in the same direction.

2.2 ADDRESSING THE WIDER ENVIRONMENT

Individual characteristics, such as personal preferences, behaviour and knowledge clearly come into play when people make choices about eating or participating in physical activity. Until relatively recently the policy response to diet and inactivity-related ill-health focused on these individual characteristics and centred mainly on educating the public about healthy eating and becoming more physically active, and providing information to enable people to make healthier choices.

It is now widely acknowledged, however, that—while education and information remain fundamental—it is not enough to focus on this approach alone. As policy makers have struggled with responses to the obesity epidemic in particular, many have been drawn to the conclusion that, while neither human metabolism nor human nature are likely to have changed fundamentally in the last 30 years, the environment we live in certainly has changed. This shift away from responsibility being placed exclusively on individuals points to a need for broader thinking about chronic disease prevention. The solutions need to come predominantly from making changes to the environment in terms of the food supply and other factors which influence what people eat and how physically active they are.

It is simply not enough to tell people to eat more healthily and to take more exercise. It is also vital to make sure that people have easy and affordable access to a healthy food supply. Equally, it is important to create safe built environments that encourage and promote active forms of transport, such as walking and cycling, and other forms of physical activity.

EATING AS AN AUTOMATIC BEHAVIOUR?

There has been a sea change in thinking about why we eat what we eat. Research increasingly suggests that eating is more influenced by environmental factors than by conscious choice. The health policy community is waking up to what food marketers have long known: it is possible to influence what people eat, without people even realising how their eating habits have been influenced.

In the US, Deborah Cohen and Thomas Farley asked why "people continue to eat more calories than they need when the consequences are so apparent, stigmatising and widely understood?"[573]

Impulse decisions can play an important role in relation to food choice. Such decisions can be influenced by learned behaviour, cultural factors and conditioned learning (e.g. where eating is associated with a particular situation, behaviour or reward). Cohen and Farley outlined how studies show that a whole host of external factors can influence what people eat. These include aspects of the food itself (the bigger the portion size the more people eat) or the eating context (people eat more at longer meals and when sharing with others). It also includes different facets of the availability and visibility of foods and other external cues (such as advertising and marketing techniques) to eat. In short, the less effort it requires to eat food, the more food is eaten. Although all of this may sound obvious, there are important implications for policy.

The authors argue that eating should be considered as an "automatic behaviour." This means that eating can be done without any conscious intent. It doesn't mean that we can't control what we eat—as with other automatic behaviours, such as smiling or frowning, it is possible to exercise control over eating but this requires mental effort.

This concept of eating, however, suggests that a different approach to changing dietary patterns is needed. It is important to acknowledge that eating takes place in a social and cultural context and to take the relationships that people have with food into account. Encouraging healthy and relaxed attitudes to food and eating from a young age may be beneficial.

It is also clear that an approach which addresses the food environment, or landscape, is necessary. Cohen and Farley suggest that appropriate policy responses include reducing portion size, limiting access to ready-to-eat foods, limiting availability of snack foods in schools and workplaces and reducing food advertising. Crucially, they also suggest that people appear to be so sensitive to changes in the food environment that barely noticeable changes might have a public health impact.

Many of these points in relation to eating as an automatic behaviour also relate to sedentariness. The way that society has developed has created circumstances which encourage lives which are increasingly sedentary rather than physically active. A multitude of factors have led to the obesogenic environment we experience today. Energy saving devices in public places, such as escalators and lifts, remove the need for physical activity, as do the labour saving devices in our homes and workplaces. Our leisure time is increasingly sedentary and spent within the home. Coordinated efforts across a range of sectors are required to address these issues and create an environment which promotes physical activity.

2.3 ADDRESSING INEQUALITY

Section 1.1.2 described the startling socio-economic gradient for health that exists across Europe. So far policy efforts have failed to close this health gap between Europe's poor and rich.

It is increasingly clear that these differences in health between socio-economic groups cannot simply be explained by differences in genetics, lifestyle factors and/or access to healthcare. The social gradient reflects unequal access to income, employment, neighbourhood circumstances and, more generally, life chances.[574] It is also now evident that it is not just the groups with lowest socio-economic status who are losing out—narrowing the health gap would lead to an improvement in health and wellbeing for all. In 2008 a major global report, from the Commission on Social Determinants of Health, emphasised that social injustice is killing on a grand scale and argued that health equity itself should become a global goal. The Commission made three over-arching recommendations:

- Improve daily living conditions;
- Tackle the inequitable distribution of power, money and resources;
- Measure and understand the problem and assess the impact of action.

In order to really tackle health inequalities, therefore, policies need to go further than addressing the social exclusion of the most disadvantaged groups. Governments need to establish general population goals to reduce health inequalities and aim to ensure that people across the social spectrum have more equal access to "health chances." This means introducing policies to narrow inequalities across the whole of society (reducing the steepness of the social gradient). It also requires policies to specifically address the needs of particular vulnerable groups such as migrants, ethnic minorities, older people and those living in poverty.

Policymakers across Europe have become increasingly concerned that these inequalities continue to exist and have, in many cases, increased in recent years. Closing the health gap is seen as an important step towards the overall goal of promoting sustainable economic growth and greater social cohesion in Europe.

Although many national governments have set out to address health inequalities, much more action is needed—fewer than half of EU countries, for example, have a policy emphasis on tackling health inequalities.[575] In 2009 the World Health Assembly urged WHO member states to tackle health inequalities with a view to closing the gap within a generation, as recommended by the Commission on Social Determinants of Health.[576] Within the EU—which is, after all, the world's largest economy, and which has some of the most sophisticated health systems and strongest social protection in the world—the issue of health inequalities both between and within countries has risen up the agenda. In October 2009, the European Commission committed to undertake a series of actions to address health inequalities in its *Communication on Solidarity in Health: Reducing Health Inequalities in the EU*.

In relation to nutrition and physical activity, there are some clear policy implications. Policies to improve diet and to promote active living must address the barriers and constraints which hinder people across the social scale (see box for examples of specific issues).

Reducing inequalities: diet and physical activity policy areas

Affordability of healthy foods and physical activity
Cost can be a significant barrier in trying to eat healthily. A diet high in fat and sugar may appear to make financial sense to someone trying to feed a family on a tight budget. Energy-dense foods, such as sugary or salty snacks, tend to cost less on a euro-per-kilocalorie basis than nutrient-dense foods, such as meat, fruit and vegetables.[577] Research in Ireland, for example, found that a single parent family with one child would have to spend 80% of the household income to purchase a healthy diet.[578]

Accessibility of healthy foods
In some countries, disadvantaged areas have become "food deserts" with very restricted access to fresh foods and to healthy options. These areas tend to have few shops and those that exist sell a limited range of products, usually at relatively high prices. Residents have poor access to transport to enable them to shop at markets or at a wider range of stores. Coupled with the higher prices of healthy foods, these constraints combine to create food poverty, making it harder for people on lower incomes to eat healthy diets.

Availability and affordability of unhealthy foods
While access to affordable healthy foods may be difficult for some groups, access to low-cost energy dense food is plentiful in many European countries.[579] For this reason, policymakers are increasingly exploring the options for economic instruments, such as taxes or subsidies, which affect the relative price of unhealthy compared to healthy foods. The introduction of measures to restrict physical access to unhealthy foods—such as planning restrictions on fast food outlets or controls on food in schools or other public institutions—is another approach under consideration.

Accessibility and affordability of physical activity opportunities
We know that socio-economic conditions have an impact of physical activity levels and that poorer groups have less free time, poorer access to leisure facilities and live in environments which are not conducive to promoting physical activity. It is important that policies focus on increasing physical activity opportunities for poorer groups. In addition to transport policy and questions of planning and infrastructure (see below) this means policies that remove financial and other barriers to participation in sports and leisure activities for people living on low incomes by, for example, lowering prices, ensuring facilities are appropriate for different age groups, genders and different ethnic groups etc.[580]

An infrastructure to encourage physical activity
If we are all to become more physically active, we need an infrastructure and built environment that is conducive to active living. In reality, many disadvantaged communities live in obesogenic environments that do little to encourage walking, cycling or other forms of activity.[581] European, national and local policies need to focus on creating neighbourhoods that facilitate walking and cycling and which are, more generally, activity-friendly. Important factors include higher development densities, planning policies to ensure that destinations such as schools and shops are accessible, traffic calming and other safety measures to ensure that people feel safe in their local environment.

2.4 OPPORTUNITIES FOR CHANGE

A wide range of policies and actions at the international, national and local levels can have an impact on what people eat and how active they are. International trade rules and agricultural policy have a direct impact on the price and availability of different foodstuffs across Europe. Taxes and subsidies (whether at the European or national levels) also affect the price, and relative affordability, of different foods. Rules concerning marketing and labelling of foods (again at both European and national levels) affect consumers' food choices and their capacity to choose healthier foods. Local planning policies can affect communities' access to affordable healthy foods. Similarly, planning and transport policies (both nationally and locally) can influence the feasibility and attractiveness of walking, cycling and other forms of physical activity.

When factors ranging from European farming policy to the portion size of an individual meal can affect nutrition, it is easy to understand why policy makers may feel daunted at the prospect of trying to improve diets and promote physical activity. Local communities may wonder how they can bring about positive change in face of the marketing might of multinational food companies or the EU Common Agricultural Policy.

Yet, the good news is that the fact that so many different factors affect what people eat and how active they are means that there is also a multitude of opportunities for action. There continues to be a role for interventions that focus on changing individual behaviour by enhancing knowledge and providing clear information. There is also, however, an array of policy options aimed at changing the health landscape and specific aspects of the environment which affect eating patterns and physical activity.

If we are to reduce the burden of diet and inactivity-related cardiovascular disease, action is required locally, nationally and internationally. Some of the policy options relate to overarching, multi-sectoral policies, while others target very specific issues. In many cases action will be needed at the national or European levels, but in other circumstances local policies are required.

2.4.1 National and local governments — key policy players

Leadership from national government

At the national level, commitment across government is an essential pre-requisite for improving diet and physical activity levels for heart healthy living. Although international cooperation is important—both at WHO and European levels—and the private sector can play a critical role, national and local governments are key policy players. Civil society at both national and local levels, and including heart foundations and others in the voluntary sector, play a key role in ensuring that governments show the real leadership that is needed, and in holding their governments to account if they do not deliver.

The "*enjoyment of the highest attainable standard of physical and mental health*"[582] and adequate food (in the meaning of nutritionally adequate diets) are fundamental human rights, and food security (including food safety) is identified as an "*underlying determinant*" essential for health. All governments that are party to the International Covenant on Economic, Social and Cultural Rights have an obligation to take steps to realise this human right to health, and ensuring equal access for all to safe and nutritious food.[583] The European Charter on Fundamental Rights which enshrines, among many others, rights relating to health care, environmental protection and consumer protection is also relevant.

A key obligation for governments is to develop a multidisciplinary and multi-sectoral food, nutrition and physical activity policy which are properly monitored and evaluated. It is important that the policy development process, and the implementation of action plans, involves a broad range of stakeholders. Between 2000 and 2008, one third of the member states in the WHO European Region had developed policies on food and nutrition.[10]

The Toronto Charter for Physical Activity has highlighted the need to elevate physical activity as a policy priority and develop, adequately resource, and implement cross-sector policies, plans and intervention programmes to increase population levels of physical activity throughout the world.[584] In most countries action will require reorienting services and funding systems to prioritise and promote physical activity. This includes developing partnerships involving health and non-health sectors and communities at multiple levels. Action to build capacity in research, practice, and training, and in public health systems will also support the promotion of physical activity.

A wide range of specific policy areas where governments should take action is outlined in the following sections. It is important to point out, however, that to be able to take a lead on nutrition and physical activity levels, countries need to have resources, capacity and some basic systems and tools in place. In order to be able to evaluate the current nutritional situation and dietary patterns, countries require national food composition tables and a national monitoring system for food consumption and dietary intake with nutritional status evaluation. To be able to implement dietary guidelines, the people on the ground (caterers, health professionals) need tools. It is also vital that countries have a pool of trained, specialised professionals who can show leadership on nutritional issues.

Building capacity in central and eastern Europe

Currently, capacity development in nutrition is a process whereby individuals, groups, institutions, organisations and societies enhance their abilities to identify and meet challenges in a sustainable manner. This is in line with the 10 principles for Capacity Development suggested by United Nations Development Programme (UNDP).[585] Capacity development (CD) in food and nutrition is much more than formal training and includes human resource development, and organisational, institutional and legal framework development with the aim of enhancing nutrition-relevant knowledge and skills to support infrastructural development.

Capacity development is a long-term, continuous process, which focuses on national priorities, plans, policies and processes.

Currently, many central and eastern European countries do not have the capacity or resources that they need to be able to develop and implement comprehensive food, nutrition and physical activity action plans.

The Network for Capacity Development in Nutrition in Central and Eastern Europe (NCDNCEE) (http://www.agrowebcee.net/ncdn/), is one of several regional professional networks developed in 2006 under the UN SCN working group (WHO and several other UN agencies are members of UN SCN), including a network for the central and eastern Europe (CEE) countries.[586]

The network has identified some nutritional challenges across the region, but also outlined the main challenges relating to nutritional policy development and implementation.[586] Furthermore, many central and eastern European countries do not have the capacity or resources that they need to be able to develop and implement comprehensive food, nutrition and physical activity action plans. The network identified the main nutritional challenges across the region, but also outlined the main challenges relating to policy development and implementation[587] and carried out an inventory of training needs.[588] Thus in the CEE Network the discussions highlighted a number of issues that could be part of a common strategy to build capacity in central and eastern Europe. These include:

- Continued development of national food, health and nutrition policies and action plans, including dietary guidelines;
- A common policy guidance from WHO and other central UN agencies to help states in their policy development on higher nutrition training as part of their capacity development efforts;
- Higher nutrition education and training at university level (including in-service training);
- Standardising nutritional monitoring in the region;
- Further capacity development and harmonisation in food composition data bases;[587,588]
- Harmonisation of the dietary recommendations;[511]
- Facilitating further development and usage of the nutritional tools, nutrition software, e-learning modules courses[589,590] developed in different European projects;
- Developing web pages for exchange and collaboration between Central and Eastern European countries (http://www.agrowebcee.net/ncdn/).

The sparse higher education and training in nutrition was highlighted as a particular area of concern by professionals in the region. This higher education and training in nutrition was considered a prerequisite for reversing the negative trend of the nutrition transition. Only a few countries in the region had specific training for nutritionists and, generally, there is inadequate knowledge of nutrition among health professionals. In 2010 this network had carried out an inventory of training needs (questionnaire sent to collaborators within NCDN network) that revealed one of the key problems related to capacity building in public health nutrition, namely a lack of basic and academic nutritional education in the majority of CEE countries. There is a similar lack of trained experts in physical activity, working among common people and apparent absence of actions (government-initiated or otherwise) to promote healthy lifestyles in CEE societies.

It is clearly important that WHO, FAO, Unicef, UNU, Cornell, Wageningen, the European Commission, and other non-European countries support the capacity development efforts in the CEE countries. WHO's regional office for Europe has already made a clear commitment to provide technical support to help member states build capacity.[10]

Local government taking the initiative

The precise division of responsibilities between national government and regional or local government will vary from country to country. Wherever that dividing line lies, regional, district, town and city councils can play an important role.

Even though in many cases national governments have overall responsibility for health policy, there are still important areas that may come under local government jurisdiction. Some examples include:

- Responsibility for pre-school care, schools and other educational institutions;
- Local planning laws, urban, rural and regional development strategies—which could have a major impact on the built environment and the food environment;
- Responsibility for leisure facilities, parks and open spaces;
- Responsibility for health and social care facilities.

There are more and more examples of local authorities taking a lead on nutrition and physical activity issues, sometimes using their powers in innovative ways. The city health department in New York, for example, used its existing powers to inspect and control restaurants and catering outlets to introduce a new requirement for calorie posting on menus and menu boards. The health department also introduced nutritional standards for food served or sold in all city agencies (including schools, care homes, prisons etc). Elsewhere, local authorities are also increasingly looking at using their local planning powers to control the food environment around schools, for example.

The potential of local and regional government to make a real difference in relation to nutrition and physical activity is now more widely recognised. In 2006, the World Health Organization awarded the French region of Aquitaine the award for promoting healthy nutrition and lifestyles, for its programme for nutrition and health of children and teenagers—a programme which combined the provision of sustainable, healthy food and the promotion of healthy eating.

Civil society – a role for national heart foundations

Civil society as a whole has an important part to play in pushing forward health policy. The fact, for example, that the community in North Karelia was very engaged in the whole issue of cardiovascular disease prevention and very supportive of, and involved in, decisive action is often cited as in important element in the initiative's success.

National heart foundations have a vital role to play within civil society. There is important work to be done in raising public awareness of the issues and mobilising public opinion on the need for government action. National heart foundations can also help to build the cross-sectoral alliances that are important for the development and implementation of national action plans. Foundations can, for example, take a lead in facilitating tri-partite discussions involving government, industry and civil society.

It is also vital that national heart foundations—along with the rest of civil society—continue to hold governments, institutions and industry to account.

The "bomb neutralisers" campaign in Finland

The Finnish Heart Association, together with the Finnish Diabetes Association, carried out an internet-based campaign for parliamentary candidates during the 2007 election campaign. The campaign was repeated during the municipal elections in 2008. The key messages of the campaign were: 1) health-related decisions are made at every level of public policy making, 2) health issues should be taken into consideration in the parliament and municipal councils, 3) health promotion, high quality health care and rehabilitation should be available for every citizen.

The campaign had two steps: bomb activation and bomb neutralisation. The bomb activation part of the campaign described the current situation and outlined which public decisions have led to this situation. The role of each individual citizen in maintaining health was also emphasised. The bomb neutralisation element of the campaign presented good practices already being carried out locally.

2.4.2 Areas for policy development — product, price, promotion and place

The following sections use the "4Ps of marketing" to outline possible areas for policy development and action. Marketers use this model, also known as the "marketing mix", to assess how well products match their target market by considering factors to do with **product, price, promotion and place**.

Product: the important policy areas to address include product reformulation, trans fats bans and labelling measures to ensure consumers can understand the nutritional content of foods.

Promotion: There are two different aspects to promotion and both are important. On the one hand, there are measures relating to the impact of modern marketing and promotional techniques on food choices and, particularly, on children. On the other hand, promotional campaigns and other techniques have tremendous potential to promote healthier diet choices. This section deals with policy issues on both sides of this coin: controls on marketing to children, rules on nutrition and health claims, and the use of marketing techniques and mass media campaigns to promote healthy options.

Price: Policy issues relevant to the price category include the use of taxes, subsidies and pricing policies to make healthier choices more affordable and less healthy choices more expensive, the impact of the Common Agricultural Policy on diets and measures to ensure that vulnerable and disadvantaged groups have access to affordable healthy foodstuffs.

Place: Several important policy issues fall under the umbrella of place. These include planning and other measures to improve access to affordable healthy food, nutritional standards for foods provided in public institutions, schools as environments to promote healthy eating and physical activity, nutritional labelling and improving access to healthy choices when people eat out, workplace initiatives, environments and infrastructure that promote active living and health services that promote healthy lifestyles.

2.4.2.1 Product

It is obvious that the nutritional quality of the food that is produced in, or imported to, Europe affects the nutritional quality of the diet. Of course, people's ease of access to foods high in fat, sugar or salt and, conversely, to fruit, vegetables and cereals has an impact on what they eat. Agriculture and trade policies have an influence on which primary foodstuffs are produced, and on the nutritional quality of some of those foodstuffs. The activities of the food manufacturing industry—and the laws that govern them—also have an enormous influence on European diets. Furthermore, the nutritional quality of meals and snacks served in catering outlets (from cafés and restaurants to hospitals and schools) is increasingly important in terms of contribution to the overall diet.

Yet, until relatively recently, this has been a neglected area for any action. Policy tended to be much more focused on changing demand (by educating the public to change eating habits) than on changing aspects of the food supply. In recent years, however, policy makers have started to focus attention on changing the nutritional quality of mainstream food and drink products. Some countries, for example, have legislated to limit the presence of harmful trans fatty acids in foods. Food authorities in various countries, along with the European Commission, have been working with the food industry to encourage reformulation of mainstream food products to reduce fat, salt and sugar contents.

Another important aspect of the "product" is the label, which should provide consumers with the information they need to be able to make healthier choices.

Using this analysis, we can also think of "physical activity opportunities" as a product. In this sense it is important that people have access to, and information about, a range of opportunities for physical activity, both in ways that integrate into daily routines and as leisure activities. These opportunities should reflect the needs and preferences of the "consumers" they are designed to reach. It is important to consider issues of availability, accessibility and affordability. Policies to ensure the quality of physical activity opportunities are important and building the evidence base on what types of physical activity interventions are most effective is a priority.

Policy Area	Recent developments	EHN Recommendations
Reformulation of food products to reduce the salt, saturated fat, and added sugar content of foods. Increasingly, attention is focusing on the importance of changing mainstream food and drink products to reduce their contribution to salt, fat and sugar intakes. It is interesting to note the potential synergy between other measures—such as improved nutrition labelling, advertising restrictions or calorie posting—and product reformulation. In the UK, for example, restrictions on advertising to children and colour-coded nutrition labelling appear to have acted as an incentive for food manufacturers to reformulate some products.[541] Similarly, part of the rationale for introducing a law requiring fast food chains to post calorie counts on menus in New York City was to encourage fast food outlets to modify the products on their menus or reduce portion size.[592] Changing the nutrient content of foodstuffs is also appropriate further back down the food chain. Agricultural production techniques can affect, for example, the fat content and the fatty acid profile of meat and dairy products. Extensive livestock production (grass-fed) has a positive effect on saturated fat content in both red meat and dairy products.	The EU strategy on nutrition, overweight and obesity (the nutrition White Paper) signalled the Commission's intention to facilitate more product reformulation. Initially, efforts at the EU level—as elsewhere—focused on salt. The EU Framework for National Salt Reduction Initiatives (the salt reduction initiative) was published in 2008. Salt reduction has been the first priority of the new High Level Group on nutrition and physical activity. The framework aims to support national plans, to enable progress to be compared, to ensure consistent messages to the food industry and to help generate momentum and action. The EU Council gave its backing to salt reduction measures, with an emphasis on reformulation, by adopting a series of conclusions in relation to actions to reduce population salt intake for better health in May 2010.[593] There are now salt reduction strategies in many countries and several countries have initially focused on efforts to reduce the salt content of bread. Some countries are pursuing a voluntary approach while others have introduced legislation. A voluntary agreement with the bakery industry in Spain, for example, succeeded in reducing the salt content of bread by more than 18% in four years.[594] The UK and Belgium have also adopted a voluntary approach. Finland introduced legislation many years ago and Portugal, which has particularly high stroke mortality rates, has launched a multi-sectoral initiative and succeeded in passing legislation to limit the sodium content of bread (having found that Portuguese bread contains almost half as much salt again as bread from six other European countries) and other processed foods, as well as a requirement to list the salt content of foods. So far, only the UK and Finland have been able to show a clear impact on population salt intakes. Attention is now turning to the need to reformulate products to reduce the fat and sugar content and to reduce portion sizes (see below). So far, the preferred approach appears to be voluntary, based on working with industry. A discussion paper presented to the High Level Group on Nutrition and Physical Activity in December 2009 made the point that although the reformulation efforts started with a focus on single nutrients (eg trans fats in Denmark, salt in UK), countries are now more likely to consider a whole product approach.	**European Commission** Efforts to reduce the fat, sugar and salt content of mainstream food and drink products should be a key priority for Europe. The European Heart Network has supported the European Commission's efforts in this area, but questions whether the proposed voluntary approach will be adequate to secure the full public health gains. EHN is also concerned that some sectors of the population may miss out on the benefits if reformulation is limited to voluntary private sector efforts. The Commission should set firm targets for progress in product reformulation and should set a priority list of products for reformulation (e.g. bread and bakery, ready prepared meals, breakfast cereals) and target levels. If the collaborative voluntary approach does not deliver results within that timescale, the Commission should not hesitate to adopt a regulatory approach and introduce rules setting maximum levels of these nutrients/ingredients for different foodstuffs. Efforts to reformulate products should be accompanied by government information campaigns that help to create consumer demand for lower salt/fat/sugar products with a higher fibre (non-starch polysaccharide) content. Agricultural production methods that improve the nutritional quality of foods should also be promoted with specific measures being taken to increase the production of vegetables and fruit and their ease of access to publicly-funded facilities. **National governments** In addition to pan-European efforts, national governments should work with the manufacturing, catering and retail sectors to bring about reductions in fat, sugar and salt contents. Governments should consider taking a mandatory approach to reformulation if a voluntary approach does not work. **Food industry** The food manufacturing, retail and catering industries should build on the progress that has already been made, and collaborate fully with national and European authorities to reduce the fat, sugar and salt content of foods.

Initiatives to reduce portion size. The growing understanding that people who are served more food eat more (regardless of other factors) has focused some attention on reducing portion size. Research shows that average portion sizes have increased in the last 10-15 years and people's energy intake increases when they are offered larger portions, as people tend not to compensate between meals.[595] Bigger portion sizes appeal to consumers because they imply better value for money and also because, over time, exposure to larger portions affects perceptions of what is an appropriate portion for one's size and level of physical activity.	Linked to the recent efforts on product reformulation, there has also been attention on the need to reduce portion sizes. The European Parliament has called on industry to review single-service portion sizes, providing a broader range of smaller portion options. More than 10 companies in Europe have made commitments to the EU Platform for Action on Diet, Physical Activity and Health that they will reduce portion size.	In parallel with initiatives to reformulate food products, the European Commission, national and local governments, along with the food industry and the voluntary sector, should facilitate, and encourage initiatives to reduce portion size.
Legislation to ban industrially produced trans fatty acids Trans fatty acids are found in commercial baked foods, fried foods, frozen foods, margarines, red meat and dairy products. Although trans fatty acids occur naturally in dairy and meat products, they are also industrially produced and added to processed foodstuffs. Given the strong evidence that consumption of trans fatty acids is a risk factor for cardiovascular disease, some national and local governments have taken the lead in regulating to restrict the amount of industrially-produced trans fats in foods.	In 2003, Denmark introduced legislation to limit the amount of partially hydrogenated oils in foods. Some local administrations were quite quick to take a lead from the Danish example, and cities such as New York and Calgary introduced legislation to limit trans fats in foods from restaurants and fast food outlets. National governments, however, have been relatively slow to follow the Danish example. In recent years, Switzerland, Austria and Iceland are among those countries that have introduced legislation to limit the trans fat content of foods and Spain has been considering a proposal. In the UK and the Netherlands, food authorities have worked with food manufacturers to agree a voluntary approach to reduce trans fats. The US and Canada have introduced mandatory labelling to warn consumers that foods contain trans fatty acids. A study for the European Parliament[596] found that the Danish ban on industrial trans fatty acids has been more effective in limiting trans fats intakes than the voluntary action or the mandatory labelling requirements. The study, commissioned for the Parliament's ENVI committee, called for a ban on industrially produced trans fats to be considered at EU level. The study was followed up by an initative by five MEPs to adopt a written declearation. However, The initiative did not achieve the required amount of signatures and fell in September 2010.	A legislative approach to reducing industrially-produced trans fatty acids has been shown to be effective. Given that trans fat intakes can vary dramatically between and within European countries, policy in this area should be guided by actual intake data of vulnerable groups and not on population averages. Measures are also needed to ensure that trans fats are not replaced with saturated fat. **European Commission** The European Commission should bring forward a proposal for an EU-wide regulation to eliminate industrially produced trans fats in foodstuffs marketed in the EU. Action at the EU level will support the functioning of the internal market while at the same time ensuring a high level of health protection for all. **National governments** In the absence of proposed EU legislation, or for countries outside the EU, national legislation to limit the presence of industrially-produced trans fatty acids in foodstuffs is recommended. **Local governments** In many countries, food sold in catering outlets is regulated at the local level (with an existing local inspection infrastructure). In the absence of national legislation, this presents the opportunity for local authorities to ban trans fats from foods served in restaurants, fast food chains, pub, cafés and other catering outlets. **Food industry** EHN favours the regulatory approach in this case, as it has been shown to be more effective. Nonetheless, the voluntary approaches in the UK and Netherlands have yielded some progress and—pending any national or European legislation—the food industry should work collaboratively with national or local food authorities to reduce the presence of trans fatty acids in foods. In particular, multi-national companies have a responsibility to ensure and demonstrate that the low levels of trans fats that they can achieve in countries where there is regulatory or policy pressure are applied uniformly across the countries where they operate.

Ensuring that consumers have easy access to meaningful information about the nutritional quality of foods. For a food and nutrition strategy to be effective it is essential that consumers can understand at-a-glance what is in their food. Clear, nutritional labelling is a crucial part of educating people about a healthy diet and how different foods contribute to it. For over 20 years public health advocates and consumer groups have been calling for rules to ensure consistent access to clear, understandable nutrition information. Consumer research shows that colour coding is an effective way to ensure this rapid understanding.[597] Nutrient profiles that enable the classification of foods that are high in fat, salt and sugar are an essential element for effective controls on food labelling and advertising.	In relation to nutrition labelling, the Commission adopted a proposal for a regulation on provision of food information to consumers in January 2008. The Commission has tried to establish nutrient profiles as required by the regulation on health and nutritional claims. The Commission's approach to nutrient profiling has come in for a great deal of criticism from public health advocates (including EHN) and consumer groups.[598]	**European Commission** Mandatory, legible, back-of-pack and front-of-pack labelling should receive first priority at the European level. EHN continues to call for a simplified front of pack scheme. This should be a "sign posting" scheme that would enable consumers to identify healthier choices at a glance. It should only contain key elements: energy, fat, saturated fat, sugars and salt. The front-of-pack scheme should be colour coded with red, yellow and green indicating high, medium and low levels of the four elements (based on percentage GDAs). **National governments** Urgent action is needed to improve the provision of nutrition information in restaurants, cafés and other catering outlets. Authorities should consider a mandatory requirement to provide nutrition information (in the form of a traffic light labelling scheme covering key nutrients) for all chains with more than 10 outlets.
Ensuring availability of fresh drinking water Policies that promote access to free, safe drinking water could contribute to reducing consumption of sugary drinks. Schools, workplaces, public institutions, local authorities and private caterers could all play a role.	In France, public health advocates have been calling for legislation to introduce a requirement for water foundations in all public places (schools, higher and further education establishments, swimming pools and sports facilities, ministries, town halls, public institutions) and transport facilities, as well as in private companies.	Policies to ensure the provision of safe unsweetened drinking water, particularly in schools, workplaces, public places and public institutions should be introduced.

2.4.2.2 Promotion

The food and soft drinks industry use a variety of techniques to grab our attention. Market research—including techniques such as focus groups to identify words and images that provoke a positive reaction and studies of shoppers' eye movements—shows that choices about what to eat and what to buy can be influenced by the way those choices are presented.

Contemporary food marketing techniques have been called the "*modern sirens, leading us inexorably to chronic diseases and sometimes to early death.*"[599] There is growing recognition that children, at the very least, should be protected from these sirens.

Food and nutrition policy also needs to promote healthier eating options. As well as appropriate use of mass media educational campaigns, policy makers need to consider how modern food marketing techniques can be used to better effect to promote healthy choices.

This section covers both policy issues that seek to mitigate the potential negative impact of modern marketing techniques on food choices and those that aim to use marketing and promotional techniques to promote healthier diets and more active living. It is important to recognise, however, the vastly different amounts of resources to these different types of promotion. The communication budget of France's national nutrition programme (PNNS), for example, is equivalent to only 0.5% of the budget that food and agriculture companies allocate to television advertising in France.[600]

Given this huge imbalance in resources, along with the fact that promotional techniques can be used to have either a positive or a negative impact, is it is clear that policy action to promote healthier choices will only be successful if it is combined with efforts to neutralise the marketing power directed at children.

Policy Area	Recent developments	EHN Recommendations
Controlling advertising of unhealthy foods aimed at children There is now widespread recognition that children should be protected from commercial influences that may influence their eating patterns. Throughout the last decade there have been growing calls for action to limit marketing of unhealthy foods to children in print, broadcast and electronic media. As well as advertising openly directed at children, there is also the issue of product placement—where food or drink products are portrayed in films or television programmes. A study in the US found that most brand placements in the movies are for energy-dense, nutrient poor foods. Six companies—PepsiCo, Coca-Cola, Nestle USA, McDonald's, Dr Pepper/Snapple Group and Burger King—account for 45% of 1,180 product placements in 138 films between 1996 and 2005.[601] There is also concern about sponsorship of sporting events—both spectator and participation sports—by foods or soft drinks high in fat, salt or sugar. Coca Cola, for example, is a major sponsor of events such as the Olympic Games and the FIFA World Cup. In the UK, for example, confectionery manufacturer Mars has sponsorship deals with football's governing bodies in England and Scotland to encourage participation in the sport. Given the global nature of many food and drink brands (and as national borders become less relevant for modern media) it is clear that international cooperation is needed to have any effect on international commercial media communications. There is, in fact, a precedent for international regulatory action to control marketing, in the form of the *International Code on the Marketing of Breast Milk Substitutes*. This is a useful precedent for international action on the marketing of foods and drinks to children.	A resolution by the World Health Assembly (Resolution WHA 60.23) requested that the Director General, among other things, takes action "*to promote responsible marketing including the development of a set of recommendations on the marketing of foods and non-alcoholic beverages to children.*" In May 2006 in Oslo, WHO convened a Forum and Technical meeting on marketing of food and non-alcoholic drinks to children. At the World Health Assembly in 2010, a new resolution (WHA63.14) was adopted, urging member states to implement the recommendations proposed by the WHO Director General to reduce the impact on children of marketing of foods high in saturated fats, trans fatty acids, free sugars or salt. The Director General is to report back on progress in implementing these recommendations to the World Health Assembly in 2012. The Directive on Audiovisual Media Services obliges EU member states and the Commission to encourage media service providers to develop codes of conduct regarding inappropriate audiovisual commercial communication relating to unhealthy foods accompanying, or included in, children's programmes. The review process that led to this new Directive is seen by many as a missed opportunity to restrict the advertising of unhealthy foods to children. The European Parliament has called on the Commission to bring forward stricter proposals if the voluntary approach is found not to be working. The Commission organised workshops on codes of conduct on advertising unhealthy foods to children in December 2009 and in October 2010 encouraging the development of national codes. In 2010 the UK took action to prohibit product placement (where items are portrayed in films or television programmes) of foods high in fat, sugar or salt in UK-made programmes.[602]	There should be no audiovisual commercial communication for foods high in fat, sugar or salt broadcast between 06h00 and 21h00. Nutrient profiling schemes can be used to identify affected food products. Audiovisual commercial communication includes surreptitious advertising, sponsorship and product placement. Restrictions on marketing of unhealthy foods to children should also encompass non-broadcast marketing techniques (eg online media etc) and the use of toy promotions to promote unhealthy foods. Controls should be introduced to prevent the promotion of links between sports or celebrities and unhealthy foods. **WHO** An international code should be developed—under the auspices of WHO—to reduce substantially the extent and impact of marketing of unhealthy foods and beverages, particularly to children. **European Commission** Measures to protect children from audiovisual commercial communication concerning unhealthy food and drinks should be a priority at European level. **National governments** Pending the introduction of an international code or EU legislation in the long-term, national governments should introduce their own restrictions to protect children from advertising for unhealthy foods. **Local government** Local authorities—including, for example, health, education and recreation departments—can also take action to protect children from commercial messages about unhealthy foods in or near schools, play areas and other places where children gather.

Mass media educational campaigns to increase demand for healthy foods and to promote physical activity. There is now widespread recognition that we cannot rely on mass media education or information campaigns alone to change dietary behaviour and activity patterns. There remains, however, a role for carefully designed campaigns, which should be supported by other policy and environmental changes. Mass media campaigns promoting physical activity that include supportive community activities, and are associated with policies addressing environmental barriers to physical activity, have been shown to be effective.[603] Other media campaigns that use one simple message, promote a national "health brand" or are based on a long-term intensive mass media campaign to promote healthy diets have been shown to be moderately effective.[603]	In its nutrition White Paper, the Commission proposed to develop and support (in cooperation with member states and relevant stakeholders) scientific information and education campaigns to raise awareness of diet and physical activity related health problems.	There is a role for the national and local governments in developing campaigns to increase demand for healthy foods and to promote physical activity. There may also be a role for the European Union to support government initiatives. It is important that any campaigns to promote healthy eating or physical activity are: • solidly embedded in a broader, multi-sectoral food and nutrition policy; • take into account research identifying criteria associated with campaigns that have been effective; and • are backed by supportive policies and broader environmental changes.
Promoting healthy options An integrated food and nutrition policy needs to increase the availability and affordability of healthier foods. It also needs to increase the demand for healthier foods by, for example, effective education campaigns, requiring clear nutrition labelling, promotional campaigns for fruit and vegetables etc. Can the so-called "modern sirens" of food marketing techniques be used to better effect to promote healthy choices? There is much to learn from the marketing techniques employed to promote foods. There is potential for in-store positioning, price promotions and other promotional techniques to be used to make it easier for people to choose healthier products.	The importance of promoting healthier options, such as fruit and vegetables, has been recognised at the international level with the launch, in November 2003, of the joint WHO and FAO Fruit and Vegetable Promotion Initiative. More recently, the European Commission's White Paper contained a commitment to use the Common Agricultural Policy (CAP) to promote healthier foods. At the national level, there are various examples of promotional initiatives to encourage consumption of healthier foods. In France, for example, the inclusion of pre-defined health messages is now compulsory in radio and television adverts for some foods and drinks, thus making use of advertising resources to communicate health messages and counteract commercial messages. Food and drink advertisers can avoid having to incorporate the health warning by paying a tax equivalent to 1.5% of the annual advertising budget for the product in question. In another example, schoolchildren in Cyprus are encouraged to eat fruit by the "Five minutes for five fruits" initiative whereby lessons are interrupted once a week for a five-minute break to eat fruit and discuss the benefits of eating fruit.	**European Commission** At the European level, sufficient funds should be made available for interventions to increase fruit and vegetable consumption. The agriculture and health sectors, which both stand to benefit from increases in fruit and vegetable consumption, should coordinate efforts to promote fruit and vegetables. CAP promotion budgets should give preference to foods of high nutritional value and incentive given to create synergies with the EU School Fruit Scheme and Most Deprived Persons schemes. Unused promotion funds should be made available for public campaigns focusing on healthy diets. Funds should not be used to promote products that are not otherwise promoted as part of healthy eating, eg alcohol. **National and local governments** Authorities at the national and local levels should explore options to promote healthier food and drink products. This should include working with the food manufacturing, retailing and catering sectors, as well as the creative industries involved in the business of persuasion, to harness the potential of marketing and promotional techniques to promote health. **Private sector** The food manufacturing, retail and catering sectors should use their know-how and the range of promotional techniques at their disposition—including product position and price promotions—to promote healthier foods. This private sector involvement should only follow on after the public health messages have been developed by the competent authorities/ government.

Rules on nutrition and health claims Controls are needed on the nutrition and health claims made for food products so that consumers can be confident that foods presented as healthy really are. Nutrient profiles that enable the classification of foods that are high in fat, salt and sugar are an essential element for effective controls on food claims.	In 2006 the EU adopted the regulation on nutritional and health claims on food. This requires the Commission to establish nutrient profiles based on scientific advice from EFSA. The Commission's approach to nutrient profiling has been fiercely criticised. EHN's analysis of the European Commission's latest approach to nutrient profiling, found that the model compared unfavourably with existing models, such as those proposed by the French, British and Australia New Zealand food standards agencies. Under the most recent Commission proposal foods such as doughnuts, pork sausages and some high sugar breakfast cereals would be eligible to make healthy claims, while some sandwiches and high fibre breakfast cereals would be ineligible for claim.	**European Commission** EHN proposes that the Commission stop seeking to develop its own model and adopts the model developed by Food Standards Australia New Zealand—this model would be fit for purpose in the European context. A model with fewer food categories, more nutrients and nutrient scoring is recommended.
Promotion of breastfeeding and ensuring appropriate marketing of breastmilk substitutes There are two aspects of promotion which are important in relation to infant feeding: • strategies which protect, promote and support breastfeeding; • controls on the promotion of commercial alternatives to breast feeding. *The Global Strategy for Infant Feeding and Young Child Feeding* calls on national governments to develop and implement a comprehensive policy on infant and young child feeding. Elements to incorporate into such strategies could include: • increasing maternity leave provisions; • legislating to help improve acceptance of breastfeeding; • providing appropriate breastfeeding facilities (local gov, employers); • ensuring access to time and private space for b/feeding during working hours; • ensuring families are given objective, accurate and up-to-date information on infant and young child feeding by independent health professionals; • establishing mechanisms for mothers to have access to skilled support to help them start and continue breastfeeding; • establishing a network of trained lay or peer counsellors in the community to provide support; • reviewing national implementation of the International *Code of Marketing of Breast Milk Substitutes* and consider additional measures if necessary. Governments that have ratified the Convention on the Rights of the Child (CRC) are bound by its provisions. The CRC: • stresses children's rights to protection from economic exploitation; • recognises the fundamental role that breastfeeding plays in fulfilling the right of every child to the highest attainable standard of health.	*The International Code on the Marketing of Breast Milk Substitutes* was adopted by the World Health Assembly (WHA) in 1981. The International Code has been reviewed every two years at the WHA. Thirteen clarifying and strengthening Resolutions have been passed, along with a *Global Strategy for Infant and Young Child Feeding* in 2002. WHA Resolutions enjoy the same status as the International Code itself. The European Commission funded a project to draw up a *Blueprint for Action for the Protection, Promotion and Support of Breastfeeding* in Europe and a EUNUTNET project to draw up standard recommendations on infant and young child feeding. Policies around employment law and labour practices are important. There is a positive association, for example, between the length of maternity leave and breastfeeding rates and duration of breastfeeding. The nordic countries have tended to adopt a package of progressive policies that protect breastfeeding (while at the same time promoting gender equality). In Sweden, for example, parents are entitled to 16 months of shared, paid parental leave (*föräldraledighet*). Parents are encouraged to share the leave equally, but one parent can take up to 420 days, with the remaining 60 days reserved for the other parent. Women's rights to return to work—on a part-time basis if they want—also have strong protection.	**EU** The EU must ensure that international conventions on infant feeding are adhered to. Europe can also play an important role in supporting member states in the implementation of sound infant and young child feeding strategies. There is also an important role for Europe in setting a framework for social and employment policies that protect breastfeeding by improving parental leave provision, requiring facilities for breastfeeding etc. EU policies must be informed by, and based on, evidence, which includes independently reviewed and independently funded research. Notably, EU Policy should support member states' right to adopt legislative measures setting the highest levels of protection, including, but not restricted to, banning the promotion of breastmilk substitutes (including follow-on formula and specialised formulas) and banning all health and nutrition claims on foods for infants and young children and pregnant and nursing mothers. **National governments** Governments should devise and implement national strategies for infant and young child feeding, which incorporate wide-ranging measures to protect, support and promote breastfeeding and reviewing on a regular basis national implementation of the International Code of Marketing of Breast Milk Substitutes and subsequent relevant WHA Resolutions.

2.4.2.3 Price

Continual advances in technology have generally meant that calories are cheaper to come by, as food has become less expensive in relation to other commodities and to income in recent decades. Similarly, the EU Common Agricultural Policy (CAP) has long been subsidising milk and beef production, thus ensuring that foods high in saturated fat have been more affordable, while fruit and vegetables have been relatively expensive.

Taxing unhealthy food: an important debate

It is well known that altering the price of foods can influence what people eat. There has been growing interest in, and debate about, the potential for governments to use economic instruments—such as taxes or subsidies—to make healthier foods more affordable and less healthy foods more expensive. The use of taxes for public health goals is not new—tobacco and alcohol are widely taxed. In both cases, using taxes to increase relative prices has been shown to reduce associated harm and, importantly, to impact on the smoking and drinking habits of young people.[604,605,606] While the very important distinctions between food on the one hand and smoking and drinking on the other must be borne in mind, it is clear that there are some lessons which can be drawn from the earlier discussions in relation to tobacco and alcohol taxation. Taxes on tobacco and alcohol, for example, are known to have a greater impact on poorer groups, prompting concerns about the impact of such regressive taxes compounding the deprivation of poorer families. In fact, modelling work on the impact of tobacco tax suggests that any loss of life expectancy attributable to tobacco tax would be 42 to 257 times less than the loss of life expectancy attributed to smoking.[607]

We know that taxes on unhealthier foods are also likely to have the greatest impact on poorer families. This is because they spend a larger proportion of their income on food and also because groups with lower socio-economic status tend to eat more of the unhealthy foods that would be targeted. This has prompted researchers and public health advocates to explore ways of mitigating the potentially negative impact of such taxes on poorer groups, while maximising the public health gains. One option is to ensure that taxes are always used in combination with subsidies on healthier foods. Another is to focus taxes on products—such as sugary soft drinks—where very similar alternative products exist as replacements. Mechanisms to make adjustments to welfare payments and benefits to reduce the economic impact of the higher taxes also merit exploration. It is clear that solutions need to be tax revenue neutral for governments.

At the more local level, pricing strategies in public institutions—such as schools, hospitals and sports facilities—may also have potential to influence what people eat in these premises. There is huge potential to have a real impact on the diets of very many European if nutritional standards and pricing strategies are used in combination to improve public sector food. Such policies could, in turn, also have an impact on the demand for particular foods—a requirement to provide vegetables and/or salads included in the price of a meal, for example, in Sweden and Finland has generated a greater market for vegetables in those countries.

The issue of price is even more important because of the strong social inequalities in cardiovascular disease that are prevalent throughout Europe. Access to affordable healthy diets in deprived areas has been identified as an important issue.

Price is also an important issue in relation to physical activity. Relative prices of transport, parking and leisure facilities may affect physical activity levels.

Policy Area	Recent developments	EHN Recommendations
The use of economic tools (taxes and subsidies) to make healthier foods more affordable and less healthy foods more expensive. It is well known that altering the price of foods can influence what people eat. In the last few years there has been growing interest in the potential for economic instruments—such as taxes or subsidies—to be used to make healthier foods more affordable and less healthy foods more expensive. Taxes have been used successfully in other areas of public health, particularly tobacco, to affect behaviour. There are concerns, however, that any taxes on unhealthy foods would hit poorer groups hardest. Similarly, higher socioeconomic groups are likely to benefit more from subsidies on healthy foods like fruit and vegetables. Because of this probable regressive nature of any so-called "junk food tax" or "fat tax", there is growing support for the idea of taxes and subsidies to be used in combination. The power of subsidies has been demonstrated by the huge dietary changes witnessed in Poland and former Czechoslovakia since the start of the market reforms in the early 1990s. In Czech Republic the abolition of consumer subsidies of up to 30-60% of retail prices led butter consumption to drop by almost two-thirds (64%) between 1989 and 1994.[22]	The European Parliament's resolution on the Commission's White Paper[608] made a number of recommendations in this area: • urged the EU, particularly the ECOFIN council, to be more flexible over member states application of lower VAT rates for necessities of a social, economic, environmental or health-oriented nature; • called on states that have not yet done so to apply a lower VAT rate to fruit and vegetables, recalling that EU rules authorise them to do this; • called for community legislation to be amended to allow fruit & vegetables to benefit from a lower rate of VAT (<5%). Some European countries have explored the use of economic instruments to influence the affordability of foods. The Romanian health minister, for example, announced plans to introduce a "junk food tax" on fast foods, cakes, confectionery, snacks, crisps and soft drinks. Funds raised by this new tax will be allocated to health programmes. A tax increase of 25% on ice cream, chocolate and sweets and increased taxes on sugary soft drinks (with a tax decrease for sugar free soft drinks) came into force in Denmark on 1 July 2010. The Danish government has also introduced a tax, which came into force in October 2011, on foods with high saturated fat contents.[609] Norway has introduced a tax on sweetened soft drinks. The UK, France and Switzerland are reported to have seriously considered introducing so-called "fat taxes" but eventually abandoned proposals, for the time being at least. At the sub-national level there are also examples of economic instruments in use. In the US, for example, state authorities have proposed taxes (such as an 18% tax on sweetened soft drinks) to raise funds and to tackle problems such as childhood obesity.	Economic instruments, such as taxes and subsidies could play an important role in promoting healthier eating. The issue of taxing unhealthy foods remains an area of intense debate and further work is needed to explore ways of mitigating the impact on poorer groups, while maximising the public health benefits. Mechanisms to explore include the combined use of taxes and subsidies, as well as compensatory welfare payments or tax exemptions. It is clear that the solutions also need to be tax revenue neutral for governments. **European Commission** The introduction of subsidies on healthy foods should be a priority for European action. The options for use of value added tax (VAT) as an economic instrument to improve health should also be explored—such as allowing member states the possibility of levying a negative VAT rate. **National governments** Governments should explore the potential for subsidies on healthy foods, which may be used in conjunction with higher taxes on unhealthy foods. Although EU member states are bound by EU rules on Value Added Tax (VAT), there is still considerable scope for action.
Pricing strategies to promote healthier food choices In addition to the imposition of taxes or subsidies at the national level, there are other ways of using price as an instrument for encouraging healthier food choices. At the local level, pricing strategies in public institutions—such as schools, hospitals and sports facilities—may also have potential to influence what people eat in these premises.	There are local examples of pricing strategies being used in a variety of ways to promote healthy choices. In the US state of Seattle, for example, schools are required to sell sugar-sweetened drinks at a higher price than water in vending machines. In Finland, for example, vegetables and salads are usually included in the price of a meal. This is reflected in the higher consumption of vegetables in young adults having lunch outside the home.[610] The private sector can also play a role. Also in Finland, a retail chain ran a campaign, called Fruit Hit, to promote consumption of fruit and vegetables. They sold fruit and vegetables at a price of 1€/kg or 1€/piece, and found that this encouraged people to buy more fruit and vegetables and to try also exotic alternatives. Reducing fruit and vegetable prices in this way promoted consumption, especially among those for whom price is one of the main criteria in composing their diet.	There is considerable potential for these instruments to be used at both macro and micro levels—from Europe-wide to individual schools or hospitals. **Local authorities** At the local level, education and health authorities (as well as other public institutions which serve or sell food) should utilise pricing strategies to promote healthier eating wherever possible.

Use of the Common Agricultural Policy to promote a healthy diet across Europe Europe's agriculture policy—initially devised to ensure self-sufficiency following the hardships of the second world war—has had a dramatic impact on the diets available to Europe's consumers and the relative affordability of different products. In simplistic terms, by heavily subsidising milk and beef production the Common Agricultural Policy (CAP) has ensured that foods high in saturated fat have been more affordable, while fruit and vegetables have been relatively expensive. There is a strong argument that the objectives of European agricultural policy should be much broader—including the aims of improving health and protecting the environment.	A major CAP reform package was agreed following the 2003 mid-term review. In 2008 the Commission published a health check for the CAP. Attention is now focused on the shape and content of the common agricultural policy after 2013. This coincides with the review of EU budget finance—including agricultural and rural development funding—from 2014 onwards. The communication on the budget review was published in November 2010.[611] The budget review stresses the need for a sustainable EU economy needs a thriving agricultural sector making its contribution to a wide variety of EU objectives— including cohesion, climate change, environmental protection, and biodiversity, health and competitiveness, as well as food security. The Commission communication "*The CAP towards 2020: Meeting the food, natural resources and territorial challenges of the future*" and consequent "*The reform of the CAP towards 2020 Consultation document for Impact Assessment*" were published in November 2010. Both highlighted new objectives of public health, chronic disease and healthy nutrition as priorities for the future of the Common Agricultural Policy. **EU School Fruit Scheme (EU SFS)**— the EU School Fruit Scheme was adopted at the end of 2008. Support worth 90M euros/year agreed. School Fruit Schemes have been implemented in 25 EU member states and efforts are being made to reduce administration burden for member states. **Most Deprived Persons Scheme (MDP)**—Recent changes in the MDP have allowed market purchases in addition to distribution of surplus stocks (intervention stocks). The European Commission adopted an amended proposal for a revised MDP scheme on 17 September 2010. The current proposal looks toward broader nutritional criteria and fruit and vegetables and vegetable based fats will be allowed under the scheme for the first time. Under the revised proposal member states, in cooperation with local service providers, would be given the flexibility to decide which food to distribute based on nutritional criteria.	**European Commission** The objectives of the Common Agricultural Policy urgently need to be brought into line with EU-wide nutrition population goals. This should be one of the key priorities for action. Food surpluses should be targeted for human consumption, especially by disadvantaged communities, in line with nutritional requirements. Market intervention for saturated fat (butter) should be phased out and intervention stocks should not be sold at sub-market prices to commercial operators. The future CAP should be based on a sound impact assessment, as a pre-condition for meeting future objectives. Health, social and environmental impact should be a central measure of the assessment process and identify policy measures that can improve diets and public health across Europe. An integrated European Food and Agriculture Policy, which works towards improving European diets in a sustainable way, should be developed. This policy should provide incentives for production of more plant-based foods and promote a shift towards more plant-based diets, with reduced consumption of meat and saturated fat and increased consumption of fruit, vegetables and whole grains.

Improving access to affordable healthy foodstuffs for vulnerable and disadvantaged groups Social inequalities in cardiovascular disease are well documented both within and between countries in Europe. Such inequalities remain a major challenge for European health and social policy. Dietary patterns and physical activity levels are known to be among the factors implicated. Options to help ensure that disadvantaged populations have access to healthy foods include: • providing food subsidies or vouchers for particular foods (eg fruit and veg) to low-income groups; • distributing food commodities; • providing free or subsidised access to catering outlets.	In *Solidarity in Health: Reducing Inequalities in the European Union*, published in October 2009, the Commission outlined the actions it will take in order to address health inequalities. The European Commission's Food Aid to Most Deprived Persons (MDP) scheme from DG AGRI has an annual budget of 500 million euros. The CAP budget for 2011 includes a pilot project to look at targeted FV subsidies for vulnerable groups (old age, pregnant women and infants, socially and economically disadvantaged groups).	European strategy and national action plans to improve diets should place particular emphasis on improving access to affordable healthy foodstuffs for lower socioeconomic groups. There may be a role for the Commission to support member states by mapping retailers' locations. (See also *Improving access to affordable healthy foods under Place*).
Economic tools (taxes, subsidies and pricing strategies) to promote physical activity Access to affordable physical activity opportunities—particularly for low income groups—is important. Financial barriers may hinder access to sports and leisure facilities and to active forms of travel. Policies and interventions which remove financial barriers to sports and leisure facilities, and to forms of active travel, are important. There are a variety of policy options on offer: • tax incentives (eg tax credits) on the purchase of bikes or other sports equipment, gym membership or children's participation in activity programmes; • subsidising sport and leisure facilities and activities; • measures to improve the relative affordability of walking or cycling compared to vehicle travel (e.g. congestion charging, parking charges). Interventions to improve affordability of physical activity opportunities should go hand-in-hand with efforts to create built and natural environments which promote physical activity and provide access to free physical activity opportunities.	The EU Physical Activity Guidelines adopted in 2008 recognise this important issue with the inclusion of a guideline that "governments should launch initiatives to coordinate and promote public and private funding devoted to physical activity and to facilitate access for the whole population."[515] The Hungarian government funds the Open Doors programme to enable sports facilities to stay open in the evenings and at weekends. Government-supported clubs also run the Moonlight programme which offers free evening or night sport activities to young people in deprived areas.[515]	Governments—at national, local and regional levels—should pursue policies and interventions which remove financial barriers to sports and leisure facilities, and to forms of active travel. These may include reducing prices, subsidising facilities, providing vouchers and/or prescribing physical activity. It should also include ensuring that appropriate facilities are provided in areas of disadvantage, that built and natural environments are designed to promote physical activity and that useable and affordable transport to travel to physical activity opportunities is offered.

2.4.2.4 Place

The physical locations where we buy or eat our food can have an important impact on our diets. Similarly, features of the environments where we live, work and play can affect how active we are.

The array of products on offer in shops and markets where we buy foods obviously has an impact on what we eat. Less obvious, perhaps, is the importance of products that are not on the shelves—access to fresh fruit and vegetables, for example, is known to be problematic in many disadvantaged communities in Europe.

An ever-growing proportion of meals in Europe are eaten outside the home. The potential influence of the wide variety of settings where people eat meals or snacks—including schools, workplaces, hospitals, leisure facilities, restaurants, pubs and cafés—is enormous.

Place is also tremendously important in determining physical activity levels. Environments to promote active living require an adequate infrastructure for walking, cycling and public transport.

Policy Area	Current situation and recent developments	EHN Recommendations
Improving access to affordable healthy food and physical activity opportunities Poor access to affordable healthier food items, such as fresh fruit and vegetables, is known to be one barrier to better nutrition for many of Europe's citizens. Measures to improve access to shops or markets selling healthier foods in under-served areas should feature as part of an integrated food and nutrition strategy. In addition, measures to make it easier for people to buy foods directly from farms can contribute to a sustainable food and nutrition strategy. Policies which improve access to physical activity opportunities are also vitally important. In recent decades communities have increasingly been designed in ways that discourage, rather than encourage, physical activity. In addition, leisure or sports facilities are scarce in some areas. In response, policies need to target: • Better provision of accessible, appropriate sports and leisure facilities; • Provision of more and safer cycle paths, cycle lanes and footpaths; • Better access to attractive and safe public open spaces.	In the nutrition White Paper the Commission acknowledges the importance of ensuring the existence of healthy options. The *WHO European Action Plan 2007-2012* calls on governments to improve the availability and affordability of fruit and vegetables and to ensure that commercial provision of food products is aligned with food-based dietary guidelines. There are numerous examples of relevant actions at the national and local levels. An initiative in convenience stores in Scotland, for example, improved access to fruit and vegetables in 600 stores in deprived areas. In the US, the local authority in South Los Angeles introduced a one-year ban on permits for fast food restaurants as part of efforts to tackle high obesity rates. The EU physical activity guidelines (2008)[515] emphasise the importance of policies to improve access to physical activity opportunities.	**European Commission** EU policies should encourage and facilitate retailers to locate in under-served areas. European agricultural policy should aim to increase access to mechanisms such as farmers' markets, community-supported agriculture, pick your own and farm-to-school initiatives. The EU should also facilitate, encourage and support policies and interventions to ensure access to affordable physical acivity opportunities. **National and local government** Planning and zoning rules should be used to ensure that all communities have access to retailers offering affordable healthy foods. Authorities should also explore the use of incentives to encourage and facilitate location of retailers in under-served areas. Possible incentives could include tax benefits and discounts loans, loan guarantees, grants to cover start up costs (e.g, refrigeration, warehousing). Strategies to create incentives for existing retailers to offer healthier choices in areas that have poor access should be explored. Similarly, they should take health considerations into account in planning decisions relating to the location of fast-food outlets and other catering establishments. National and local governments should develop strategies to increase and improve mechanisms for people to buy directly from farms and to support community food growing. Options include community allotments, school growing schemes, temporary leases to allow growing on unused land and a community land bank to act as a broker between landowners and groups wanting land for growing food. Local, regional and national governments should implement strategies to ensure provision of accessible, affordable and appropriate physical activity opportunities.

Improving the nutritional quality of food served and/or sold in public institutions (sports and leisure facilities, government offices, universities, facilities for older people, prisons, detention centres, hospitals and other public services). (See below for schools). In the same way that hospitals, educational establishments and government buildings took the lead in establishing smoke-free public places, public sector bodies have the potential to make considerable improvements to diets across Europe. There is a wide variety of ways in which public institutions could take action. These include: • applying nutritional standards to all food sold or provided; • devising procurement guidelines which ensure that health criteria are taken into account; • improving availability of affordable healthier choices (including by pricing strategies); • restricting availability of unhealthy food and drinks; • introducing smaller portion sizes; • limiting advertising or commercial information for unhealthier foods.	Some national and local administrations have sought to improve the nutritional quality of food provided in public institutions. The potential impact of action on the nutritional quality of public sector food is enormous—in the UK alone, for example, nearly 1 billion meals are served annually in schools, the health service and prisons.[612] The health department in New York City has adopted a mandatory approach. Nutrition standards for all food sold or served in city agencies (including schools, centres for older people, homeless shelters, child care centres, after school programmes, prisons, public hospitals and parks) were introduced in 2008.	Ensuring that food sold or served in public institutions is coherent with population-wide dietary goals should be a key priority for action at the European level. This should apply to all foods served in public institutions, whether it is sold directly or by an external provider under contract. These efforts to improve the nutritional quality of public food should be consistent with other policies towards a sustainable food supply—promoting procurement of local and sustainable produce—and could be an important economic driver for an improved food supply. **European Commission** The Commission should support and encourage member states in the application of nutritional standards for food sold or served in public institutions. A first step could be a Europe-wide review of the existing quality standards and procurement practices for food provided in public institutions. This could be done, for example, under the auspices of the High Level Group as a cooperative measure. **National and local governments** Authorities at the national and/or local level should act to introduce nutritional standards for foods sold or served in public institutions. Public procurement guidelines should ensure that health criteria are taken into account. The application of nutritional standards should be accompanied by pricing strategies to improve the relative affordability of healthier items, introduction of smaller portion sizes, restrictions on commercial information relating to foods high in fat, sugar or salt and rules requiring public institutions to provide safe drinking water.

Schools and pre-school facilities as health-promoting environments that encourage and facilitate healthy eating and active living

Schools and pre-school facilities play a vital role in protecting and promoting the health of children. Their role includes equipping children and young people with the knowledge and skills they need to be able to eat healthy diets and live active lives. In addition, children spend a large part of their waking lives in schools and the meals and snacks they eat in school, or during the school day, are an important part of their diets

Food

Comprehensive, high-intensity, multi-component school-based interventions on diet and physical activity have been shown to be effective.[603]

A whole-school approach to promoting healthy eating can include:

- applying nutritional standards to *all* food provided or sold in schools;
- ensuring children are educated about food and nutrition and equipped with practical food skills;
- increasing access to drinking water;
- restricting commercial information;
- restricting access to unhealthy foods outside the school premises.

Physical activity

Similarly, components of a whole-school approach to promoting physical activity can include:

- increasing time spent in physical education;
- increasing amount of actual physical activity in PE classes;
- increasing opportunities for extra-curricular activity;
- encouraging community use of sports facilities;
- reducing screen time in childcare facilities.

Within the EU, schools are an area of member state competence. Nonetheless, the Commission's nutrition White Paper reiterated the crucial role that schools play and emphasised that there is already firm evidence that locally focused interventions, with very wide ownership, targeting children under 12 can be effective in changing long-term behaviour. The White Paper also emphasised that it is important that schools remain a "protected environment" and that any partnerships with the private sector should be undertaken in a "transparent and non-commercial way."

At the end of 2008 the EU adopted a School Fruit Scheme with support worth 90M euros/year agreed.

Across Europe, there are many examples of school-based interventions and educational policies aimed at improving nutrition and physical activity levels among children and young people.

Many countries have taken action to improve the quality of food provided in schools, and access for children from all socio-economic backgrounds. Finland, for example, has been providing free school lunches in primary schools since 1948—a measure which has since between extended to secondary education and universities.

Other countries are taking action to restrict access to foods high in fat, sugar and salt in schools. France has banned vending machines from schools and Latvia has banned the sale of certain types of food and soft drinks in pre-school facilities and schools.

There are also examples of national action to improve access to healthier foodstuffs in schools. Norway and England have schemes to provide some school-children with a free piece of fruit or vegetables. Denmark, Italy and Portugal all have, or are planning to introduce, a fruit break in schools. Primary schools in Wales have been providing free school breakfasts since 2004.

Some local authorities have taken action to support health-promoting policies within the school. The London Borough of Waltham Forest, for example, proposed planning regulations that would prevent hot food takeaway shops from opening within 400m of the boundary of a school or youth centre. In an alternative approach to restricting pupils' access to fast food outlets, Glasgow City Council in Scotland is restricting secondary school pupils from leaving school during lunchtime.

There are also examples of education departments acting to restrict children's exposure to commercial information in schools. Schools in Scotland, for example, were issued with official guidance requiring them to discuss any sponsorship contracts valued at more than £3,000 with local authorities.

Similarly, there are examples of initiatives to increase physical activity in schools. In Denmark, for example, the number of physical education (PE) lessons have been doubled, along with provision of new equipment and training for teachers.[316]

The creation of health promoting schools, that adopt a whole-school approach to health, should be encouraged.

In relation to food, this should include:

- ensuring that children have the education and practical skills necessary to enable them to eat a healthy diet by including both nutrition and food skills in the school curriculum;
- ensuring that food provided in schools complies with high nutritional quality standards (whether meals, or from vending machines or other outlets);
- ensuring that energy dense and high-salt snacks, high-sugar soft drinks and confectionery that is high in fat or sugar are not available in schools;
- the development of policies (whether by schools, education departments or local authorities) which limit children's access to outlets selling unhealthy foods during the school day;
- ensuring access to free drinking water for all pupils;
- a prohibition on commercial communications for foods high in fat, sugar or salt aimed at children and young people in schools, as elsewhere.

The impact of these measures could be greatly enhanced by introducing policies to provide free school meals to all pupils.

In terms of promoting physical activity, the whole-school approach should encompass:

- increasing the number of hours of physical education and activity in the school day, with a recommended minimum number of hours per week;
- establishing guidance and incentives for schools and local government to improve the environment around schools to encourage walking and cycling;
- establishing safe routes to schools from neighbouring communities;
- facilitating and improving the quality of physical education;
- promoting opportunities and practices to build activity in and around the school day (including before and after school clubs);
- establishing safe zones around all schools where walking and cycling are prioritised and car travel is much more difficult.

Measures to enable people to make healthier choices when they eat out (in the commercial catering sector) More and more meals are eaten or prepared outside the home—including in restaurants, cafés, pubs, fast-food outlets, service stations, entertainment venues or parks etc. In some countries, people obtain up to a quarter of their food energy from food eaten outside the home. There is considerable scope for improving diets across Europe by: • improving the nutritional quality of food served in commercial catering outlets; • improving provision of nutrition information to consumers at the point where they make choices about what to eat; • improving the availability of healthier choices; • providing nutrition education to catering staff and restaurauteurs to enable them to modify cooking practices and promote healthy eating. There is potential synergy between measures to improve the provision of nutrition information about foods and the availability of healthier options.	At the national level, there are examples of action to improve the provision of information about the nutritional quality of food eaten outside the home. In England, for example, the Food Standards Agency is working with sectors of restaurant industry on a voluntary basis to encourage provision of nutrition info on menus. In the US, various local jurisdictions have legislated to require fast food outlets to post nutritional information (most commonly calories) on menus and menu boards. To pre-empt the introduction of many different versions of the law, some of the largest fast food companies have announced plans to voluntarily introduce menu calorie posting. Initiatives may also target particularly at-risk groups. In Sweden, for example, an intervention to promote healthier eating among lorry drivers combined the use of incentives (lottery tickets for healthier choices), education (via truck stop staff) and promoting a healthier "meal of the day" option at truck stops. A study in one of the participating truck stops found a 26% reduction (280 kg) in gross fat usage in four months.[614]	Improving provision of nutrition information on food eaten outside the home should be a priority for action in European countries. Authorities should introduce legislation to require nutrition information to be displayed on menus and menu boards for all restaurants or catering chains with more than 10 outlets. This information needs to be readily understandable and available at the point when people decide what to eat (i.e. on menus, menu boards or product description tags). A traffic light labelling scheme covering key nutrients is recommended. National and/or local authorities throughout Europe should work with the catering sector—from major fast-food chains to small local restaurants—to encourage and support them in the provision of healthier choices, to encourage the use of oils high in polyunsaturated or monounsaturated fats rather than hard fats high in saturated fats, introducing smaller serving sizes and providing clear nutrition information. For smaller catering establishments (with fewer than 10 outlets) where a regulatory approach may not be appropriate, incentive approaches (such as award schemes) should be used to encourage them to provide healthier menu items and to provide nutrition information.
Actions in the workplace to improve diet and physical activity It is clear that the places where we work have an important impact on our health and lifestyles. Factors such as what we eat during our time at work, how physically active we are at work, and how we travel to and from work are all relevant. There is, however, relatively little recognition of the importance of workplace health promotion. This is despite that fact that workplace health and safety policies have been a driving force behind some of the major public health gains of recent years—concerns about employee health and safety (and potential litigation), for example, helped to smooth the way for legislation to create smoke-free public spaces. Multi-component programmes promoting healthy eating and/or physical activity in the workplace that have been shown to be effective include: • provision of healthy food and drinks in the workplace; • provision of space for fitness or prompts to encourage people to use the stairs; • involving workers in the planning and implementation of interventions; • involving workers' families; • provision of individual health behaviour change strategies and self-monitoring.[603]	WHO has designated the workplace a key setting for health promotion in the 21st Century.[615] The European Union has recognised the important role of the workplace in influencing the health of workers and their families. In 2002, the Commission published a Communication on a Community strategy on health and safety at work. The nutrition White Paper called on businesses to support the development of healthy lifestyles in the workplace and to work with employee organisations to develop cost-effective ways for companies of different sizes to promote healthy lifestyles.	WHO, the European Commission and national governments should continue to encourage and support employers, trade unions and other employees' organisations to work together to promote and facilitate healthy eating and physical activity in the workplace. **Private and public sector employers** Every workplace should have a healthy work-life balance policy, developed collaboratively with employees and their representatives. This policy should ensure that food served or sold in the workplace is of a high nutritional quality. It should also ensure that employees have access to clear information on the nutritional composition of all food sold or served in the workplace. Employers should introduce measures to encourage employees to incorporate physical activity into their daily life. These could include: • encouraging employees to walk, cycle or use other forms of transport involving physical activity to travel part or all of the way to and from work; • helping employees to be physically active during the day (eg providing prompts to encourage stair use, encouraging short walk breaks etc); • providing information about cycling and walking routes.

Creation of environments that promote active living To increase physical activity levels, communities need access to an infrastructure that encourages and facilitates active living. This includes: • increasing access to outdoor recreational facilities and public open spaces (cost, opening hours, ease of access); • locating schools within walking distance of residential areas; • improving access to public transport; • using planning rules to promote mixed-use development; • enhancing personal safety in areas where people could be physically active. In addition, there are a number of policies that can help to create an infrastructure conducive to walking and cycling as modes of transport. These include: • better facilities for cycling (cycle paths and lanes, bike parking and bike stations, provision of showers at workplaces, bike sharing schemes, bike racks on buses and allowing bikes to be taken on other public transport); • measures to make walking safer and more attractive (well-lit pavements on both sides of every road, refuge islands in the middle of wide streets, clearly-marked well-lit crossings and pedestrian-activated crossings); • measures to reduce volume and speed of traffic in residential areas (traffic calming across residential neighbourhoods, congestion charging, car free zones). Research suggests that while individual measures do work, the best results to increase cycling have been found when communities have implemented an integrated package of different strategies (infrastructure, promotion, planning, restrictions on car use).[616]	The 2007 White Paper stated that the Commission's belief that "*Member states and the EU must take pro-active steps to reverse the decline in physical activity levels in recent decades brought about by numerous factors.*" The Commission supports sustainable urban transport actions and considers walking and cycling projects to be a key component. A Green Paper on urban transport was published in 2007, and an Action Plan on Urban Mobility was issued in September 2009. The Netherlands and Germany (both countries with relatively high cycling rates) have invested heavily in facilities for cycling. Between 1978 and 1996 the already extensive network of bike paths in the Netherlands more than doubled, while the extent of the German bikeway network tripled between 1967 and 1995.[617]	**European Union** The EU should ensure that European development or structural funding is conditional on projects being able to enhance the infrastructure for promoting active living. **National and local governments** Authorities at the national and local levels should implement a wide range of measures to encourage and facilitate walking, cycling and other forms of physical activity as part of people's daily routines. These can include: • development of an integrated transport strategy that emphasises walking and cycling; • improving provision of local parks and green places; • provision of maps and guides on good places to walk and cycle; • provision of clear signs to enable people to measure the distance they walk/cycle; • improving access to sports and leisure facilities for all sectors of the community (eg free crèches, discounted access, late night sessions etc); • ensuring stairs are prominent, accessible and well-lit in new buildings; • encouraging town planners to provide facilities that can be walked to and around, and to promote development of areas which have a mixture of residential, commercial and public services; • taking action to enhance personal safety in areas where people could be physically active.
Health service involvement in promoting healthy lifestyles The health sector is another setting which can have an influence on what we eat and how active we are. In some cases this impact is direct—the food served to inpatients in hospitals and other people in care (see section on public institutions). In other cases the influence is less direct—where health professionals give advice and support to encourage healthy lifestyles. There is considerable scope to improve the role that health services play in promoting healthy dietary and physical activity patterns.	The WHO European Action Plan proposed actions to engage primary health care staff in nutrition assessment and the provision of counselling on diet, food safety and physical activity. The EU White Paper proposed that the professional bodies of health professionals act to strengthen the training of health professionals in relation to nutrition and physical activity related risk factors for ill health, with particular emphasis on a preventive approach. In Finland, for example, the Neuvokas Perhe (Smart Family) programme aims to strengthen family-based lifestyle guidance by providing health professionals in maternity and child welfare clinics with tools, knowledge and support to be able to promote good dietary and physical activity habits.	**European Union** The European Commission should take steps to improve and enhance the role of health services in prevention and early detection of diseases. This should include a review of how effective health services across the EU are in promoting prevention and in early detection of diseases. The inclusion of healthy lifestyle promotion in the curricula of health professionals across the EU should be reviewed. Developing the training and further education opportunities for health professionals is an important aspect of capacity building in central and eastern European countries, where such opportunities are sparse currently.

2.5 ENSURING A HEALTHY, SUSTAINABLE FOOD SUPPLY

2.5.1 The relationship between CVD prevention and measures to protect the environment

It is no longer possible to consider food, nutrition and physical activity in isolation of today's major social and environmental challenges. As we advocate policies to bring about changes in diet and physical activity levels, we must ensure that these policies do not undermine efforts to protect the environment and the planet's scarce resources. Rather, policy makers must make the most of any synergies that could be created by tackling the health, environmental and food security agendas and they need to be aware of any tensions that may arise from these aims.

There is a growing awareness that human beings are having a major impact on the physical environment and that this, in turn, is already having a major impact on human health and well being including rates of CVD in some populations. Measures taken to improve public health, including those aimed at preventing CVD, may have a beneficial effect on the environment but there may also be some areas of conflict—such as when public health advice is to encourage fish consumption, while the world is faced with declining fish stocks.

This section first outlines some of the major changes to the environment at this time. Some of these changes can be attributed to how we consume food and to the way we move around the planet and so are connected with the current and recommended diets and levels of physical activity that are discussed in the rest of this position paper.

Secondly, it looks as some of the effects of changes to the environment on health and in particular CVD.

Thirdly, it looks at how measures to protect the environment may have an impact on population-based interventions to promote human health and vice versa.

2.5.2 Major changes to the environment at this time

There are well known and growing concerns about anthropogenic (human-generated) climate change. The reports of the Intergovernmental Panel on Climate Change (IPCC)[618] demonstrate unequivocally that the climate is changing and most of the change is caused by human activity. The latest (2007) report concludes that "most of the observed increase in globally averaged temperatures since the mid-20th century is very likely due to the observed increase in anthropogenic greenhouse gas concentrations." These "green house gases" (GHGs) include, among others, carbon dioxide, methane and nitrous oxide.

The food system is a major contributor to GHGs. For impacts associated with the whole of the supply chain—from agriculture through to consumption—it has been estimated that the food sector in its entirety accounts for around 30% of GHG emissions.[619-621] For the European Union, it has been estimated that agriculture alone contributes 9% of GHG emissions.[622] Livestock for meat and dairy production are the most important source of GHG emissions associated with food production—being responsible for an estimated 18% of global CO^2 emissions.[621]

But climate change is not the only human-generated environmental problem: there is also the accelerating depletion of environmental resources necessary for human existence. There are serious pressures on the availability of land and water for food production. Over 70% of the world's water consumption is used in agriculture and the rising demand for food to feed the world's growing population contributes to the pressures on fresh water supplies. Changing dietary patterns are also important—meat production requires 8–10 times more water than cereal production.[623] Land resources are also under stress leading to soil erosion, loss of fertility, desertification and salinisation which can, in turn, lead to food shortages.

Modern intensive farming is also currently highly dependent on fossil fuels. These are used in a range of agricultural practices from running tractors to making artificial fertilisers—which is one of the reasons why agriculture contributes significantly to GHG emissions. Food manufacture, packaging, storage and transport also depend largely on fossil fuels.

The ever increasing use of fossil fuels means that consumption from ever diminishing stocks threatens to outstrip production. Even some minerals necessary for human food production, such as phosphorous, are in danger of running out.

The over-exploitation of natural resources extends to that of wild animals and plants that have traditionally been sources of food, such as fish. The accelerating depletion of natural resources also leads to problems with the decline in biodiversity—the dramatic loss of plant varieties, for example, leaves us dependent on a narrow range of varieties and, as a result, more vulnerable to pests and disease.

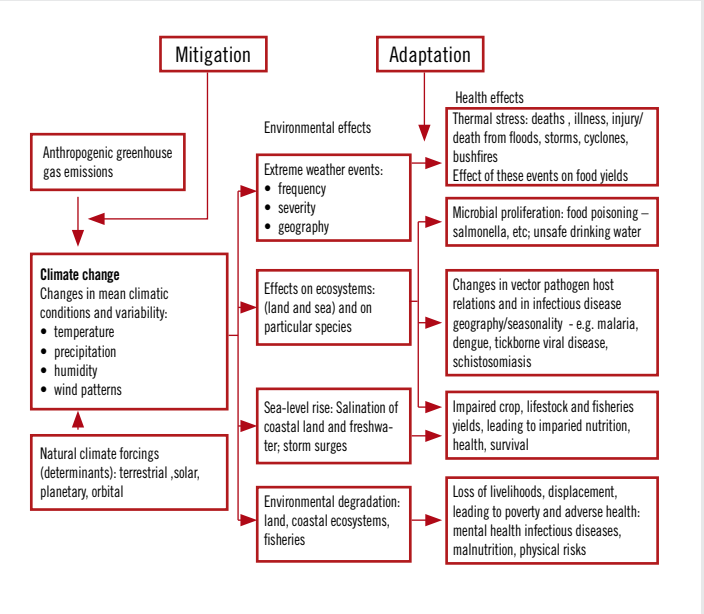
2.5.3 Effects of environmental change on human health

These changes to the environment present a real threat to health. Climate change is already having an effect on human health—a relatively conservative assessment published by WHO is 2003, for example, estimated that climate change had been responsible for the loss of 5.5 million disability adjusted life years (DALYs) in the year 2000.[624]

These environmental changes are likely to impact on health to an ever increasing degree if current trends

in the use of natural resources continue. The potential mechanisms through which climate change may impact on those different aspects of health are illustrated in Figure 44.

FIGURE 44 PATHWAYS FOR THE IMPACT OF CLIMATE CHANGE ON HEALTH



Note: Mitigation refers to true primary prevention (reducing greenhouse gas emissions). Adaptation (a form of late primary prevention) entails interventions to lessen adverse health effects.
Source: McMichael et al, Lancet[625]

Although in some areas climate change is predicted to bring some health benefits (due to changes in malaria transmission or to fewer deaths from exposure to cold), the IPCC projects that the negative impact will outweigh any benefits, especially in developing countries.

While most of the current effects of environmental change are having an impact on the health of people in low and middle income countries (only 8,000 of the 5.5 million DALYs lost in 2000 were in developed countries) no country will be immune. And it would be a mistake to think that chronic diseases are not affected. Ambient temperature rises can directly affect health by, for example, precipitating heart attacks.[626]

In Europe the heatwave of 2003 caused up to 70,000 deaths, mainly from respiratory and cardiovascular causes[627] and one study estimates that half of these excess deaths are due to anthropogenic climate warming.[625]

2.5.4 SYNERGIES AND TENSIONS BETWEEN MEASURES AIMED AT PROMOTING HEALTH AND AT PROTECTING THE ENVIRONMENT

It is generally thought that measures to protect the environment will be synergistic with measures to promote human health but this may not always be the case.

There is, as noted elsewhere in this position paper, considerable scientific consensus on what constitutes a healthy diet and optimal levels of physical activity. In this position paper we propose goals for diet and physical activity that would promote public health in European populations based on that consensus (see section 1.3.11).

There has also been some attempt on the part of national and international agencies to come to an agreement about what constitutes a 'sustainable' diet and level of physical activity.

2.5.4.1 Synergies and tensions in promoting a healthy, sustainable diet

A sustainable diet can be defined as one that promotes health, environmental sustainability, economic sustainability and social equalities (e.g. in income and education). But this begs the question of what such a diet would precisely look like in terms of foods and nutrients. The question is not merely academic. A greater understanding of what constitutes a sustainable diet has implications for, and might affect, the policy recommendations outlined in Section 2.4 of this position paper, such as those relating to the labelling and marketing of foods.

Those interested in defining a sustainable diet have generally focused on the environmental impacts of current and future consumption patterns, particularly in relation to GHG emissions, resource depletion, land use change and species diversity, whilst recognising that sustainability has other dimensions.

Some national government bodies in Europe have already issued or prepared official guidance on a sustainable

diets—notably the Swedish National Food Administration in 2009,[628] the German Council for Sustainable Development in 2010[629] and the UK Sustainable Development Commission in 2011.[630]

The Swedish National Food Administration—in its guidance—divided its recommendations by food group: meat; fish; fruits and vegetables; cereals, rice and potatoes; cooking fats and water. The guidance points out, for example, that "meat—beef, lamb, pork and chicken—is the foodstuff with the greatest impact on the environment" and "that from a health perspective, there is also no reason to eat as much meat as we do today" and so, "by all means, eat meat, but reduce the amount." It also suggests choosing fish and shellfish from stable stocks, seasonal and locally grown fruit and vegetables, etc.

The German Council for Sustainable Development offer advice for a wide range of day-to-day consumer decisions including food shopping. It argues that for optimal health, social, economic and environmental benefits, "your shopping basket should contain above all:

- Healthy food products;
- Organic products;
- Seasonal fruit and vegetables grown locally;
- Less meat and fish;
- Fair-trade products and
- Beverages in recyclable packaging units."

In the UK the Sustainable Development Commission (SDC) (an independent body that advises the Government) has also produced a series of recommendations for attaining a sustainable diet.[630] These are not dissimilar from those produced by the German Council for Sustainable Development and the Swedish National Food Administration but in contrast to these two other government bodies sought to develop these recommendations in a systematic way. To do this the SDC first set out a series of framework recommendations—drawn from previous sources—and then assessed these framework recommendations for their impact on four aspects of sustainability: health, environmental sustainability, economic sustainability and social equalities using the available literature. The table below shows the results of that analysis:

Table 32 Summary of impacts of framework guidelines for sustainable and healthy diets from the UK Sustainable Development Commission

	Framework guideline	Public health	Environmental sustainability	Economic sustainability	Social inequalities
1	Consume less food and drink	+	+	–	+
2	Accept different notions of quality	o	+	–	o
3	Accept variability of supply	±	±	o	±
4	Shop on foot or over the internet	o	+	o	o
5	Cook and store food in energy conserving ways	o	+	o	o
6	Prepare food for more than one person and for several days	o	+	o	o
7	Reduce food waste	+	+	–	o
8	Reduce consumption of meat and dairy products	±	±	–	+
9	Reduce consumption of food and drinks with low nutritional value	+	+	–	+
10	Reduce consumption of bottled water	o	+	o	o
11	Increase consumption of organic food	o	±	+	–
12	Eat seasonal, field grown fruit and vegetables	–	+	–	o
13	Eat fish from sustainable stocks	–	±	o	+

+ some evidence of positive impacts
– some evidence of negative impacts
± some evidence of both positive and negative impacts
o no evidence of impacts

Source: http://www.bis.gov.uk/assets/bispartners/foresight/docs/food-and-farming/synthesis/11-628-c8-changing-consumption-patterns.pdf

This table raises a series of questions relating to the synergies and tensions between recommendations made about food for various reasons. For instance the table shows that the evidence indications that eating merely "seasonable, field grown fruit and vegetables" or just "fish from sustainable stocks" would likely have a negative effect on health. The reason for this is that if consumers just followed these recommendations they would eat less fruit, vegetables and fish than they currently do leading for example to an increased risk of CVD. Similarly the Commission considers that there is (on balance) no evidence of an impact on health from eating organic food yet there is some evidence that a greater consumption of organic food would be good for the environment.

On the basis of this analysis the SDCs final recommendations were that:

Highest priority changes, with a significant and immediate impact on "sustainable diets", and where health and different facets of sustainability complement each other, are:

- reducing consumption of meat and dairy products;
- reducing consumption of food and drink of low nutritional value, i.e. fatty and sugary foods (and tea, coffee and alcohol);
- reducing food waste.

Medium priority changes, which would have a significant positive effect on sustainability but where gains in one area might have negative impacts in other areas, are:

- increasing consumption of fruit and vegetables, particularly seasonal and field grown;
- consuming fish from sustainable stocks only;
- increasing consumption of foods produced with respect for wildlife and the environment e.g. organic food.

Lower priority changes, which would make a smaller impact on sustainability, but with largely complementary effects across health and the different facets of the sustainability, are:

- reducing energy input by shopping on foot or over the internet, and cooking and storing food in energy-conserving ways;
- drinking tap water instead of bottled water.

These might be compared with the summary table of population goals (Section 1.3.11) in this paper. It might be noted that there are various similarities and differences. Firstly, in that the goals in this paper are quantified while the SDC's are not, and that the former are largely for nutrients and food components with some foods (e.g. fruit and vegetables) whilst the SDC's are for foods and types of foods based on their methods of production.

On the other hand there are similarities. The goals in this position paper translate into a shift towards a plant-based diet, with higher intakes of vegetables, fruits and whole grains and lower meat and dairy intakes. This shift is in line with at least two of the priorities outlined above (reducing consumption of meat and dairy products; increasing fruit and vegetable consumption).

This means that there is likely to be considerable scope for policy synergy. For example, the inclusion of agricultural emissions in New Zealand's "emissions trading scheme" could have a positive impact on both livestock emissions and saturated fat consumption.[631] Similarly, a shift in demand towards a more plant-based diet could also be beneficial in terms of food security and resource use since the widespread use of cereals as animal feeds—including to meet the fast growing demand for meat and dairy products among the expanding middle classes of middle-income countries—has contributed to recent rises in commodity prices and, thus, to widespread food insecurity.

2.5.4.2 Synergies and tensions in promoting physically active lives

Many of the proposals outlined in section 2.4 of this paper relate to policies to encourage and facilitate creation of an environment that promotes active living. This includes the promotion of active travel, by policies that encourage the integration of walking and cycling into daily routines. Given that environmental policies seek to reduce transport-related greenhouse gas emissions (transport accounted for almost a quarter of emissions from global energy use in 2004), there are potential synergies.

A reduction in motor vehicle use, for example, could reduce a number of health risk factors—namely, urban air pollution, physical inactivity and road traffic danger. Woodstock and colleagues examined the health impacts of different urban transport scenarios in London and Delhi, concluding that a combination of active travel and lower-emission vehicles would bring the greatest health benefits.[632] This scenario would reduce the number of DALYs lost by 7,439 in London, with a reduction in years lost to ischaemic heart disease of 10-19%. By 2030 these same scenarios would result in a 60% reduction in transport-related CO^2 emissions since 1990 in London (compared to a 4% increase according to the business-as-usual scenario).

Their analysis highlighted, however, the importance of careful implementation of such policies. Although the reduction in cars on the roads would reduce the risk of road traffic accidents, as more people walk and cycle, and for longer distances, there would be more exposure to the remaining road traffic risk. It is vital, therefore, that policies focus on creating safe urban environments for active travel. This can include enforcement of speed limits, controls for heavy goods vehicles and cycle routes which give cyclists priority at junctions. Urban planning policies are also important—mixed-use developments which enable homes, schools, shops and workplaces to be situated close to one another reduce the distances people need to travel to school, work, shops or other services.

2.5.5 Conclusions

The diet and physical activity population goals proposed in this paper are formulated from the perspective of preventing cardiovascular diseases but we have sought to take some note of environmental perspectives e.g. in setting the goals for n-3 fatty acids and making recommendations about fish.

EHN is mindful that we can no longer consider diet and physical activity in isolation of the major environmental challenges facing our planet. That is why this section highlights new initiatives aimed at developing recommendations for a diet that is both healthy and sustainable.

There is a growing evidence base to support diet and physical activity population goals and policy approaches that would promote environmental sustainability. We anticipate that future editions of this report will need to take more note of this evidence base.[727]

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and stroke
european heart network

www.ehnheart.org
European Heart Network
Rue Montoyer 31
B – 1000 Brussels
Belgium
Telephone: +32 2 512 91 74
Fax : +32 2 503 35 25
Email: info@ehnheart.org