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Transforming European food and drink policies for cardiovascular health

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European Heart Network

The European Heart Network (EHN) is a Brussels-based alliance of heart foundations and likeminded non-governmental organisations throughout Europe, with member organisations in 25 countries. EHN plays a leading role in the prevention and reduction of cardiovascular diseases, in particular heart disease and stroke, through advocacy, networking, capacity-building and patient support, so that they are no longer a major cause of premature death and disability throughout Europe.

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Executive Summary

Despite considerable progress in tackling cardiovascular disease (CVD), it remains the leading cause of death and a major cause of illness and disability for men and women in Europe. Dietary risks are responsible for around half of the death and disability caused by CVD at an estimated cost of €102 billion in the European Union (EU) alone (Chapter 1).

Since the European Heart Network (EHN) published its last paper on *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe* in 2011, there have been many major developments in both the scientific arena and the policy landscape.

A review of the recent scientific developments and an in-depth look at some of the recent media reports of 'controversies' on diet and health show that, generally speaking, the evidence on the links between diet and CVD has strengthened, rather than weakened, in the last few years (Chapter 2). This paper presents a set of population goals, revised to take the new evidence into account (Chapter 2.1); the goals provide clear pointers towards a cardiovascular health-promoting diet for Europe.

Taken together, these population goals should translate to a cardiovascular health-promoting diet that has a low energy density;[i] this is important for weight maintenance, and for the prevention of overweight and obesity. A cardiovascular health-promoting diet means a shift from an animal-based diet to a more plant-based diet. It includes vegetables, fruit and berries in abundance. Whole grain products, nuts and seeds, fish, pulses, low-fat dairy products are also important, as are non-tropical vegetable oils in modest amounts. This dietary pattern limits consumption of red meat, processed meat products and foods or drinks which are low in vitamins, minerals and dietary fibre and/or high in free sugars, saturated/trans fats or salt. A diverse and balanced diet covers the need for nutrients, and food supplements are rarely needed. It now falls to governments to translate these population goals into clear guidance about foods, taking into account the typical diet in the country.

In a perfect world people would buy and eat different foods to reflect this evidence and advice, and markets would respond to the changes in demand (Chapter 3). In today's complex food systems, however, the 'market' does not function perfectly and there are many other forces – often powerful – driving the food supply in addition to consumer demand. Major economic and policy drivers determine what food is produced, what is imported and how foods are marketed. Many of these global and external factors are well beyond the reach of individual governments, posing real challenges for policymakers. The complex picture also means, however, that there are many different points along the food chain where policymakers can take action to improve diets.

In the six years since EHN's last paper was published, there is more recognition of how important it is for governments to take action to improve the food supply and food environments. While some countries have adopted binding or voluntary measures, a great deal more progress is urgently needed to implement effective food and drink policies for preventing diet-related CVD (Chapter 4).

To that end, three overarching recommendations and three clusters of specific recommendations are proposed (see figure on following page).

EHN calls for rapid and full implementation of these recommendations in order to realise the vision for every European – irrespective of the place or socio-economic circumstances into which they are born – to be able to live free from avoidable diet-related CVD, and thus be able to have a productive working life and many years of active retirement free from cardiovascular ill-health or disability.

i Energy density is the amount of energy (calories) per gram of food.

EHN recommendations for food and drink policies for cardiovascular health

Implement policies to tackle cardiovascular health inequalities in Europe

Ensure that robust mechanisms for nutrition governance are in place and fit-for-purpose

Develop an integrated health and environment approach to food systems and promote health-environment win-wins in food-based dietary guidelines

Food supply-side recommendations

- Establish a global food convention
- Reform agricultural and food policy to align with public health priorities
- Ensure trade and investment policies protect and promote public health

Food demand-side recommendations

- Use taxes and/or subsidies
- Implement regulatory controls on marketing of unhealthy foods
- Adopt a nutrient profile for regulation of claims, mandatory simplified front-of-pack nutrition labelling and menu labelling

Food composition recommendations

- Set legal limits for levels of industrially-produced trans fats
- Establish nutrition standards for food in schools, hospitals and other public institutions
- Implement wide-reaching ambitious food reformulation programmes

Introduction

Europeans are living longer than ever before and there has been dramatic progress in tackling premature deaths from heart disease and stroke in recent decades. Cardiovascular disease (CVD), however, remains a leading cause of death and ill-health in Europe – accounting for nearly half (45%) of all deaths in the European region.[1i] Many of these deaths deprive people of their retirement years resulting in suffering and loneliness for their partners and families. Yet, the true burden of CVD is much greater, frequently starting before the age of retirement. Heart disease and stroke are leading causes of illness and disability, responsible for 64.7 million years lost to death or disability in Europe every year, nearly a quarter (23%) of the total. Uneven progress in tackling CVD is also a major contributor to the health gaps – both between and within countries – that separate Europe's poorer and wealthier populations and result in marked geographical differences. In addition to the human loss and suffering, this burden brings potentially devastating social and economic costs in terms of increased healthcare needs, lost productivity and substantial extra social care. Furthermore, as average healthy life expectancies – how long people can expect to live without any disability – have not kept pace with rising retirement ages in many countries, there are enormous social and economic challenges ahead.

Unhealthy diets, overweight and obesity are major contributors to heart disease and stroke. Dietary risks are responsible for half of the deaths and disability caused by CVD across Europe. In the European Union alone, diet-related death and disability costs an estimated €102 billion every year.

The good news is that much of cardiovascular disease can be prevented and dietary risk factors are avoidable – the case for investing in prevention is compelling. Policies and actions to reduce exposure to dietary risk factors can, and do, work. For example, where mandatory upper limits on industrially-produced trans fatty acids have been introduced, they have been shown to greatly reduce exposure to these harmful fats, sometimes with evidence of a fall in hospitalisation for cardiovascular events. Government-led salt reduction campaigns in several countries have resulted in meaningful reduction of salt levels in foods leading to demonstrably lower average sodium intakes in some cases. Such interventions have lead to real, measurable improvements in cardiovascular health.

In 2011, the European Heart Network (EHN)'s paper *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe* proposed a series of population goals for diet and physical activity, along with a raft of recommendations for policy action. In the six years since the paper was published, some countries have acted on a number of these recommendations, by adopting binding or voluntary measures to help their citizens eat more healthily. Recognition of the need for governments to play an important role in improving the food supply and food environments – as well as providing consumers with information and education – continues to grow. There appears to be growing political and public acceptability of government interventions such as taxes on sugary drinks or restrictions on marketing of unhealthy foods to children. In addition, the urgency of tackling non-communicable diseases (NCDs) and improving diets has achieved greater formal recognition, both globally and regionally.

Nonetheless, much more progress is needed. If current trends continue, many European countries will miss agreed global targets to halt the rise in obesity or diabetes, reduce salt intakes, increase breastfeeding or reduce physical inactivity by 2025. One in three 11 year olds in the European region is overweight.[ii] Breastfeeding rates in the region are the lowest in the world. Ten years on from the introduction of the first mandatory upper limits on industrially produced trans fats in Denmark – and despite evidence that mandatory limits are more effective than a voluntary approach – only a handful of European countries have introduced mandatory limits and the European Commission is only now considering an EU-wide legal limit. More than a quarter of a century since health advocates first called for clear front-of-pack colour-coded nutrition labelling, most people in Europe still have to both decipher and calculate/interpret nutrient panels on the back of food packaging if they want any meaningful nutritional information about foods they buy (and we know that only a small proportion of consumers actually try now to do so given the complexity of the information). Despite the progress in some countries with product reformulation to reduce salt levels, it is disappointing that some voluntary schemes set under-ambitious targets and do not cover a wide enough range of products and that there has not been enough progress or innovation on reducing total fat, saturated fat and sugar levels in foods. Despite widespread implementation of restrictions, mainly voluntary, on advertising of foods high in fats, sugar or salt during children's television programming, millions of European children continue to be exposed to intense advertising during family viewing and, increasingly, through new forms of marketing online and through social media. While many countries have taken action to improve food provided in schools and preschool institutions, too many children are still able to access unhealthy meals and snacks at school. Similarly, it remains common for hospitals and other government-supported institutions to continue to serve or sell foods, confectionery and soft drinks that can contribute to ill health.

i This report covers the European region, as defined by the World Health Organization. References to the European region, therefore, refer to the 53 countries in the wider European region. Where possible, figures are given for both the European region and the European Union.

ii http://www.euro.who.int/en/health-topics/noncommunicable-diseases/obesity/data-and-statistics

It is clear, therefore, that member states and European institutions continue to face considerable challenges in implementing effective policies for preventing diet-related CVD. Moreover, governments and institutions throughout the region are being simultaneously challenged by broader issues, such as environmental sustainability and how to ensure public health is protected when trade agreements are in place.

EHN's vision states that every European has a right to a life free from avoidable cardiovascular disease. This recognises that all Europeans should be able to have a productive working life *and* many years of healthy, active retirement. The specific vision for this paper is as follows:

Every European – irrespective of the place or socio-economic circumstances into which they are born – has a right to a life free from avoidable diet-related cardiovascular disease.

The mission of this paper, therefore, is to:

- Define a cardiovascular health promoting diet, in the current European context, and set out specific population goals to help achieve that diet;
- Identify key areas for policy action to enable and support adoption of a cardiovascular health-promoting diet and, ultimately, achieve the vision outlined above.

More specifically, the objectives of the paper are to:

1. Describe and explain the burden of diet-related CVD – including its social, economic and societal impact – and the ways in which this burden is changing.
2. Analyse developments in science since the publication of EHN's 2011 paper, including reviewing new and emerging evidence on nutrients, foods and drinks, addressing issues that have attracted much media attention and identifying desirable healthy dietary patterns.
3. Describe the complex food systems that shape diets across Europe and explore in more depth some of the specific drivers of these systems.
4. Describe the current policy landscape and examine uptake of the nutrition-related policy recommendations made in EHN's 2011 paper and other progress in this area.
5. Identify a package of key evidence-based effective policies for promoting sustainable food systems and achieving the vision of every European – irrespective of the place or socio-economic circumstances into which they are born – having a right to a life free from avoidable diet-related CVD.

The recommendations in this paper cover effective policies to be implemented at the international, EU, national and local community levels.

Unlike the 2011 paper, this paper does not cover the science and policy options relating to **physical activity**. This is a policy area that warrants scientific scrutiny and careful consideration of effective policy actions in its own right and EHN will consider it separately together with its Physical Activity Expert Group. It is important to stress, however, that there are numerous interactions between physical activity and nutrition. The population goals that are proposed are defined to apply to current European populations, and these predominantly have low levels of physical activity. All efforts to reduce energy intake and the energy density of diets need to be accompanied by efforts to increase energy expenditure by wide-ranging policies that enable, support and promote physical activity.

While the paper makes a comment on **alcohol** consumption in relation to cardiovascular health, it is beyond the scope of this paper to review policy options for reducing excess alcohol consumption.

1 Diet and cardiovascular disease – why action is needed

Key points

- Despite progress in tackling cardiovascular disease (CVD), it remains the leading cause of death for men and women in Europe[i] and a leading cause of illness and disability.
- CVD accounts for 45% of all deaths, equivalent to 3.9 million deaths each year in the European region and 1.8 million deaths annually (37% of all deaths) in the European Union (EU).
- As Europeans are living longer, more people are living with CVD despite falling CVD death rates. In 2015, more than 85 million people in Europe were living with CVD and almost 49 million people were living with CVD in the EU.
- Over the past 25 years, the absolute number of CVD cases has increased in Europe and in the EU, with increases in the number of new CVD cases evident in most countries.
- CVD is responsible for the loss of 64.7 million years to death or disability in Europe, equivalent to 23% of all disability-adjusted life years (DALYs) lost across the region. Within the EU, 26 million DALYs were lost as a result of CVD, around 19% of the total DALYs lost.
- In addition to the human loss and suffering, this burden brings devastating social and economic costs – costing the EU economy €210 billion a year in healthcare, lost productivity and informal care costs.
- In many European countries average healthy life expectancy – how long people can expect to live without any disability – is lower than the statutory retirement age, posing enormous social and economic challenges and depriving people of healthy, active retirement years.
- The burden of death and disability due to CVD is very unevenly spread, both between and within countries in the European region, with a higher burden typically found in Central and Eastern European countries and strong socio-economic gradients within countries.
- Dietary risks are responsible for 56% of all the DALYs lost to CVD in Europe. These risks are also responsible for 49% of the DALYs lost to CVD in the EU, at an estimated annual cost of €102 billion.

1.1 Leading cause of death in Europe

Despite dramatic reductions in rates of premature death related to cardiovascular disease (CVD) in Europe, it remains the leading cause of death for men and women in the region.[i] CVD accounts for 45% of all deaths, causing 3.9 million deaths each year (Figure 1 and Figure 2). It is also the main cause of death within the European Union (EU) – where it is responsible for 37% of all deaths, equivalent to 1.8 million deaths each year (Figure 3 and Figure 4). In absolute numbers, more women die from CVD than men. This is due to the fact that the population of women is greater than the population of men. However, in all European countries for which data are available, age-standardised mortality rates[ii] are higher in males than in females.

CVD is the main cause of death in women in all but two European countries and is the main cause of death in men in all but 12 countries. The two most common forms of CVD – coronary heart disease[iii] and stroke – are, respectively, the most common and second most common causes of death in both the European region and the EU.

i This report covers the European region, as defined by the World Health Organization. References to 'Europe' or 'the European region', therefore, refer to the 53 countries in the wider European region. Where possible, figures are given for both the European region and the European Union.

ii Age-standardisation adjusts crude mortality rates to remove the influence of different population age structures, and hence allows more meaningful comparisons to be made between countries and over time.

iii In this paper we use coronary heart disease (CHD) and ischaemic heart disease interchangeably.

Figure 1 Leading causes of death among men in the European region Source: European Cardiovascular Statistics, 2017[1]

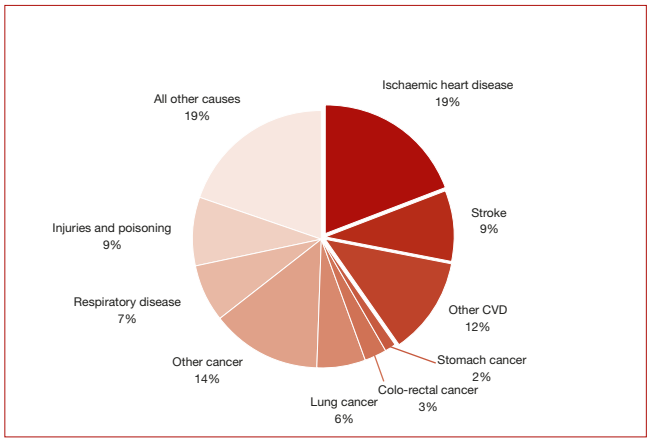


Figure 2 Leading causes of death among women in the European region Source: European Cardiovascular Statistics, 2017[1]

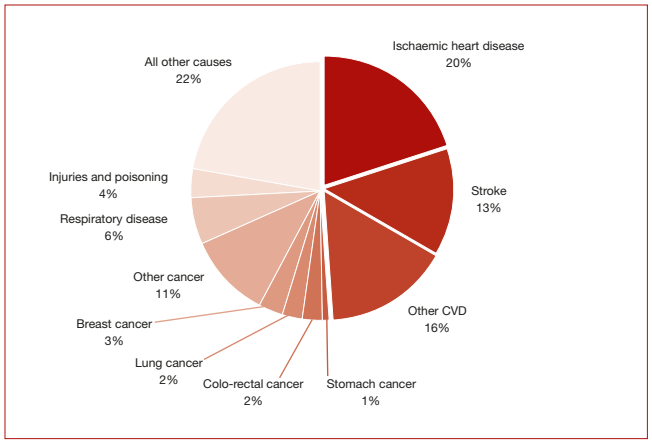


Figure 3 Leading causes of death among men in the European Union Source: European Cardiovascular Statistics, 2017[1]

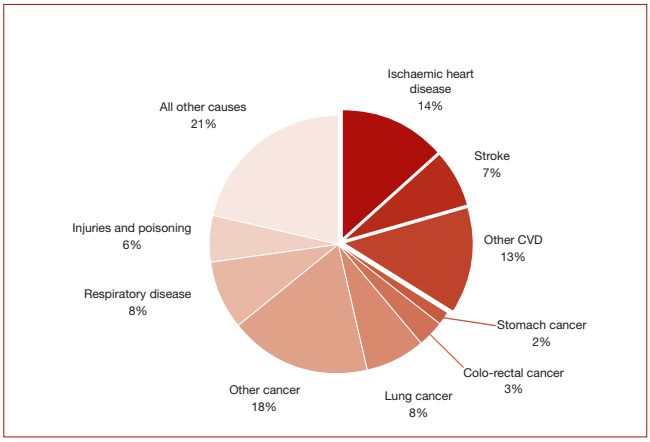
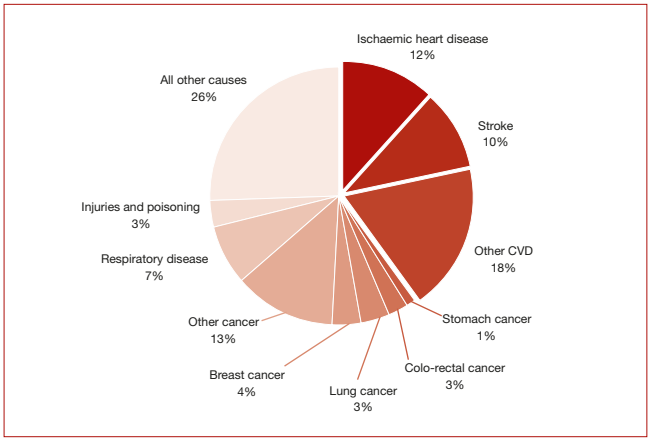


Figure 4 Leading causes of death among women in the European Union Source: European Cardiovascular Statistics, 2017[1]



1.1.1 A major cause of premature death

It is not uncommon for the extraordinary burden of CVD mortality to be dismissed as an inevitable side-effect of the fact that Europeans now live longer than ever before. Contrary to this 'if you live long enough, something is going to get you' argument – and despite great progress in extending average life expectancy – CVD is also a leading cause of premature death.

It is a harsh reality that many cardiovascular deaths occur in people who have yet to reach, or have recently reached, retirement age. For men, CVD is the main cause of death before the age of 65 in Europe, responsible for 31% of deaths. In women under 65, CVD is the second largest single cause of mortality, accounting for 26% of all deaths (Figure 5 and Figure 6). In the EU, CVD is the second largest cause of death before the age of 65, responsible for 24% and 16% of deaths in men and women of this age respectively (Figure 7 and Figure 8).

According to OECD figures, CVD accounts for between 12% and 25% of all potential years of life lost in men before the age of 70 in European countries.[2]

Figure 5 Deaths under 65 by cause, men, latest available year, European region Source: European Cardiovascular Statistics, 2017[1]

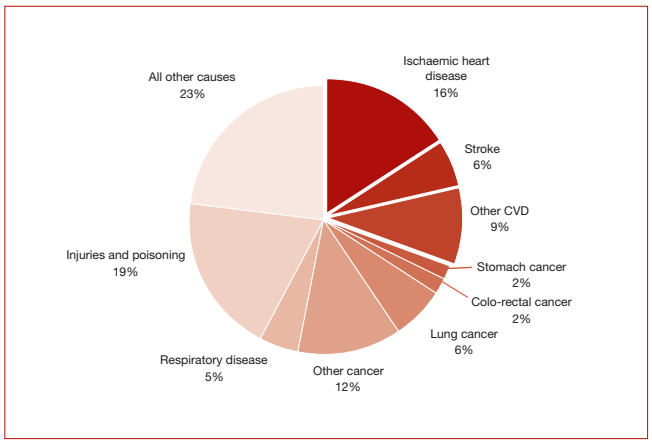


Figure 6 Deaths under 65 by cause, women, latest available year, European region Source: European Cardiovascular Statistics, 2017[1]

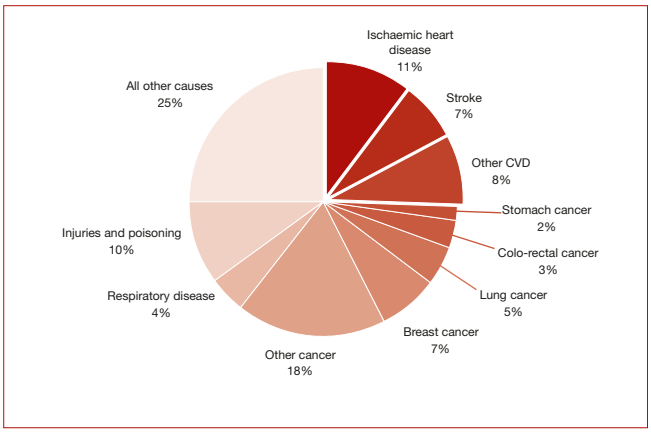


Figure 7 Deaths under 65 by cause, men, latest available year, European Union Source: European Cardiovascular Statistics, 2017[1]

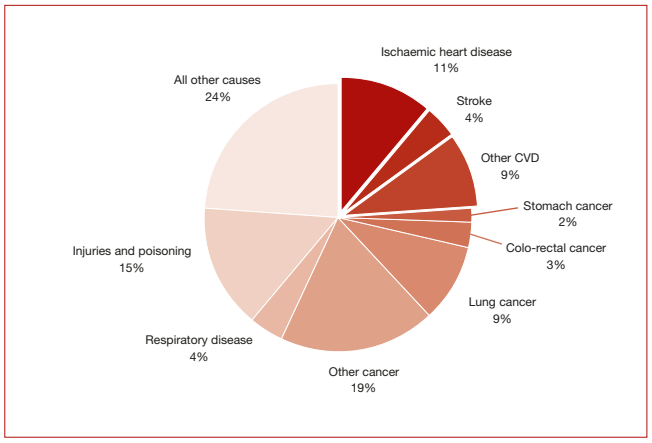
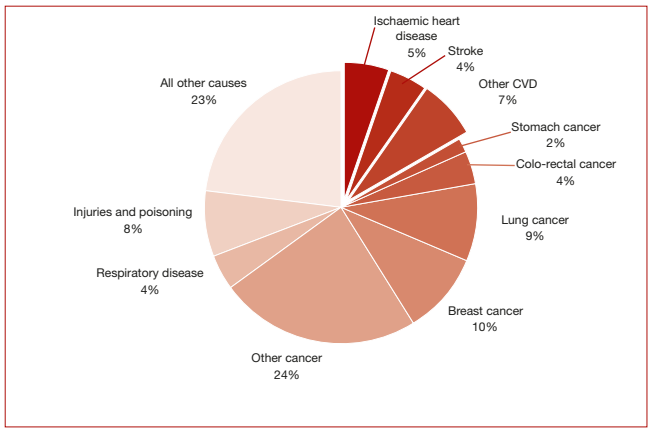


Figure 8 Deaths under 65 by cause, women, latest available year, European Union Source: European Cardiovascular Statistics, 2017[1]

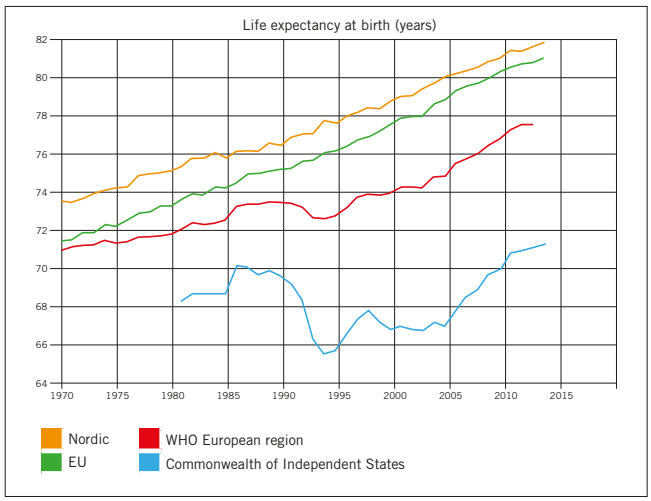


1.1.2 Inequalities across the European region

There continue to be stark inequalities in health across Europe, with a difference of at least 11 years between the lowest life expectancy and the highest national life expectancy across the region.[3]

The situation in Central and Eastern Europe is particularly worrying. There is, for example, a dramatic enduring and continuing gap in life expectancy between, for example, the Nordic countries and the Commonwealth of Independent States (Figure 9).[3] Since 1960, life expectancy gains in the World Bank's Europe and Central Asia region – which covers the central and eastern countries in the European region – have been the lowest in the world, and current life expectancies in the region are similar to those in Western European countries (EU-15) during the 1960s.[4] The main cause of this life expectancy gap is CVD.[4]

Figure 9 Life expectancy at birth in the European region, 1970–2014 Source: WHO/Europe, European HFA Database, July 2016



Comparing the CVD mortality burden across individual European countries reveals substantial variation, with a higher burden typically found in Central and Eastern European countries compared to that in Northern, Southern and Western countries. This is evident across both EU and non-EU member states. Within the EU, the proportion of all deaths due to CVD ranges from 23% in France to 60% in Bulgaria among men, while in women, the burden ranges from 25% in Denmark to 70% in Bulgaria. Outside the EU, the CVD mortality burden varies from 24% in Israel to 59% in Ukraine among men, and from 25% in Israel to 75% in Ukraine among women.

Strong geographical disparities are apparent, with relatively high rates observed in Eastern and Central Europe (particularly post-Soviet states) and lower rates in Northern, Western and Southern Europe. For example, for both sexes in the EU, the age-standardised death rate for coronary heart disease (CHD) in the latest available year is lowest in France (77 deaths per 100 000 in males; 32 deaths per 100 000 in females) and highest in Lithuania (700 deaths per 100 000 in males; 429 deaths per 100 000 in females). Outside the EU, the lowest death rates are found in Israel (115 deaths per 100 000 in males; 67 deaths per 100 000 in females) whilst the highest rates are found in Ukraine (1 102 deaths per 100 000 in males; 429 deaths per 100 000 in females) (Figure 10).

Death rates for stroke are higher in Eastern and Central regions than in Northern, Southern and Western regions (Tables 1 and 2). For example, among EU countries, they range from 53 per 100 000 in France and Luxembourg to 353 per 100 000 in Romania in males and from 42 per 100 000 in France to 281 per 100 000 in Bulgaria in females. Outside the EU, the lowest death rates from stroke are found in Switzerland (51 deaths per 100 000 in males; 47 deaths per 100 000 in females) while the highest rates are found in TFYR Macedonia (383 deaths per 100 000 in males; 345 deaths per 100 000 in females) (Figure 11).

Figure 10 Age-standardised death rates from CHD, men, latest available year, European region Source: European Cardiovascular Statistics, 2017[1]

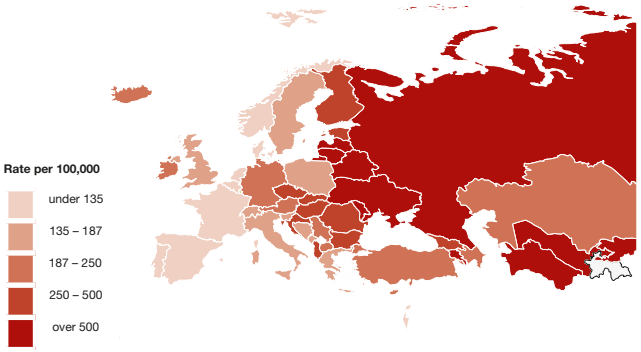


Figure 11 Age-standardised death rates from stroke women, latest available year, European region Source: European Cardiovascular Statistics, 2017[1]

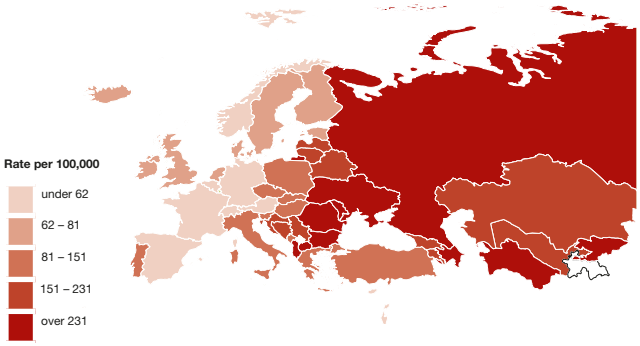


Table 1 Age-standardised death rates from CHD, all ages, by sex, latest available year, European region

Males		Females	
	Deaths per 100 000		Deaths per 100 000
San Marino	35	France	32
France	77	San Marino	32
Netherlands	90	Spain	45
Portugal	98	Netherlands	45
Spain	102	Luxembourg	45
Belgium	113	Portugal	49
Luxembourg	115	Belgium	51
Israel	115	Cyprus	64
Denmark	124	Israel	67
Norway	126	Denmark	67
Montenegro	138	Greece	68
Switzerland	141	Norway	68
Greece	145	Montenegro	72
Italy	148	Switzerland	74
Cyprus	157	Italy	83
Sweden	174	Slovenia	86
Slovenia	175	United Kingdom	87
Poland	176	Poland	90
United Kingdom	177	Sweden	94
Bosnia & Herzegovina	185	Germany	101
TFYR Macedonia	188	TFYR Macedonia	103
Germany	189	Ireland	117
Serbia	194	Serbia	125
Ireland	226	Iceland	125
Austria	226	Bosnia & Herzegovina	132
Iceland	238	Austria	132
Malta	240	Finland	137
Turkey	244	Kazakhstan	140
Kazakhstan	249	Turkey	145
Azerbaijan	250	Bulgaria	145
Bulgaria	251	Malta	163
Albania	256	Georgia	169
Georgia	266	Azerbaijan	178
Finland	269	Albania	183
Croatia	341	Estonia	211
Romania	364	Croatia	244
Czech Republic	388	Romania	250
Estonia	388	Czech Republic	253
Slovakia	465	Hungary	315
Hungary	479	Slovakia	321
Latvia	584	Latvia	331
Armenia	637	Lithuania	429
Uzbekistan	688	Armenia	446
Lithuania	700	Russian Federation	466
Russian Federation	790	Belarus	505
Republic of Moldova	898	Uzbekistan	508
Kyrgyzstan	984	Republic of Moldova	717
Belarus	1 011	Ukraine	727
Ukraine	1 102	Kyrgyzstan	747

Table 2 Age-standardised death rates from stroke, all ages, by sex, latest available year, European region

Males		Females	
	Deaths per 100 000		Deaths per 100 000
Switzerland	51	France	42
France	53	Switzerland	47
Luxembourg	53	Israel	48
Israel	57	Spain	52
Austria	62	Luxembourg	52
Spain	62	San Marino	55
Germany	65	Austria	56
Norway	66	Norway	59
Netherlands	68	Germany	59
Belgium	69	Belgium	60
Cyprus	70	Sweden	62
United Kingdom	70	Denmark	64
Ireland	71	Netherlands	65
Sweden	73	Estonia	66
Denmark	79	Cyprus	66
Malta	84	United Kingdom	68
Iceland	88	Ireland	70
Finland	89	Iceland	72
Estonia	93	Finland	76
Italy	96	Malta	78
San Marino	109	Italy	83
Poland	117	Poland	91
Czech Republic	124	Portugal	99
Montenegro	126	Slovenia	104
Portugal	128	Czech Republic	104
Greece	139	Slovakia	123
Turkey	140	Turkey	124
Slovenia	144	Hungary	128
Armenia	159	Greece	136
Hungary	173	Montenegro	147
Slovakia	173	Bosnia & Herzegovina	157
Bosnia & Herzegovina	184	Uzbekistan	158
Serbia	210	Belarus	160
Croatia	210	Armenia	162
Uzbekistan	210	Croatia	173
Lithuania	219	Kazakhstan	175
Kazakhstan	225	Georgia	175
Belarus	239	Lithuania	179
Georgia	244	Serbia	187
Albania	295	Latvia	231
Latvia	296	Ukraine	231
Romania	297	Romania	241
Ukraine	305	Turkmenistan	259
Turkmenistan	314	Kyrgyzstan	277
Azerbaijan	345	Republic of Moldova	279
Kyrgyzstan	351	Albania	280
Bulgaria	353	Bulgaria	281
Republic of Moldova	354	Russian Federation	318
TFYR Macedonia	383	Azerbaijan	342
Russian Federation	415	TFYR Macedonia	345

Over the past 30 years, mortality rates from CHD have been declining in most Northern and Western European countries in both men and women. Long-term trends in Central and Eastern countries have been less consistent, however, with sharp decreases followed by increases and then further decreases in countries such as Ukraine and Russia, and more gradual increases followed by decreases in other countries such as Romania. Since around 2000 to 2005, age-standardised death rates from CHD have been falling in the majority of European countries, including those in Central and Eastern regions (Figures 12-13). Comparing the percentage difference in death rates from 2003 and the latest available year, the rate of decline among men in EU countries varied from 13% in the Czech Republic to 54% in the Netherlands and from 8% in the Czech Republic to 57% in Estonia among women. In non-EU countries, declines in age-standardised death rates from CHD between 2003 and the most recent year ranged from 12% among both men and women in Ukraine to 76% and 79% among men and women respectively in Kazakhstan. Only in Kyrgyzstan (not shown on figures) were death rates from CHD higher in the most recent year (2012) than in 2003.

Similar trends in age-standardised mortality rates are seen for stroke, with steady declines occurring since the 1980s in most Northern, Southern and Western European countries compared to more recent decreases in Central and Eastern European countries (Figures 14 and 15). Within the EU, the percentage difference in age-standardised mortality rates between 2003 and the latest available year ranged from 14% in Bulgaria to 73% in Estonia among men and from 11% in Ireland to 75% in Estonia among women. In non-EU countries, the percentage difference over the same period varied from 4% in Albania to 56% in Armenia 73% among men and from 6% in Macedonia to 75% in Kazakhstan among women. Only in Azerbaijan in both sexes and Albania in women was the age-standardised death rate from stroke in the most recent year higher than that in 2003 (not shown on figures).

Figure 12 Age-standardised death rates/100 000 from CHD, men, 1980 to 2015, selected European countries Source: European Cardiovascular Statistics, 2017[1]

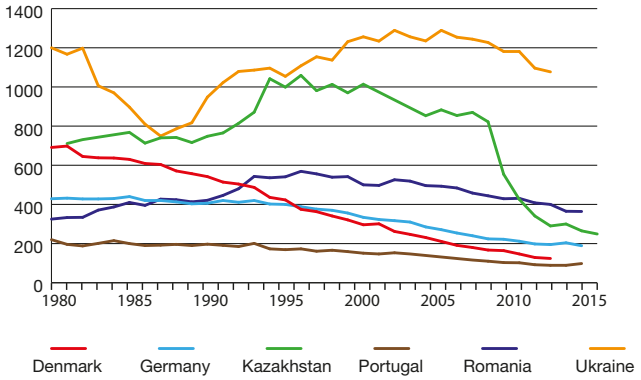


Figure 13 Age- standardised death rates/100 000 from CHD, women, 1980 to 2015, selected European countries Source: European Cardiovascular Statistics, 2017[1]

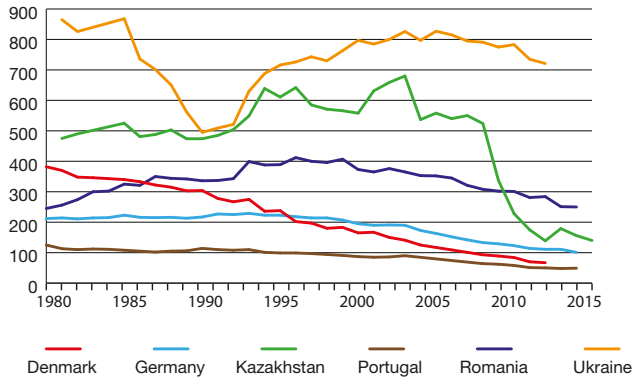


Figure 14 Age-standardised death rates/100 000 from stroke, men, 1980 to 2015, selected European countries Source: European Cardiovascular Statistics, 2017[1]

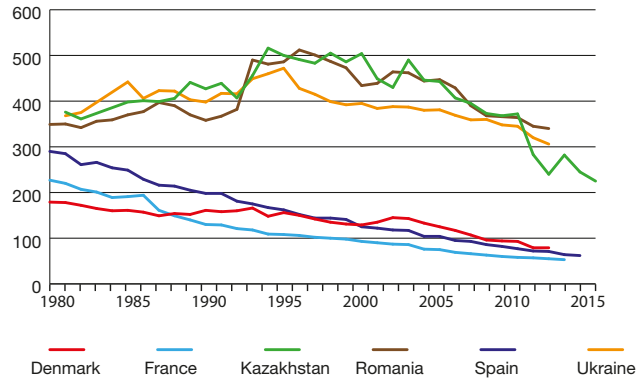
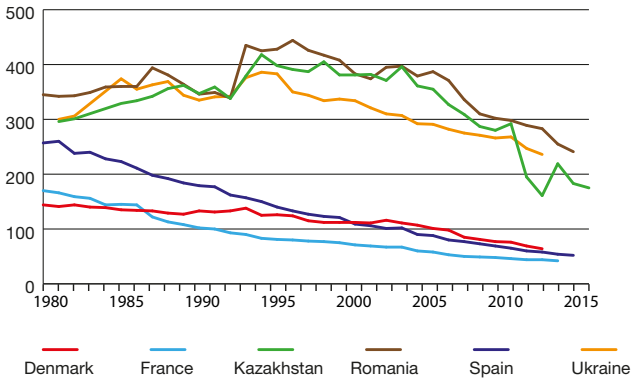


Figure 15 Age standardised death rates/100 000 from stroke, women, 1980 to 2015, selected European countries Source: European Cardiovascular Statistics, 2017[1]



1.1.3 A huge burden of illness and disability

In addition to the loss and suffering due to preventable deaths, for millions of people CVD means living with illness and disability for prolonged periods. In contrast to some commonly held misconceptions about heart disease and stroke resulting in quick and painless deaths, CVD is a major cause of years lived with disability.

In 2015, there were just under 11.3 million new cases of CVD in Europe as a whole – 5.4 million among men and 5.8 million among women – and 6.1 million new cases of CVD in the EU, where prevalence numbers for women are also higher than for men, meaning there are more women living with CVD than men.[iv] Between 1990 and 2015 most European countries reported an increase in the number of new CVD cases with increases of nearly 100% in some countries.

In 2015, more than 85 million people in the European region were living with CVD and almost 49 million people were living with CVD in the EU. Over the past 25 years, the absolute number of CVD cases has increased in Europe and in the EU, with increases in the number of new CVD cases found in most countries.

In 2015, CVD accounted for 23% of all the years lost to death or disability in the European region, responsible for the loss of 64.7 million disability-adjusted life years (DALYs) (Figure 16). For the EU, more than 26 million DALYs were lost as a result of CVD, around 19% of the total DALYs lost (Figure 17).

Figure 16 Disability-adjusted life years lost by cause, 2015, European region Source: European Cardiovascular Statistics, 2017[1]

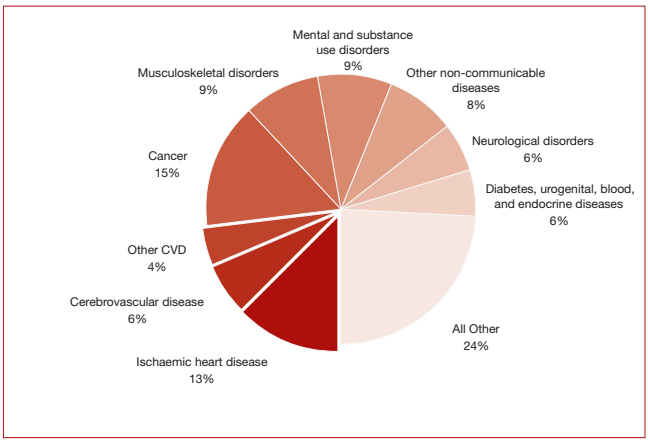
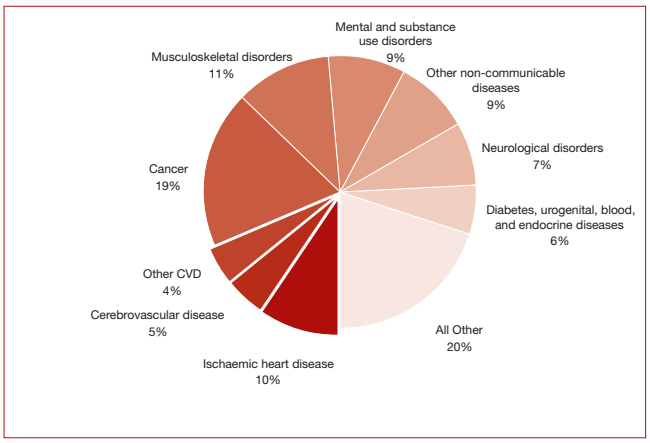


Figure 17 Disability-adjusted life years lost by cause, 2015, European Union Source: European Cardiovascular Statistics, 2017[1]



1.1.3.1 The economic cost of CVD

In addition to the human loss and suffering associated with this burden of CVD, there are also potentially devastating social and economic costs. Overall, CVD is estimated to cost the EU economy almost €210 billion per year – of this, around 53% (€111 billion) is due to direct health care costs, 26% (€54 billion) is due to productivity losses and 22% (€45 billion) to the informal care of people with CVD (Table 3).

Table 3 Total cost of CVD, heart disease and stroke, 2015, European Union

	CVD		Heart disease		Stroke	
	€ billions	% of total	€ billions	% of total	€ billions	% of total
Direct healthcare costs	€110.9	53%	€18.9	32%	€20.1	44%
Productivity loss due to mortality	€31.6	15%	€13.8	23%	€5.4	12%
Productivity loss due to morbidity	€22.6	11%	€6.0	10%	€4.0	9%
Informal care costs	€45.1	21%	€20.6	35%	€15.9	35%
TOTAL	**€210.2**		**€59.3**		**€45.4**	

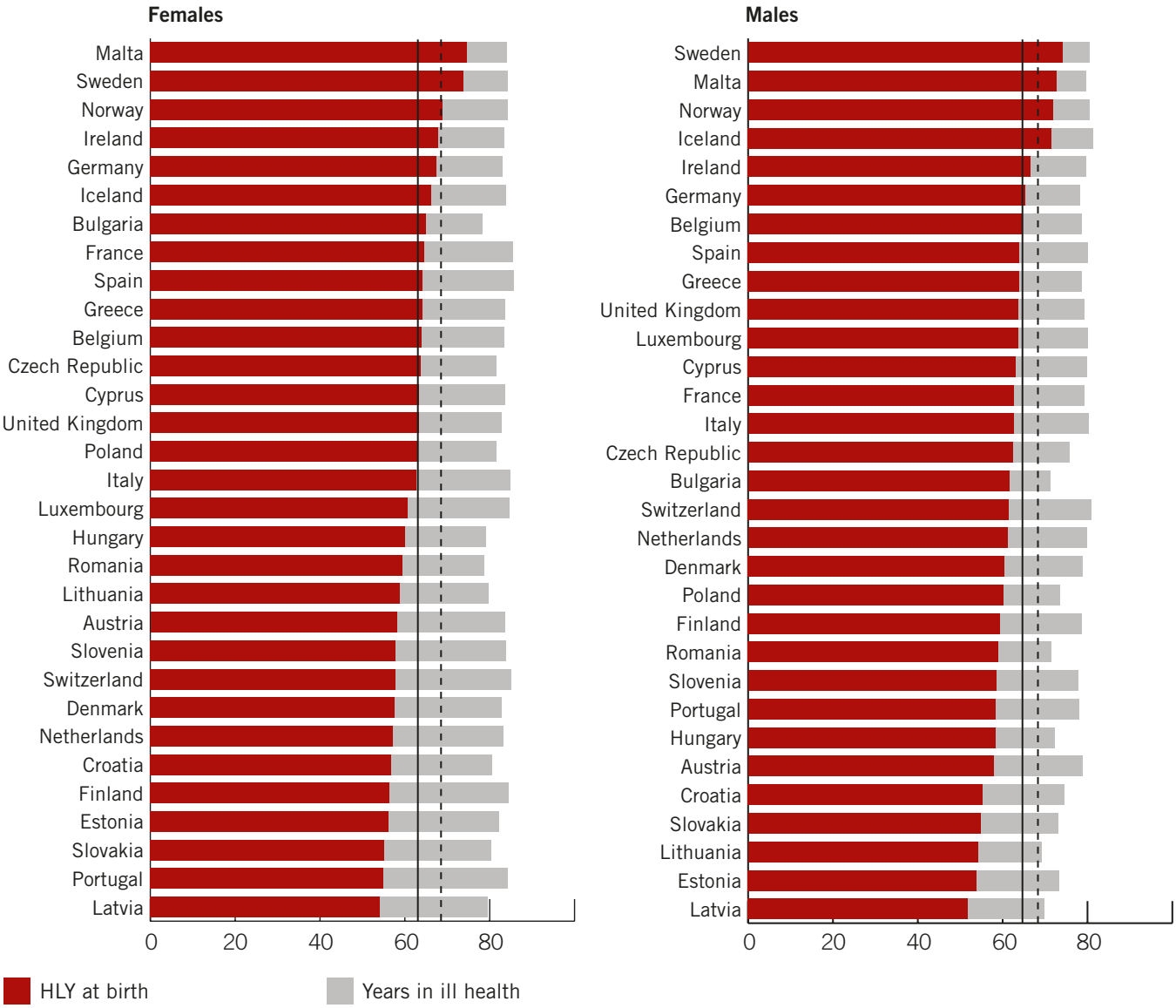
Note: The total CVD column includes heart disease and stroke as well as other forms of CVD.

iv Age standardised prevalence, though is higher in men then in women, suggesting that the disability burden is greater for men than for women

1.1.3.2 Social implications of CVD

The true social implications of the CVD burden reach beyond the human and economic costs of health care, lost productivity and care costs. Demographic changes and the tremendous achievements in extending life expectancy mean that in OECD countries the average number of people over the age of 65 for every 100 people of working age has doubled from 14 in 1950 to 28 in 2015.[5] This ratio is predicted to increase to 35 in 2025 and reach 51 by 2050.[5] In order to finance the pensions and care needs of these ageing populations, European governments are raising statutory retirement ages. Substantial numbers of the population, however, already have some disability at these new or proposed retirement ages. Figure 18 shows that, for the vast majority of European countries, average healthy life expectancy is below 70 for both men and women. In 11 countries, the healthy life expectancy of women is under 60, and in 12 countries men can expect to live healthily for less than 60 years. The average retirement age for OECD countries was 65.5 years in 2015[5] and in many EU member states the retirement age will be increased to 67 or 68 years within the next 10 years.[v] Furthermore, given the health inequalities that exist throughout Europe, these average figures are likely to mask a picture that is very much worse in poorer socioeconomic groups.

Figure 18 Healthy life expectancy (HLY), years lived in ill health and current and future retirement ages in some European countries



Notes:

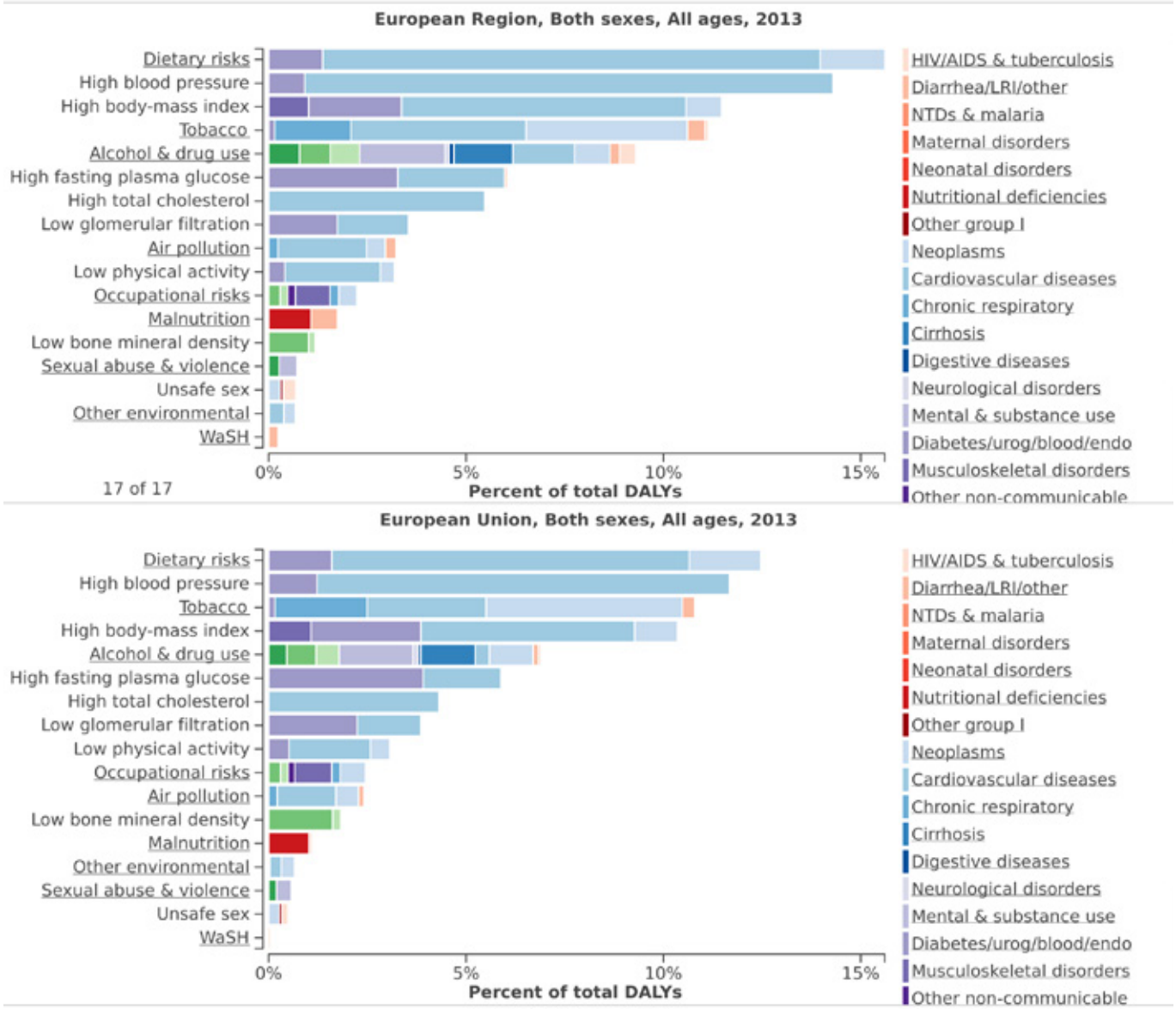
- Average retirement age for OECD countries in 2014 was 64 years for men and 63 for women (see solid lines). Retirement ages for people entering the labour market at 20 are rising to 67 or older for both men and women (dotted lines).[2]
- Healthy life expectancy and years in ill health data from: EUROSTAT, 2015, and retirement ages from OECD.
- Due to no data being available for Switzerland in 2015, data from 2014 has been used.

v http://www.etk.fi/en/the-pension-system-2/the-pension-system/international-comparison/retirement-ages/#toggle-id-1

1.2 Dietary contribution to burden of death and disability

Poor diet is a leading contributor to ill-health and premature death. In Europe on average, and in the EU, of the four major behavioural risk factors (diet, low physical activity, smoking, alcohol use) dietary factors made the largest contribution to the population-level CVD mortality burden in both sexes in 2015.[vi] A cluster of dietary risks[vii] accounts for 1.12 million CVD deaths among men in the European region and more than 476 000 male CVD deaths in the EU. For women, 1.13 million CVD deaths in the European region and more than 458 000 CVD deaths in the EU were attributable to the cluster of dietary risks factors.[viii] These dietary risk factors account for 56.2% of male CVD deaths in 2015 in the European region and 50.4% in the EU. For women, 48.3% of female CVD deaths in 2015 in the European region and 41.5% of such deaths in the EU were attributable to dietary risk factors.

Figure 19 Risk factor contributions to the burden of disease in the European region and the European Union Source: Institute for Health Metrics and Evaluation, GBD Compare, Viz Hub



vi At the individual level, smoking makes the greatest contribution to increasing the risk of CVD mortality, but the greater population prevalence of poor diet makes this risk factor the most meaningful at the aggregate level.

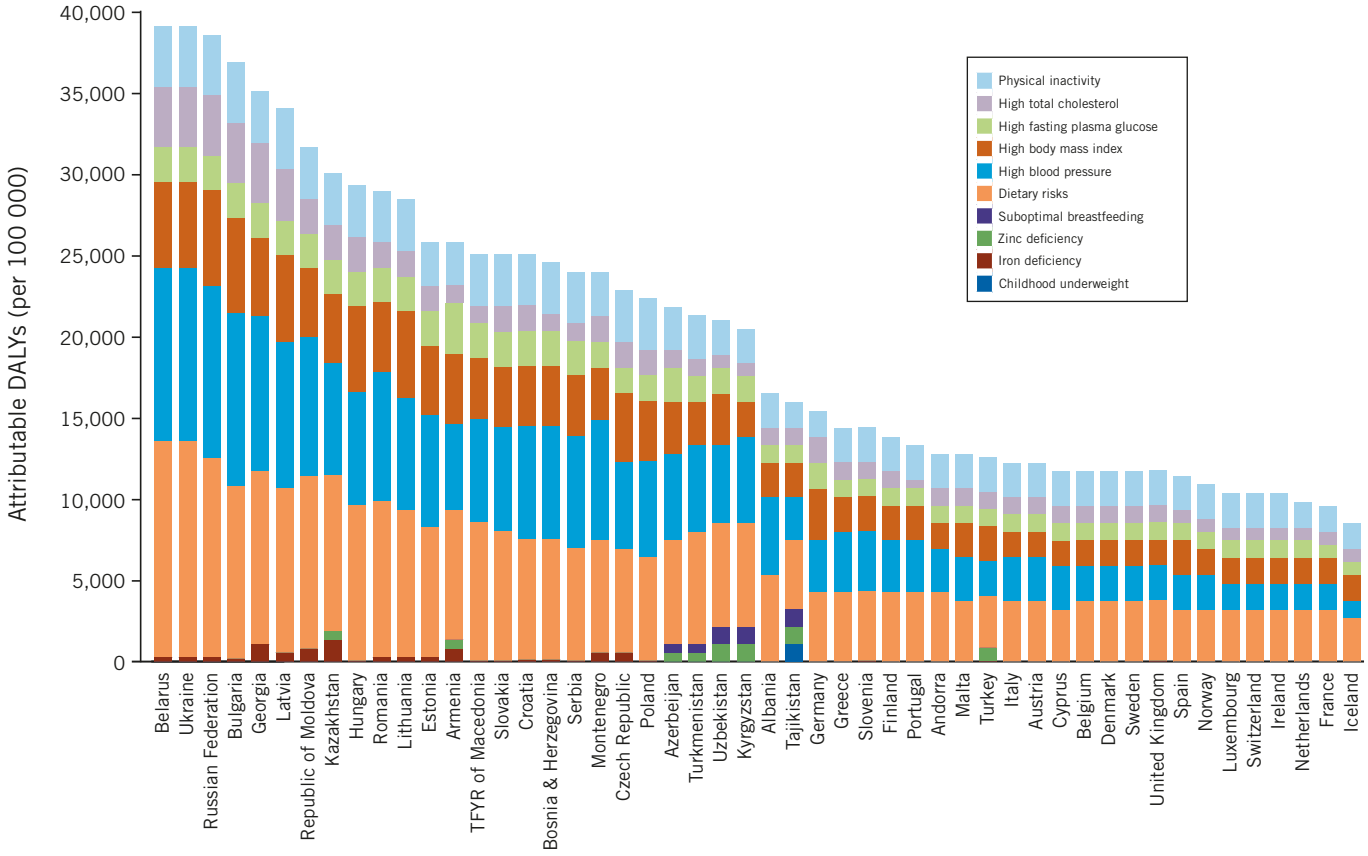
vii The cluster of dietary risks includes: high sodium; low fruit; low whole grains; low vegetables; low nuts and seeds; high processed meat; low fibre; low omega-3; low polyunsaturated fatty acids; high trans fat; suboptimal calcium; low milk; high red meat and high sweetened beverages.

viii Data from the Global Burden of Disease database (2015) https://vizhub.healthdata.org/gbd-compare/.

When the burden of ill-health is also included, dietary risk factors are the greatest contributor to the burden of death and disability, expressed as DALYs lost, in Europe and in the EU (Figure 19).

Figure 20 shows the contribution of risk factors to non-communicable diseases (NCDs) for 50 countries in Europe. It illustrates clearly that, not only is the overall burden of NCDs heavier in the Eastern part of the European region, but that DALYs contributed by dietary risk factors (in orange) are also considerably higher in Eastern European countries – with dietary risk factors accounting for 10 000 or more DALYs per 100 000 of those lost to NCDs in Belarus, Ukraine, Russian Federation, Bulgaria, Georgia, Moldova and Kazakhstan compared to well below 5 000 in most of Western Europe. Diet and physical inactivity are also the major, if not only, reasons why high blood pressure, high cholesterol, high body mass index and high blood glucose occur and contribute to the national burden.

Figure 20 DALYs attributed to risk factors related to NCDs for countries in the European region Source: Global Burden of Disease Study 2010 (GBD 2010. Results by Risk Factor 1990–2010 - Country Level. Seattle, United States Institute for Health Metrics and Evalution (IHME) 2013



1.2.1 Dietary contribution to the burden of cardiovascular disease in Europe

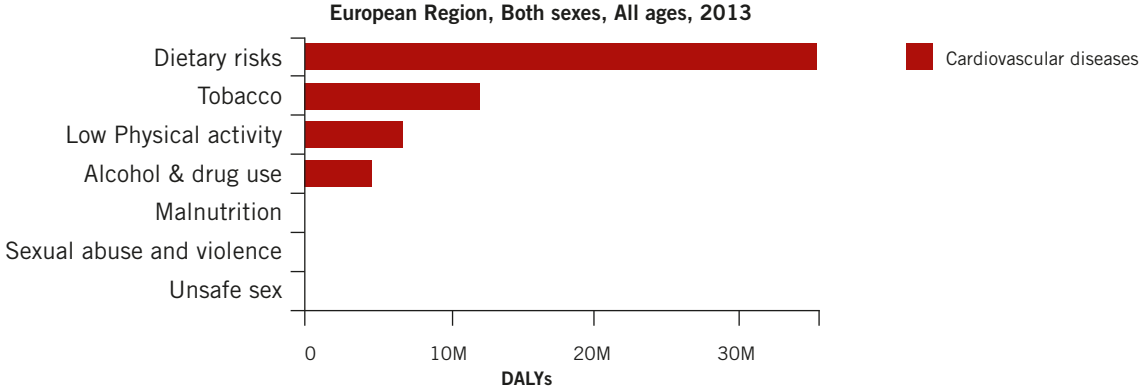
Specifically in relation to CVD, dietary risks accounted for 56.1% of total DALYs lost to CVD (men and women combined) in the European region in 2015. In the EU, 49.3% of DALYs lost to CVD in 2015 were attributable to dietary risk factors.

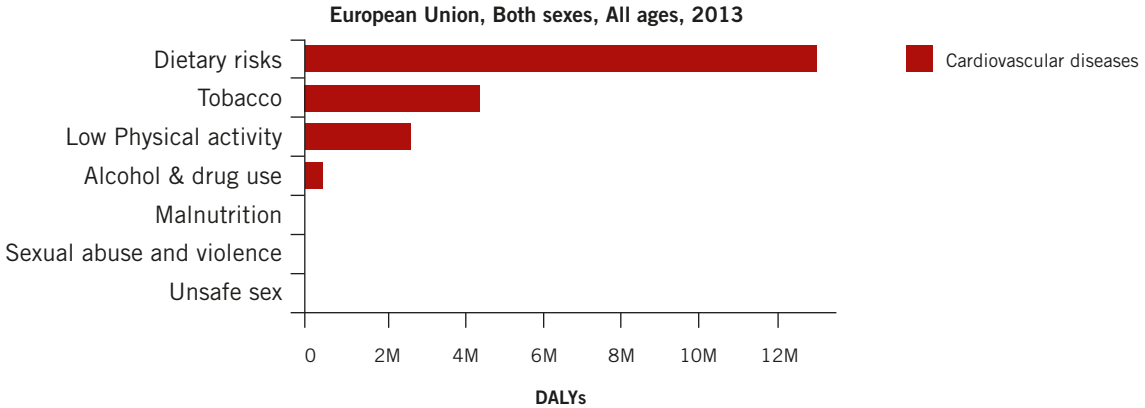
For men, 59.7% of DALYs were lost to CVD in the European region in 2015, and 53.8% in the EU. Among women in the European region, 51.5% of DALYs lost to CVD were attributable to dietary risks in 2015, while 43.4% of the burden of CVD among women in the EU was attributable to dietary risks. [1]

Figure 21 shows that the number of DALYs lost to CVD in 2013 attributable to dietary risk factors is much greater than those accounted for by other behavioural risk factors.

Dietary factors account for 56% of the DALYs lost to cardiovascular disease in the European region and 49% in the EU. The total economic cost of the burden of diet-related cardiovascular disease could, therefore, be considered to be 49% of the total annual economic costs of CVD in the EU, equivalent to €102 billion.

Figure 21 Behavioural risk factor contributions to the burden of CVD in the European region and the European Union

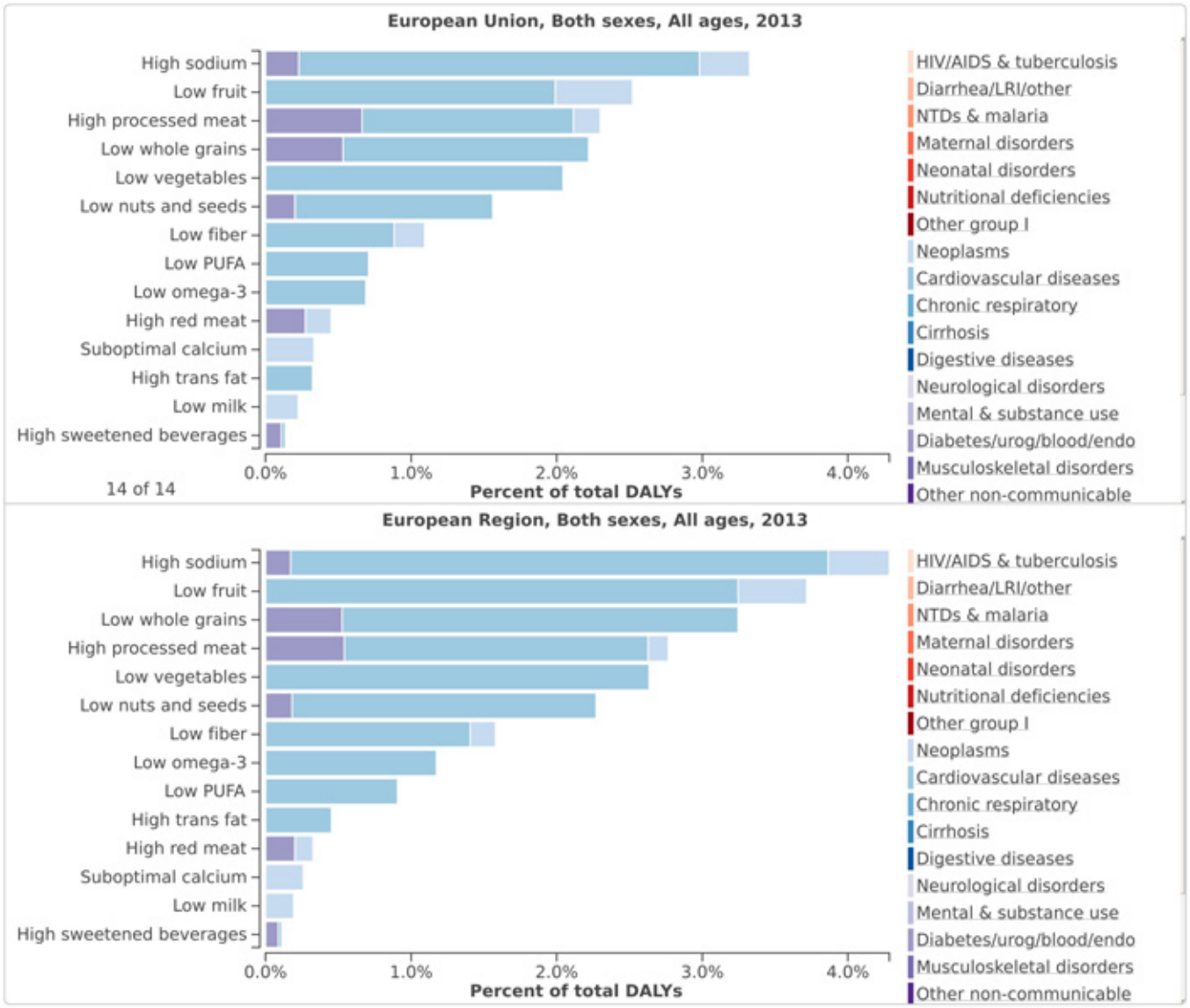




1.2.2 Contribution of specific dietary risk factors to the burden of disease

Within the dietary risk factors group, the contribution of individual dietary risk factors to DALYs lost to all causes (Figure 22) including CVD is estimated. When only DALYs lost to CVD are considered the Global Burden of Disease analysis finds the leading risk factors are diets that are high in sodium, low in fruit, low in whole grains and low in vegetables.

Figure 22 Contribution of specific dietary risk factors to DALYs, all causes, in the European Union and the European region Source: Institute for Health Metrics and Evaluation, GBD Compare, Viz Hub



1.3 Clear case for investing in prevention

Cardiovascular disease is to a large extent preventable and, given the economic burden that it currently presents – in addition to the suffering it causes, the potential economic gains are enormous. Globally, the direct and indirect costs of CVD were estimated at US$863 billion in 2010, predicted to rise to US$1.04 trillion by 2030.[7] The lost output between 2011 and 2030 from NCDs, including CVD, has been estimated at US$ 47 trillion.[7] The economic burden of life lost due to all NCDs was estimated at US$ 22.8 trillion in 2010 and is projected to reach US$ 43.3 trillion by 2030. [7]

In Europe, the total costs of CVD alone were estimated to be €210 billion in 2015 for the EU, including €111 billion in health care costs, €45 billion in providing informal care and €54 billion in productivity losses. There is clearly, therefore, an overwhelming case for investing in prevention.

We know from experience that prevention can work. Historical changes in dietary patterns that occurred for economic or political reasons – as happened in Poland, for example, in the early 1990s – have provided evidence of the dramatic effects that are possible in a relatively short timescale. The differences that exist between countries also point to lower rates of CVD being possible.

As set out in section 1.2, 56% of all DALYs lost to CVD in Europe are attributed to dietary risk factors. Within the EU, dietary risk factors account for 49% of DALYs lost to CVD at an estimated cost of €102 billion. Reducing exposure to these risks, as well as smoking, therefore, offers great potential to reduce the death and disability caused by CVD.

A population-based approach – which aims to reduce exposure to risk factors across the whole population rather than only targeting high-risk individuals – offers the greatest promise. This greater benefit has been recognised for the last 35 years.[8] Yet efforts to ensure that high-risk individuals can access treatment have not been matched by a similar coherent, pervasive and intense focus on the key preventive measures needed. Improvements in access to medicines to lower risk factors (e.g. blood cholesterol or raised blood pressure) have been seen across Europe in recent years – between 2000 and 2013 the use of antihypertensive drugs and of cholesterol-lowering drugs increased in all European OECD countries for which data are available.[1] The smallest increase in the use of cholesterol-lowering drugs over that period was 50% (in France), with some countries experiencing very dramatic increases. In Estonia, for example, there was a 29-fold increase. While this improved access to medications is welcome, such pharmaceutical approaches to prevention remain relatively costly and warrant stronger population-based efforts.

As part of a population-wide approach, actions to reduce exposure to dietary risks have tremendous potential, since around half of the DALYs lost to CVD are attributable to dietary risks. There is a growing body of evidence that interventions relating to nutrition are not only good value for money (cost-effective in terms of how much investment is needed to save a DALY) but that they actually result in net cost savings.[9,10]The economic costs of implementing, for example, taxes on unhealthy foods[9] or measures to reduce salt levels in processed foods[10] are outweighed by the resulting economic benefits. It pays in both health and economic terms, therefore, to take preventive measures. The following sections review the science underpinning diet-related actions and the evidence for their effectiveness.

References

1. Wilkins, E. *et al. European Cardiovascular Disease Statistics 2017. European Heart Network* (2017).
2. Organisation for Economic Co-operation and Development (OECD). *OECD Health Data 2012: Definitions, Sources and Methods*. (2012).
3. World Health Organization Regional Office for Europe. The European health report 2015 Targets and beyond – reaching new frontiers in evidence - Highlights. (2015).
4. Smith, O. & Nguyen, S. N. *Getting better: improving health system outcomes in Europe and Central Asia* (2013).
5. OECD. *Pensions at a Glance 2013: OECD and G20 Indicators*. (2013).
6. OECD. *Pensions at a glance 2015. OECD and G20 indicators.* (2015).
7. Bloom, D. *et al. The Global Economic Burden of Noncommunicable Diseases*. 8, (2011).
8. Rose, G. Strategy of Preventive Medicine. 171 (2008).
9. Cecchini, M. *et al.* Tackling of unhealthy diets, physical inactivity, and obesity: Health effects and cost-effectiveness. *Lancet* 376, 1775–1784 (2010).
10. Vos, T. Assessing Cost-Effectiveness in Prevention. *Assess. Cost-Effectiveness Prev.* (2010).

2 Food, drink and cardiovascular disease: the science

Key points

- The scientific evidence base for recommending a cardiovascular health-promoting diet has generally strengthened in the last six years.
- Population goals for foods and nutrients have been reviewed and updated to take into account recent research findings. Specific target goals – with a *direct* impact on cardiovascular disease (CVD) outcomes – are recommended for fruit and vegetables, saturated fats, trans fats, dietary fibre and salt. Marker goals – which have an *indirect* impact on CVD outcomes and are indicators of a good dietary pattern for CVD prevention – are proposed for free sugars, total fat, total carbohydrate, sugar-sweetened beverages and body mass index.
- The population goals need to be adapted into food-based dietary guidelines at the national level. In general, a cardiovascular health-promoting diet means a shift from an animal-based diet to a more plant-based diet. It includes vegetables, fruit and berries in abundance. Whole grain products, nuts and seeds, fish, pulses and low-fat dairy products are also important, as are non-tropical vegetable oils in modest amounts. This everyday dietary pattern also limits consumption of red meat, processed meat products and foods or drinks with low content of vitamins, minerals and dietary fibre and/or a high content of free sugars, saturated/trans fats or salt.
- Apparent controversies about dietary recommendations often stem from a limited understanding, or misrepresentation, of the science or methodological issues relating to associations between diet and health outcomes.
- Careful unpicking of two apparent controversies – relating to salt and saturated fat – reveals that the evidence for the messages to limit salt/sodium consumption and to replace saturated fat with unsaturated fats or fibre-rich complex carbohydrates remains robust. Recent research on the associations between consumption of free sugars, and more specifically sugar-sweetened beverages, reinforces the evidence for the recommendation to limit consumption. There is a growing evidence base on the impact that nutrition during early life – pre-conception and during pregnancy, infancy and early childhood – can have on later health outcomes, including cardiovascular risk factors. This means that both maternal nutrition status and infant/young child feeding are important.

Following a detailed two-year review of the evidence base on foods, nutrients and cardiovascular disease (CVD), EHN's 2011 report proposed a series of population goals in order to inform and monitor policy. These goals were proposed on the basis of a series of specially commissioned expert papers which were reviewed by EHN's Nutrition Expert Group. These in-depth reviews – which provide a valuable summary of the overall evidence base – were included in the 2011 report (the full version is available from http://www.ehnheart.org/publications-and-papers/publications.html).

For this edition, the Expert Group, supported by special advisers, has reviewed those population goals on the basis of major new evidence that has emerged in the last few years. In addition, a small number of expert reviews have been commissioned to summarise new important evidence on a few issues which have been the subject of debate and particular media interest in recent years.

2.1 Healthy food and drink patterns

While Chapter 2.2 considers the evidence on specific foods, nutrients and ingredients, it is important to remember that foods and drinks are not consumed in isolation and the overall dietary patterns of foods and drinks consumed are important. The different aspects of the diet relate to one another in a variety of ways and there could potentially be synergies and cumulative effects.

A number of specific dietary patterns have been the subject of considerable scientific investigation. While much research has focused on deconstructing the diets and identifying the most important elements of dietary patterns associated with health, there is a growing evidence base on the health outcomes associated with the overall dietary patterns and the degree of adherence to specific patterns.

The most well-known and most frequently researched dietary pattern is the Mediterranean diet, which comprises high intakes of fruits, vegetables, legumes, wholegrain products, fish and unsaturated fatty acids (especially olive oil) and low consumption of (red) meat, dairy products and saturated fats. A 2010 meta-analysis of prospective cohort studies found that greater adherence to a Mediterranean diet was associated with a 10% lower cardiovascular incidence or mortality and an 8% lower all-cause mortality.[1] A randomised controlled trial (RCT) in high-risk individuals found that following a Mediterranean dietary pattern over five years was associated with a 29% lower CVD risk than those on control diets.[2] Research on secondary prevention, in people who have already suffered a cardiovascular event, also suggests that a Mediterranean-style diet reduces the risk of recurrent heart disease.[3] A consistent feature of the Mediterranean diet has been a small amount of regular alcohol, but there is no evidence that the health benefits of this dietary pattern are due to alcohol.

The Dietary Approaches to Stop Hypertension (DASH) dietary pattern is high in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, beans and nuts and is low in fats, sweets, sugar-sweetened drinks and red meat. It is low in saturated fats and sodium, and rich in potassium, calcium, magnesium, fibre and protein. The original DASH

trial[4] demonstrated that the DASH dietary pattern lowered blood pressure and LDL-cholesterol levels, resulting in reduced CVD risk, compared to typical US diets, and these findings were confirmed in the DASH-Sodium trial.[5] Later, the OmniHeart Trial, found that replacing some of the carbohydrates in DASH with the same amount of either protein (with an emphasis on protein from plant sources) or unsaturated fats resulted in bigger falls in blood pressure and LDL-cholesterol.[6]

The Healthy Nordic Diet emphasises food items typical for the food culture in the Nordic countries. This dietary pattern includes natural fibre-rich foods such as vegetables (e.g. dark-green leaves, fresh peas and beans, cabbage, onions, root vegetables, and fruiting vegetables such as peppers and tomatoes), pulses, fruits, berries, nuts, seeds and whole grains as well as fish and seafood, rapeseed oil, vegetable oil-based fat spreads, and fat-free and low-fat dairy products.[7] Intervention studies suggest that changing from a typical diet in Nordic countries to the Healthy Nordic Diet (according to the Nordic Nutrition Recommendations) is associated with cardiovascular benefits.[7]

The US Dietary Guidelines for Americans 2015–2020 concluded that there is strong evidence that healthy eating patterns are associated with a reduced risk of CVD and that higher intakes of vegetables and fruits, and whole grains, have consistently (though not quite as consistently for whole grains) been identified as characteristic of healthy eating patterns.[8] Other characteristics of healthy eating patterns (albeit identified with less consistency) include fat-free or low-fat dairy, seafood, pulses and nuts, along with lower intakes of meats, processed poultry, sugar-sweetened foods (particularly drinks) and refined grains. The healthy eating pattern recommended to the US population includes a variety of vegetables from all the sub-groups (including pulses), fruits (especially whole fruits), grains (at least half of which are whole-grains), fat-free or low-fat dairy, a variety of protein foods (including seafood, lean meats, poultry, eggs, pulses), nuts, seeds, soy products and oils. This pattern limits saturated and trans fats, added sugars and sodium.

2.1.1 What do EHN proposed population goals mean for food and drink patterns?

In Chapter 2.2 EHN proposes specific population goals that are beneficial for prevention of CVD. Taken together, the proposed goals represent a cardiovascular health-promoting dietary pattern for present day Europe.

In summary, the energy density of a cardiovascular health-promoting diet for the current European population – which predominantly has low levels of physical activity – should be low, which is important for weight maintenance, and for prevention of overweight and obesity. A cardiovascular health-promoting diet means a shift from an animal-based diet to a more plant-based diet. It includes vegetables, fruit and berries in abundance. Whole grain products, nuts and seeds, fish, pulses and low-fat dairy products are also important, as are non-tropical vegetable oils in modest amounts. This everyday dietary pattern also limits consumption of red meat, processed meat products and foods or drinks with low content of vitamins, minerals and dietary fibre and/or a high content of free sugars, saturated/trans fats or salt.

The precise details of how these population goals translate to food-based dietary guidelines – which include advice on specific foods to eat or to avoid – will depend on the specific national context and prevailing dietary patterns. Such food-based dietary guidelines should also take into account seasonal availability, sustainability and other socio-economic factors.

2.2 Summary of latest science on foods, nutrients and cardiovascular disease

Table 4 summarises the revised population goals for foods and other related goals, while Table 5 summarises the revised population goals for nutrients and other components.

These goals represent a recommended average intake or level for the population as a whole; they are not dietary guidelines for individuals. The goals are intended to provide guidance on a desirable everyday diet and should not be interpreted, or presented, as meaning that individuals should *never* deviate from them. Furthermore, since the report is based on a population-level approach, it does not take account of genetic variations in how individuals respond to dietary risk factors or individual susceptibility to disease. There is a growing body of evidence in this field and, as a result, increasing scope for clinical services to provide personalised nutrition counselling.

Furthermore, the process of defining population goals is, to some extent, a matter of judgement. When there is a clear 'dose-response' relationship between an element of the diet and risk of CVD, for example, the population goal seeks to identify a point on the dose-response curve where the risk of CVD is reduced but which is also practical and acceptable in the contemporary European context.

The primary purposes of the population goals are:

- To enable policymakers to identify the gaps between actual and recommended dietary patterns,
- To inform the setting of policy priorities, and
- To facilitate monitoring of progress.

Goals on nutrients, foods and other factors

A few goals for specific foods or other factors are given in Table 4, but a greater number of goals are set out for specific nutrients in Table 5. There is a growing body of food-based research exploring the associations between specific foods and health outcomes. Evidence on such associations is not, however, of the same quality as the evidence on the associations between specific nutrients and health outcomes. Hence, this paper has a greater emphasis on population goals for nutrients.

It is, however, extremely important that national policymakers translate the nutrition goals into food-based dietary guidelines in a way that is appropriate to each national context, taking into account eating habits and food systems in the country.

Intermediate and long-term goals

In some cases, two different sets of population goals are proposed:

- **Intermediate targets** based on an assessment of current dietary patterns in Europe and including 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 the pragmatic constraints that feed into the intermediate targets can be overcome.

In other cases, it is considered so important to start working towards the long-term goal immediately that no intermediate target is proposed.

Goals adapted to the European context

The goals take into account the reality of current European populations, which, in general, comprise substantial proportions of overweight or obese individuals, with high levels of physical inactivity, who currently consume diets that are energy dense. The relationship between physical activity and nutrition is complex, and physical activity can influence energy intake and appetite control, as well as affecting energy expenditure. It is important to recognise that, while this paper does not deal specifically with physical activity, the population goals are influenced by current physical activity levels and policies to promote cardiovascular health-promoting diets need also to be accompanied by policy action to facilitate, encourage and support physical activity.

Specific 'target' and 'marker' goals

For these reasons two distinct types of population goal are proposed:

- Specific **target goals** that have a *direct* impact on CVD outcomes, independently of other aspects of the diet. The goal for saturated fat, for example, is a specific target goal with a direct impact on cardiovascular outcomes as described in section 2.3.3 and set out in Table 5;
- Other **marker goals** that have an *indirect* impact on CVD outcomes and are indicators of a good dietary pattern for CVD prevention. For example, some goals are important because of their impact on the energy density of the diet and, therefore, important for the prevention of unhealthy weight gain in the context of a population with low levels of physical activity. The goal on total fat, for example, is a marker goal of an optimal dietary pattern for cardiovascular health.

Policymakers need to develop policies and food-based dietary guidelines towards achieving *both* the specific target goals and the marker goals, in order to achieve optimal dietary patterns for cardiovascular health.

There is now greater awareness that how people achieve the dietary goals is also important – in other words, what foods people use to replace nutrients or components that guidelines recommend reducing, such as saturated fat. The overall pattern of food intake (the dietary pattern) is important. Both dietary guidelines and policy options, therefore, need to be carefully designed to take into account these possible substitution issues. It is important, for example, that fats are not replaced by refined carbohydrates. Equally, trans fatty acids should not be replaced with saturated fats. In policy terms, for example, it is important that taxes do not inadvertently increase consumption of other – untaxed – unhealthy foods.

Table 4 Proposed population goals for foods and other related goals[i]

FOOD/ OTHER GOALS	POPULATION GOALS[ii]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
FOODS			
Fruit and vegetables	**Intermediate:** More than 400 g/ day **Long-term:** More than 600 g/day	The evidence that higher intakes of fruits and vegetables are protective against premature death[9] and, more specifically, against cardiovascular deaths[9] and the risk of stroke[10] has strengthened since our previous report. This includes processed fruit and vegetables, including frozen, canned, etc. Consumption of potatoes or fruit juice does not count towards the fruit and vegetable goal. This is a specific **target** goal; increasing fruit and vegetable intakes is also important when replacing fat intakes.	WHO continues to recommend intakes of at least 400 g of fruit and vegetables combined daily. EHN maintains the proposed interim population goal of at least 400 g per person daily and the case for the longer-term goal of 600 g daily – in line with the World Cancer Research Fund's recommendations for prevention of cancer – is supported by increasingly strong evidence.
Sugar-sweetened drinks *[Beverages containing added caloric sweeteners such as sucrose or high-fructose corn syrup.]*	**Intermediate:** Decrease as much as possible **Long-term:** Virtually zero	See scientific comment on free sugars below. Much of the new information that has become available on the potential association between consumption of sugars and the risk of cardiovascular disease in recent years relates to the contribution of SSBs (See Chapter 2.3.2). Because the dose-response relationships so far do not suggest a lower threshold, the recommendation should be to limit free sugars consumption as much as possible in order to obtain the largest benefits. Sugar-sweetened beverages are major contributors to free sugars consumption and limiting consumption of these products as much as possible is recommended, especially in those struggling to maintain a healthy weight. This goal does not relate to fruit juices. However, in order to meet the goals for free sugars (which do include sugars from fruit juices) consumption of fruit juices should also be low (See Sugars goal in Table 5). This is a **marker goal** for an optimal dietary pattern for cardiovascular health.	Several recent national guidelines have recommended reducing (Germany[11]) minimising or limiting (France,[12] Nordic countries) consumption of sugar-sweetened beverages. In the Netherlands the recommendation is to limit consumption of sugar-containing beverages, including fruit juices, as much as possible. In its 2016 statement on added sugars and cardiovascular disease risk in children and adolescents, the AHA recommends that children and adolescents limit their intake of SSBs to 1 or fewer 8-oz (=237 ml) beverages per week.[13]

i These goals are for population averages, not dietary goals for individuals.

ii Some goals are broken down into intermediate and long-term goals. See further explanation in Section 2.2, which precedes these tables.

FOOD/ OTHER GOALS	POPULATION GOALS[ii]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
OTHER RELATED GOALS			
Body mass index	**Intermediate:** Average BMI of less than 23 for adults **Long-term:** Average BMI of 21 for adults	A healthy weight is recommended in order to improve the cardiovascular risk profile by preventing raised blood pressure and dyslipidaemia and reducing the risk of developing type 2 diabetes. These goals (unchanged since 2011) reflect average BMI for the whole population. It is important to be clear that recommendations may be different for people with particular clinical conditions, such as heart failure. There is also evidence that optimal weight in people aged over 70 is higher than in the young and middle-aged[14] and it should be noted that being underweight is also associated with health risks in older people. Body mass index is only one measure of body composition or 'fatness'. The ratio of waist to hip circumference and simple waist circumference are also used. WHO considers that a waist circumference of $\geq$ 94 cm in men and $\geq$ 80 cm in women represents increased risk and $\geq$ 102 in men and $\geq$ 88 in women represents substantially increased risk.[14,15] None of these anthropometric measures are able to give a precise estimate of the proportions of lean and fat tissue or the distribution of body fat, but there is convincing evidence for all of them that there is a moderate or strong relationship to CVD risk and they are valuable population markers of body composition. The relationship between smoking and body composition presents difficulties in interpretation of data on CVD. Smokers tend to have lower BMI than non-smokers (except for very heavy smokers) and so lower BMI may appear to be associated with increased risk of smoking-related CVD, because of confounding by smoking. Because it is difficult to ascertain exposure to smoking precisely, residual confounding is possible even if researchers try to control for smoking. Furthermore, smokers tend to have a greater waist circumference (more visceral adiposity) even though they tend to have a lower BMI. Moreover, low BMI may be mainly due to lean tissue loss rather than fat loss, possibly as a result of pre-existing disease or a marker of disease severity. This makes it difficult to fully explain the complex interactions between body size, shape and composition, smoking and CVD risk. This is a **marker goal** of a healthy pattern of diet and lifestyle for cardiovascular health.	This is in line with WHO's recommendation for a population median BMI range of 21–23. 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'.

Table 5 Proposed population goals for nutrients and other components[iii]

NUTRIENT/ DIETARY COMPONENT	POPULATION GOALS[iv]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
NUTRIENTS AND OTHER COMPONENTS			
Saturated fat	**Intermediate goal:** Less than 10% of food energy for the general population and less than 7% of food energy for a population at a high risk for AVD, less than one-third of total fat **Long-term goal:** 7% of dietary energy, and less than one-third of total fat	There is convincing evidence that partial replacement of SFA with unsaturated fat, especially PUFA and complex carbohydrates, decreases the concentrations of both total and LDL cholesterol and the risk of AVD. Saturated fats should make up less than one-third of total fat intake, while unsaturated fats (total of mono- and polyunsaturated fats) should be at least two-thirds of total fat intake. The recent controversy about saturated fat is due to issues of research methodology (see Chapter 2.3.3). One key issue is the fact that there are substantial (as much as four- to five-fold) differences in individual responses to changes in intakes of key SFAs. This means that cohort studies that do not measure blood cholesterol levels – but only measure SFA intakes – may not be sensitive enough given the range of saturated fat intakes within a population to show the relationship between SFA and heart disease. Another important issue is publication of papers that rely on studies which do not take into account what has replaced saturated fat in the diet. Replacing SFA with simple carbohydrates has unfavourable effects, but lower fat, high fibre diets are associated with consistent benefit. There is general consensus that the intake of SFA should be less than 10% of dietary energy and less than 7% for a population at high risk for AVD. The saturated fat target is a **specific target with a direct impact** on CVD outcomes. Saturated fats should be replaced with unsaturated fats, particularly PUFA, and fibre-rich complex carbohydrates.	Current dietary recommendations from the Nordic Nutrition Council, American College of Cardiology/American Heart Association and the European Society of Cardiology highlight the value of limiting intakes of SFA and TFA and having a moderate intake of unsaturated fat within the context of a healthy dietary pattern including fibre–rich carbohydrates to reduce the levels of risk factors, mainly LDL-cholesterol concentration, and the subsequent incidence of heart disease.[7,16,17] The general recommendation that unsaturated fats should contribute at least two thirds of total fatty acids in the diet is in line with the 2012 Nordic Nutrition Recommendations. WHO is in the process of updating its guideline on fats, including the 2003 recommendation for not more than 10% of total energy from saturated fat.[18]

iii These goals are for population averages, not dietary goals for individuals.

iv Some goals are broken down into intermediate and long-term goals. See further explanation in Section 2.2, which precedes these tables.

NUTRIENT/ DIETARY COMPONENT	POPULATION GOALS[iv]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
Trans fatty acids (TFAs)	Not more than 0.5% of energy from TFAs, of which 0% should be from industrially-produced TFAs	Based on experimental and many observational studies a high intake of TFA is considered very deleterious in terms of its effect in inducing a hazardous blood lipid profile and the risk of AVD. There is general consensus that the intake of TFA should be as low as possible. This is a **specific target with a direct impact** on CVD outcomes. It is important that trans fatty acids are replaced with unsaturated fats or fibre-rich complex carbohydrates.	The European Food Safety Authority's Panel on Dietetic Products, Nutrition and Allergies recommends that trans fatty acid intakes should be 'as low as possible'.[19] WHO is in the process of updating its guideline on fats, including the 2003 recommendation for less than 1% of total energy from trans fatty acids.[18]
Total fat	About 25% of food energy	Reducing the total fat to 25% – together with a lower free sugar intake and an increase in vegetable and fruit consumption to more than 400 g/d – will substantially reduce dietary energy density and contribute to minimising weight gain in the context of widespread physical inactivity. This total fat goal is sufficient to enable an adequate intake of essential fatty acids and vitamin E. This proposed **marker goal** for fat intake is, therefore, based on the need to limit the energy density of the diet for inactive European populations and thus reduce the risk of weight gain and diabetes and, therefore, the longer term risk of coronary heart disease. Total fat should be partially replaced with fibre-rich, complex unrefined carbohydrates, rather than refined carbohydrates. Unsaturated fats (total of mono- and polyunsaturated fats) should be at least two-thirds of total fat intake. There is insufficient evidence to set a precise recommendation for the ratio of n-6 polyunsaturated acids to n-3 polyunsaturated fatty acids.	This is in keeping with the long-term goal in EHN's 2011 paper for 20–25% energy from total fat. WHO is in the process of updating its guideline on fats, including the 2003 recommendation for less than 15–30% of total energy from total fat.[18] In its 2000 report on obesity WHO proposed 20–25% energy for sedentary individuals and societies.[20]
Total carbohydrate	**Intermediate:** More than 55% of energy **Long-term:** Up to 65% of energy	The goal for total carbohydrate is obtained by calculating the remaining portion of food energy, if total fat and protein are consumed according to the goals presented here. It is important that the carbohydrate be derived principally from whole-grain cereals, fruit, berries, vegetables and legumes. Refined cereal products should be replaced with whole grain products. This goal is a **marker** of a healthy dietary pattern.	National or sub-regional population goals for total carbohydrates range from 45 E% to 65 E% (Nordic Nutrition Council,[21] US,[8] UK,[22] EU[23]). The goals proposed 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%).[18]

NUTRIENT/ DIETARY COMPONENT	POPULATION GOALS[iv]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
Free sugars *[Defined as mono-saccharides and disaccharides added to foods and beverages by the manufacturer cook or consumer, and sugars naturally present in honey, syrups, fruit juices and fruit juice concentrates]*	**Intermediate:** Less than 10% of energy **Long-term:** 5% of energy	While EHN's previous recommendation related to added sugars, this recommendation relates to free sugars (see definition, left), in line with WHO. Importantly, this includes sugars in honey, syrups and fruit juices. Over the past six years a considerable amount of new information has become available on the potential association between consumption of sugars and the risk of CVD, and the effects are most evident when sugar-sweetened drinks and sugars are consumed in excess, i.e. when accompanied by weight gain. Adiposity increases with increasing consumption of sugars or sugar-sweetened beverages, but weight-gain independent effects may also be present (See Chapter 2.3.2). Evidence suggests that the increased risk is mainly associated with fructose-containing sugars, but the role of non-fructose containing sugars is less well studied and has not been fully elucidated. Although study results are not always consistent, a linear dose-response relationship between intake and risk is suggested by most studies. Confounding by other aspects of a Western diet cannot be fully excluded. Because the dose-response relationships so far do not suggest a lower threshold, the recommendation should be to limit free sugars consumption as much as possible in order to obtain the largest benefits. Limiting consumption of major contributors to free sugars consumption as much as possible should be recommended, especially in those struggling to maintain a healthy weight. This is a **marker goal** for an optimal dietary pattern for cardiovascular health.	EHN's interim and long-term goals take into account current intakes and are broadly in line with WHO's strong recommendation for all individuals to reduce intake of free sugars to less than 10% of total energy intake (including alcohol) in both adults and children to prevent unhealthy weight gain and dental caries. WHO also suggests a further reduction of the intake of free sugars to below 5% of total energy intake for further health benefits.[24] French, Nordic and Dutch dietary guidelines recommend limiting consumption of various foods rich in sugar and/or added sugar.[468] In England the recommendation is to limit free sugar intake to less than 5% of average energy intake from 2 years upward and to minimise sugar-sweetened beverage consumption in children and adults[7]. In the US, the Dietary Guidelines Advisory Committee concluded that a healthy dietary pattern is low in sugar-sweetened foods and drinks.[9] The American Heart Association recommends not more than 100 kcal per day from added sugars for women and no more than 150 kcal per day from added sugars for men, equivalent to about 5% of energy.[11] In its 2016 statement on added sugars and CVD risk in children and adolescents, the American Heart Association states that it is reasonable to recommend that children and adolescents consume ≤ 25 g (100 kcal) of added sugars per day.[10]
Fibre* * Using the AOAC definition	At least 12.6 g dietary fibre* per 1000 kcal (3 g per MJ energy)	This goal refers to natural fibre-rich foods, including whole grain cereals, pulses, vegetables, fruits and berries, nuts and seeds. These goals are solely based on the AOAC definition of dietary fibre. EHN's previous goals included a goal for non-starch polysaccharides (a different definition of fibre). Since the AOAC definition has been adopted by Codex and the EU, EHN has simplified the goals by focusing on this definition. The goal for fibre is both a **specific target** with a direct impact on cardiovascular health *and* a **marker** of a healthy dietary pattern for cardiovascular health.	Recent recommendations for fibre are in the region of 25–30 g of fibre per day for adults (UK,[22] US,[8] Nordic Nutrition Council[7]). The proposed EHN goal is coherent with these other recommendations on the basis of an assumed daily energy intake of about 2,500 kcal (26 g for the intermediate and 31 g for the long-term goal).

NUTRIENT/ DIETARY COMPONENT	POPULATION GOALS[iv]	EXPLANATORY COMMENT	OTHER INTERNATIONAL OR NATIONAL RECOMMENDATIONS
Salt	Less than 5 g of salt (2 g of sodium) per day	Higher salt intake is causally related to higher blood pressure, and a small and sustained reduction in salt intake causes a fall in blood pressure in almost everyone across the whole range of blood pressure (although there will be individual variation in the extent of the response). High blood pressure contributes to strokes and heart attacks and a fall in blood pressure is associated with their reduction (and the effect is related to the size of the fall in blood pressure). Natural experiments in different countries, direct experiments in primates, migration studies in humans, results from most prospective cohort studies in human populations and some randomised clinical trials support the concept that a moderate reduction in salt intake in the population would result in a reduction of strokes and heart attacks. Studies that suggested lower salt intakes might be associated with increased risk of CVD events suffer from measurement errors that would introduce fatal biases (errors) in the results and, hence, erroneous results (See Chapter 2.3.4). Well-conducted prospective studies – with sufficient statistical power and in which sodium excretion is accurately measured and where the study population does not include people who are already unwell – support a graded, positive and linear relationship between sodium intake and both CVD and all-cause mortality. This is a **specific target** goal, directly related to cardiovascular outcomes.	The proposed target is in line with the World Health Organization guideline of less than 5 g of salt (2 g of sodium) per day[25] and with the global NCD target of achieving a 30% reduction in consumption by 2025.[26]

Notes to Tables 4 and 5:

E%: When the goals are expressed as a percentage of food energy (E%), this represents the proportion of the total calorie intake from all food and drink consumed **excluding alcohol**.

Protein: Although a goal for protein intakes is not necessary in relation to the prevention of CVD, a balance of fat, carbohydrate and protein is important. WHO and FAO recommend from 10% up to 20% of dietary energy should come from protein of reasonable quality.[27]

Total energy (calories): Intake should be adequate to support growth and development, as well as physical activities, and to reach and maintain desirable body weight and **micronutrient intakes** should be adequate to ensure health, according to existing recommendations for different population groups.

Saturated fat: There is considerable media interest in whether some types of saturated fat – such as dairy fat or coconut oil – are less 'unhealthy' than others. Scientists have been exploring the associations between specific individual saturated fatty acids and health outcomes. There is not enough evidence to justify population goals for individual fatty acids, because most of the significant sources of saturated fat compose of fatty acids of various chain length.

The odd-chain fatty acids pentadecanoic acid (C15:0) and heptadecanoic acid (C17:0) are often thought to be biomarkers of dairy fat intakes. The levels of these fatty acids in the

blood appear to be inversely associated with the risk of CVD and diabetes. There is evidence, however, that these two fatty acids may not be specific biomarkers of dairy fat – levels of pentadecanoic acid and heptadecanoic acid are not always clearly associated with dairy fat intakes [28,29] and may be associated with intake of fat from fish[28], other type of animal origin[29] or dietary fibre intake.[30] It also remains important, as with saturated fat in general, to define which other nutrients are used to replace dairy fat and coconut oil in the diet.

Breastfeeding: No population goal for breastmilk or breastfeeding is included in the table. WHO recommends exclusive breastfeeding for six months followed by complementary feeding and continued breastfeeding for up to two years or beyond, and countries in the European region have signed up to WHO's global target to increase rates of exclusive breastfeeding for six months up to at least 50% by 2025. Some national authorities in Europe advise that complementary feeding can sometimes be introduced at four months while acknowledging the importance of ideally complying with the WHO guideline.

Water: Although it is not included in the tables, water is essential for adequate hydration of the body and an adequate water intake is vital. Estimated requirements 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.[31] 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, and are more critical for children and older people. 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: No population goal is proposed for folate from food. EHN's Expert Group considers that inclusion of any recommendations for particular foods specifically because of their folate content or for folic supplements is not warranted for CVD prevention. Folate intake should be adequate to ensure health, according to existing recommendations for different population groups. Optimal B-vitamin status can be achieved with a heart healthy diet that includes leafy green vegetables, whole grain foods, lean meat and low-fat dairy products.

Antioxidants and polyphenols: No population goal is proposed for antioxidants and polyphenols. EHN's Nutrition Expert Group considers that a cardiovascular health-promoting diet provides abundant antioxidants and confirmed its earlier conclusion that there is not currently sufficient conclusive evidence to justify making any public health recommendation for particular foods specifically because of their content of antioxidants or polyphenols. In particular, EHN does not recommend taking supplements because there is evidence of no benefit and at high levels there is evidence of harm. Supplements are no remedy for a poor diet.

Phytosterols (plant sterols and stanols): EHN does not propose a population goal for phytosterols (plant sterols and stanols), because these are only meant for people with high blood cholesterol levels. The 2016 European Guidelines on cardiovascular disease prevention in clinical practice conclude that functional foods containing phytosterols are effective in lowering LDL-C levels by an average of 10% when consumed in amounts of 2 g/day and that the cholesterol-lowering effect is in addition to that obtained with a low saturated fat/high fibre diet or use of statins.[14]

Alcohol: Consumption of three or more alcoholic drinks (10 g of alcohol in a drink[v]) per day is associated with increased CVD risk. Moderate alcohol consumption (one or two drinks per day) has been associated with a lower risk of CVD than in people who drink no alcohol at all, but the possibility for confounding cannot be excluded. Furthermore, recent research has shed doubt on this association (with non-drinkers having the lowest risks for cardiovascular outcomes). We cannot conclude from this that alcohol is protective and, therefore, cannot recommend that people consume alcohol for cardiovascular benefit. Some national guidelines (e.g. Denmark, Estonia) state that there is no safe level of alcohol consumption on the basis of increased risk of cancer and other conditions.[vi]

Pulses: Regular consumption of dietary pulses (the dried seeds from the legume family such as beans, chickpeas, lentils and peas) is recommended by some authorities and pulses are a very important component of the diet for vegetarians and vegans. Pulses have a higher protein content than cereals, are rich in calcium, iron, zinc, folate and pro-vitamin A and are more affordable than meat and dairy protein sources. They are a significant source of dietary fibre and have a low glycaemic index. There is some emerging evidence that daily consumption of a 130 g serving of pulses can reduce LDL cholesterol levels[32] and that higher pulse intakes are associated with lower risk of coronary heart disease, reduced blood pressure and obesity.[33]

Colonic flora: A cardiovascular health-promoting diet will include plentiful dietary fibre. While there is a lot of interest in colonic flora (microorganisms in the gut) and the possible implications for nutrition, EHN's Nutrition Expert Group considers the emerging data is not yet complete enough to make any firm recommendations.

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v The official definitions of how much alcohol is in a 'drink' or a 'unit' vary between countries. The definition in this paper is 10 g of alcohol (ethanol).

vi http://www.iard.org/policy-tables/drinking-guidelines-general-population/

2.3 New and emerging evidence

Diet and health topics continue to make headline news on a recurring basis and a number of nutrition 'controversies' have received considerable media coverage in recent years. This section addresses some of those issues in more detail, in order both to inform the proposed population goals and to provide a resource for EHN members who are regularly asked to comment on such topics.

2.3.1 Controversies in analyses of diet and the risk of cardiovascular disease

Controversy about dietary recommendations often stems from a limited understanding, or misrepresentation, of the science of associations between diet and health outcomes. The following sections unpick those issues in relation to sugars, fat and salt. To help explain those specific issues, more general points and underlying principles are set out here.

The strongest evidence comes from systematic reviews of well-conducted randomised controlled trials (RCTs), in which the effects of the replacement of one dietary component with another have been assessed. Non-randomised RCTs, cohort studies, case-control studies and cross-sectional studies are weaker and involve less direct evidence, as explained by the Scottish Intercollegiate Guidelines Network (SIGN), (see sign.ac.uk), the Center for Nutrition Policy and Promotion (CNPP) (see cnpp.usda.gov) and the National Institute for Health and Care Excellence (NICE) (see nice.org.uk).

Observational studies, including cohort studies, simply provide some indication of an association. These are subject not only to errors in dietary methodology but also differences in individual responsiveness to different components of the diet so they are intrinsically only suggestive if they show any evidence at all. RCTs assess the direct impact of a dietary change and therefore are much more valuable. Observational studies are important in creating hypotheses for further research. They have also a role in studies examining hard endpoints, like CHD or cancer, but it is important to keep in mind the form of evidence as the studies often provide indications of only a relationship and not a direct effect.

The challenges regarding dietary data include the different types of methodology for collecting data – e.g. food records, food frequency questionnaires and dietary recalls – as well as the subsequent reference to a dietary compositional database used to estimate dietary intake of nutrients from foods. In epidemiological studies the timing of the collection of dietary data is also very significant. There are studies in which the most recent dietary intake data is decades old. Many chronic diseases take decades to develop, so long follow-up is crucial, but dietary intake should be analysed repeatedly in these studies. The database to be used for analysing dietary intake must be relevant for the population to be studied and the marked individual differences in responsiveness to the same diet in individuals within the populations still need to be borne in mind.

The use of high-quality biomarkers increases the reliability of the dietary intake data, e.g. the fatty acid composition of plasma lipids. However, there are also some irrelevant or controversial biomarkers used, e.g. the odd-chain saturated fatty acids as an indicator of dairy fat intake since they are present also in fish.[34–36] In long-term studies the changes in the overall level of risk factors and treatment practices affect the results. This is very true regarding CVD, as has become evident, for example, in Finland where cardiovascular risk factors in the general population have fallen markedly over the years.[37,38]

2.3.2 Dietary sugars and the risk of cardiovascular disease: an update

In its paper *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe*, published in 2011, the European Heart Network has set an interim population goal for consumption of sugars of less than 10% of energy from added sugars and to reduce as much as possible consumption of sugar-sweetened drinks. More ambitious longer-term goals were formulated as less than 5% of energy from added sugar and zero consumption sugar-sweetened drinks.[39]

This section reviews the recent scientific literature about the role of dietary sugars in CVD and their potential mechanism(s) since 2010. Between 2010 and 2016 a considerable number of additional cross-sectional, observational and randomised cross-over studies on the relationship between the consumption of sugars and cardiometabolic health and its underlying mechanism(s) have been published. In addition, extensive reviews and meta-analyses have been performed, some of which have served as background information for (inter)national dietary recommendations. The majority of studies concerned sugar-sweetened beverage consumption and fructose consumption, with less attention for total or added sugars and the potential difference between liquid and solid forms of sugar.

In this paper, first the recent dietary recommendations on consumption of sugars in the general population is reviewed. Next a summary of the systematic reviews and meta-analyses is presented and the results of any additional studies, which were not included in these systematic reviews. The last section is devoted to some newer evidence with respect to the mechanisms underlying the relationship between consumption of sugars and CVD risk.

2.3.2.1 Definitions

Dietary sugars are glycaemic carbohydrates and consist of all mono- and disaccharides. 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 all mono- and disaccharides combined. Sugars can occur naturally in foods or can be added. According to the European Food Safety Agency (EFSA)[23] 'added sugars' refers to sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar

preparations used as such or added during food preparation and manufacturing. Sugar alcohols (polyols) such as sorbitol, xylitol, mannitol, and lactitol, are usually not included in the term sugars, although they are partly metabolised. The World Health Organization (WHO) uses the term 'free sugars' rather than 'added sugars'.[40] Free sugars are defined as monosaccharides and disaccharides added to foods and beverages by the manufacturer, cook or consumer, and sugars naturally present in honey, syrups, fruit juices and fruit juice concentrates.[40] This definition combines non-milk extrinsic sugars and added sugars. Extrinsic sugars are naturally occurring sugars not located in the cellular structure of foods. Sugar-sweetened beverages (SSBs) are beverages containing added caloric sweeteners such as sucrose or high-fructose corn syrup (HFCS).

2.3.2.2 Dietary recommendations

Since 2010, new dietary recommendations have been published in many European countries, such as France[12], Germany[11], the Nordic countries,[21] England[22] and the Netherlands.[41] Also in the US new dietary guidelines were formulated.[42] In addition, a scientific statement about consumption of sugars in children and adolescents was been published by the American Heart Association in 2016.[13] WHO published a guideline for sugar intake in 2015.[40] A short summary of these recommendations with respect to intake of sugars is given below. It should be realised that these diet recommendations aim to maintain or improve overall health in the general population and do not specifically address CVD risk. However, apart from dental caries, most of the recommendations for consumption of sugars are based on its relationship to the risk of obesity and type 2 diabetes, which also affect CVD risk.

In France it is recommended that the consumption of sugar and foods rich in sugar such as sugar-sweetened beverages, jams, chocolate, pastries, sugar-containing desserts, ice cream[12] is limited. The German Nutrition Society recommends a reduction in the consumption of sugar-sweetened beverages.[11] In the Nordic countries the advice is to limit intake of sweetened beverages and foods with added sugars.[43] In the Netherlands the recommendation is to limit consumption of sugar-*containing* beverages, including fruit juices, as much as possible. The consumption of foods with high sugar content, such as candy, cookies and pastries, should also be limited.[41] In England the recommendation is to limit free sugar intake to less than 5% of average energy intake for age groups from 2 years upward and to minimise SSB consumption in children and adults.[22] It was estimated that a reduction of free sugar intake to 5% of energy intake would lead to a moderately lower weight in the majority of the population.[22] WHO proposes that all individuals should consume less than 10% and in appropriate national circumstances less than 5%.[24] The latter recommendation (<5%) is based on the prevention of dental caries. WHO recommends this reduced intake of free sugars throughout the life course.[24] In the US, the Dietary Guidelines Advisory Committee concluded that a healthy dietary pattern is low in sugar-sweetened foods and drinks.[44] The American Heart Association (AHA) concluded in 2009 that a prudent upper limit of consumption of sugars would be half of the discretionary calorie allowance that can be accommodated within the appropriate energy intake level of an individual to achieve or maintain a healthy weight. Thus, most American women should eat or drink no more than 100 kcal per day (or ≈ six teaspoons) from added sugars, and most American men should eat or drink no more than 150 kcal per day from added sugars[45] which in practice means no more than about 5% of energy intake. In its 2016 statement on added sugars and CVD risk in children and adolescents, the AHA recommends that children and adolescents limit their intake of SSBs to one or fewer 8-oz (=237 ml) beverages per week and states that it is reasonable to recommend that children and adolescents consume ≤ 25 g (100 kcal) of added sugars per day which is again a limit of about 10% in younger children and about 5% in adolescents.[13]

Although some of the recommendations above do not specify upper limits of intake of sugars, they do recommend maintenance of a healthy weight. In the context of a nutritionally balanced diet there is little room for energy-dense foods and drinks, containing a high sugar or fat content, if a healthy weight is to be maintained. This is the background for the quantitative advice given. However, it has also been argued that restricting the intake of added sugar and advocating increased fruit and vegetable consumption, as in most recommendations, may not alter total sugar intake.[46]

2.3.2.3 Evidence for an association between sugars and cardiovascular disease

Different aspects of the association between consumption of sugars and CVD have been studied and reviewed over the past six years. Here only outcomes based on prospective cohorts and RCTs are discussed; cross-sectional studies are not included. In addition, the results of prospective studies should be interpreted with care, because they may be subject to reporting bias, and residual confounding. While the strongest evidence is usually obtained from well-designed and conducted RCTs, these also have their limitations. RCTs are usually of relatively short duration, include small numbers of participants with varying baseline characteristics and may also suffer from compliance issues. When evaluating the evidence relating to consumption of sugars with CVD mortality and risk factors, which is reviewed below, these limitations should be kept in mind.

2.3.2.4 Sugars and cardiovascular disease mortality

Few studies on the association between consumption of sugars and CVD mortality are available. Paganini-Hill and colleagues analysed the association between SSB consumption and all-cause mortality in the Leisure World Cohort Study including 13 624 elderly participants and 11 386 deaths.[47] The relative risk (RR) was 1.02 (95% confidence interval (CI) 0.92, 1.13) for cola and 1.03 (95% CI 0.92, 1.16) for other sugar-sweetened soft drinks, comparing >1 can/week with no consumption. Tasevska and colleagues investigated the association between consumption of sugars and CVD mortality in the NIH-AARP Diet and Health Study, a cohort of US adults aged 51–70 years at baseline.[48] The cohort included 353 751 participants and 10 894 subsequent cardiovascular events. No significant

associations of total sugars, added sugars, total fructose, added fructose, total sucrose and added sucrose with CVD mortality were found. However, when consumption of sugars was divided in solid and liquid sources, there was evidence that sugars from beverages, and especially fructose from beverages, were associated with an increased CVD mortality (Hazard Ratio (HR) 1.14 (95% CI 1.02, 1.28) in women and HR 1.13 (95% CI 1.05, 1.22) in men when comparing the lowest with the highest quintile of intake). Yang et al analysed the association between added sugars and CVD mortality in the NHANES III cohort, a nationally representative sample of US adults, with 11 733 participants and 831 CVD deaths.[49] The adjusted HR was 2.03 (95% CI 1.26, 3.27) comparing the highest with the lowest quintile of consumption of added sugars. HRs were 1.30 (95% CI 1.09, 1.55) and 2.75 (95% CI 1.40, 5.42), respectively, when participants who consumed 10.0% to 24.9% or 25.0% or more calories from added sugars were compared with those who consumed less than 10.0% of calories from added sugars.

Summary on sugars and cardiovascular disease mortality

Although the latter two studies may be suggestive of an association between consumption of sugars and CVD mortality, the available evidence is currently too limited and still too heterogeneous to draw conclusions about the causal relationship of such an association.

2.3.2.5 Sugars and coronary heart disease

In 2012, Hauner et al reviewed the literature on the association between consumption of sugars and CHD.[11] They concluded that, due to the low number of studies, there was insufficient evidence for a significant association between the intake of mono- and disaccharides or the consumption of sugar-sweetened beverages and the risk of CHD. A systematic review by Sonestedt and colleagues,[50] which informed the Nordic Nutrition Recommendations, and the report of the SACN (2012)[22] committee in the UK, came to similar conclusions. Subsequently, Huang et al[51] in 2014 and Xi et al[52] in 2015 both performed a systematic review and meta-analysis of prospective cohort studies on the association between SSB consumption and the risk of CHD. Both included four studies with 194 664 participants and 7 396 cases. Xi and colleagues used a fixed effect model to estimate the relative risk of CHD, which is unlikely to be correct. Huang et al used a more appropriate random effects model. Comparing the lowest with the highest category of SSB consumption in each study cohort a RR of 1.17 (95% CI 1.07, 1.28) was found without significant heterogeneity between studies ($I^2 = 0\%$). The risk of CHD increased by 16% with each additional serving/day of SSBs.[51] Additionally, in 2015, Li and colleagues analysed data from the Nurses Health Study and the Health Professionals Follow-up Study (127 536 participants, 7 667 incident cases of CHD) and found that intake of carbohydrates from refined starches and added sugars was positively associated with the risk of CHD (HR 1.10, 95% CI 1.00, 1.21).[53] This category mainly included foods with relatively high glycaemic index, such as potatoes, refined grains and added sugars from beverages and foods.

Summary on sugars and coronary heart disease

The number of studies on the association between incident CHD and consumption of sugars is small (n=5). Moreover, they mainly focus on SSB consumption and studies on the association between consumption of added sugars and CHD risk are currently lacking. More studies are clearly needed before a reliable conclusion on the association between free or added sugars and CHD can be made.

2.3.2.6 Sugars and stroke

Xi and colleagues also did a meta-analysis of studies on the association between SSB consumption and stroke.[52] Six prospective cohorts with 259 176 participants and 10 011 cases of stroke were included. Using an inappropriate fixed effect model, the highest intake of SSB was marginally associated with the risk of total stroke (RR 1.10, 95% CI 1.00, 1.20) compared with the lowest level, with little evidence of heterogeneity ($I^2 = 43\ \%$). With a more appropriate mixed effects model the association would probably not have been significant.

Summary on sugars and stroke

So far there is no evidence that higher SSB consumption increases the risk of stroke. There are no studies on the association between total consumption of sugars and stroke.

2.3.2.7 Sugars and hypertension

Hypertension is a major risk factor for CVD and the effects of sugars on hypertension has been the topic of cohort studies and RCTs.

Systematic reviews and meta-analyses

Four meta-analyses[22,52,54,55] and four systematic reviews[11,50,56,57] have been performed on dietary sugars and blood pressure and/or risk of incident hypertension. The reviews by Hauner et al[11] and Sonestedt et al,[50] both in 2012, included four studies and both reported no significant association between consumption of sugars and risk of hypertension.

In 2014 Keller and colleagues included five prospective cohorts and one RCT and concluded that there was a direct association between SSB consumption and a change in blood pressure.[57] The 2014 review by Malik et al included six prospective cohort studies and six cross-sectional studies with a total of 409 707 participants.[56] The conclusion of this review was also that SSB consumption was positively associated with blood pressure and risk of hypertension.

In 2015 Jayalath and colleagues meta-analysed three studies on five prospective cohorts (240 508 participants and 79 251 cases) with respect to SSB consumption and risk of hypertension.[55] The RR was 1.12 (95% CI 1.06, 1.17; $I^2 = 62\%$) comparing the highest quantile (≥ one serving/d) with the lowest (none). The authors indicated that collinearity of SSB consumption with other components of a Western diet could not be excluded. The meta-analysis by Xi and

colleagues included one additional cohort and used a different analysis of the SUN cohorts (incident hypertension instead of the metabolic syndrome criteria for blood pressure).[52] In total 259 176 participants and 10 011 cases were included. The highest intake of SSB was positively associated with the risk of hypertension (RR 1.10, 95% CI 1.06, 1.15; $I^2 = 47$ %) compared with the lowest level of intake.

Randomised clinical trials on consumption of sugars and blood pressure were reviewed by the Scientific Advisory Committee on Nutrition (SACN) in the UK in 2012)[22], Ha et al in 2012[58] and Te Morenga and colleagues in 2014.[54] The meta-analysis of SACN was based on three RCTs and concluded that there was no evidence for an effect of increased consumption of sugars on systolic blood pressure (SBP) or diastolic blood pressure (DBP).[22] The systematic review and meta-analysis by Ha specifically looked at the effect of fructose consumption on BP in RCTs. Thirteen isocaloric (n = 352) trials were included. Fructose intake in isocaloric exchange for other carbohydrates significantly decreased DBP (mean difference (MD) -1.54 (95% CI -2.77, -0.32; $I^2 = 47\%$) and mean arterial pressure (MD -1.16 (95% CI -2.15, -0.18; $I^2 = 97\%$). There was no significant effect of fructose on systolic blood pressure (MD -1.10 (95% CI -2.46, 0.44; $I^2 = 31\%$).[58] Te Morenga et al included 12 trials with 324 participants in their meta-analysis. Overall no effect of increased intake of sugars on SBP was found (MD 1.1 mm Hg (95% CI -1.0, 3.2; $I^2 = 67\%$), but there was a significant effect on DBP (MD 1.4 mm Hg (95% CI: 0.3, 2.5; $I^2 = 41\%$). When only trials with a duration of 8 weeks or longer were included, the effects were more pronounced (for SBP 6.9 mm Hg (95% CI 3.4,10.3) and for DBP 5.6 mm Hg (95% CI 2.5, 8.8). Studies funded by the sugar industry (n=5) generally reported less pronounced effects of sugar on blood pressure and excluding these studies from the analysis strengthened the hypertensive effects of higher sugar intakes.[54]

Additional studies

Several additional studies, not included in the reviews summarised above, were identified. In 2010 Perez-Poso and colleagues studied the effect of adding a high dose of 200 g fructose/d in liquid form to the habitual diet but avoiding SSBs in 74 normotensive individuals during two weeks.[59] Ambulatory SBP increased by 7 ± 2 mmHg and DBP by 5 ± 2 mmHg (P = 0.004 and 0.007, respectively). Body weight increased by 0.6 ± 0.2 kg, P = 0.003. In an RCT by Teunissen-Beekman et al in 2012 94 overweight participants were randomised to a group consuming three glucose (maltodextrin)-containing drinks (60g glucose/d) or three isocaloric protein drinks/day during four weeks.[60] Body weight did not change during the intervention in either group. After four weeks SBP and DBP measured in the clinic/office were 4.9 ±1.7 mmHg (P = 0.005) and 2.7 ± 1.3 mmHg (P = 0.05) higher in the glucose group. Ambulatory daytime SBP was also 4.6 ± 1.7 mm Hg higher in the glucose group (P = 0.006), whereas ambulatory daytime DBP did not differ between groups (P = 0.37). Lowndes and colleagues performed a randomised controlled trial in 2014 in which 65 overweight and obese individuals were placed on a eucaloric (weight stable) diet for 10-weeks with sucrose- or HFCS-sweetened, low-fat milk at 10% or 20% of calories.[61] Blood pressure did not change and no differences in blood pressure changes were found among groups. In 2015 Raatz and colleagues compared the effects of 50 g/d of honey, sucrose or HFCS for two weeks in 55 adults.[62] SBP was unchanged, whereas DBP was lowered, but there were no significant differences among treatments. Lustig et al, in 2016, studied the effect of reducing consumption of sugars from the habitual 28% of total energy intake to 10% by starch substitution during nine days in 43 children.[63] No comparison with a control treatment was included in this study. SBP did not change (-1.4 mmHg (95% CI -4.9, 2.1)) over the 10 days, DBP decreased significantly by 4.9 mmHg (95% CI -8.1, -1.8). Blood pressure changes were adjusted for the reduction in weight of 0.9 ± 0.2 kg (P<0.001) that occurred over the 10-day intervention. Given the time course of the weight change it was considered unlikely that the children were in a negative energy balance at the time of the post-intervention measurements. However, since no control group was included, these results should be interpreted with care.

Summary on sugars and hypertension

Overall, the results seem to suggest that the risk of hypertension may increase with increased long term consumption of SSBs. RCTs on the BP effect of changes in consumption of sugars suggest that lowering intake of sugars may lower blood pressure, especially when maintained over a longer period of time, but the effect size appears highly variable and will need to be further explained. The association is not nearly as clear-cut as the relationship between salt intakes and blood pressure (See Chapter 2.3.2).

2.3.2.8 Sugars and type 2 diabetes

Type 2 diabetes is an important risk factor for CVD and the association between sugars and type 2 diabetes has been addressed in a number of studies.

Systematic reviews and meta-analyses

Three systematic reviews and meta-analyses on the association between consumption of sugars and risk of type 2 diabetes haven been published since 2010.[64–66] All three focus on the role of SSBs. The review by Malik and colleagues included eight prospective cohort studies with a total of 310 819 participants and 15 043 subsequent cases of incident type 2 diabetes.[64] A RR of 1.26 (95% CI 1.12, 1.41; $I^2 = 66\%$) comparing the highest (most often one to two servings per day) with the lowest intake category (most often none or less than one serving/month) in each study was found. A serving was defined as a can of 330 ml. Greenwood et al[65] did a meta-analysis in 2014 of six prospective studies, only partly overlapping with those reviewed by Malik in 2010. The RR from the linear dose–response meta-analysis was 1·20 (95% CI 1.12, 1.29) per 330 ml/d of SSBs with substantial heterogeneity between the cohorts ($I^2 = 80$ %). The last meta-

analysis by Imamura and colleagues in 2015 was based on 17 prospective cohorts (38 253 cases/10 126 754 person years).[66] The analysis showed a RR of 1.18 (95% CI 1.08, 1.28; I^2 = 89%) per serving/day of SSB. After adjustment for differences in adiposity, the association was attenuated, RR 1.13 (95% CI 1.06, 1.21; I^2=79%) per serving/day of SSB. Various sensitivity analyses supported the positive association between SSBs and type 2 diabetes. However, the association of fruit juice consumption with incident type 2 diabetes, based on 13 prospective cohorts, was not significant (RR 1.05 (95% CI 0.99, 1.11) per serving/day of fruit juice; I^2=58 %), and the RR estimate was unstable in sensitivity analyses.

Additional studies

One additional study investigated the association between intake of different types of sugars and the risk of type 2 diabetes in the EPIC-Norfolk cohort involving 749 individuals with diabetes compared with a randomly selected sub-cohort of 3 496 participants aged 40–79 years.[67] Dietary intakes of total carbohydrates, starch, sucrose, lactose or maltose were not significantly related to diabetes risk after adjustment for confounders. After additional adjustment for energy intake, however, higher intakes of fructose were inversely associated with incident type 2 diabetes (HR 0.88 (95% CI 0.78, 0.99).[67]

Summary on sugars and type 2 diabetes

The meta-analyses on the association between SSB consumption and risk of incident type 2 diabetes suggest a 20–25% increase in risk per serving (around 350 ml) per day of SSB, which seems to be at least partially mediated by increased adiposity. The only study that looked at total consumption of sugars and its different components, on the other hand, found an inverse association between a higher consumption of fructose (as percentage of total energy intake) and the risk of incident type 2 diabetes. It is currently unclear how these findings can be reconciled.

2.3.2.9 Sugars and body weight or adiposity

Obesity is an important risk factor for hypertension, type 2 diabetes and CVD. Over the last five years a considerable number of new studies on the association between sugars and obesity have been published.

Systematic reviews and meta-analyses on added sugars

In a review of the scientific literature published in 2012, underpinning the German recommendations on consumption of sugars, Hauner et al concluded that there was insufficient evidence for a role of sucrose or added sugars in increasing the risk of obesity in adults and children.[11] The association between SSBs and obesity risk was considered possible, especially in overweight children and adolescents. In the UK SACN concluded in 2012 that studies provided conflicting evidence concerning the relationship between sweetened beverages and BMI, with US studies tending to find small but positive associations and European studies tending to report no evidence of a statistical association.[22] In the same year Te Morenga and colleagues published a systematic review and meta-analysis on intake of free sugars and its relationship with adiposity in children and adults.[68] This review was commissioned by WHO and was the basis for the WHO recommendation on sugars. Exposure was based on total consumption of free sugars, but also on specific sources such as sucrose, glucose, fructose, or sugar-sweetened beverages. In five randomised trials of adults without strict control of energy intake (ad libitum) a reduced intake of dietary free sugars was associated with a decrease in body weight (MD 0.80 kg, 95% CI 0.39,1.21) compared with no change in free consumption of sugars, with no evidence of heterogeneity (I^2=17%). Increased intake of dietary free sugars in the context of an ad libitum diet was associated with significantly greater weight gain (MD 0.75 kg (95% CI 0.30, 1.19)) compared with no change in consumption of free sugars in adults. Heterogeneity among studies was significant (I^2 =82%). This analysis was based on 10 studies. In contrast, when free sugars were (partly) replaced by isocaloric amounts of other carbohydrate or other macronutrient sources (12 studies), there was no evidence for a change in body weight (MD 0.04 kg (95% CI -0.04, 0.13; P=0.3) (I^2=32%).[68] Overall, Te Morenga concluded that in the context of ad libitum diets, higher intake of free sugars or SSBs is associated with higher body weight. When energy intakes are controlled then the effect is negligible implying that, the effects of sugar appear to be mediated by changes in energy intake and that there is no selective fructose effect, because isocaloric exchanges are ineffective.[68] The same conclusion was drawn in a systematic review and meta-analysis on the association between fructose intake and body weight by Sievenpiper and colleagues in 2012.[69]

Additional studies on added sugars

Two additional prospective cohort studies, not included in the review by Te Morenga were identified. Pollock et al in 2012 investigated the association between fructose consumption and visceral and subcutaneous abdominal fat mass in 559 adolescents.[70] After adjustment for total fat mass and other relevant factors, fructose consumption was positively associated with visceral fat mass, but not with subcutaneous abdominal fat mass. Lee and colleagues in 2015 analysed the effects of an increase in consumption of added sugars on changes in waist circumference (WC) and BMI z-score in 9 to 10-year old girls over one year.[71] Each additional 4 g/day of liquid added sugars was associated with a 0.22 mm increase in WC (P < 0.001) and a 0.002 increase in BMI z-score (P = 0.003). Each 4 g/day of solid added sugar was associated with a 0.13 mm increase in WC (P = 0.03) and a 0.001 increase in BMI z-score (P = 0.03). There was no association between an increase in naturally occurring sugars and changes in BMI z-score or waist circumference.[71]

Systematic reviews and meta-analyses on SSBs

The specific role of consumption of sugar-sweetened beverages has been the subject of a number of additional systematic reviews and meta-analyses[72–76] since 2010. In addition two reviews of reviews on this topic have been published.[77,78]

The systematic review by Woodward-Lopez et al in 2010 included 24 prospective cohorts: 16 showed a positive association between SSB intake and body weight and eight showed no association.[72] In addition, five RCTs on increased SSB intake and four on reduced SSB intake were reviewed. The results of studies on the reduction in SSB intake were equivocal, but those on the increase in SSBs were all positive, i.e. increased consumption led to increased body weight. The results of this review were largely confirmed in the meta-analysis of Malik and colleagues in 2013.[74] Malik included 15 prospective cohorts in children (174 252 children) and seven in adults (25 745 adults). In children the BMI change was 0.06 kg/m^2 (95% CI 0.02, 0.10) per one daily serving increment of SSBs with significant heterogeneity among studies; in adults the weight change was 0.12 kg (95% CI 0.10, 0.14) per one daily serving increment of SSBs also with significant heterogeneity. Malik et al also did a meta-analysis of five RCTs in children (2 772 children) on the effect of reducing SSB consumption and of five RCTs in adults (292 adults) on the effect of increasing SSB intake. In children there was a non-significant BMI change of -0.17 (95% CI -0.39, 0.05) per one daily serving reduction of SSBs with significant heterogeneity among studies. More BMI reduction was found in substitution studies than in studies with an education approach. Noteworthy is the result of the DRINK study, in which masked replacement of a can of SSB (104 kcal) with a sugar-free beverage per day over 18 months led to a one kg smaller increase in weight among 11–12 year old school children.[79] In adults a weight change of 0.85 kg (95% CI 0.50, 1.20) per one daily serving increment of SSBs was found with no significant heterogeneity among studies. Based on these analyses Malik concluded that SSB consumption promotes weight gain in children and adults.[74]

Another meta-analysis, only including RCTs, was performed initially by Mattes et al in 2011[73] and then updated by Kaiser and colleagues in 2013.[75] The latter meta-analysis included seven RCTs on increased and eight on reduced SSB intake. Of these, seven were not included in the meta-analysis by Malik.[74] Conversely, two trials in the meta-analysis by Malik were not included by Kaiser. The trials on increased intake showed a standardised mean difference (SMD) of 0.28 (95% CI 0.12, 0.44; I^2 = 48%), favouring an increase in weight. The studies on reduced intake of SSBs showed a non-significant SMD of 0.06 (95% CI -0.01, 0.13; I^2 = 59%). When only studies in overweight or obese individuals were included, the SMD became statistically significant (SMD 0.25 (95% CI 0.13, 0.38; I^2 = 49%)). Based on their meta-analysis Kaiser et al concluded that, although the evidence suggests that a beneficial effect of a reduction in SSB may be demonstrable in some populations, the effect size is small and of equivocal statistical significance.[75] In 2015 a systematic review on SSBs and obesity risk was published by Pereira.[76] Twelve prospective cohorts and five RCTs on reduction of SSB consumption in children were included. Pereira concluded that the totality of evidence points to an increased risk of weight gain with higher SSB consumption, but that the heterogeneity among studies and the methodological limitations of both observational and experimental studies makes it difficult to establish the strength of the association.

Additional studies on SSBs

Some additional studies not included in the systematic reviews and meta-analyses summarised above were identified. An RCT by Lowndes et al in 2014 confirms that increased sugar-containing beverage consumption leads to significant weight gain (approximately 1 kg over 10 weeks) in adults, independent of the type of sugar (HFCS, sucrose, lactose).[80] In the prospective Framingham Third Generation cohort with 1003 participants, higher SSB intake at baseline was not associated with change in BMI (P_{trend} = 0.87), but it was associated with higher visceral adipose tissue volume at 6-y follow-up (P_{trend} <0.001) after adjustment for multiple confounders including change in body weight.[81] Massougbodji and colleagues in 2014 investigated whether the scientific quality and other study characteristics were associated with conclusions of reviews on the causal relation between SSBs and body weight.[77] The investigators included five meta-analyses, three systematic reviews and 12 non-systematic reviews. Scientific quality scores were unrelated to conclusions, but industry-funded reviews (n=4) were more likely to suggest that the evidence for a causal relation was weak in contrast to the other reviews that generally considered the evidence to be well-founded.

In 2015 Keller and colleagues also published a review of reviews on the relationship between SSBs and adiposity in children and adolescents.[78] They included 13 reviews and meta-analyses. Nine reviews concluded that there was a direct relation between SSB consumption and weight gain, overweight, and obesity; four did not. They indicated that even the two meta-analyses with the highest scientific quality score (by Malik et al[74] and Kaiser et al,[75] see above) came to discrepant conclusions, which could be related to the inclusion criteria applied but also to the funding source of the authors (non-industry and industry respectively).[77]

Summary on sugars and adiposity

The reviews and meta-analyses on the effect of sugars on adiposity in majority conclude that adiposity increases with increasing consumption of sugars or SSBs. The effects of a reduction in SSB consumption in RCTs in children are heterogeneous, which may be related to the intervention method (education or provision of beverages). No RCT data on the effect of a reduction in SSB or sugars consumption on body weight or adiposity as such in adults are available. In isocaloric exchange studies no effects of reducing or increasing consumption of sugars on adiposity are seen, suggesting that the association is mediated by increased energy intake.

2.3.2.10 Sugars and fatty liver

A recent meta-analysis has suggested that non-alcoholic fatty liver disease (NAFLD) is associated with an increased risk of incident cardiovascular disease.[82] In reviews by Tappy and Le in 2012[83] and Vos et al in 2013,[84] the potential contribution of dietary fructose consumption to the development of NAFLD was discussed. Tappy and Le concluded that short-term fructose overfeeding may increase hepatic triglycerides without reaching the levels seen in

NAFLD. Vos et al concluded that fructose is associated with increasing hepatic fat, inflammation, and possibly fibrosis in humans. However, whether fructose alone can cause NAFLD or only when consumed excessively in the setting of insulin resistance, a positive energy balance, and a sedentary lifestyle is unclear.[84] In a recent four week RCT by Jin et al 24 overweight adolescents who had hepatic fat >8% and who were regular consumers of sweet beverages were randomised to isocaloric fructose only or glucose only beverages.[85] After four weeks, there was no significant change in hepatic fat or body weight in either group.

Currently there is therefore not enough evidence to draw conclusions about the association between consumption of sugars and NAFLD.

2.3.2.11 Sugars and lipids

Increased plasma concentrations of lipids and lipoproteins are associated with increased cardiovascular disease risk.[86] Several recent systematic reviews and meta-analyses have addressed the potential association between sugars and plasma lipids.

Systematic reviews and meta-analyses

In 2012 Hauner et al concluded that the results of studies on the association between the intake of sugars and LDL-cholesterol and HDL-cholesterol concentration were inconsistent.[11] They also concluded that an association between fructose intakes < 100 g/d and plasma triglycerides was improbable, but that higher intakes increase plasma triglycerides. Sonestedt and colleagues in 2012 concluded that too few studies were available for a conclusion.[50] SACN reported no effect of consumption of sugars on total, HDL- or LDL-cholesterol or triglycerides. Subsequently, two meta-analyses have reported on the association between consumption of sugars and blood lipids.[54,87]

Te Morenga and colleagues in 2014[54] undertook a meta-analysis of 37 RCTs comparing the effect of higher with lower levels of free consumption of sugars on various blood lipid parameters. Higher compared with lower free sugar intakes significantly raised triglyceride concentrations (MD 0.11 mmol/L (95% CI 0.07, 0.15; I^2 = 73%), total cholesterol (MD 0.16 mmol/L (95% CI 0.10, 0.24; I^2 = 74%), low-density lipoprotein cholesterol (MD 0.12 mmol/L (95% CI 0.05, 0.19; I^2 = 73%), and high-density lipoprotein cholesterol (MD 0.02 mmol/L (95% CI 0.00, 0.03; I^2 = 36%). The effects were most pronounced in isocaloric exchange studies.[54] These findings are in contrast with findings of two previous systematic reviews and meta-analyses that specifically examined the effects of fructose consumption compared with consumption of other carbohydrates.[88,89] A more recent meta-analysis on this topic by Chiavaroli et al in 2015 included RCTs of at least one week duration that studied the effect of fructose consumption compared to a control carbohydrate on lipids.[87] Fifty-one isocaloric trials (n=943), in which fructose was provided in isocaloric exchange for other carbohydrates, and eight hypercaloric trials (n=125), in which the fructose was supplemented in excess, were included. Fructose had no effect on LDL-cholesterol, non-HDL-cholesterol, triglycerides, or HDL-cholesterol in isocaloric trials. However, in hypercaloric trials fructose raised triglycerides (MD 0.26 mmol/L (95% CI 0.11, 0.41; I^2 = 66%). These results suggest that the increase in triglycerides in the hypercaloric studies depended on the energy surplus when feeding fructose-containing diets. Furthermore, the investigators considered that the trials were limited by short follow-ups and low quality scores.[87]

Additional studies

In 2014 Lowndes and colleagues published the results of an RCT in which intakes of isocaloric amounts of HFCS, sucrose and placebo during 10 weeks were compared in 65 overweight individuals.[61] The type of sugar did not affect the lipid response to the diets. Lustig and colleagues in 2016 studied the effect of reducing consumption of sugars from the habitual 28% of total energy intake to 10% by starch substitution during nine days in a non-controlled trial in 43 children.[63] Fasting triglycerides were reduced from 1.4 ± 0.9 to 1.0 ± 0.5 mmol/L (P=0.002), LDL from 2.4 ± 0.6 to 2.1 ± 0.6 mmol/L (P=0.003) and plasma HDL from 1.2 ± 0.2 to 1.0 ± 0.2 mmol/L (P<0.001). When adjusted for weight loss, these changes were no longer significant. Given the fact that no control group was included, these results should be interpreted with care.

Summary on sugars and lipids

The studies on the effect of consumption of sugars on lipids show highly variable outcomes and currently no clear conclusions can be drawn. It also remains unclear whether any effects are mediated by changes in adiposity, since one meta-analysis suggests that the plasma lipid increasing effects are mainly seen in studies in which energy balance is maintained, whereas the other meta-analysis concludes that the effects are due to increased energy intake rather than the sugars (fructose) consumption per se. High intakes of fructose can be associated with increased triglyceride concentrations, but the relevance of this finding for policymaking and specifying national average dietary targets or optimum goals is unclear.

2.3.2.12 Mechanisms

Several mechanisms have been suggested for the association between consumption of sugars and CVD risk. There is clear evidence that consumption of sugars and especially SSB consumption may lead to excess energy intake and gain of body mass and fat mass (see above). How much of the increased cardiovascular risk by dietary sugars is explained by adiposity and its associated cardiometabolic disturbances, such as the cluster of risk factors representing the metabolic syndrome (abdominal obesity, atherogenic dyslipidemia, raised blood pressure, insulin resistance with or without glucose intolerance, proinflammatory state and a prothrombotic state[90]), has not fully been elucidated, but other mechanisms have been suggested as well.

Rosset et al recently reviewed the potential mechanisms underlying the effects of fructose-containing sugars on CVD.[91] A major focus was on a potential role for

hyperuricemia in fructose-induced cardiovascular risk. High fructose intake induces increases in plasma uric acid levels, both acutely[92] and chronically.[93] As potential mechanisms increased synthesis of uric acid by the liver or reduced renal uric acid excretion were suggested. Hyperuricemia might induce endothelial dysfunction and indirectly insulin resistance by activating the NALP3 inflammasome. In addition, hyperuricemia may activate lipogenic enzymes. A link between elevated uric acid levels and increased subsequent rates of CVD events has been suggested and there is increasing evidence that it may be an independent risk factor.[94] Nevertheless the role of diet in promoting hyperuricaemia with the induction of subsequent cardiovascular events is still unclear.[91,95]

Hyperglycemia and high insulin levels have been suggested to lead to endothelial dysfunction.[96] Mechanisms may involve inhibition of oxidative stress-induced dysregulation of nitric oxide homeostasis and formation advanced glycation end products (AGEs).[97] Indeed, Teunissen-Beekman et al in 2015 found lower endothelial dysfunction z-scores (based on plasma concentrations of cellular adhesion molecules (ICAM, VCAM and E-selectin) and the clotting protein Von Willebrand factor) when glucose consumption (60 g/d in the form of maltodextrin) was isocalorically replaced by protein consumption for four weeks.[98] Also fructose consumption has been shown to induce expression of cellular adhesion molecules.[99] In addition, fructose may contribute to a prothrombotic state by induction of Tissue Factor, sometimes termed thromboplastin.[99]

A review by Ares and colleagues in 2015 put forward the hypothesis the fructose may interact with renal salt handling, which is important for blood pressure regulation.[100]

Finally, some recent reports suggest that polymorphisms in certain genes may influence the susceptibility of individuals to the detrimental cardiometabolic effects of SSBs.[101–103]

2.3.2.13 Conclusion

Over the past six years a lot of new information has become available on the potential association between consumption of sugars and the risk of CVD, especially on the contribution of SSBs. The effects are most evident when sugars are consumed in excess, i.e. when accompanied by weight gain. However, some weight-gain independent effects may also be present. Evidence suggests that the increased risk is mainly associated with fructose-containing sugars, but the role of non-fructose containing sugars is less well studied and has not been fully elucidated. Although study results are not always consistent, a linear dose-response relationship between intake and risk is suggested by most studies. However, confounding by other aspects of a Western diet cannot be fully excluded. Well-controlled studies on the effects of reducing the intake of sugars on cardiovascular risk are relatively scarce, with the strongest evidence for a beneficial effect of reducing SSB consumption on adiposity in children from two well-controlled RCTs and for some lowering of blood pressure. Limiting the consumption of free or added sugars, and especially SSBs, is likely to have positive effects on adiposity, although the effect will be moderate. Based on the meta-analysis of Malik and colleagues in 2013, adults who consume one serving of SSB per day have on average a 0.22 kg (95% CI 0.09, 0.34) greater increase in body weight after one year than those not consuming any SSBs.[74] Nevertheless, limiting free sugars consumption may have a beneficial, although small, impact on cardiovascular health of populations. Because the dose-response relationships so far do not suggest a lower threshold, the recommendation should be to limit free sugars consumption as much as possible in order to obtain the largest benefits. Major contributors to free sugars consumption are soft drinks including fruit juices and dairy products with added sugars, sweets, candies, cakes and cookies. Limiting consumption of such food products as much as possible should be recommended, especially in those struggling to maintain a healthy weight.

2.3.3 Fats: the role of the quality of dietary fat on atherosclerotic vascular diseases

In 2011, EHN concluded that a moderate amount of dietary fat with an emphasis on the quality of fat is significant for maintaining health and recommended the following: total fat less than 30 % of energy intake (E%), saturated fat (SFA) less than 10 E%, trans fat (TFA) less than 1 E%, polyunsaturated fat 6–11 E% including alpha-linolenic acid (ALA) 1–2 E%, and monounsaturated fatty acids 8–13 E%. This summary concentrates on the recent evidence on the field.

2.3.3.1 Introduction

Recently, there has been some debate regarding the significance of the amount and quality of dietary fat in the prevention and treatment of diseases, especially atherosclerotic vascular diseases (AVD), e.g. coronary heart disease (CHD).[vii] There are meta-analyses confirming the current dietary recommendations that decreasing the intake of SFA is of benefit with regard to the prevention of AVD,[104] whereas some controversial meta-analyses show no benefit in this regard.[105] In particular, replacing SFA partly with polyunsaturated fatty acids (PUFA) has been shown to decrease the risk of AVD.[53,104,106,107] There are recent data, that individual SFAs may have different effects on the risk of diabetes.[108] It has been suggested that SFAs from some dairy products would not increase the risk of AVD or stroke,[109] but recent data do not support this, since individual SFAs present in dairy fat have been shown to be associated with AVD.[110] The results are similar for dairy fat in general.[111]

These controversies have gained a lot of publicity in the media and even the credibility of the current dietary recommendations has been questioned in some studies or comments. It is important to understand the basis of the

vii Throughout this paper we often refer to coronary heart disease (CHD), a term that is well recognised by general audiences. In this sub-chapter the term atherosclerotic vascular disease (AVD) is used. AVD includes, but is not limited to, CHD. The term is used in this sub-chapter in order to be precise about the research findings where the research was conducted on the broader range of conditions grouped together under the term AVD.

current recommendations and understand the limitations of the controversial evidence.

2.3.3.2 The amount of fat and energy density

The evidence from many different analyses including prospective cohort studies indicates that total fat intake is not associated with the risk of AVD in Caucasian populations.[107] The original detailed analyses in Keys et al's Seven Country Study[112] made it clear that there was no relationship between the intake of total fat and the prevalence of heart disease in different communities and that the type of fatty acids in the dietary fat was the key to raising blood cholesterol concentration and thereby increasing the risk of heart disease.

With the escalating rates of obesity and diabetes with their longer-term effect in amplifying AVD rates there is a need for greater focus on the dietary factors that may promote excessive weight gain and this is where the importance of the total fat content has become apparent.

Progressive weight gain requires that the amount of dietary energy consumed exceeds the body's total use of energy both for normal body maintenance and physical activity thereby leading to an accumulation of energy in the body both as fat and lean tissue. So when physical activity declines it is necessary to reduce total food energy intake to maintain body weight. This then requires ether conscious reductions in food consumption or reliance on the recognised, but far from robust, satiety mechanisms of food intake control. Given the progressive and widespread reduction in physical activity in Europe as work and other physical demands on workers were mechanised, with computers often dominating the workplace, and with private car transport often satisfying general transport needs in much of Europe, it is perhaps not surprising that obesity has escalated over the last 30–40 years. Furthermore, with the progressive fall in food costs and the increase in the general availability of attractive food, snacks and soft drinks throughout the day backed up by intense multiple forms of marketing it is easy to argue that these factors affecting food intake may be the primary determinants of weight gain (See Chapter 3).

Given this context of multiple factors affecting energy balance and the marked daily variation in food energy consumed as well as the usually more modest change in daily exercise, discerning significant changes in intake or output relevant to weight changes is not straightforward. We also cannot measure simultaneously both food intake and energy output with sufficient accuracy to discriminate the cause of a 0.3 to 1% (i.e. a 10 to 20 kcal change) in average intake or expenditure over say a month or more to reflect the energetic equivalence of the usual 0.5 to 1.0kg per year magnitude of weight gain in adults.

Discriminating dietary factors that intrinsically affect intake therefore requires careful analysis where it is necessary to distinguish between other environmental factors that influence intake. The amount of fat in the food does tend to increase the sensory mouth feel of a food with an intake promoting effect and fat has less of an effect in inducing satiety than either protein (the most powerful inducer of satiety[113] or carbohydrate.[114] These effects suggest, therefore, that the total amount of fat may promote weight gain by permitting more calories to be consumed inadvertently. This was originally called passive overconsumption. There are now major studies with systematic reviews showing that total dietary fat does promote weight gain[115], but a series of meticulously controlled dietary studies shows that it is the high energy/gram content of fat which affects markedly the energy density of the diet[116,117] and it is the energy density of the diet which readily leads to unrecognised overeating. So the lower the physical activity level becomes, the lower the energy density of the diet needs to be to maintain energy balance and avoid weight gain. Thus, severe inactivity requires a low-fat (e.g. 20% of energy) low sugar diet to lower the energy density of the diet sufficiently to limit weight gain whereas if adults are very active e.g. cycling to work and in their daily activity then a 40% total fat (low sugar) diet can still allow energy balance to be maintained.[118] Crude analyses of national diets in relation to national obesity rates showed a clear positive relationship to a nation's dietary fat content.[119] Further trials where the subjects were again unaware of the real purpose of the assessments then showed that the key to maintaining weight and energy balance when inactive was not the total fat content *per se* but the reduction in the general energy density of the diet[116,117] and this could be achieved by lowering either the refined carbohydrate content e.g. as sugar, or by lowering the fat content but preferably both.[120] The consumption of a fibre-rich, unrefined carbohydrate, and vegetable and fruit rich diet also helps as now documented in many trials.[22] So unrecognised changes which increase the energy density of the diet proved to be an important determinant of weight gain[121] and more recently analyses of energy density in communities have also been related to the prevalence of weight gain or obesity.[122]

The choice of a level of fat intake for body weight maintenance is therefore affected both by the degree of physical activity in a community and the extent to which other factors affecting the dietary energy density are changed. Thus a high fibre, whole-grain-rich diet with vegetables and fruit together with a lower sugar intake allows a slightly higher fat intake to be consumed without weight gain Furthermore the diabetes prevention problems in Finland, which require appreciable weight loss, require a low-fat diet (as well as appreciable and defined increases in physical activity). So the total fat goal is set at below 30%[123] and in practice about 25% with an explicit need to eat far more fibre-rich food and more vegetables and fruit. The DASH diet where a lower fat intake led to a fall in blood pressure involved a reduction in fat intake from 37% to 26%[4] and Ferro–Luzzi's integrated data on the fat intake of middle-aged Italian men on the Mediterranean in the EURATOM studies[124] in the early 1960s showed their intake to be 28% which also fits with the chemical analyses undertaken on the Mediterranean diet even in Greece (28% fat) as well as in Italy (26% fat) in the 1950s by Keys and colleagues as part of the Seven Country studies.[125] About a fifth of these middle-aged men were already overweight or obese despite heavy manual work.[126]

Thus, given the data from meticulous studies on limiting both hypertension and diabetes and the linear relationship between the fat intake and the propensity to weight gain,[115] a fat intake of 25% accompanied by at least a 60% fibre rich carbohydrate diet with over 400 g/d of vegetables and fruit is consistent with an appropriate reduction in the risk of weight gain and diabetes and therefore the longer term risk of CHD. This 25% fat value is also consistent with the reduced energy density of the diet required by those who have been obese and then manage to maintain their reduced weight long term.[127,128]

2.3.3.3 The quality of fat

Risk factors

The quality of dietary fat has major impact on serum total and LDL-cholesterol concentration.[14,107] Furthermore, modern genetic studies strongly suggest that LDL-cholesterol is causally related to atherosclerosis.[129–131] Partial replacement of SFA and TFA by cis-MUFA and PUFA decreases convincingly serum LDL cholesterol concentration without affecting HDL-cholesterol concentration,[17,107,132] and the total-cholesterol to HDL-cholesterol ratio improves as well. If total fat intake is markedly reduced HDL-cholesterol concentration tends to decrease and the concentration of triglycerides tends to increase.[132] However, if a fibre-rich diet is eaten the overall lipid changes are beneficial in that total and LDL cholesterol concentrations as well as triglyceride concentrations are lower.

According to a summary by Mensink and colleagues[133] replacing 1 E% of TFA with 1 E% SFA, cis-MUFA, or PUFA decreases the total-cholesterol to HDL-cholesterol ratio by 0.31, 0.34, and 0.67 units, respectively. The effects of ruminant and industrially produced TFA do not seem to differ in their effects on serum lipid profile.[134,135]

There are data that the dietary fatty acid matrix may affect the hypercholesterolemic effect of dairy fat. Thus Tholstrup et al[136] showed a more remarkable increase in serum total and LDL-cholesterol concentrations by butter than cheese with standardised dietary amount of SFA.

Partial replacement of SFA or carbohydrates by cis-MUFA may have favourable effect on insulin sensitivity and fasting plasma insulin concentration.[107] Furthermore, recent observational studies show that the high amount of PUFA (e.g. linoleic acid) in different plasma fatty acid fractions is associated with better insulin sensitivity.[137,138]

The effect of the quality of dietary fat on blood pressure may depend on the total amount of dietary fat. In a randomised long term trial those with lower intakes of SFA from infancy had lower blood pressure.[139] However, in a study with 40 E% fat no difference between MUFA and SFA was found[140], whereas in the KANWU study a beneficial effect of MUFA on blood pressure was more pronounced in subjects with a fat intake below 37 E%.[141] However, in a recent systematic review, no conclusion could be drawn regarding the effect of the quality of dietary fat on blood pressure.[107]

One of the striking features of metabolic studies is the substantial, i.e. 4–5 fold differences, between individuals in their blood total and LDL cholesterol concentration responses to a standardised change in intake of the key SFAs. This means that although blood cholesterol concentrations are related to future CHD in cohort studies with a clear relationship to the development of heart disease whereas cohort dietary studies of SFA intakes rarely show this relationship. It is not just the errors in dietary assessment but the intrinsic differences in individuals' responsiveness that obscure the importance of reducing SFA intake for the whole population and explain why the relationships found across societies with very different SFA intakes show such a clear relationship to heart disease as originally observed by Keys and colleagues. Modest reductions in SFA intake achieved in community trials will induce only a modest effect on the average blood cholesterol concentration of the group so may not lead to a clearly evident reduction in CHD.

Atherosclerotic vascular diseases (AVD)

In a meta-analysis of 28 cohort studies and 16 RCTs SFA did not affect the risk of AVD independent of the intake of unsaturated fat or a healthy dietary pattern.[142] However, in RCTs where unsaturated fatty acids, especially PUFA, were substituted for SFA, AVD events were reduced by 14–19% corresponding to about 10% reduced risk for each 5 E% increase in PUFA intake.[104,106] As expected, the effect was more pronounced in longer studies (>2 years) and in men and in younger subjects where the relative risks are higher. In a pooled analysis of prospective cohort studies there was a 20% decreased risk of AVD in both men and women when 5 E% of PUFA was substituted for SFA.[143] In a pooled analysis of different types of studies a substitution of 1 E% PUFA for SFA reduced the risk of AVD by ≥2–3%.[144] In a systematic review it was concluded that there is convincing evidence that partial replacement of SFA with PUFA decreases the risk of AVD, especially in men.[107] Recent data from prospective cohort studies show that those on a higher intake of unsaturated fats, especially PUFA, and/or high quality carbohydrates compared with those on a higher SFA intake had a lower risk of CHD.[53]

The effect of replacing SFA with carbohydrates seems to depend on to the quality of carbohydrates. In general, replacement of SFA with carbohydrates has shown no beneficial effect on the risk of AVD.[143] Replacement of SFA with carbohydrates with high glycaemic index was associated with an increased risk of myocardial infarction whereas there was no association when SFA was replaced with carbohydrates with low glycaemic index.[145] The importance of the quality of carbohydrates has been confirmed by recent evidence.[53] Diets that are rich in dietary fibre, i.e. with complex unrefined carbohydrates in the diet, are associated with lower rates of heart disease.[22] This led the UK government expert SACN committee to recommend a major increase in dietary fibre intake and implies that lower fat, higher carbohydrate diets need to be fibre rich to obtain a reduced risk of CVD. High fibre diets were also found to reduce the risk of type 2 diabetes and so will also reduce the long-term risk of CVD. When diets are also relatively rich in the beta–glycan components of fibre then dietary trials

demonstrate a lowering of total cholesterol, LDL cholesterol and triglyceride concentrations in the blood consistent with the effects in lowering CVD.[22]

Regarding individual SFAs (C12:0-C18:0) there are minor differences in their association with AVD. Zong et al. (2016) have reported that 1 E% decrease in the intake of C12:0-C18:0 was associated significantly with reduced risk of CHD with PUFA, whole grain carbohydrates and plant protein as the replacements.[110] A similar decrease in the intake of C16:0 and an increase in the intake of MUFA were also significantly associated with reduced risk of AVD. There is some evidence from case-control studies that plasma levels of odd-chain fatty acids pentadecanoic acid (C15:0) and heptadecanoic acid (C17:0) are inversely associated with the risk of cardiovascular disease.[146,147] These fatty acids are often thought to be biomarkers of dairy fat intake but the association with dairy fat intakes is not always clear.[28] Levels of these acids may be associated with intake of fat from fish[28], other type of animal origin [29] and/or may be synthesised in the body from dietary fibre.[30]

When the sources of fat are considered then recent data from three US cohort studies show that intakes of vegetable sources of fat and PUFA rather than animal fat including dairy fat, are associated with a lower risk of CVD.[111]

In observational studies, TFA is associated with increased risk of AVD.[142,148] This is also in line with the marked effect of TFA on LDL cholesterol concentration and on the LDL-cholesterol/HDL-cholesterol ratio.[134,135,149] The source, i.e. natural vs. industrially produced, does not seem to have an impact on the metabolic effects.[134,135] Furthermore, it is important to keep in mind that the most abundant sources of TFA are also rich in SFA.[143]

The role of MUFA in affecting the risk of CVD has been unclear. In general epidemiological studies subjects eating more MUFAs rather than SFAs did not seem to have a lower risk of AVD,[143] whereas in long-term prospective cohort studies MUFA mostly associates with a decreased risk of AVD when compared with SFA or high glycaemic index carbohydrates richer diets.[132,150] In a recent systematic review the apparent favourable effect of cis-MUFA on AVD was considered unlikely to reflect a direct effect of MUFA intakes rather than the benefit of not eating SFAs.[107] In a recent analysis of large USA cohort studies of medical professionals, isocaloric differences of 5 E% MUFA rather than SFA was associated with a 13 % lower total mortality and a 27% lower mortality for 5% higher PUFAs rather than SFAs was 27%.[151] Population analyses giving variable results regarding MUFA may relate to MUFA intakes being also strongly linked to SFA intakes in many 'Western' countries, where olive oil is not used in abundance,[152] and in earlier studies the additional effect of TFAs also confused the picture.[143]

There are no data on the optimum ratio of n-6-to-n-3 PUFA, but it has generally been considered prudent to avoid replacement of SFAs with n-6 PUFA only (LA), and to include some n-3-PUFA (e.g. ALA) as well.[153] Nevertheless, some data do not suggest that avoiding LA is beneficial.[151] The role of n-6 PUFA has been considered controversial, but a recent systematic review concluded that the proportion of total PUFA, n-6 PUFA, and linoleic acid (LA, C18:2n-6) in plasma lipids has a favourable association with the risk of AVD.[107]

Regarding n-3 fatty acids, i.e. eicosapentaenoic acid (EPA, C20:5n-3) and docosahexaenoic acid (DHA, C22:6n-3) of mostly animal origin and ALA (C18:3n-3) of plant origin, they have favourable associations with the AVD risk.[107] An intake of 200–250 mg/d of EPA + DHA has been shown to be associated with some benefit whereas higher intakes are associated with no additional observed benefit.[154] A very low intake (<0.06 g/d) of EPA+DHA is associated with an increased risk of AVD in one study.[155] Regarding the source of these fatty acids it may be important whether they originate from fish or from supplements.[156] ALA intake has been shown to be associated with a decreased risk of AVD.[142,157–159]

2.3.3.4 Conclusions

There is convincing evidence that partial replacement of SFA with unsaturated fat, especially PUFA, and complex carbohydrates decreases the concentrations of both total and LDL cholesterol and the risk of AVD. Based on experimental and many observational studies a high intake of TFA is considered very deleterious regarding the serum lipid profile and the risk of AVD. Replacing SFA with simple carbohydrates has unfavourable effects as well but lower-fat, high-fibre diets are associated with consistent benefit. The dietary recommendations for reducing the levels of important risk factors, mainly LDL-cholesterol concentration, and the subsequent incidence of AVD highlight the value of limiting intakes of SFA and minimising intake of TFA, partly through eliminating industrially produced TFA, and having a moderate intake of unsaturated fat [7,16,17] within the context of healthy dietary pattern including fibre–rich carbohydrates.

Based on the current evidence it is important to emphasise the value of moderating the total amount of fat, i.e. to about 25 E%, to avoid the risks of weight gain related to too high an intake of dietary fat, e.g. excess energy intake and usually a lower than recommended intake of dietary fibre. This amount of total fat is sufficient to enable an adequate intake of essential fatty acids, namely LA and ALA, as well as vitamin E. There is a general consensus regarding the intake of SFA, i.e. it should be less than 10 E% for the general population and less than 7 E% for a population at high risk for AVD. There is also a wide consensus regarding the intake of TFA, i.e. it should be as low as possible. Due to this high level of consensus there is no reason for EHN to make an exception regarding these aims. In addition, it is important to emphasise the need for long term energy balance and avoiding weight gain and obesity. This re-emphasises the importance of the overall quality of the diet including a low sugar intake (i.e. a healthy dietary pattern), as in current recommendations.[7,16,17]

2.3.4 Salt – the benefits of population salt reduction: update of evidence since 2011

In its 2011 paper *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe*, EHN concluded that average daily salt intake among adults in Europe was probably around 10 g per day and proposed an intermediate population goal for maximal daily consumption of 5 g/day and a more ambitious longer term target of 4 g/day. This document reviews the new scientific evidence on this issue.

2.3.4.1 Key points

- Salt[viii] is causally related to blood pressure (BP): the higher the salt intake, the higher the BP, an effect which can be seen from birth.
- A sustained reduction in salt intake (up to 50% of what we eat now) causes a fall in BP in almost everyone across the whole range of BP, although individuals will respond more or less, depending on factors like age, ethnicity, initial levels of BP and body weight.
- High BP contributes to strokes and heart attacks and a reduction in BP is associated with their reduction. The magnitude of the effect is related to the size of the fall in BP.
- It is logical to consider, therefore, that a moderate reduction in salt intake in the population, through a modest reduction in BP, will result in a reduction in the incidence of strokes and some reduction in heart attacks.
- Natural experiments in different countries, direct experiments in primates, migration studies in humans, results from most prospective cohort studies in human populations and some randomised clinical trials support this concept.
- Some prospective observational studies have suggested that lower salt intake might be associated with 'increased' risk of CVD events. These studies, however, suffer from measurement errors that would introduce fatal biases in the results and, hence, erroneous conclusions.
- Well-conducted prospective studies – with sufficient statistical power and in which sodium excretion is accurately measured as an index of sodium intake and with the exclusion from the study population of people who are already unwell – support a graded, positive and linear relationship between sodium intake and both CVD and all-cause mortality.
- The World Health Organization currently therefore recommends targets of 5 g of salt (2 g of sodium) per day with a global target, as part of the global NCD action plan, to achieve a 30% reduction from current consumption by 2025.
- Sodium intakes exceed the recommended levels in almost all countries, so that virtually all populations would benefit from sodium reduction, supported by enhanced surveillance.
- Global actions are underway globally to reduce average population salt consumption.
- Population salt reduction is amongst the most cost-effective public health initiatives for reducing the burden of CVD.
- Population salt reduction programmes are feasible and effective (and are therefore considered 'preventive' imperative), cost-saving in all settings ('economic' imperative), powerful, rapid and equitable ('political' imperative).

2.3.4.2 Consideration About Targets

From anthropological accounts, evidence in primates and contemporary data from hunter-gatherer human populations still living in remote areas of the world where there is no access to salt, a consumption of as little as 1g of salt (0.4g of sodium) per day is compatible with a healthy life.

Salt consumption in the world varies, but (apart from the isolated populations mentioned above) no country in the world consumes less than 5 g of salt (2.0 g of sodium) per day.

Quality evidence from RCTs of the blood pressure lowering effect of salt reduction suggests a dose-response effect including as little as 5 g of salt (2.0 g of sodium) per day.

Prospective observational and modelling studies suggest a dose-dependent beneficial effect on cardiovascular outcomes.

Based on the best available evidence so far, long-term targets have been set to limit average population intakes to <5 g of salt (2.0 g of sodium) per day. However, it is acknowledged that, although rapid reductions are feasible, population salt reduction must be achieved gradually to allow adaptation of taste preference and an increase in consumer acceptance and demand. Many countries exceed these salt targets several fold.

Short- and medium-term targets have been set by the United Nations (UN) High Level Group for a 30% reduction in population salt consumption by 2025.

2.3.4.3 Background – Need To Update The Previous EHN Report

The 2011 paper *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe* extensively reviewed the role of salt and its effects on human health and concluded:

- The precise amount of daily salt intake among adults in Europe is not known, but probably lies around 10 g per day (*see update in present document*),
- Falls in salt intake reduce blood pressure, with clear evidence of a fall in cardiovascular morbidity and mortality (*see update in present document*),
- The mechanisms are still unexplained,

viii In this document salt (NaCl sodium chloride) and sodium are used to refer to sodium intake. Please note the following conversion: 2.5g (2,500mg) of salt = 1.0 (1 000mg) of sodium.

- A maximal daily consumption of 5 g/day seems reasonable for adults (with a longer term ambitious goal of 4 g/day). Policies for a progressive decrease are needed (*see update in present document*).

Since 2011, various publications have become available, ranging from new meta-analyses of RCTs, cohort studies in populations and patients' groups, new methodologies for assessing salt intake, modelling studies on effectiveness and costs, and policy documents. Critically, public health policies have been developed at the global level, led by a UN General Assembly Resolution in 2011 and by further analyses[162,163] World Health Organization (WHO) action plans and the initiatives of numerous international health agencies and implementation plans have been undertaken for population reductions in salt consumption for the prevention of cardiovascular disease.

Current recommended targets specify an intake of *5g of salt (2g of sodium) per day* with an action plan for a *30% reduction from current consumption by 2025*[1]. However, despite a large level of general scientific consensus, dissenting voices from industry and from some members of the scientific community have created a 'controversy'. The main criticisms are: a) a low salt intake may not lower blood pressure in everyone; b) a lower salt intake, as suggested by guidelines, may cause harm by increasing cardiovascular mortality; c) there is not sufficient evidence to justify current policies.

The following update will summarise new evidence, will respond to these criticisms and will offer updated conclusions.

2.3.4.4 Global Salt Consumption

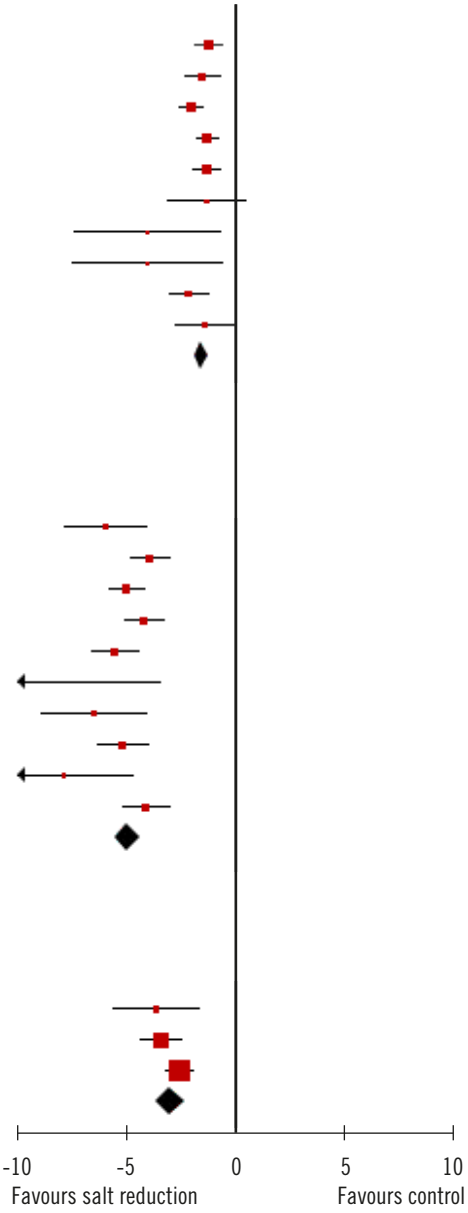
The Global Burden of Disease Nutrition and Chronic Disease Expert Group (NutriCoDE) has recently published two reports on comprehensive and comparable estimates of sodium intake globally[164] and estimates of global attributable deaths from cardiovascular causes above levels of 5 g salt (2 g of sodium) per day,[165] i.e., the World Health Organization recommended targets.[160] Sodium intakes exceed the recommended levels (often by three fold) in almost all countries with small differences by age and sex.[161] Furthermore, 1.65 million deaths from cardiovascular causes that occurred in 2010 were attributed to sodium consumption above the reference level of 5 g of salt (2.0 g of sodium) per day.[165]

2.3.4.5 Salt And Blood Pressure

Salt is causally related to blood pressure (BP), the higher the salt intake, the higher the BP, with an effect being seen from birth.[166] A small and sustained reduction in salt intake (up to 50% of what we eat now) causes a fall in BP in almost everyone across the whole range of BP, although individuals will respond more or less, depending on factors like age, ethnicity, their initial levels of BP and their body weight. These facts have been proven over and over again and summarised in repeated systematic reviews and meta-analyses of small and large clinical trials in people with and without high BP. Figure 23 shows the collective estimates of all meta-analyses published to date on the effect of salt reduction on BP in adults[167–176] The meta-analyses differ by the time of their analysis, so in the available studies, there are differences in the inclusion criteria (short-term studies of <4 weeks versus longer-term studies of >4 weeks), the proportion of normotensive and hypertensive participants, the study designs (cross-over, parallel group, blinded and open designs) and the proportion of relevant subgroups (gender, age and ethnic group). Despite differences between studies, the range of the pooled and weighted estimates of an effect are all in favour of salt reduction. Furthermore, their 95% confidence intervals are compatible with each other, indicating consistency, with differences between them likely due to random variation. Furthermore, when using very 'short-term salt restriction' trials with very large changes in salt intake (unlikely to be comparable to 'longer-term more moderate salt reduction' procedures) it has been argued that changes in metabolic and hormone variables that occur may be harmful.[169,172–174] These changes are due to rapid and transient activations of sympathetic adrenergic activity and haemoconcentration, not detected in longer-term and moderate salt reduction trials.[170,175,176] Finally, in 2015, a randomised, placebo-controlled crossover study was published.[164] It examined the effect of sodium or potassium supplementations on blood pressure in 36 participants, whose diet was fully controlled for the duration of the study. The subjects were provided with 2,500 kcal per day, 2 g of sodium and 2 g of potassium per day, and their systolic blood pressure was between 130 and 159 mmHg. In the sodium arm, participants were given 3 g of added sodium (equivalent to 7.5 g of salt per day) for 4 weeks. Twenty-four hour (24h) urinary sodium excretion increased, on average, by 98 mmol per day (~2.3 g of sodium or ~5.8 g of salt per day) compared to placebo. During sodium supplementation, office, 24h and central blood pressures all increased significantly compared to placebo (7.5/3.3 mmHg, 7.5/2.7 mmHg and 8.5/3.6 mmHg, respectively).

Figure 23 Forest-plot summarising the results of published meta-analyses of randomised clinical trials of the effects of salt reduction on systolic blood pressure. Results are reported as standard mean difference and 95% confidence intervals (CIs)

Study or subgroup	Std. Mean Difference	SE	Studies Total	Participants Total	Weight	Std. Mean Difference IV, Random 95% CI	Year
1.1.1 Normotensive							
Graudal 1998	-1.2	0.3061	56	2581	16.2%	-1.20 [-1.80, -0.60]	1998
Midgley 1998	-1.5	0.4113	28	2035	10.8%	-1.50 [-2.31, -0.69]	1998
He 2002	-2.03	0.2704	17	734	18.8%	-2.03 [-2.56, -1.50]	2002
Graudal (W) 2003-8	-1.27	0.25	57	5096	20.4%	-1.27 [-1.76, -0.78]	2008
Graudal (W) 2011	-1.27	0.3112	71	7299	15.9%	-1.27 [-1.88, -0.66]	2011
Graudal (A) 2011	-1.27	0.9184	3	393	2.7%	-1.27 [-3.07, -0.53]	2011
Graudal (B) 2011	-4.02	1.7092	7	506	0.8%	-4.02 [-7.37, -0.67]	2011
He (B) 2013	-4.02	1.7449	3	412	0.8%	-4.02 [-7.44, -0.60]	2013
He (W) 2013	-2.11	0.4694	12	1901	8.9%	-2.11 [-3.03, -1.19]	2013
Aburto 2013	-1.38	0.6939	7	3067	4.6%	-1.38 [-2.74, -0.02]	2013
Subtotal (95% CI)			261	24024	100.0%	-1.55 [-1.86, -1.24]	
Heterogeneity: Tau2= 0.06; Chi2= 12.18, df = 9 (P= 0.20); I2 = 26%							
Test for overall effect: Z = 9.84 (P < 0.00001)							
1.1.2 Hypertensive							
Midgley 1998	-5.9	0.9541	28	966	7.0%	-5.90 [-7.77, -4.03]	1998
Graudal 1998	-3.9	0.4592	58	2161	14.9%	-3.90 [-4.80, -3.00]	1998
He 2002	-4.96	0.4031	11	2220	16.2%	-4.96 [-5.75, -4.17]	2002
Graudal (W) 2003-8	-4.18	0.4592	58	3391	14.9%	-4.18 [-5.08, -3.28]	2008
Graudal (W) 2011	-5.48	0.5357	76	4903	13.3%	-5.48 [-6.53, -4.43]	2011
Graudal (A) 2011	-10.21	3.4541	8	477	0.8%	-10.21 [-16.98, -3.44]	2011
Graudal (B) 2011	-6.44	1.2296	9	674	4.8%	-6.44 [-8.85, -4.03]	2011
He (W) 2013	-5.12	0.5867	17	623	12.3%	-5.12 [-6.27, -3.97]	2013
He (B) 2013	-7.83	1.597	5	0	3.1%	-7.83 [-10.96, -4.70]	2013
Aburto 2013	-4.06	0.5612	24	2273	12.8%	-4.06 [-5.16, -2.96]	2013
Subtotal (95% CI)			**294**	**17688**	**100.0%**	**-4.93 [-5.52, -4.33]**	
Heterogeneity: Tau2= 0.41; Chi2= 18.57, df = 9 (P = 0.03); I2 = 52%							
Test for overall effect: Z = 16.17 (P < 0.00001)							
1.1.3 All							
Grobbee 1984	-3.6	1	13	584	10.6%	-3.60 [-5.56, -1.64]	1984
Midgley 1998	-3.4	0.4592	56	3021	35.7%	-3.40 [-4.3, -2.50]	1998
Geleijnse 2003	-2.54	0.3163	40	0	53.7%	-2.54 [-3.16, -1.92]	2003
Subtotal (95% CI)			**109**	**3605**	**100.0%**	**-2.96 [-3.63, -2.28]**	
Heterogeneity: Tau2= 0.12; Chi2= 2.95, df = 2 (P = 0.023); I2 = 32%							
Test for overall effect: Z = 8.60 (P < 0.00001)							



In conclusion, the results of these analyses, despite different interpretations at the time of their publication, all agree on the following:

1. Salt intake is one of the major determinants of BP in populations and individuals;
2. A reduction in salt intake causes a dose-dependent reduction in BP;
3. The effect is seen in both sexes, in people of all ages and ethnic groups, with all starting BPs, and is detectable for measurements made in the office, over a continuous 24h period and when measuring central BPs.

Similar results have been described in children.[177,178]

Salt Sensitivity

A moderate reduction in salt intake reduces BP in most but not all individuals. The effect on BP varies largely from person to person.[179] Salt sensitivity has a variety of determinants, including race and ethnicity[180], age[181], body mass index,[182–184] and diet quality, as well as associated disease states (e.g. hypertension, diabetes, and renal dysfunction). It is also partially under genetic control, as these more salt responsive individuals, whether considered 'normotensive' or 'hypertensive'; tend to have a positive family history of hypertension.[185] The BP response to a moderate change in salt intake is normally distributed.[186] Many experimental models have been used for the past 40 years to attempt an individual characterisation of so-called salt sensitivity.

These methods have included BP responses to (1) acute and large changes in salt intake, with or without diuretic-induced volume depletion and (2) moderate changes in salt intake over days in normotensive volunteers,[187] patients, or the general population.[188] They also included the response of the renin-angiotensin-aldosterone system[188–190] and the clearance of endogenous lithium, a non-invasive method for assessing segmental renal tubular sodium handling[190] and considered a proxy for salt sensitivity.[191,192] Measures of salt sensitivity are associated with more severe cardiovascular risk factor profiles,[184,193–195] and they are also negative prognostic indicators.[184,193] Although less easy to detect, salt sensitivity is also present in normotensive people. In a small clinical study with a long-term follow-up, normotensive salt-sensitive individuals had a cumulative mortality as high as that of hypertensive patients.[196]

In the last year two comprehensive reviews of the topic have been published.[197,198] Both independent appraisals of the available evidence agree on the following points:

1. The paradigm of 'salt-sensitivity' has important pathophysiological meaning in understanding individuals' variation in BP response to changes in salt intake;
2. Knowledge gaps suggest further research in the area;
3. Due to important limitations (lack of uniformity of assessment across studies, lack of an established method of assessment, lack of reproducibility of current methods, variable definitions) the concept of 'salt sensitivity' is not useful to the practising physician in clinical practice;

the concept is not relevant or useful in the design and implementation of public health policies based on a moderate reduction in population salt intake, and aiming at a modest shift to the left in the average distribution of salt consumption and BP, with a recognised ensuing reduction in cardiovascular outcomes.[199,200]

Salt And Mortality

High BP contributes to strokes and heart attacks and a reduction in BP is associated with their reduction. The magnitude of the effect is related to the size of the fall in BP. It is therefore conceivable that a moderate reduction in salt intake in a population, through a reduction in BP, would result in a reduction of strokes and heart attacks. The collective evidence from systematic reviews of prospective longitudinal studies indicates that a lower salt intake is associated with a lower incidence of fatal and non-fatal cardiovascular events, in particular stroke.[176,201] This is also supported by a meta-analysis of the few RCTs available to date that have measured fatal and non-fatal outcomes.[202]

Since 2011, analyses of prospective observational studies have suggested, in some cases, that lower salt intake might be associated with an 'increased' risk of CVD events, in particular heart failure. These studies have been the object of intense scrutiny due to numerous methodological issues present in observational studies that would introduce fatal biases (errors) in the results and, hence, erroneous conclusions.

A comprehensive account of these issues has been published by the American Heart Association.[203] These issues are reported below (Table 6).

Table 6 Methodological issues in the assessment of prospective observational studies of salt consumption and cardiovascular outcomes

Domain 1	**Errors with the greatest potential to alter the direction of association**
	Systematic error in sodium assessment • Lower risk of errors: 24h urine collections for sodium analyses, but some collections have no quality assurance, and do not exclude incomplete collections • Higher risk of errors in methodology: e.g. the use of other general 24h urine collections obtained for other purposes, all dietary assessments, spot and overnight urine collections
	Reverse causality • Lower risk of errors: when participants are recruited from the general population and those with pre-existing CVD excluded • Intermediate risk: when sick populations are not excluded or included despite claims to the contrary; when there is the presence of other CVD risk factors; use in specifically sick populations • Higher risk: specific types of sick populations (e.g.: heart failure, kidney disease, diabetes); removal of sick participants from the analysis changes the direction of the subsequently observed association
Domain 2	**Errors with some potential to alter the direction of association**
	Potential for residual confounding • Incomplete adjustment: not including two or more factors such as age, sex, race, socioeconomic status, cholesterol, BMI or weight, smoking, diabetes; if diet-based assessments of sodium intake are made then the neglect of total calorie intake; if urine-based, adjusting by weight, BMI or assessed creatinine excretion • Imbalance across sodium intake levels: with age difference across sodium groups >5 years; imbalance in sex or race distribution across sodium groups >20% • Inadequate follow-up: low level of follow-up (<80%) or when the quality for outcome assessment is uncertain.
Domain 3	**Errors with the potential to lead to a false null result**
	Random error in sodium assessment • Lower risk of error: more than four 24h urine assessments on average; Food Frequency Questionnaires • Intermediate risk: between two and four 24h urine collections, or corrections for regression dilution bias; dietary reports • Higher risk: urine collection <24 h or single 24h urine collection; single dietary recall or 1-day food record
	Insufficient power • Less than 80% power to detect a 10% reduction in relative risk for every standard deviation difference in sodium intake
	Studies using same data but reporting divergent results • NHANES I: analyses in the same age group with the same follow-up by different authors – both inverse and positive associations reported with the use of different selections, adjustments etc. • NHANES III: analyses in different age groups with different follow-up by different authors – both inverse and positive associations reported when again using different analytical approaches.

2.3.4.6 Population-based Cohort studies

Stolarz-Skrzypek et al[204] followed up 3 681 participants without CVD who were members of families that were randomly enrolled in the FLEMENGHO and EPOGH studies in Belgium. They measured baseline salt consumption with a single 24h urine collection and recorded all-cause and cardiovascular mortality for a median period of 7.9 years. Vital status was obtained in all participants; 219 deaths were recorded, of which 84 deaths were cardiovascular. Cardiovascular mortality (p=0.02), but not all-cause mortality (p=0.10), was higher in the group with a lower urinary sodium excretion, when adjusted for confounders. The authors conclude that lower sodium excretion was associated with higher CVD mortality and that the results do not support current recommendations of a generalised reduction in sodium intake at the population level.

Comment. The study presents several weaknesses: (a) the lower sodium group had not only lower urinary volume excretion but also lower urinary creatinine and potassium excretion, suggesting incomplete collections in that group;[205–207] (b) the lower sodium group had paradoxical higher proportion of low socio-economic participants, in contrast with the expectations of a higher salt intake in that group;[208–210] (c) the only statistically significant finding was for CV deaths (n=50 in the low sodium group), in contrast with no significant effects when fatal and non-fatal events were considered together. In particular, there was no effect on stroke events.[211]

O'Donnell et al[212] obtained morning fasting urine samples from 101 945 persons taking part in the Prospective Urban Rural Epidemiology (PURE) study, a cohort study that enrolled and followed up 156 424 persons, aged 37–70

years from 628 rural and urban communities in 17 low-middle- and high-income countries. Salt intake was inferred from estimated 24h urinary sodium excretion calculated with the Kawasaki formula applied to a morning fasting urine sample. They recorded all-cause and major CV deaths for a mean follow-up of 3.7 years. A composite outcome occurred in 3 317 participants. There was a J-shaped association between estimated sodium excretion and CV events: a higher estimated sodium excretion of ≥7 g (>17.5 g of salt) per day was associated with increased risks of death and major CV events, with a stronger effect among people with 'hypertension'. On the other hand, an estimated sodium excretion that was below 3 g (7.5 g of salt) per day was also associated with an increased risk of the composite outcome.

Comment. a) the estimated sodium excretion using morning fasting spot urines with Kawasaki or other equations is unreliable and biased, as demonstrated by several studies,[213–216] including validations within the PURE study;[217,218] b) the sodium study within the PURE study may be affected by selection bias: the sodium study, in fact, included only 65% of the participants; there were fewer from India (5 v 18%) and many more from China (42% v 30%). Moreover, a high proportion of participants had pre-existing ill-health (hypertension, blood pressure medication, pre-existing CHD and CVD); c) the lower sodium group was grossly unbalanced compared to the higher sodium group: it was older and had fewer men, fewer Asians and smokers with more Africans and non-Asians and urban persons; they also had a lower BP, a higher LDL-cholesterol, a history of CVD and diabetes, lower level of physical activity and higher medication use (suggesting reverse causality); d) the lower sodium excretion group (<3 g/day or <7.5 g of salt/day) was unable to discriminate within the range of recommended targets for populations and, therefore, was not informative for policy making.

Joosten et al[219] followed up 7 543 participants free of cardiovascular and kidney disease in the PREVEND study in Gröningen, the Netherlands. They measured baseline salt consumption by two 24h urine collections at baseline and recorded the occurrence of fatal and non-fatal CHD events for a median period of 10.5 years. 452 CHD events were recorded. In the entire cohort, there was no statistically significant association between estimated salt intake and CHD event rate (HR=1.07, 95% CI 0.98-1.18). However, higher sodium excretion was associated with an increased CHD risk amongst subjects with hypertension or with increased NT-proBNP concentrations. No trend for increased risk on low sodium excretion was detected.

Comment. Given the small number of events, the study might have lacked statistical power to statistically detect a small increase in risk in the overall cohort (average 7%). Also, it is apparent from the figure (although not reaching statistical significance) that there was a linear trend between urinary sodium excretion and adjusted risk, with no indication of a J-shaped relation at lower levels of urinary sodium.

Cook et al[220] followed up pre-hypertensive participants during an extended post-trial surveillance in TOHP II (10 years follow-up) and TOHP I (15 years follow-up). 193 fatal and non-fatal cardiovascular events were recorded amongst the 2 275 participants not in a sodium reduction intervention group. Multiple (3-to-7 per individual) 24h urine collections were obtained throughout. There was a linear 17% increase in risk of CVD events per 1 g/day increase in sodium excretion. No J-shaped trend was observed at lower sodium excretion.

Comment. This study design overcomes major methodological challenges of prior studies and, in spite of the relatively small event rate, does detect an overall benefit of lower sodium intake with no evidence of non-linear effects. Assessment of sodium is strengthened by a multiple collections carefully controlled for completeness.

Pfister et al[221] followed up 9 017 men and 10 840 women (age 39–79 years) participating in the EPIC-Norfolk prospective study in the UK. They estimated baseline salt consumption on a casual spot urine sample obtained at baseline using the Tanaka formula for estimating daily sodium excretion and then recorded 1 210 fatal (n=137) and non-fatal (1,073) incident cases of heart failure (702 men and 508 women) during a mean follow-up of 12.9 years. There was a U-shaped association between estimated urinary sodium and heart failure risk. The risk associated with high sodium was attenuated when adjusting for blood pressure whereas the risk associated with low sodium was attenuated when adjusting for pre-existing disease.

Comment. The study suffers from the following methodological flaws: a) the biased assessment of sodium consumption due to the use of casual spot urines and application of the Tanaka formula; b) a clear selection bias in the low sodium group representing sicker individuals (reverse causality); c) the latter point is supported by the diluting effect of risk in the low sodium group when adjusted for pre-existing disease.

Kalogeropoulos et al[222] followed up 2 642 older adults (71–80 years) without prevalent CVD at baseline participating in a community-based prospective cohort study in Pittsburgh and Memphis, USA (Health ABC Study). They measured baseline salt consumption by a Food Frequency Questionnaire and recorded CVD (n=1 981) and heart failure (HF) (n=2 628) mortality over 10 years of follow-up. In total, 881 had died, 572 of CVD and 398 of HF. The results show no significant association between FFQ-determined dietary sodium intake and mortality from CVD or HF.

Comment. The article was the subject of several comments in the media and in the scientific literature. A variety of sources agreed on the main criticisms: there was a systematic error in the assessment of sodium intake with the use of imprecise methods, such as FFQ; there was a potential for reverse causality possibly due to selection bias with the lower sodium group.[223–225]

Lamelas et al[226] reports from a sub-sample (in four South American countries) of the PURE Study. They obtained morning fasting urine samples from 17 033 persons from Argentina, Brazil, Chile and Colombia. They estimated 24h urinary sodium and potassium excretion from a morning fasting urine sample, using the Kawasaki formula. All-cause mortality and major CVD was the primary outcome with a

median follow-up of 4.7 years. A composite CVD outcome occurred in 568 participants (417 deaths, 143 had a CVD event, and 148 a myocardial infarct (MI), 102 had a non-fatal stroke and 41 developed HF). There was a possible J-shaped association between estimated sodium excretion and composite event: a higher estimated sodium excretion (≥7 g/day) was associated with increased risks of death and major CV events, but the effect was attenuated by adjustments. On the other hand, the reported increased risk at lower sodium excretion was never statistically significant.

Comment. This study design suffers from all the limitations reported for the main PURE Study. Specifically, a) the estimated average 24 sodium excretion using a morning fasting spot urines measure recalculated with the Kawasaki or other equations is unreliable and biased, as demonstrated by several studies,[213–216] including validations within the PURE study[217,218] itself; b) the lower sodium excretion group (<3 g/day or <7.5 g of salt/day) was unable to discriminate within the range of previously recommended salt targets of 3 g, 5 g or 6 g/day and, therefore, is not informative for policymaking; c) the analysis of the South American sub-sample is also inconclusive due to a lack of statistical power.

Cook et al[227] followed up pre-hypertensive adults during an extended post-trial surveillance in the TOHP II study with on average a 25.7 year follow-up and in the TOHP I analysis of 22.4 years of follow-up. A total of 77 and 174 deaths respectively occurred amongst the unique 2 974 participants not in a sodium reduction intervention group. Multiple (three to seven per individual) 24h urine collections were obtained throughout. There was a direct linear association between average sodium intake and mortality (HR: 1.12 per 1g sodium/day [95% CI: 1.00 to 1.26], p=0.05). No J-shaped trend was observed at lower sodium excretion.

Comment. This study design overcomes major methodological challenges of prior studies and, in spite of relatively small event rates, detects an overall benefit of lower sodium intake with no evidence of non-linear effects. The validation of the assessment of sodium excretion is strengthened by the use of multiple urine collections carefully controlled for completeness.

2.3.4.7 Patients' groups

Ekinci et al[228] followed up 638 patients with type 2 diabetes attending a single diabetes clinic. They measured baseline salt consumption with a median number of two 24h urine collections and recorded all-cause and cardiovascular mortality for a median period of 9.9 years. Vital status was obtained in 620 patients; 175 deaths were recorded, of which 75 deaths were of cardiovascular cause. Both all-cause (p=0.017) and cardiovascular (p=0.026) mortality were higher in the group with a lower urinary sodium excretion, when adjusted for confounders. The authors conclude that intervention studies are necessary to establish causality and whether it is appropriate to advocate salt reduction in these patients.

Comment. Patients in the lower tertile of estimated sodium excretion were at greater risk of death. They were older, had a longer duration of diabetes and were more likely taking insulin, beta-blockers, and less likely to take ACE inhibitors, and their GFR was lower than in the other sodium groups. This raises the question whether the results were affected by 'reverse causality', due to the selection of sicker patients who were therefore more likely to die in the low sodium group.

Thomas et al[229] followed up 2 807 patients with type 1 diabetes without end stage renal disease (ESRD) in a nationwide multicentre study (FinnDiane Study). They measured baseline salt consumption by a single 24h urine collection and recorded all-cause mortality (n=217) and ESRD incidence (n=126) for a median period of 10 years. There was a U-shaped relationship between sodium excretion and both mortality and ESRD. The authors call for caution before applying salt restriction universally.

Comment. The methodology for measuring compliance and completeness of urine collections are not reported nor are there the mean values presented. From what can be gathered from the published data the risk of incomplete urine collections was highly likely. Reverse causality and residual confounding are also likely explanations of the findings.

O'Donnell et al[230] followed up two cohorts of patients (n=28 880) included in the ONTARGET and TRANSCEND trials which are RCTs of anti-hypertensive therapy in high risk patients. They estimated 24h urinary sodium and potassium excretion from a morning fasting urine sample, using the Kawasaki formula, and recorded CV death, myocardial infarction (MI), stroke, and hospitalisation for congestive heart failure (CHF) for a median follow-up of 56 months. A composite cardiovascular outcome occurred in 4 729 participants, including 2 057 CV deaths, 1 412 non-fatal MI, 1 282 non-fatal stroke, and 1 213 developed CHF. There was a J-shaped association between estimated sodium excretion and CV events.

Comment. a) there was an inaccurate estimate of sodium intake based on spot urine samples with the use of the Kawasaki equation. The method has been extensively assessed for validity and it has been unequivocally shown to be biased and unable to characterise individuals' sodium excretion (intake),[213–216] including the authors' validation in a subsequent study[217,218] (see above); b) the population group studies was made of old and sick patients with pre-existing conditions (70% hypertensive, 37% with diabetes, 48% with previous MI, 21% with a previous stroke or transient ischaemic attack (TIA) and 3% with atrial fibrillation); c) patients were often on multiple medications (overall 29% were on diuretics, 41% of whom were in the lowest sodium group). The latter two points strongly points to a 'reverse causality' bias as a likely explanation of the mortality findings.

Saulnier et al[231] followed up a prospective inception cohort of 1 439 French patients with type 2 diabetes, in whom the median duration of follow-up was 70 months (SURDIAGENE Study). They report a non-linear relationship between urinary sodium and cardiovascular mortality.

Comment. This is a report of results in a letter with no details provided. The original publication[232] does not report the

methodology for assessing sodium excretion. This report is therefore difficult to assess in terms of quality.

Singer et al[233] followed up a cohort of 3 505 hypertensive individuals participating in a worksite hypertension programme. They measured daily urinary sodium excretion with a single 24h urine collection, and obtained mortality data for a mean follow-up period of 18.6 years. Overall there were 1 013 deaths (399 cardiovascular). In adjusted models sodium intake was not significantly associated with cardiovascular mortality. The authors suggest that the inconsistent results cast doubt on whether a single measurement can reliably predict mortality over a prolonged follow-up period.

Comment. This study is inconclusive due to lack of statistical power. However, the authors' conclusion has merit.

Äijälä et al[234] followed up a cohort of 1 405 participants in a population-based study of treated hypertensive patients and matched normotensives. 716 of them completed a food diary from which to calculate dietary sodium consumption and 690 without previous CVD were included (329 men and 361 women). They obtained information on fatal and non-fatal CVD events for a mean follow-up period of 19 years. Overall there were 109 events. In adjusted models sodium intake was significantly associated with increased cardiovascular events, both in men and women.

Comment. This is a small study, the assessment of dietary sodium is inaccurate and the selection of participants (cases and matched control) suggest that they are not fully unselected cases needed for an unbiased analysis.

Mills et al[235] followed up a cohort of 3 757 patients with chronic kidney disease (CKD) from seven locations in the USA. They assessed baseline urinary sodium excretion from a cumulative calibrated measure based on three 24h urine collections and obtained information on non-fatal composite CVD events (CHF, MI and stroke) for a median follow-up of 6.8 years. 804 composite events (575 of CHF, 305 of MI and 148 of stroke) occurred. The study showed a significant linear association between calibrated 24h urinary sodium excretion and composite CVD events with no evidence of non-linear effects.

Comment. This study design overcomes major methodological challenges of prior studies and, in spite of relatively small event rate, detects an overall benefit of lower sodium intake with no evidence of non-linear effects. Assessment of sodium in strengthened by a calibration of multiple collection carefully controlled for completeness.

Mente et al[236] reported on a pooled analysis of about 134 000 individuals (about 63 500 with hypertension) taken from four independent studies (i.e. PURE, EPIDREAM screnees, ONTARGET and TRANSCEND). They estimated 24h urinary sodium and potassium excretion from a morning fasting urine sample, using the Kawasaki formula, and recorded the primary outcome as a composite of death, myocardial infarction (MI), stroke and heart failure (HF). They reported data for normotensive and hypertensives separately. Hypertension was defined if untreated when baseline BP was ≥140/90mmHg or if participants were prescribed anti-hypertensive drugs at baseline. In hypertensives, higher sodium intake of >7 g (>17.5 g of salt) per day and lower sodium intake <3 g (7.5 g of salt) per day were associated with increased risk compared to those with 4–5 g of sodium (10–12.5 g of salt) per day, whereas in normotensive participants, higher sodium intake was not associated with increased risk but lower sodium intake was.

Comment. This study suffers from flaws that have been repeatedly addressed in previous reports but ignored in the present study. Three areas: i) inappropriate assessment of exposure with spot urines (as extensively explained above); ii) reverse causality by including sick participants that are over-represented in the 'lower' sodium group; iii) the artificial split of participants into normotensive and hypertensive which is not biologically plausible and reduces the statistical power of analysis and leads to paradoxical and implausible results such as the findings of an 'inverse' relationship between BP levels and cardiovascular outcomes in normotensives, irrespective of their estimated salt intake (Figure 3, bottom panel of original publication[236]). A comprehensive critique of this approach can be seen in published correspondence.[237,238]

2.3.4.8 Assessment of Salt Intake By Urinary Sodium Excretion

Salt intake is extremely variable between individuals as well as from day-to-day in the same person. Therefore, even a single measurement of the daily amount of sodium excreted in the urine (often regarded as the 'gold' standard for assessing individuals' salt consumption) may be inadequate.[239] In a well-conducted physiological study single 24-hour urine collections at intakes ranging from 6 to 12 g salt per day are not suitable to detect a 3 g difference in individual salt intake. Repeated measurements of 24h urinary sodium improve precision, suggesting multiple 24h urine collections over time are necessary to assess a person's salt intake.[239]

On the other hand, there is great interest in replacing 24h urinary sodium with easier methods to assess dietary sodium. A recent systematic review included 1 380 130 participants from 20 studies. The main statistical method for comparing 24h urine collections with alternative methods was the use of a correlation coefficient. Spot, timed, and overnight urine samples were subject to greater intra-individual and inter-individual variability than 24h urine collections. There was a wide range of correlation coefficients between 24h urine sodium and other measures of sodium excretion.[240] Subsequently, numerous validation studies have been published, comparing 24h urine collections with estimates of daily sodium excretion from spot urines extrapolated with the application of different formulae. The results have been analysed and compared using Bland-Altman plots. There is a global consensus from a variety of population analyses that spot urines (irrespective of the formulas used to estimate daily consumption) lead to biased estimates of 24h urinary sodium excretion with overestimates at lower levels and underestimates at higher levels.[213–218]

2.3.5 Dietary cholesterol

As with the previous EHN paper, no specific population goal is proposed for dietary cholesterol. There is, however, considerable public confusion about dietary cholesterol – which in previous decades was a focus for dietary advice. This section, therefore, provides a brief explanation for the lack of a population goal on dietary cholesterol either in this report or in most government and WHO recommendations.

Although there is strong evidence that lower intakes of dietary cholesterol are associated with reduced cardiovascular risk, and moderate evidence that such eating patterns are associated with reduced risk of obesity this reflects the fact that in Western diets dietary cholesterol is found in animal foods which tend to be high in fat and saturated fat so dietary cholesterol in these analyses serves as a marker of the nature of the diet.[8] There is evidence, however, that dietary cholesterol can increase blood cholesterol but its effect is modest compared with the impact of saturated fatty acids on the blood cholesterol content; usually the majority of circulating cholesterol is assessed as having been synthesised by the liver in response to dietary fatty acids but there are differences in the response of individuals to dietary cholesterol which also relates to their individual responsiveness to saturated fat.[241] There are, therefore, wide variations in how individuals respond to dietary cholesterol.

There is no need to include a specific population-level recommendation on dietary cholesterol. This is because, first, the magnitude of the effect of saturated fat is much greater than that of dietary cholesterol and, second, foods with higher dietary cholesterol levels also tend to have high saturated fat levels. Thus, diets lower in saturated fat levels will also be lower in dietary cholesterol.

There are some foods – including egg yolks and some shellfish – that are higher in dietary cholesterol but not saturated fats. Consuming one additional egg daily will raise the ratio of total cholesterol to HDL-cholesterol by 0.040 units, implying an increased risk of heart attack of around 2%.[242] While eggs do contribute micronutrients to the diet, their contribution to dietary cholesterol is much greater[242] and current average consumption of eggs and other foods rich in dietary cholesterol should not rise.

2.3.6 Red and processed meat

EHN's population goals have generally focused on nutrients and ingredients rather than foods (with some exceptions such as fruit and vegetables and sugary drinks). Translation of population goals into food-based dietary guidelines needs to take into account the existing eating habits and food environment in each country.

In the period since the previous EHN paper there has been a surge in media and public interest in the health effects of eating meat, particularly since WHO's International Agency for Research in Cancer classifications of red meat as *probably carcinogenic* and processed meat as *carcinogenic* made international headline news in October 2015.[243]

This section summarises the relationship between meat and CVD, with reference also to associations between meat and other NCDs. In doing so, there may be some overlap with other sections (e.g. on fats).

2.3.6.1 Meat and cardiovascular disease

Whereas most of the fat in plant-based foods is unsaturated (either monounsaturated or polyunsaturated), meat is high in saturated fat. Furthermore it is only animal-derived products that contain dietary cholesterol. Therefore, consumption of meat – particularly meat products high in saturated fat – have been linked with an increased risk of CVD since the Seven Countries Study by Ancel Keys in the late 1970s and early 1980s identified a strong association between average CVD rates and average blood cholesterol levels. Average blood cholesterol levels were in turn found to be positively associated with average levels of dietary cholesterol and of saturated fat and possibly negatively associated with average levels of unsaturated fat (though this was less clear).

The results of this cross-country comparison were quickly augmented by observational studies on individuals and more latterly by experimental studies (including RCTs) and thereby relationships between blood cholesterol levels and the likelihood of coronary heart disease (CHD) were shown to be causal and not just associations.[115,244,245]

Research carried out since the early 1980s on the relationship between different types of fat intake and risk of CVD has shown it to be more complicated than previously thought (see section 2.2.2 on fats). Nonetheless, the basic story has remained essentially the same for the last 20 or 30 years. High intakes of saturated fat increase the risk of CVD, particularly if coupled with high levels of sugar and other refined carbohydrates in the diet, and a reduction in the average saturated fat intake in most countries would generate substantial health benefits. Given that meat is an important source of saturated fat there would seem – on this basis alone – to be an obvious case for reducing the intake of saturated fat from meat.

There is currently some debate about whether the fat composition of meat products can be enhanced in relation to its effects on health by different feeding practices. For example it is certainly the case that n-3 polyunsaturated fat levels in beef can be increased by high forage-based diets.[246] This is likely to mean that the meat from grass-fed cattle will be slightly healthier than from non-grass-fed cattle. However, it seems unlikely that the slightly improved fat composition of beef from grass-fed animals (compared for example with beef produced more intensively) has any major impact on the health of its consumers.

It is also important to consider that much of the meat consumption in European countries is in the form of processed meat products (bacon, ham, sausages, ready meals, etc.). Salt is used as a preservative and flavour enhancer in most processed foods so consumption of these processed foods can lead to an excessive consumption of sodium.

An important systematic review of the evidence for the direct relationship between red and processed meat consumption

and the risk of CVD has recently been updated.[247] It suggests that the effects of red meat consumption on risk of CHD vary depending on processing. In meta-analyses of prospective studies, higher risk of CHD is seen to be associated with processed meat consumption but a smaller increase or no risk is seen with unprocessed meat consumption. Differences in sodium content (~400% higher in processed meat in the reviewed studies) appear to account for about two-thirds of this risk difference. A similar meta-analysis shows that consumption of processed red meat and, to a lesser extent, unprocessed red meat is associated with increased risk of stroke.[248]

The strong association between processed meat consumption and cardiovascular health found in these meta-analyses is mirrored in a dose-response meta-analysis of nine prospective cohort studies with all-cause mortality as an outcome.[249] The researchers found increased risk of all-cause mortality for processed meat consumption and total red meat consumption, and a non-significant increased risk associated with non-processed red meat consumption. Whereas previous meta-analyses had compared health risks in groups categorised as 'high' or 'low' meat consumers, Larsson and Orsini quantified meat consumption in the included studies and considered how risk changes at different consumption levels. They found that all-cause mortality increases at all levels of processed meat consumption, and they found a very low threshold (10 g/d) at which risk begins to increase for total red meat consumption.[249]

2.3.6.2 Meat and other NCDs

Possible associations between meat consumption and other NCDs have also been examined. In relation to cancer, the World Cancer Research Fund (WCRF) has examined many possible relationships between meat consumption and cancer and the one relationship that it describes as 'convincing' is the relationship between red and processed meat intake and colorectal cancer.[250] The International Agency for Research on Cancer (IARC) similarly concluded recently that red meat is 'probably carcinogenic to humans' and that the association between red meat consumption and cancer is clearest for colorectal cancer, but associations were also seen for pancreatic cancer and prostate cancer. IARC also classified processed meat as 'carcinogenic to humans' based on sufficient evidence that consumption of processed meat causes colorectal cancer.[243]

In relation to obesity, there is little evidence for any direct relationship with meat consumption. Some meat products are energy dense and WCRF suggests that the evidence linking the consumption of large amounts of energy dense foods with overweight and obesity is 'probable'. WCRF also indicates, however, that the evidence linking meat product consumption with overweight and obesity is 'limited and inconclusive'.[250] On the other hand there is some evidence that red meat consumption may be associated with increased risk of diabetes, with, yet again, higher risks for processed red meats[247] and suggestions that factors other than fatty acids (e.g. the haem content of the meat and/or dietary cholesterol) may contribute, but these findings are all based on associations.

2.3.6.3 Official recommendations about meat consumption

In 2002 WHO advised people to moderate consumption of preserved meat to reduce the risk of cancer[18] and this message was confirmed after the 2015 IARC publication which classified processed meat as carcinogenic.[243] Some European authorities have also recently recommended reducing intakes of red and processed meat.[7,251–254]

It has also begun to be suggested that a reduction in meat consumption would be good for the environment as well as health. In 2015 the Swedish government, for example, reaffirmed its recommendation to eat less red and processed meat on both health and sustainability grounds. The National Food Agency recommends eating less than 500 g of red and processed meat per week (equivalent to 600–750 g raw meat).[252] Despite growing awareness of the impact of food production, and therefore consumption, on environmental sustainability other authorities have been slow to follow the Swedish Government's example.

2.3.6.4 Conclusion

In conclusion, it does seem likely that the negative consequences of eating large amounts of meat in European countries outweigh the positive benefits. It seems clear that if meat products are to be eaten on a routine basis, those products that are lower in saturated fat and salt, e.g. lean, fresh meat rather than processed meat, are preferable for cardiovascular health. Furthermore, on the basis that it seems that red and processed meats eaten in excess, rather than white and unprocessed meats, increase the risk of disease, it is advisable to eat less of the red and processed meats and replace these (particularly, if necessary, in high meat consumers) with white and unprocessed meat products.

The issue of meat consumption and health also needs to be set in the context of the important question about the impact of meat consumption (in terms of quantity and type of meat) on the health of the planet. (See Chapter 3.4 for further discussion on these issues).

2.3.7 Nutrition in early life

There is a growing evidence base on the impact that nutrition during early life – pre-conception and during pregnancy, infancy and early childhood – can have on later health outcomes, including cardiovascular risk factors. This means that both maternal nutrition status and infant/young child feeding are important.

Maternal nutrition status – before and during pregnancy – can influence growth and development and later health outcomes of the child.[255,256] More specifically if a pregnant woman has suboptimal nutritional status, particularly during the period of embryogenesis, this may induce changes in the way genes are expressed in the offspring (epigenetic programming) and how these genes respond to environmental factors later in life.

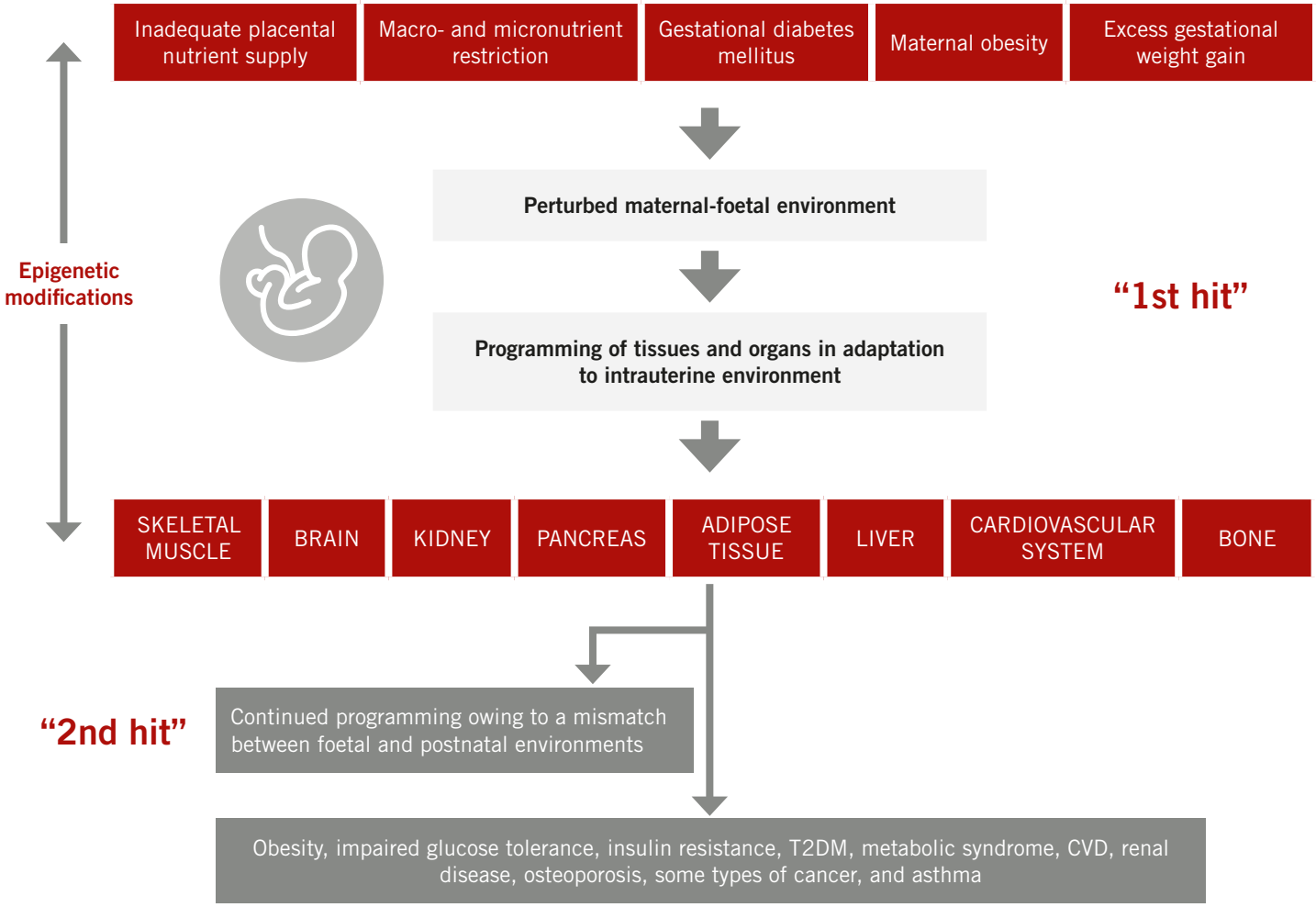
A 2016 review on maternal nutrition by the WHO Regional Office for Europe summarised recent evidence on nutritional programming and intergenerational effects.[255] The majority of research into this issue of 'programming' during early life has focused on the impact of maternal undernutrition – low protein intakes and other nutrient restrictions in mothers are associated with the nutritional epigenetic programming of obesity in their offspring – potentially leading to a lifelong vulnerability to obesogenic environments. In other words, if a woman is undernourished before or during pregnancy her offspring have a higher risk of overweight or obesity if they are later exposed to an environment of plentiful food. Low birth weight and intrauterine growth restriction are strongly associated with later type 2 diabetes, abdominal adiposity, abnormal lipid metabolism, obesity, hypertension, CVD and increased risk of death from CHD. There is abundant evidence that there is a graded relationship between birth weight and later health outcomes, throughout the normal birth weight range.

More recently, research has explored the impact of maternal overnutrition (excess intake with weight gain leading to overweight or obesity) on epigenetics and the later propensity to NCDs. Studies show that both deprivation and excess in-utero change normal growth patterns and increase the risk of obesity, diabetes and metabolic disorders later in life. Thus, maternal obesity is associated with health problems for women and their offspring. If a woman is obese before or after conception, she is at greater risk of pregnancy-related complications, gestational hypertension and diabetes, and these risks are, in turn, associated with babies being large for their gestational age and with changes in how they metabolise sugar and fats. The BMI of the mother prior to pregnancy is associated with higher risk of obesity throughout the lifespan and related metabolic disorders. There is also some evidence to suggest that a high-fat diet and excess energy intake during pregnancy (independent of maternal obesity) can lead to changes that increase risk of CVD in offspring. Furthermore, there is some evidence that programming of taste preferences can take place during pregnancy if the maternal diet is high in sugar and/or fat[257,258] and that the more variable the mother's diet during pregnancy or breastfeeding the greater later acceptance of new foods/flavours in the child.[259]

Although understanding of the mechanisms involved in these changes is growing, the mechanisms and the extent of the impact of such programming are not yet fully clear. Figure 24 illustrates some of the pathways involved in the programming of obesity and NCDs.

Figure 24 Nutritional stressors involved in metabolic programming of obesity and NCDs

Source: Adapted from World Health Organization Regional Office for Europe, 2016.255



Tackling an intergenerational cycle of malnutrition seems therefore important when addressing socio-economic inequalities in health. Interventions to improve maternal nutrition status, therefore, may be an effective contributor to reduce health inequalities in future generations.

Optimal infant and young child nutrition is also important for the prevention of CVD, through protection against cardiovascular risk factors such as overweight, obesity and diabetes later in life.[260,261] WHO recommends early initiation of breastfeeding (within the first hour of birth), on-demand exclusive breastfeeding from birth until six months of age followed by timely introduction of appropriate complementary feeding and continued breastfeeding up to two years of age or beyond. A supportive policy environment is essential to promote and protect breastfeeding and appropriate complementary feeding and to protect against cardiovascular risk factors and to nurture healthy food preferences in young children (See Chapter 4).

References

1. Sofi, F., Abbate, R., Gensini, G. F. & Casini, A. Accruing evidence on benefits of adherence to the Mediterranean diet on health : an updated systematic review and meta-analysis 1, 2. *Am. J. Clin. Nutr.* 92, 1189–1196 (2010).
2. Estruch, R. *et al.* Primary prevention of cardiovascular disease with a Mediterranean diet. *N. Engl. J. Med.* 368, 1279–90 (2013).
3. Kris-etherton, P., Eckel, R. H. & Howard, B. V. Lyon Diet Heart Study Benefits of a Mediterranean-Style, National Cholesterol Education Program/American Heart Association Step I Dietary Pattern on Cardiovascular Disease AHA Science Advisory. *Clin. Sci.* 8721, 1823–1825 (2001).
4. Appel, L. *et al.* A Clinical Trial of the Effects of Dietary Patterns on Blood Pressure. *NEJM* 336, 1117–1124 (1997).
5. Sacks, F. *et al.* Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med* 344, 3–10 (2001).
6. Appel, L. J. *et al.* Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA* 294, 2455–2464 (2005).
7. Nordic Council of Ministers. *Nordic Nutrition Recommendations 2012. Integrating nutrition and physical activity. Nordic Nutrition Recommendations 2012* (2012). doi:10.6027/Nord2014-002
8. U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020 Dietary Guidelines for Americans. 8th Edition. (2015).
9. Wang, X. *et al.* Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. *Br. Med. J.* 349, g4490 (2014).
10. Hu, D., Huang, J., Wang, Y., Zhang, D. & Qu, Y. Fruits and Vegetables Consumption and Risk of Stroke: A Meta-Analysis of Prospective Cohort Studies. *Stroke* 45, 1613–1619 (2014).
11. Hauner, H. *et al.* Evidence-based guideline of the German Nutrition Society: carbohydrate intake and prevention of nutrition-related diseases. *Ann Nutr Metab* 60 Suppl 1, 1–58 (2012).
12. Programme National Nutrition Santé. *La santé vient en mangeant. Le guide alimentaire pour tous.* (2011).
13. Vos, M. B. *et al.* Added Sugars and Cardiovascular Disease Risk in Children: A Scientific Statement From the American Heart Association. *Circulation* 136, (2016).
14. Piepoli, M. F. *et al.* 2016 European Guidelines on cardiovascular disease prevention in clinical practice. *Eur. Heart J.* 28, 2375–414 (2016).
15. World Health Organization. Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation. *World Heal. Organ.* 8–11 (2008). doi:10.1038/ejcn.2009.139
16. Eckel, R. H. *et al.* AHA/ACC guideline on lifestyle management to reduce cardiovascular risk. A report of the American College of Cardiology / American Heart Association task force on practice guidelines. *Circulation* 129, S76–99 (2014).
17. Piepoli, M. F. *et al.* European Guidelines on cardiovascular disease prevention in clinical practice (version . The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by represe. *Eur Hear. J* 37, 2315–2381 (2016).
18. WHO. Diet, nutrition and the prevention of chronic diseases. *World Health Organ. Tech. Rep. Ser.* 916, i–viii-1-149-backcover (2003).
19. EFSA Panel on Dietetic Products Nutrition and Allergies. Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated fatty acids, monounsaturated fatty acids, trans fatty acids, and cholesterol. *EFSA J.* 8, 1461 (2010).
20. World Health Organization. *Obesity: Preventing and managing the global epidemic. Report of a WHO consultation.* (2000).
21. Nordic Council of Ministers. Nordic Nutrition Recommendations 2012. (2012).
22. Scientific Advisory Committee on Nutrition (SACN). *Carbohydrates and Health.* (2015).
23. European Food Safety Authority. Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre. *EFSA J.* 8, 1–77 (2010).
24. World Health Organization. *Guideline: Sugars intake for adults and children. World Health Organisation* (2014).
25. World Health Organization. Sodium intake for adults and children. (2012).
26. World Health Organization. *Global action plan for the prevention and control of noncommunicable diseases 2013–2020.* (2013).
27. WHO/FAO/UNU Expert Consultation. Protein and amino acid requirements in human nutrition. *World Health Organ. Tech. Rep. Ser.* 1–265 (2007). doi:ISBN 92 4 120935 6
28. Lankinen, M. & Schwab, U. Biomarkers of dairy fat. *Am. J. Clin. Nutr.* 101, 1101–1102 (2015).
29. Ratnayake, W. Concerns about the use of 15:0, 17:0, and trans-16:1n–7 as biomarkers of dairy fat intake in recent observational studies that suggest beneficial effects of dairy food on incidence of diabetes and stroke. *Am J Clin Nuir* 101, 1102–1103 (2015).
30. Weitkunat, K. *et al.* Odd-chain fatty acids as a biomarker for dietary fiber intake: a novel pathway for endogenous production from propionate. *Am. J. Clin. Nutr.* 105, 1544–1551 (2017).
31. European Food Safety Authority. *Dietary reference values for water. Scientific opinion of the panel on dietetic products, nutrition and allergies.* (2008).
32. Ha, V. *et al.* Effect of dietary pulse intake on established therapeutic review and meta-analysis of randomized controlled trials. *Can. Med. Assoc. J.* 186, 252–262 (2014).
33. Rebello, C. J., Greenway, F. L. & Finley, J. W. Whole grains and pulses: A comparison of the nutritional and health benefits. *J. Agric. Food Chem.* 62, 7029–7049 (2014).
34. Ozogul, Y., Ozogul, F., Cicek, E., Polat, A. & Kuley, E. Fat content and fatty acid compositions of 34 marine water fish species from the Mediterranean Sea. *Int J Food Sci Nutr* 60, 464–475 (2009).
35. Aggelousis, G. & Lazos, E. S. Fatty acid composition of the lipids from eight freshwater fish species from Greece. *J Comp Anal* 4, 68–76 (1991).
36. Saadatian-Elahi, M. *et al.* Plasma phospholipid fatty acid profiles and their association with food intakes: results from a cross-sectional study within the European Prospective Investigation into Cancer and Nutrition. *Am Nutr* 89, 331–346 (2009).
37. Vartiainen, E. *et al.* Thirty-five-year trends in cardiovascular risk factors in Finland. *Int J Epidemiol.* 39, 504–518 (2010).
38. Valsta, L. *et al.* Männistö S, Explaining the 25-year decline of serum cholesterol by dietarty changes and use of lipid-lowering medication in Finland. *Public Health Nutr.* 13, 932–938 (2010).
39. European Heart Network. *Diet, physical activity and cardiovascular disease prevention in Europe.* (2011).

40. World Health Organization. *Guideline: sugars intake in adults and children*. (2015).
41. Voedingscentrum. *Richtlijnen Schijf van Vijf*. (2016).
42. US Dept Health and Human Services & US Dept Agriculture. Scientific Report of the 2015 Dietary Guidelines Committee. 4, 1–11 (2015).
43. Nordic Nutrition Recommendations. *Integr. Nutr. Phys. Act. 5th Ed. Nord. Counc. Minist. Nord 002* 2014 SRC, (2012).
44. Dietary Guidelines Advisory Committee. *Scientific Report of the 2015 Dietary Guidelines Advisory Committee*. (2015).
45. Johnson, R. K. *et al.* Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation* 120, 1011–1020 (2009).
46. Sanders, T. A. B. How important is the relative balance of fat and carbohydrate as sources of energy in relation to health? *Proc Nutr Soc* 75, 147–153 (2016).
47. Paganini-Hill, A., Kawas, C. H. & Corrada, M. M. Non-alcoholic beverage and caffeine consumption and mortality: the Leisure World Cohort Study. *Prev Med* 44, 305–310 (2007).
48. Tasevska, N. *et al.* Sugars and risk of mortality in the NIH-AARP Diet and Health Study. *Am J Clin Nutr* 99, 1077–1088 (2014).
49. Yang, Q. *et al.* Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* 174, 516–524 (2014).
50. Sonestedt, E., Overby, N. C., Laaksonen, D. E. & Birgisdottir, B. E. Does high sugar consumption exacerbate cardiometabolic risk factors and increase the risk of type 2 diabetes and cardiovascular disease? *Food Nutr Res* 56, (2012).
51. Huang, C., Huang, J., Tian, Y., Yang, X. & Gu, D. Sugar sweetened beverages consumption and risk of coronary heart disease: a meta-analysis of prospective studies. *Atherosclerosis* 234, 11–16 (2014).
52. Xi, B. *et al.* Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. *Br J Nutr* 113, 709–717 (2015).
53. Li, Y. *et al.* Saturated Fats Compared With Unsaturated Fats and Sources of Carbohydrates in Relation to Risk of Coronary Heart Disease: Cohort Study. *J Am Coll Cardiol* 66 SRC-G, 1538–1548 (2015).
54. Te Morenga, L. A., Howatson, A. J., Jones, R. M. & Mann, J. Dietary sugars and cardiometabolic risk: systematic review and meta-analyses of randomized controlled trials of the effects on blood pressure and lipids. *Am J Clin Nutr* 100, 65–79 (2014).
55. Jayalath, V. H. *et al.* Sugar-sweetened beverage consumption and incident hypertension: a systematic review and meta-analysis of prospective cohorts. *Am J Clin Nutr* 102, 914–921 (2015).
56. Malik, A. H., Akram, Y., Shetty, S., Malik, S. S. & Yanchou Njike, V. Impact of sugar-sweetened beverages on blood pressure. *Am J Cardiol* 113, 1574–1580 (2014).
57. Keller, A., Heitmann, B. L. & Olsen, N. Sugar-sweetened beverages, vascular risk factors and events: a systematic literature review. *Public Heal. Nutr* 18, 1145–1154 (2015).
58. Ha, V. *et al.* Effect of fructose on blood pressure: a systematic review and meta-analysis of controlled feeding trials. *Hypertension* 59, 787–795 (2012).
59. Perez-Pozo, S. E. *et al.* Excessive fructose intake induces the features of metabolic syndrome in healthy adult men: role of uric acid in the hypertensive response. *Int J Obes* 34, 454–461 (2010).
60. Teunissen-Beekman, K. F. *et al.* Protein supplementation lowers blood pressure in overweight adults: effect of dietary proteins on blood pressure (PROPRES), a randomized trial. *Am J Clin Nutr* 95, 966–971 (2012).
61. Lowndes, J. *et al.* The effect of normally consumed amounts of sucrose or high fructose corn syrup on lipid profiles, body composition and related parameters in overweight/obese subjects. *Nutrients* 6, 1128–1144 (2014).
62. Raatz, S. K., Johnson, L. K. & Picklo, M. J. Consumption of Honey, Sucrose, and High-Fructose Corn Syrup Produces Similar Metabolic Effects in Glucose-Tolerant and -Intolerant Individuals. *J Nutr* 145, 2265–2272 (2015).
63. Lustig, R. H. *et al.* Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. *Obesity* 24, 453–460 (2016).
64. Malik, V. S. *et al.* Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care* 33, 2477–2483 (2010).
65. Greenwood, D. C. *et al.* Association between sugar-sweetened and artificially sweetened soft drinks and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. *Br J Nutr* 112, 725–734 (2014).
66. Imamura, F. *et al.* Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction. *BMJ* 351, h3576 (2015).
67. Ahmadi-Abhari, S. *et al.* Dietary intake of carbohydrates and risk of type 2 diabetes: the European Prospective Investigation into Cancer-Norfolk study. *Br J Nutr* 111, 342–352 (2014).
68. Morenga, L., Mallard, S. & Mann, J. Te. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ e7492* 346 SRC-, (2013).
69. Sievenpiper, J. L., de Souza, R. J. & Mirrahimi, A. Effect of fructose on body weight in controlled feeding trials: a systematic review and meta-analysis. *Ann Intern Med* 156, 291–304 (2012).
70. Pollock, N. K. *et al.* Greater fructose consumption is associated with cardiometabolic risk markers and visceral adiposity in adolescents. *J. Nutr.* 142, 251–257
71. Lee, A. K. Sugars and adiposity: the long-term effects of consuming added and naturally occurring sugars in foods and in beverages. *Obes. Sci. Pract.* 1, 41–49 (2015).
72. Woodward-Lopez, G., Kao, J. & Ritchie, L. To what extent have sweetened beverages contributed to the obesity epidemic? *Public Health Nutr.* 14, 499–509
73. Mattes, R. D., Shikany, J. M., Kaiser, K. A. & Allison, D. B. Nutritively sweetened beverage consumption and body weight: a systematic review and meta-analysis of randomized experiments. *Obes Rev* 12, 346–365 (2011).
74. Malik, V. S., Pan, A., Willett, W. C. & Hu, F. B. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. *Am. J. Clin. Nutr.* 98, 1084–1102
75. Kaiser, K. A., Shikany, J. M., Keating, K. D. & Allison, D. B. Will reducing sugar-sweetened beverage consumption reduce obesity? Evidence supporting conjecture is strong, but evidence when testing effect is weak. *Obes. Rev.* 14, 620–633
76. Pereira, M. A. Sugar-sweetened and artificially-sweetened beverages in relation to obesity risk. *Adv Nutr* 5, 797–808 (2014).
77. Massougbodji, J., Le Bodo, Y., Fratu, R. & De Wals, P. Reviews examining sugar-sweetened beverages and body weight: correlates of their quality and conclusions. *Am. J. Clin. Nutr.* 99, 1096–1104
78. Keller, A. & Bucher Della Torre, S. Sugar-Sweetened Beverages and Obesity among Children and Adolescents: A Review of Systematic Literature Reviews. *Child. Obes.* 11, 338–346
79. de Ruyter, J. C., Olthof, M. R., Kuijper, L. D. & Katan, M. B. Effect of sugar-sweetened beverages on body weight in children: design and baseline characteristics of the Double-blind, Randomized INtervention study in Kids. *Contemp Clin Trials* 33, 247–257 (2012).
80. Lowndes, J., Sinnett, S., Yu, Z. & Rippe, J. The effects of fructose-containing sugars on weight, body composition and cardiometabolic risk factors when consumed at up to the 90th percentile population consumption level for fructose. *Nutrients* 6, 3153–3168 (2014).
81. Ma, J. *et al.* Sugar-Sweetened Beverage Consumption Is Associated With Change of Visceral Adipose Tissue Over 6 Years of Follow-Up. *Circulation* 133, 370–377 (2016).
82. Targher, G., Byrne, C. D., Lonardo, A., Zoppini, G. & Barbui, C. Non-alcoholic fatty liver disease and risk of incident cardiovascular disease: A meta-analysis. 65, 589–600 (2016).
83. Tappy, L. & Lê, K.-A. Does fructose consumption contribute to non-alcoholic fatty liver disease? *Clin. Res. Hepatol. Gastroenterol.* 36, 554–560
84. Vos, M. B. & Lavine, J. E. Dietary fructose in nonalcoholic fatty liver disease. *Hepatology* 57, 2525–2531

85. Jin, R. *et al.* Dietary fructose reduction improves markers of cardiovascular disease risk in Hispanic-American adolescents with NAFLD. *Nutrients* 6, 3187–3201
86. Report, A. I. F. & Panel, I. I. I. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment final report. *Circulation* 106, 3143–3421 (2002).
87. Chiavaroli, L., Livesey, G., Taylor, R., Hulshof, T. & Howlett, J. Effect of fructose on established lipid targets: a systematic review and meta-analysis of controlled feeding trials. *Hear. Assoc 4 e001700 Glycemic response Heal. Syst. Rev. metaanalysis relations between Diet. glycemic Prop. Heal. outcomes Am Nutr S* 87, 258S–68 (2008).
88. Livesey, G., Taylor, R., Hulshof, T. & Howlett, J. Glycemic response and health – a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. *Am J Clin Nutr* 87, 258S–268S (2008).
89. Sievenpiper, J. L. *et al.* Heterogeneous effects of fructose on blood lipids in individuals with type 2 diabetes: systematic review and meta-analysis of experimental trials in humans. *Diabetes Care* 32, 1930–1937
90. Grundy, S. M. *et al.* Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Arterioscler. Thromb. Vasc. Biol.* 24, e13–8
91. Rosset, R., Surowska, A. & Tappy, L. Pathogenesis of Cardiovascular and Metabolic Diseases: Are Fructose-Containing Sugars More Involved Than Other Dietary Calories? Curr Hypertens Rep ; : 44. 18, (2016).
92. Carran, E. L., White, S. J., Reynolds, A. N., Haszard, J. J. & Venn, B. J. Acute effect of fructose intake from sugar-sweetened beverages on plasma uric acid: a randomised controlled trial. *Eur Nutr* 70, 1034–1038 (2016).
93. Bruun, J. M., Maersk, M., Belza, A., Astrup, A. & Richelsen, B. Consumption of sucrose-sweetened soft drinks increases plasma levels of uric acid in overweight and obese subjects: a 6-month randomised controlled trial. *Eur J Clin Nutr* 69, 949–953 (2015).
94. Borghi, C. The role of uric acid in the development of cardiovascular disease. *Curr Med Res Opin* 31 Suppl 2, 1–2 (2015).
95. Caliceti, C., Calabria, D., Roda, A. & Cicero, A. F. G. Fructose Intake, Serum Uric Acid, and Cardiometabolic Disorders: A Critical Review. *Nutrients* 9, (2017).
96. Mapanga, R. F. & Essop, M. F. Damaging effects of hyperglycemia on cardiovascular function: spotlight on glucose metabolic pathways. *Am J Physiol Hear. Circ Physiol* 310, H153–73 (2016).
97. Mah, E. & Bruno, R. S. Postprandial hyperglycemia on vascular endothelial function: mechanisms and consequences. *Nutr. Res.* 32, 727–740
98. Teunissen-Beekman, K. F. M. *et al.* Dietary proteins improve endothelial function under fasting conditions but not in the postprandial state, with no effects on markers of low-grade inflammation. *Br. J. Nutr.* 114, 1819–1828
99. Cirillo, P. *et al.* Fructose induces prothrombotic phenotype in human endothelial cells : A new role for 'added sugar' in cardio-metabolic risk. *J. Thromb. Thrombolysis* 40, 444–451
100. Ares, G. R. & Ortiz, P. A. Direct renal effects of a fructose-enriched diet: interaction with high salt intake. *Am Regul Integr Comp Physiol* 309, R1078–81 (2015).
101. Qi, Q. *et al.* Sugar-sweetened beverages and genetic risk of obesity. *N. Engl. J. Med.* 367, 1387–1396
102. Olsen, N. J. *et al.* Interactions between genetic variants associated with adiposity traits and soft drinks in relation to longitudinal changes in body weight and waist circumference. *Am. J. Clin. Nutr.* 104, 816–826
103. Zheng, Y. *et al.* Sugar-sweetened beverage intake, chromosome 9p21 variants, and risk of myocardial infarction in Hispanics. *Am. J. Clin. Nutr.* 103, 1179–1184
104. Hooper, L. *et al.* Effects of total fat intake on body weight. *Cochrane Database Syst Rev* 8, CD011834.b (2015).
105. Siri-Tarino, P. W., Sun, Q., Hu, F. B. & Krauss, R. M. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *Am. J. Clin. Nutr.* 91, 535–546 (2010).
106. Mozaffarian, D., Micha, R. & Wallace, S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *PLoS Med.* 7, e1000252 (2010).
107. Schwab, U. *et al.* Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing type-2 diabetes, cardiovascular disease, and cancer: a systematic review. *Food Nutr Res 25145* 58, (2014).
108. Forouhi, N. G. *et al.* Differences in the prospective association between individual plasma phospholipid saturated fatty acids and incident type 2 diabetes: the EPIC-InterAct case-cohort study. *lancet. Diabetes Endocrinol.* 2, 810–818 (2014).
109. Astrup, A. A changing view on saturated fatty acids and dairy: from enemy to friend. *The American journal of clinical nutrition* 100, 1407–1408 (2014).
110. Zong, G. *et al.* Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies. *BMJ (British Med. Journal)* 355, i5796 (2016).
111. Chen, M. *et al.* Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. *Am. J. Clin. Nutr.* 104, 1209–1217 (2016).
112. Keys, A. *Seven countries. A multivariate analysis of death and coronary heart disease.* (Harvard University Press, 1980).
113. Johnstone, A. M. Safety and efficacy of high-protein diets for weight loss. *Proc Nutr Soc* 71, 339–349 (2012).
114. Hopkins, M., Gibbons, C., Caudwell, P., Blundell, J. E. & Finlayson, G. Differing effects of high-fat or high-carbohydrate meals on food hedonics in overweight and obese individuals. *Br J Nutr* 115, 1875–1884 (2016).
115. Hooper, L., Martin, N., Abdelhamid, A. & Davey Smith, G. Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst. Rev.* (2015).
116. Stubbs, R. J., Harbron, C. G. & Prentice, A. M. Covert manipulation of the dietary fat to carbohydrate ratio of isoenergetically dense diets: effect on food intake in feeding men ad libitum. *Int Relat Metab Disord* 20, 651–660 (1996).
117. Bell, E. A. & Rolls, B. J. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am. J. Clin. Nutr.* 73, 1010–1018
118. Stubbs, R. J., Harbron, C. G., Murgatroyd, P. R. & Prentice, A. M. Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. *Am Nutr* 62, 316–329 (1995).
119. Bray, G. A. & Popkin, B. M. Dietary fat intake does affect obesity! *Am. J. Clin. Nutr.* 68, 1157–1173 (1998).
120. Poppitt, S. D. & Prentice, A. M. Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite* 26, 153–174 (1996).
121. Prentice, A. M. & Jebb, S. A. Fast foods, energy density and obesity: a possible mechanistic link. *Obes. Rev.* 4, 187–194 (2003).
122. Rouhani, M. H., Haghighatdoost, F., Surkan, P. J. & Azadbakht, L. Associations between dietary energy density and obesity: A systematic review and meta-analysis of observational studies. *Nutrition* 32, 1037–1047 (2016).
123. Absetz, P. *et al.* Type 2 diabetes prevention in the 'real world': one-year results of the GOAL Implementation Trial. *Diabetes Care* 30, 2465–2470 (2007).
124. Ferro-Luzzi, A. & Branca, F. Mediterranean diet, Italian-style: prototype of a healthy diet. *Am J Clin Nutr* 61, 1338S–1345 (1995).
125. Ferro-Luzzi, A., James, W. P. T. & Kafatos, A. The high-fat Greek diet: a recipe for all? *Eur. J. Clin. Nutr.* 56, 796–809 (2002).
126. Fidanza, F., Puddu, V., Veccio, A. & Keys, A. Men in rural Italy. *Acta Med Scand* S460, 116–146 (1996).
127. Klem, M., Wing, R., Lang, W., McGuire, M. & Hill, J. Does weight loss maintenance become easier over time? *Obes Res* 8, 438–444 (2000).
128. Raynor, H. A. *et al.* Van Dietary energy density and successful weight loss maintenance. *Eat Behav* 12, 119–125 (2011).

129. Teslovich, T. M. *et al.* Biological, clinical and population relevance of 95 loci for blood lipids. *Nature* 466, 707–713 (2010).
130. Waterworth, D. M. *et al.* Genetic variants influencing circulating lipid levels and risk of coronary artery disease. *Arter. Thromb Vasc Biol* 30, 2264–2276 (2010).
131. Deloukas, P. *et al.* Large-scale association analysis identifies new risk loci for coronary artery disease. *Nat Genet* 45, 25–33 (2013).
132. Joris, P. J. & Mensink, R. P. Role of cis-Monounsaturated Fatty Acids in the Prevention of Coronary Heart Disease. *Curr Atheroscler Rep* 18, (2016).
133. Mensink, R. P., Zock, P. L., Kester, A. D. M. & Katan, M. B. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am. J. Clin. Nutr.* 77, 1146–1155 (2003).
134. Brouwer, I. A., Wanders, A. J. & Katan, M. B. Effect of animal and industrial trans fatty acids on HDL and LDL cholesterol levels in humans – a quantitative review. *PLoS One* 5, e9434 (2010).
135. Gebauer, S. K., Destaillats, F., Dionisi, F., Krauss, R. M. & Baer, D. J. Vaccenic acid and trans fatty acid isomers from partially hydrogenated oil both adversely affect LDL cholesterol: a double-blind, randomized controlled trial. *Am J Clin Nutr* 102 SRC-, 1339–1346 (2015).
136. Tholstrup, T., Hoy, C.-E., Andersen, L. N., Christensen, R. D. K. & Sandstrom, B. Does fat in milk, butter and cheese affect blood lipids and cholesterol differently? *J. Am. Coll. Nutr.* 23, 169–176 (2004).
137. Forouhi, N. G. *et al.* Association of Plasma Phospholipid n-3 and n-6 Polyunsaturated Fatty Acids with Type 2 Diabetes: InterAct Case-Cohort Study. *PLoe100 doi101371journalpmed100 eCollection* 13, (2016).
138. Imamura, F. *et al.* Effects of Saturated Monounsaturated Fat, and Carbohydrate on Glucose-Insulin Homeostasis: Review and Meta-analysis of Randomised Controlled Feeding Trials. *PLoS Med.* 13, e1002087 (2016).
139. Niinikoski, H. *et al.* Blood pressure is lower in children and adolescents with a low-saturated-fat diet since infancy: the special turku coronary risk factor intervention project. *Hypertension* 53, 918–924 (2009).
140. Bos, M. B. *et al.* Effect of a high monounsaturated fatty acids diet and a Mediterranean diet on serum lipids and insulin sensitivity in adults with mild abdominal obesity. *Nutr. Metab. Cardiovasc. Dis.* 20, 591–598 (2010).
141. Rasmussen, B. M. *et al.* Effects of dietary saturated, monounsaturated, and n-3 fatty acids on blood pressure in healthy subjects. *Am Nutr* 83, 221–226 (2006).
142. Skeaff, C. M. & Miller, J. Dietary fat and coronary heart disease: summary of evidence from prospective cohort and randomised controlled trials. *Ann Nutr Metab* 55, 173–201 (2009).
143. Jakobsen, M. U. *et al.* Major types of dietary fats and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *J Am Clin Nutr* 89, 1425–1433 (2009).
144. Astrup, A. *et al.* The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010? *Am J Clin Nutr* 93, 684–688 (2011).
145. Jakobsen, M. U. *et al.* Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *Am. J. Clin. Nutr.* 91, 1764–1768 (2010).
146. Warensjo, E. *et al.* Biomarkers of milk fat and the risk of myocardial infarction in men and women: a prospective, matched case-control study. *Am. J. Clin. Nutr.* 92, 194–202 (2010).
147. Pfeuffer, M. & Jaudszus, A. Pentadecanoic and Heptadecanoic Acids: Multifaceted Odd-Chain Fatty Acids. *Adv. Nutr.* 7, 730–734 (2016).
148. Imamura, F. *et al.* Novel circulating fatty acid patterns and risk of cardiovascular disease: the Cardiovascular Health Study. *Am J Clin Nutr.* 96, 1252–1261 (2012).
149. Brouwer, I. A. *Effects of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis.* (2016).
150. Schwingshackl, L. & Hoffmann, G. Monounsaturated fatty acids and risk of cardiovascular disease: synopsis of the evidence available from systematic reviews and meta-analyses. *Nutrients* 4, 1989–2007 (2012).
151. Wang, Q. *et al.* Impact of Nonoptimal Intakes of Saturated, Polyunsaturated, and Trans Fat on Global Burdens of Coronary Heart Disease. *J. Am. Heart Assoc.* 5, e002891 (2016).
152. Sundstrom, J. *et al.* Dyslipidemia and an unfavorable fatty acid profile predict left ventricular hypertrophy 20 years later. *Circulation* 103, 836–841 (2001).
153. Ramsden, C. E., Hibbeln, J. R., Majchrzak, S. F. & Davis, J. M. n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials. *Br* 104, 1586–1600 (2010).
154. Trikalinos, T. A. *et al.* Concordance of randomized and nonrandomized studies was unrelated to translational patterns of two nutrient-disease associations. *Epidemiol* 65, 16–29 (2012).
155. Strom, M., Halldorsson, T. I., Mortensen, E. L., Torp-Pedersen, C. & Olsen, S. F. Fish, n-3 fatty acids, and cardiovascular diseases in women of reproductive age: a prospective study in a large national cohort. *Hypertens. (Dallas, Tex. 1979)* 59, 36–43 (2012).
156. Wu, J. H. Y. *et al.* Omega-3 fatty acids and incident type 2 diabetes: a systematic review and meta-analysis. *Br. J. Nutr.* 107 Suppl, S214–27 (2012).
157. Pan, A. *et al.* alpha-Linolenic acid and risk of cardiovascular disease: a systematic review and meta-analysis. *Am J Clin Nutr* 96, 1262–1273 (2012).
158. Mozaffarian, D. *et al.* Interplay between different polyunsaturated fatty acids and risk of coronary heart disease in men. *Circulation* 111, 157–164 (2005).
159. Farvid, M. S. *et al.* Dietary linoleic acid and risk of coronary heart disease: A systematic review and meta-analysis of prospective cohort studies. *Circulation* 130, 1568–1578 (2014).
160. World Health Assembly. *Sixty-sixth World Health Assembly, follow-up to the Political Declaration of the High-Level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases.* (2013).
161. Powles, J., Fahimi, S. & Micha, R. Global, regional and national sodium intakes in 1990 and 2010 a systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide. *BMJ Open* 3, (2013).
162. Hendriksen, M. A. H., van Raaij, J. M. A., Geleijnse, J. M., Breda, J. & Boshuizen, H. C. Health gain by salt reduction in europe: a modelling study. *PLoS One* 10, e0118873 (2015).
163. Webb, M. *et al.* Cost effectiveness of a government supported policy strategy to decrease sodium intake: global analysis across 183 nations. *BMJ* 356, i6699 (2017).
164. Gijsbers, L. *et al.* Effects of sodium and potassium supplementation on blood pressure and arterial stiffness: a fully controlled dietary intervention study. *J. Hum. Hypertens.* 29, 592–598 (2015).
165. Mozaffarian, D. *et al.* Global sodium consumption and death from cardiovascular causes. *N. Engl. J. Med.* 371, 624–634 (2014).
166. Cappuccio, F. P. & Capewell, S. Facts, issues and controversies in salt reduction for the prevention of cardiovascular disease. *Funct Food Rev* 7, 41–61 (2015).
167. Grobbee, D. E. & Hofman, A. Does sodium restriction lower blood pressure? *Br. Med. J. (Clin. Res. Ed).* 293, 27–29 (1986).
168. Midgley, J. P., Matthew, A. G., Greenwood, C. M. & Logan, A. G. Effect of reduced dietary sodium on blood pressure: a meta-analysis of randomized controlled trials. *JAMA* 275, 1590–1597 (1996).
169. Graudal, N. A., Galløe, A. M. & Garred, P. Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride: a meta-analysis. *JAMA* 279, 1383–1391 (1998).
170. He, F. J. & MacGregor, G. A. Effect of modest salt reduction on blood pressure: a meta-analysis of randomized trials. Implications for public health. *J. Hum. Hypertens.* 16, 761–770 (2002).

171. Geleijnse, J. M., Kok, F. J. & Grobbee, D. E. Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomised trials. *J. Hum. Hypertens.* 17, 471–480 (2003).
172. Graudal, N. A., Hubeck-Graudal, T. & Jurgens, G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. *Cochrane Database Syst Rev* CD004022 (2011).
173. Graudal, N. A., Hubeck-Graudal, T. & Jürgens, G. Effects of low-sodium diet vs. high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). *Am. J. Hypertens.* 25, 1–15 (2012).
174. Jurgens, G. & Graudal, N. A. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride. *Cochrane Database Syst Rev* 1, CD004022 (2004).
175. He, F., Li, J. & MacGregor, G. Effect of longer term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomized trials. *Br Med J* 346, f1325 (2013).
176. Aburto, N. *et al.* Effect of lower sodium intake on health: systematic review and meta-analyses. *Br Med J* 346, f1326 (2013).
177. He, F. J. & MacGregor, G. A. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. *Hypertension* 48, 861–869 (2006).
178. He, F. J., Marrero, N. M. & Macgregor, G. A. Salt and blood pressure in children and adolescents. *J. Hum. Hypertens.* 22, 4–11 (2008).
179. Campese, V. M. Salt sensitivity in hypertension. Renal and cardiovascular implications. *Hypertension* 23, 531–550 (1994).
180. Luft, F. C. *et al.* Salt sensitivity and resistance of blood pressure. Age and race as factors in physiological responses. *Hypertension* 17, I102–8 (1991).
181. Cappuccio, F. P., Markandu, N. D., Carney, C., Sagnella, G. A. & MacGregor, G. A. Double-blind randomised trial of modest salt restriction in older people. *Lancet (London, England)* 350, 850–854 (1997).
182. Rocchini, A. P. *et al.* Insulin and renal sodium retention in obese adolescents. *Hypertension* 14, 367–374 (1989).
183. He, J. *et al.* Dietary sodium intake and subsequent risk of cardiovascular disease in overweight adults. *JAMA* 282, 2027–2034 (1999).
184. Strazzullo, P. *et al.* Altered renal sodium handling in men with abdominal adiposity: a link to hypertension. *J. Hypertens.* 19, 2157–2164 (2001).
185. Luft, F., Miller, J., Weinberger, M., Christian, J. & Skrabal, F. Genetic influences on the response to dietary salt reduction: acute salt loading or salt depletion in humans. *J Cardiovasc Pharmacol* 12, S49–55 (1988).
186. Weinberger, M., Miller, J., Luft, F., Grim, C. & Fineberg, N. Definitions and characteristics of sodium sensitivity and blood pressure resistance. *Hypertension* 8, II127–34 (1986).
187. Cappuccio, F., Markandu, N., Sagnella, G. & MacGregor, G. Sodium restriction lowers high blood pressure through a decreased response of the renin system – direct evidence using saralasin. *J Hypertens* 3, 243–7 (1985).
188. Barba, G. *et al.* Renal function and blood pressure response to dietary salt restriction in normotensive men. *Hypertension* 27, 1160–4 (1996).
189. Cappuccio, F. *et al.* Renal tubular sodium handling and plasma atrial natriuretic peptide, renin activity and aldosterone in untreated men under normal living conditions. *Eur J Clin Invest* 21, 40–46 (1991).
190. Folkerd, E. *et al.* Clearance of endogenous lithium in humans: altered dietary salt intake and comparison with exogenous lithium clearance. *Am J Physiol* 269, F718–22. (1995).
191. Chiolero, A., Maillard, M., Nussberger, J., Brunner, H. & Burnier, M. Proximal sodium reabsorption: an independent determinant of blood pressure response to salt. *Hypertension* 36, 631–7 (2000).
192. Cwynar, M., Stompor, T., Barton, H. & Grodzicki, T. Endogenous lithium clearance: a diagnostic method of assessing sodium sensitivity in hypertension. Methodological and clinical implications. 2014; 72: 1–7. *Kardiol. Pol.* 72, 1–7 (2014).
193. Cappuccio, F., Strazzullo, P., Siani, A. & Trevisan, M. Increased proximal sodium reabsorption is associated with increased cardiovascular risk in men. *J Hypertens* 14, 909–14 (1996).
194. Cappuccio, F., Strazzullo, P., Farinaro, E. & Trevisan, M. Uric acid metabolism and tubular sodium handling. Results from a population-based study. *JAMA* 270, 354–9 (1993).
195. Barbato, A. *et al.* Metabolic syndrome and renal sodium handling in three ethnic groups living in England. *Diabetologia* 47, 40–6 (2004).
196. Weinberger, M., Fineberg, N., Fineberg, S. & Weinberger, M. Salt sensitivity: pulse pressure and death in normal and hypertensive humans. *Hypertension* 37, 429–32 (2001).
197. Galletti, F. & Strazzullo, P. The blood pressure-salt sensitivity paradigm: pathophysiologically sound yet of no practical value. *Nephrol. Dial. Transplant.* 31, 1386–1391 (2016).
198. Elijovich, F. *et al.* Salt Sensitivity of Blood Pressure: A Scientific Statement From the American Heart Association. *Hypertension* 68, e7–e46 (2016).
199. Rose, G. Strategy of Preventive Medicine. 171 (2008). doi:10.1093/acprof:oso/9780192630971.001.0001
200. Rose, G. Sick individuals and sick populations. *Int J Epidemiol* 14, 32–38 (1985).
201. Strazzullo, P., D'Elia, L., Kandala, N.-B. & Cappuccio, F. P. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. *BMJ* 339, b4567 (2009).
202. He, F. J. & MacGregor, G. A. Salt reduction lowers cardiovascular risk: meta-analysis of outcome trials. *Lancet* 378, 380–382 (2011).
203. Cobb, L. K. *et al.* Methodological issues in cohort studies that relate sodium intake to cardiovascular disease outcomes: a science advisory from the American Heart Association. *Circulation* 129, 1173–1186 (2014).
204. Stolarz-Skrzypek, K. *et al.* Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. *JAMA* 305, 1777–1785 (2011).
205. He, F. J., Appel, L. J., Cappuccio, F. P., de Wardener, H. E. & MacGregor, G. A. Does reducing salt intake increase cardiovascular mortality? *Kidney Int.* 80, 696–698 (2011).
206. Campbell, N. R. C., Cappuccio, F. P. & Tobe, S. W. Unnecessary controversy regarding dietary sodium: a lot about a little. *Can. J. Cardiol.* 27, 404–406 (2011).
207. Campbell, N., Correa-Rotter, R., Neal, B. & Cappuccio, F. P. New evidence relating to the health impact of reducing salt intake. *Nutr. Metab. Cardiovasc. Dis.* 21, 617–619 (2011).
208. Ji, C., Kandala, N.-B. & Cappuccio, F. P. Spatial variation of salt intake in Britain and association with socioeconomic status. *BMJ Open* 3, (2013).
209. Ji, C. & Cappuccio, F. P. Socioeconomic inequality in salt intake in Britain 10 years after a national salt reduction programme. *BMJ Open* 4, e005683 (2014).
210. Cappuccio, F. P. *et al.* Geographic and socioeconomic variation of sodium and potassium intake in Italy: results from the MINISAL-GIRCSI programme. *BMJ Open* 5, e007467 (2015).
211. Labarthe, D. R. & Briss, P. A. Urinary sodium excretion and cardiovascular disease mortality. *JAMA* 306, 1084–5; author reply 1086 (2011).
212. O'Donnell, M. J., Mente, A. & Rangarajan, S. Urinary sodium and potassium excretion, mortality, and cardiovascular events. *New Engl J Med* 371, 612–23 (2014).
213. Campbell, N. Validation and comparison of three formulae to estimate sodium and potassium excretion from a single-morning fasting urine compared to 24-h measures in 11 countries. *J. Hypertens.* 32, 2499–2500 (2014).
214. Wang, C.-Y. *et al.* Urinary excretion of sodium, potassium, and chloride, but not iodine, varies by timing of collection in a 24-hour calibration study. *J. Nutr.* 143, 1276–1282 (2013).
215. Cogswell, M. E. *et al.* Validity of predictive equations for 24-h urinary sodium excretion in adults aged 18–39 y. *Am. J. Clin. Nutr.* 98, 1502–1513 (2013).
216. Ji, C., Miller, M. A., Venezia, A., Strazzullo, P. & Cappuccio, F. P. Comparisons of spot vs 24-h urine samples for estimating population salt intake: validation study in two independent samples of adults in Britain and Italy. *Nutr. Metab. Cardiovasc. Dis.* 24, 140–147 (2014).

217. Mente, A. *et al.* Validation and comparison of three formulae to estimate sodium and potassium excretion from a single morning fasting urine compared to 24-h measures in 11 countries. *J. Hypertens.* 32, 1005–14; discussion 1015 (2014).

218. Peng, Y. *et al.* Validation and Assessment of Three Methods to Estimate 24-h Urinary Sodium Excretion from Spot Urine Samples in Chinese Adults. *PLoS One* 11, e0149655 (2016).

219. Joosten, M. M. *et al.* Sodium excretion and risk of developing coronary heart disease. *Circulation* 129, 1121–1128 (2014).

220. Cook, N. R., Appel, L. J. & Whelton, P. K. Lower levels of sodium intake and reduced cardiovascular risk. *Circulation* 129, 981–989 (2014).

221. Pfister, R. *et al.* Estimated urinary sodium excretion and risk of heart failure in men and women in the EPIC-Norfolk study. *Eur. J. Heart Fail.* 16, 394–402 (2014).

222. Kalogeropoulos, A. P. *et al.* Dietary sodium content, mortality, and risk for cardiovascular events in older adults: the Health, Aging, and Body Composition (Health ABC) Study. *JAMA Intern. Med.* 175, 410–419 (2015).

223. Yi, B., Titze, J. & Choukèr, A. Dietary Sodium Intake and Risk of Cardiovascular Disease. *JAMA Intern. Med.* 175, 1578–1579 (2015).

224. Liu, Z. & Zhang, X. Dietary Sodium Intake and Risk of Cardiovascular Disease. *JAMA Intern. Med.* 175, 1579 (2015).

225. Science Media Centre. *Expert reaction to paper on salt intake, mortality and cardiovascular disease.* (2016).

226. Lamelas, P. M. *et al.* Association of Urinary Sodium Excretion With Blood Pressure and Cardiovascular Clinical Events in 17,033 Latin Americans. *Am. J. Hypertens.* 29, 796–805 (2016).

227. Cook, N. R., Appel, L. J. & Whelton, P. K. Sodium Intake and All-Cause Mortality Over 20 Years in the Trials of Hypertension Prevention. *J. Am. Coll. Cardiol.* 68, 1609–1617 (2016).

228. Ekinci, E. I. *et al.* Dietary salt intake and mortality in patients with type 2 diabetes. *Diabetes Care* 34, 703–709 (2011).

229. Thomas, M. C. *et al.* The association between dietary sodium intake, ESRD, and all-cause mortality in patients with type 1 diabetes. *Diabetes Care* 34, 861–866 (2011).

230. O'Donnell, M. *et al.* Urinary sodium and potassium excretion and risk of cardiovascular events. *JAMA* 306, 2229–38 (2011).

231. Saulnier, P.-J., Gand, E., Hadjadj, S. & SURDIAGENE Study Group. Sodium and cardiovascular disease. *N. Engl. J. Med.* 371, 2135–2136 (2014).

232. Hadjadj, S. *et al.* Prognostic value of the insertion/deletion polymorphism of the ACE gene in type 2 diabetic subjects: results from the Non-insulin-dependent Diabetes, Hypertension, Microalbuminuria or Proteinuria, Cardiovascular Events, and Ramipril (DIABHYCAR), Diabete d. *Diabetes Care* 31, 1847–1852 (2008).

233. Singer, P., Cohen, H. & Alderman, M. Assessing the associations of sodium intake with long-term all-cause and cardiovascular mortality in a hypertensive cohort. *Am. J. Hypertens.* 28, 335–342 (2015).

234. Äijälä, M. *et al.* Dietary sodium intake and prediction of cardiovascular events. *Eur. J. Clin. Nutr.* 69, 1042–1047 (2015).

235. Mills, K. T. *et al.* Sodium Excretion and the Risk of Cardiovascular Disease in Patients With Chronic Kidney Disease. *JAMA* 315, 2200–2210 (2016).

236. Mente, A. *et al.* Associations of urinary sodium excretion with cardiovascular events in individuals with and without hypertension: a pooled analysis of data from four studies. *Lancet* 388, 465–475 (2016).

237. Cappuccio, F. P. Sodium and cardiovascular disease. *Lancet* 388, 2112 (2016).

238. Cappuccio, F. P. & Campbell, N. R. C. Population Dietary Salt Reduction and the Risk of Cardiovascular Disease: A Commentary on Recent Evidence. *J. Clin. Hypertens.* 19, 4–5 (2017).

239. Lerchl, K. *et al.* Agreement between 24-hour salt ingestion and sodium excretion in a controlled environment. *Hypertens. (Dallas, Tex. 1979)* 66, 850–857 (2015).

240. Ji, C. *et al.* Systematic review of studies comparing 24-hour and spot urine collections for estimating population salt intake. *Rev. Panam. Salud Publica* 32, 307–315 (2012).

241. Katan, M. *et al.* Congruence of individual responsiveness to dietary cholesterol and to saturated fat in humans. *J Lipid Res* 29, 883–92 (1988).

242. Weggemans, R. M., Zock, P. L. & Katan, M. B. Dietary cholesterol from eggs increases the ratio of total cholesterol to high-density lipoprotein cholesterol in humans: A meta-analysis. *Am. J. Clin. Nutr.* 73, 885–891 (2001).

243. Bouvard, V. *et al.* Carcinogenicity of consumption of red and processed meat. *Lancet Oncol.* 16, 1599–1600 (2015).

244. Skeaff, C. M. & Miller, J. Dietary fat and coronary heart diseases: Summary of evidence from prospective cohort and randomised controlled trials. *Ann. Nutr. Metab.* 55, 173–201 (2009).

245. de Souza, R. J. *et al.* Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 351, h3978 (2015).

246. Givens, D. I. The role of animal nutrition in improving the nutritive value of animal derived foods in relation to chronic disease. *Proc. Nutr. Soc.* 64, 395–402 (2005).

247. Micha, R., Michas, G. & Mozaffarian, D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes – an updated review of the evidence. *Curr. Atheroscler. Rep.* 14, 515–524 (2012).

248. Kaluza, J., Wolk, A. & Larsson, S. C. Red meat consumption and risk of stroke: a meta-analysis of prospective studies. *Stroke.* 43, 2556–2560 (2012).

249. Larsson, S. C. & Orsini, N. Red meat and processed meat consumption and all-cause mortality: a meta-analysis. *Am. J. Epidemiol.* 179, 282–289 (2014).

250. World Cancer Research Fund/American Institute for Cancer Research. *Food, nutrition, physical activity and the prevention of cancer: A global perspective.* (2009).

251. Health Council of the Netherlands. *Dutch dietary guidelines 2015.* (2015).

252. Swedish National Food Agency. *Find your way to eat greener, not too much and be active.* (2015).

253. Scientific Advisory Committee on Nutrition. *Iron and Health 2010.* (2010).

254. EAT GOOD The Finnish Dietary Guidelines.

255. World Health Organisation Regional Office for Europe (Institution). Good Maternal Nutrition The best start in life. (2016).

256. WHO. Report of the Commission on Ending Childhood Obesity. *World Heal. Organ.* (2015).

257. De Cosmi, V., Scaglioni, S. & Agostoni, C. Early Taste Experiences and Later Food Choices. *Nutrients* 9, E107 (2017).

258. Trout, K. & Wetzel-Effinger, L. Flavor learning in utero and its implications for future obesity and diabetes. *Curr Diab Rep* 12, 60–6 (2012).

259. Mennella, J. A., Jagnow, C. P. & Beauchamp, G. K. Prenatal and Postnatal Flavor Learning by Human Infants. *Pediatrics* 107, E88–E88 (2001).

260. Horta, B. L., de Mola, C. L. & Victora, C. G. Long-term consequences of breastfeeding on cholesterol, obesity, systolic blood pressure, and type-2 diabetes: systematic review and meta-analysis. *Acta Paediatr.* n/a-n/a (2015). doi:10.1111/apa.13133

261. Yan, J., Liu, L., Zhu, Y., Huang, G. & Wang, P. P. The association between breastfeeding and childhood obesity: A meta-analysis. *BMC Public Health* 14, 1267 (2014).

3 Sustainable food systems for cardiovascular health

Key points

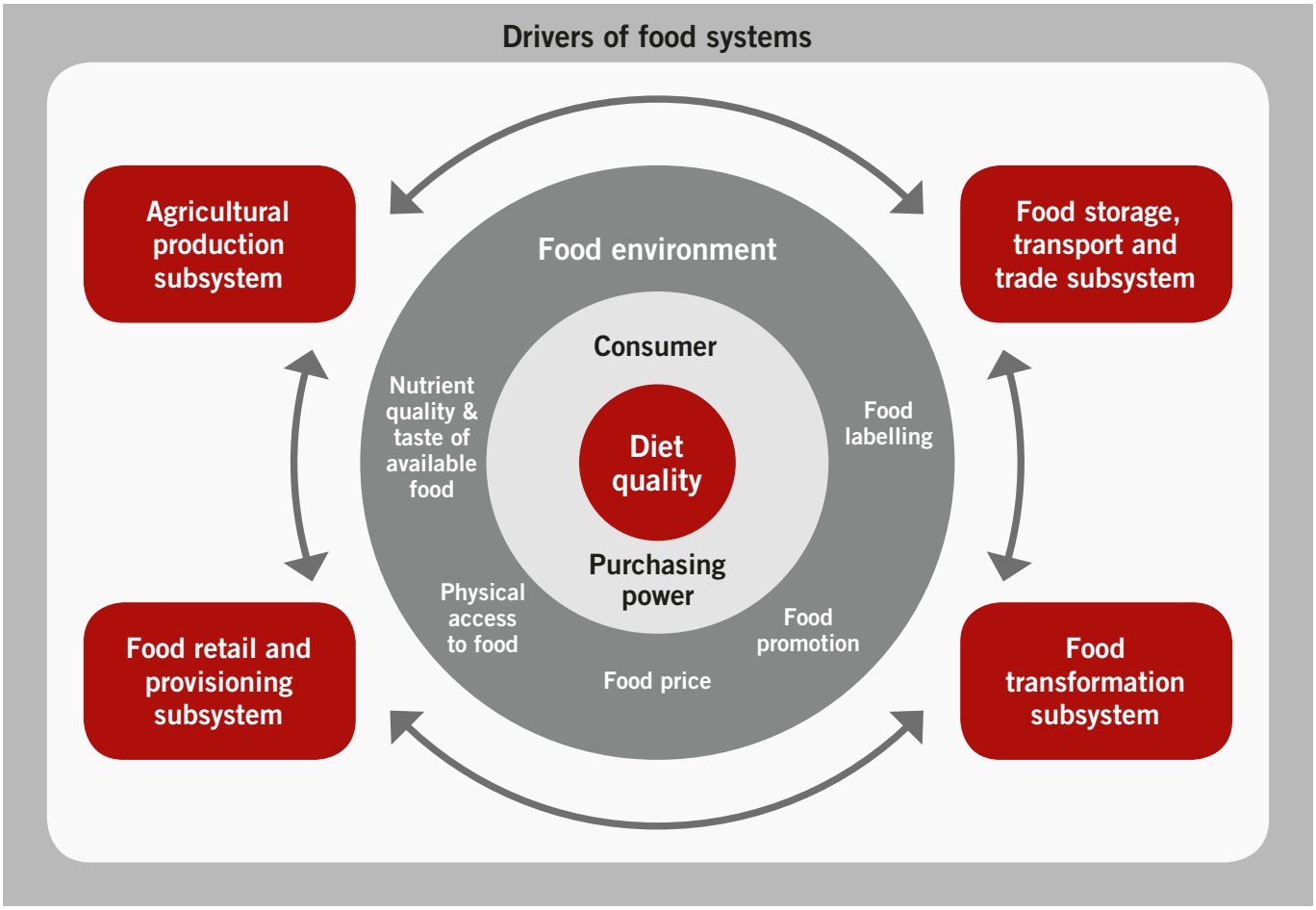
- Today's food systems are intricate – with long food chains that involve many different actors – and external factors that have an important influence on what is produced, how it is sold and at what price. This complexity presents significant challenges for policymakers. Many of the global and external drivers are well beyond the unilateral control of particular national or regional authorities, let alone individuals. Education and information campaigns alone will be unable to bring about the dietary change that is required.
- This complicated 'big picture' also presents opportunities – there are many different entry points along the food chain where policymakers can take action to enable and encourage healthy sustainable diets.
- The EU's Common Agricultural Policy (CAP) has undoubtedly helped shape current food environments and dietary patterns both within and outside the EU. Radical rethinking of the CAP could contribute greatly to creating an environment conducive to positive dietary changes.
- Trade and investment agreements can impact on the food and nutrition environment by directly impacting on the relative price and availability of unhealthy foods and by constraining the policy space available to governments to implement strong public health nutrition policies. There is a need to strengthen consideration of nutrition issues in trade policymaking.
- Commercial food promotion is one environmental characteristic driving consumption of foods high in fat, sugar or salt (HFSS foods). Despite the initiation of voluntary self-regulatory regimes on marketing to children in Europe, as well as statutory regulation in some countries, evidence implies that current approaches are inadequate, particularly given the shift towards youth-targeted digital marketing, including highly targeted, personalised marketing. Decisive policy action is needed to protect consumers from the ubiquitous marketing of unhealthy foods.
- Global environmental change is both a driver and an outcome of food systems. Food system activities have considerable environmental impact, including on climate change, land use and water use. Modelling suggests that climate change will negatively impact on diet-related health overall.
- There is considerable overlap between consuming healthier diets and achieving higher levels of sustainability and an integrated health and environment approach to food systems is needed. Health-environment win-wins need to be promoted through dietary guidelines and broader policy approaches, which go beyond influencing individual choice, are required.

In a perfect world the evidence-based population goals outlined in Chapter 2 would be swiftly translated by governments into clear dietary guidelines, and then people would change their food choices to reflect the latest advice. This would lead to growing demand for healthy products while demand for less healthy products would decline, then markets would respond to these demand signals by producing more healthier foods and less unhealthy food products. Such changes in production would then lead to a drop in prices for healthier foods, while unhealthy foods would become more expensive.

In reality, however, the reasons why we eat what we eat are many and complex. In today's intricate food systems the market functions imperfectly and there are multiple forces driving the food supply in addition to consumer demand. The distance between production and consumption has been growing. Food chains that traditionally were short – bringing fresh foods from farms to tables – are now long, involve many different actors and often provide ultra-processed foods. In practice, the actions of large agri-food companies and, to a lesser extent, government policies and public sector investment play an important role in driving production. Many factors on the supply side of the market – what is produced, how it is sold and at what price – are powerful drivers of consumption. These aspects of the food system have an impact on the food environment, and elements of the environment – coupled with individual preferences – impact on consumer decisions about what to eat.

Figure 25, from a 2016 report of the Global Panel on Agriculture and Food Systems for Nutrition[1], shows one graphic representation of food systems and how they impact on and interact with food environments, individuals and diet quality.

Figure 25 Links between diet quality and food systems. Reproduced with kind permission from: Global Panel on Agriculture and Food Systems for Nutrition (2016).[1]



Agricultural production subsystem: primary production of foods and related inputs, including production of arable crops, horticulture, animals and fish. **Food storage, transport and trade subsystem**: system in which food is handled, treated, stored, packed, moved, transported and traded. **Food transformation subsystem:** foods are transformed into final products (including marketing). **Food retail and provisioning subsystem:** Moves products into the hand of the consumer (markets, informal retail, street vendors, supermarkets and small stores).

It is now increasingly recognised that current food systems are flawed and fail to deliver affordable healthy diets to all who need them. In order to tackle malnutrition and improve the diets of populations, therefore, an integrated approach to transforming food systems is needed and governments have promised to take such action. In the Rome Declaration on Nutrition, issued at the Second International Conference on Nutrition (ICN2) in 2014, governments pledged to 'enhance sustainable food systems by developing coherent public policies from production to consumption and across relevant sectors to provide year-round access to food that meets people's nutrition needs and promote safe and diversified healthy diets.'[2]

Within the EU, these broader aspects of food systems are also on the common agenda. The discussion paper 'Food of the Future – The Future of Food'[i] presented by the Netherlands during its Presidency of the European Union in the first half of 2016 highlighted these issues.

At the end of 2016, the European Commission Joint Research Centre (JRC) published a report on 'Delivering on EU Food Safety and nutrition in 2050 - Future challenges and policy preparedness'.[ii] The report, which was presented at a conference on 'The future of food in the EU' (organised by the JRC and the trio council presidency of the Netherlands, Slovakia and Malta) contains four future scenarios, and, depending on the scenario, provides challenges for the food and nutrition system in 30 to 50 years from now. Furthermore, in 2018 the Austrian Presidency of the Union will focus on improving health through transformation of the food system/ food value chain as one of its priorities.

i http://www.aieaa.org/sites/default/files/NL%20-%20food-of-the-future-en.pdf
ii https://ec.europa.eu/jrc/en/publication/eur-scientific-and-technical-research-reports/delivering-eu-food-safety-and-nutrition-2050-future-challenges-and-policy-preparedness

To help translate the commitments of the Rome Declaration into concrete action, the Second International Conference on Nutrition also adopted a Framework for Action that recommends a set of 60 policy options and strategies.[3] The Rome Declaration and the Framework for Action have since been endorsed by the UN General Assembly, WHO and FAO. The UN declared a Decade of Action on Nutrition (2016-2025) and countries are now being supported to implement measures to adjust food systems to sustainably support healthy diets and improved nutrition for all.[4]

In highlighting the complexity of the 'big picture', Figure 25 points to the challenges facing European policymakers. It is easy to understand why education and information campaigns alone will be unable to bring about the dietary change that is required. Many of the global and external drivers are well beyond the unilateral control of particular national or local governments – let alone individuals. What this complex picture also reveals, however, is that policymakers – at international, national or local levels – have many different entry points for action within the food system. Figure 26 shows some examples of these potential entry points to improve diets along the food value chain. Policy action needs to harness these opportunities to create an integrated food chain – from farm to table – that enables and encourages healthy, sustainable diets.

The Global Panel on Agriculture and Food Systems for Nutrition outlines a decision-making tool to help policymakers assess 'diet gaps' in the food system in a particular country or local context and to identify available policy options to achieve healthy diets (See Appendix 1).

Another tool to help policymakers identify appropriate opportunities for action is the Food Environment Policy Index (Food EPI).[6] Developed by the International Network for Food and Obesity/NCDs Research, Monitoring and Action Support (INFORMAS), the Food EPI is a tool for:

- Identifying and prioritising actions needed to address critical gaps in government policies;
- Comparing the extent of implementation of government policies between countries;
- Tracking progress over time.

The tool has been applied in a number of countries including New Zealand, England, Thailand, Malaysia, Vietnam, Singapore, South Africa, Mexico, Chile, Guatemala, Canada and Australia.[6] The conceptual framework of Food-EPI (Figure 27) highlights that policy actions need to be backed by infrastructure support, including political leadership and robust governance.

Figure 26 Opportunities to improve nutrition and diets along the food value chain Reproduced with kind permission from: Fanzo et al, 2017.5

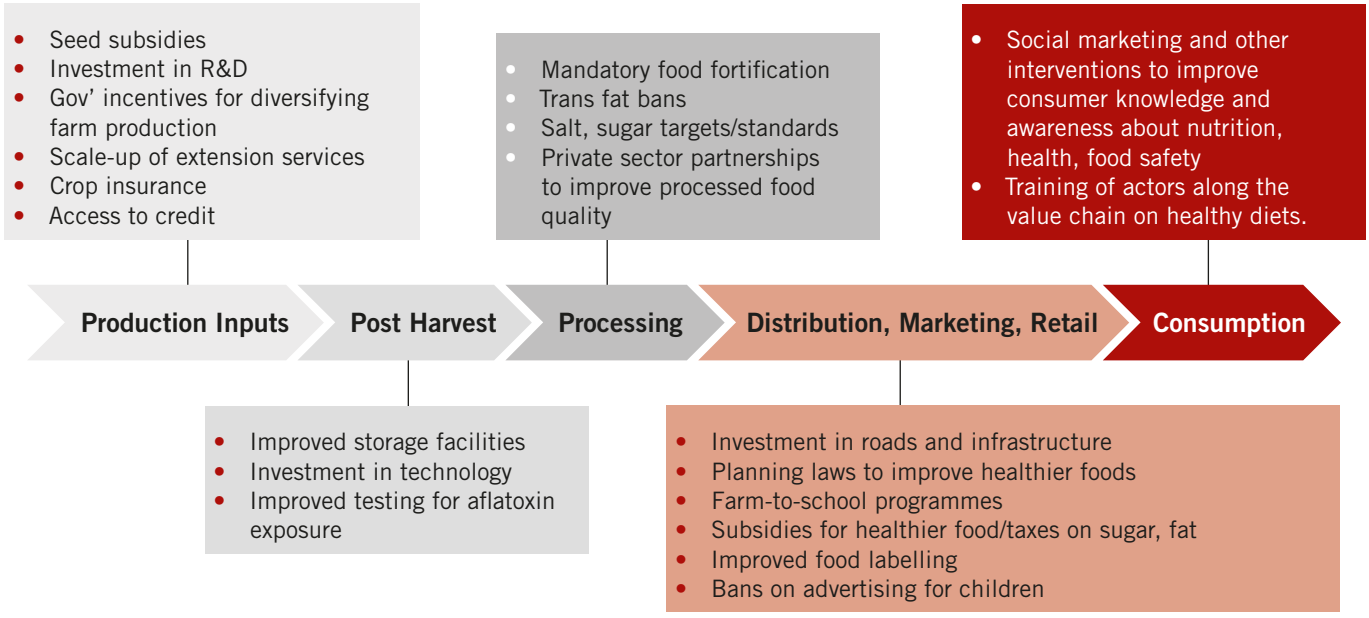
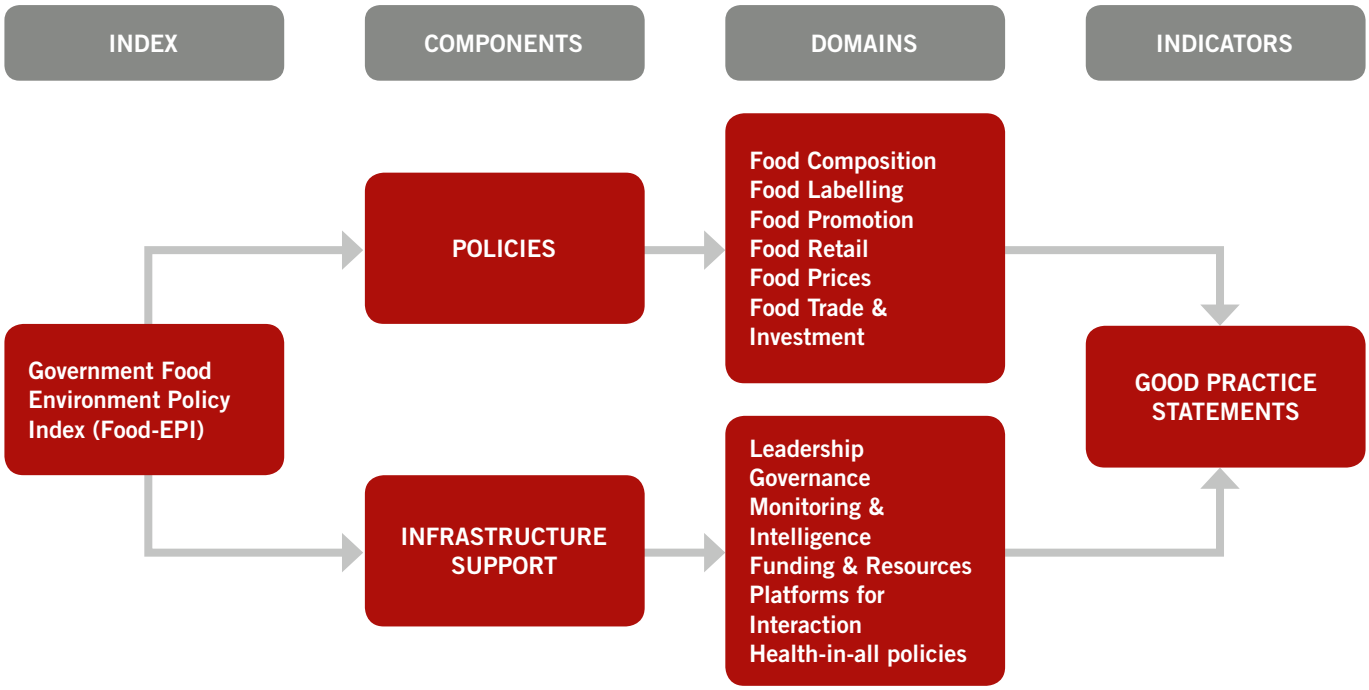


Figure 27 Conceptual framework for the Food Environment Policy Index (Food-EPI) tool Source: Swinburn et al, 2013, reproduced in Food Foundation[6]



Chapter 4 will explore the potential entry points for policymakers and identify the key priorities for policy action for achieving cardiovascular health-promoting diets in a European context.

The remainder of this chapter explores in more depth four specific issues that are particularly relevant to the challenges of creating sustainable European food systems for cardiovascular health:

- **Agriculture and supply side issues** – What are we producing in Europe?
- **Trade** – Impact of trade and investment agreements on food and nutrition
- **Food promotion** as a driver of consumption patterns
- **Food-environment links** – Matching cardiovascular health promotion with a sustainable food system

3.1 Agriculture and supply side issues: What are we producing/importing?

The EU is the biggest global exporter and importer of food and drink, with exports worth €98 billion and imports worth €72 billion in 2015.[7] The main items imported into the EU are animal feed, exotic products, wine, sugar, tobacco, and fruit and vegetables coming mainly from Brazil, Argentina, the USA, and China. The main exports of the EU are alcoholic drinks, animal products, animal feed preparations and smoking products. The main recipient countries of EU exports are the USA, Russia, Japan, Norway, Canada and Switzerland.

As set out in Chapter 1, diet is the leading cause of the global burden attributed to obesity and noncommunicable diseases (NCDs) such as cardiovascular disease (CVD), diabetes and cancer.[8–11] There has been a paradigm shift in post-World-War II diets from plain diets towards less sustainable and less healthful diets with currently people consuming too much total energy, fat, sugar, and salt, while living less physically active lives, across the world.[12,13]

As described in the first part of this chapter, the issue of dietary choices and how these choices might lead to the development of NCDs is complex with many different factors involved.[14,15] One of the key factors affecting dietary choices is undoubtedly the ready availability of some foods (with choices affected by the type of food, its quality and price). Agricultural policies therefore impact on dietary choices since they influence food availability. This chapter examines in more detail the Common Agricultural Policy (CAP), which is at the centre of the agri-food system in Europe.

3.1.1 Background

The CAP was the cornerstone of the European Economic Community (EEC) established by the 1957 Treaty of Rome.[16] This followed a period immediately after the Second World War when there were widespread food shortages, devastating conditions of poverty, poor housing and unemployment in Europe. In response to this, governments decided to

follow the acclaimed UK war–time policies of ensuring food security and fostering agriculture and food production with a specific bias towards generating a greater supply of cheaper meat, milk, butter, fats and oils. These commodities had been shown by Corry Mann to promote the growth of stunted children and to help supply sufficient food energy for both children[17] and adults,[18] before and during the war.[19] The importance of food security then became a key pillar of national security and both sides of the European 'Iron Curtain' regarded national food production as a key priority for survival.

The CAP was then introduced after an agreement between the six countries forming the former EEC, now known as European Union (EU), and had three main objectives; 1) to create a stable common market, 2) to provide affordable food to all the citizens of the six countries of EEC, and 3) to provide a better standard of living for the farmers of the EEC.[16] The emphasis on the adequacy of incomes for farmers also related to the need to foster farming in rural areas, which had become relatively depopulated during the huge wartime industrial effort. So, although cheaper food and promoting farmers' incomes were somewhat contradictory, two mechanisms were put in place to achieve them.

The first mechanism concerned the target price. Each product was allocated with a target price and products of the same kind entering the market from outside these six countries were allocated a 'penalty' tariff to ensure that the agreed EEC target price would not be challenged by lower-price imported products.

The second mechanism was triggered when the target price was not achieved for specific products within the EEC. If a product's market price fell below the target-price, then the countries would purchase all the lower priced produce of the specific product.[16] By removing all the below-target-price items from the supply chain, this prevented a further fall in price. New country members adopted the same agricultural policies when they joined the EU.[16] In keeping with these policies, there was a huge input to research funding for agriculture with many countries subsidising not only animal production and plant research but all the detailed, practical and technical advice to farmers as well as paying part or all the costs of new farm facilities, e.g. buildings, milking facilities, sheds etc. Cereal growing for animal feeding also became a top European priority, when it was discovered that feeding sheep and cattle cereals, rather than grass, markedly increased their growth rates and milk production. Sugar beet production was also introduced because it proved easy to grow in temperate climates and had soil enhancing qualities as well as producing desirable sugar to increase food energy supply. Safeguarding the importation of sugar from Europe's overseas territories and former colonies was also seen as important. Marketing boards were also created to help the farmers sell their burgeoning production and public institutions, e.g. schools and government establishments, ensured that the food served, often for free or at subsidised prices, included an ample supply of meat, full-fat milk, butter and animal and vegetable fats.[20]

This Europe-wide agriculture/food policy was extraordinarily successful, with farmers becoming more prosperous, rural communities better sustained and meat, butter, milk and fats and oil production rising steadily. However, what then emerged because of these European governments' actions and the EEC's pricing and purchasing policies was a surplus of all these products. This then led to the storage by the EEC of food 'mountains', despite special measures to increase consumption and to subsidise the European export of these products at lower prices. It was also beginning to be recognised that simply linking payments to production allowed those farmers with the largest production/land to receive automatically most of the payments, leaving small-scale farmers more vulnerable.[16]

In Central and Eastern Europe, which had been incorporated into the Eastern Bloc in the immediate post–war years, agriculture and food priorities remained heavily influenced by the Second World War experience of semi-starvation. So it was considered that, not only was local food production a national priority for security reasons, but that meat and milk production was critical to feed the increasing populations of the Soviet Union. Therefore, in keeping with the Communist policies of public ownership and collective work, farms were collectivised in most countries and even in countries where 85% of farms retained their individual ownership (e.g. Poland) these farmers were neglected and all government subsidies, research etc. was focused on the collective farms. Russian nutrition experts also took on board the need for animal protein to promote the growth and well-being of children and to sustain the physical capacity of adults. However, they not only provided a clear food production and pricing strategy for the whole for the Soviet Union[21] but also interpreted the evidence on animal protein needs to be two to three times higher than the newer Western thinking.[22] This meant that far greater cereal production was considered important for beef and milk production, but for climatic and other reasons relating to inefficient agriculture, cereal production was inadequate for the perceived needs of the Soviet Union so they were forced to buy cereals on the world market and, in practice, from the US. This led to the KGB spying to find technologies to improve cereal production, with the CIA playing a major role in predicting future world cereal prices, and the need to use precious Soviet foreign currency reserves for cereal purchases.[23] These prices depended on the likely cereal production in a swathe of countries from Ukraine through southern Russia into the east, spanning the extreme southern portions of Siberia, as well as cereal production in the mid-West of the United States.

By that stage it had become evident that food was not only important for children's growth and adult's working capacity but that some ingredients in the diet could lead to the newly recognised societal problem of heart disease. It was demonstrated that saturated fat increased blood cholesterol levels and that raised blood cholesterol levels markedly increased the risk of heart disease. It was also soon recognised that the average blood cholesterol level in the population was rising steadily and saturated fat intakes had risen markedly in response to all the government measures to promote butter, milk, and fat-rich meat production. Based on these concerns, Norway introduced, in 1962, a whole

series of agriculture and pricing policy measures to reduce saturated fat consumption and to increase vegetable and fruit production.[24] Later communities in Kuopio, Finland, demanded action as the death rates of middle-aged men was reaching epidemic proportions. This then led to the now famous North Karelia initiative to change the community's eating patterns based on individual and community action.

The changing nature of agriculture also led to the realisation in the West (but not the Soviet Union) that, with the marked increase in mechanised farming, fewer farmers were required. So, the European Mansholt Plan was introduced in 1968 as the first reform in CAP, which provided incentives for farmers to leave their farming businesses.[25] Food companies were also beginning to change their policies on witnessing the medical concern about dietary saturated fat. So, Unilever in the Netherlands developed a polyunsaturated margarine, Flora, to help people reduce their blood cholesterol levels, but there were few if any links between societal health analyses and agricultural policies. As the food surpluses continued,[26] individual countries took their own initiatives – in the UK, for example, the then Prime Minister declared in 1982 that a drastic change in the subsidising and nurturing of farmers was required. This approach, however, was unusual and by now the expanded European agriculture and food industries lobbied intensively. They were helped by the Mediterranean countries, where poverty and low production levels persisted and prevalence of heart disease remained low. The massive production of excess butter, full fat milk and fatty meats meant that each industrial component of the food chain was deploying their marketing techniques, lowering their prices and using all available measures to sell their commodities and products. From the 1980s onwards, the food industry also began to apply a variety of techniques to improve the general availability and marketing of very attractive flavour-enhanced foods, snacks and drinks, helped by the food industry's collaboration with the Monell Centre in the US.[iii] They also focused on the Mediterranean and low-income countries that had not 'benefited' from all the variety of attractive high fat, sugary and salty foods available in Northern Europe and America. So, the diet in the Mediterranean countries began to change markedly[27] and the prevalence of obesity and diabetes began to rise at the same time as efforts began to sell low-fat milks and spreads, to limit the fat in meats and to promote healthy foods. In practice, however, this involved diverting the residual fat into ice cream, baked products and snacks.

The second European agriculture reform finally took place many years later, in 1992, and was initiated after international complaints expressed in the Uruguay Round of world trade talks about the way that CAP was manipulating prices and the mass availability of different food commodities.[28] The changes then made included the extension of milk quotas to limit milk production, policies for set-aside land to limit the total amount of crops produced, and reductions in the level of institutional prices for cereals and beef. Farmers for the first time received direct payments independent of their production but based on land and per head of livestock owned. Additional CAP funds were also made available for programmes to assist the development of rural areas and for schemes where farmers pursued environmentally-friendly agricultural practices in return for additional payments.[29] The 1992 reforms were the first ones with an environmental element attached to them. Yet all these reforms were still being accompanied by ever-greater rates of food production, falling food prices and agricultural policies geared to benefiting the food chain producers and processors and with little regard to their health implications. Paradoxically, the availability of fruit and vegetables was restricted for many years while the EU decided to pay for the destruction of fruit and vegetable stocks to raise prices and improve financial returns to Mediterranean farmers. Such policies exacerbated the rising health problems of the European population associated with limited fruit and vegetable intakes. Yet there was already clear evidence that consumers were particularly sensitive to the price changes in vegetables and fruit and reduced their purchases when prices rose.[30]

In 2000, further European agricultural reforms extended the 1992 measures with further reductions in institutional prices and an increase in the rural development and agro-environmental schemes.[31] In 2003, more reforms were agreed but mainly on specific sectors of agriculture, such as sugar (introduced in 2005) and fruit and vegetables (introduced in 2007). In 2004, 10 new Central European member states with significant agricultural industries but very poor population health joined the EU. This, combined with further international pressures for trade liberalisation, led to the introduction of the 'single farm payment'.[31] This was a set amount paid annually to each farm to encourage farmers to make their production decisions based on market demand and production costs.[31] The latest CAP reform took place in 2013, focusing mainly on strengthening rural development, direct payments to farmers and encouraging environmentally friendly production of agricultural products[32] followed by the abolition of milk quotas on 31 March 2015.[33]

3.1.2 The CAP, nutrition and NCDs

Throughout its lifetime, the CAP has come under heavy criticism mainly because of its significant financial cost relating to the farming industry, which is an ever-smaller component of the economic and industrial sector in each EU country. In the 1970s and 1980s, the CAP absorbed about two-thirds of the European Commission's entire annual budget, so European taxpayers were paying higher taxes than would have been the case in the absence of farm support. By setting target and intervention prices substantially above the prices prevailing on world markets, the cost of food for European consumers also rose.[34] CAP has also been criticised at an international level because farmers in developing nations could not compete with the import levies and with the lower price of the excess European produce exported to low and middle-income countries.

As described above, at the time of its introduction the CAP was not designed primarily to address any nutritional issues

iii See: http://www.monell.org/support_sponsorship/corporate_sponsorship/

but rather to encourage a resurgence in the devastated rural economies after the Second World War. The focus on priorities for subsidies, however, did take into account original public health nutritional analyses of the need for hungry people to have more food energy, whether as fat or sugar, and for children's growth animal protein was already recognised as beneficial. The first discussions on the lack of modern nutritional considerations emerged in the early 1960s in Scandinavia. It was only in the mid 1980s, due the epidemic of heart disease in Europe since the Second World War, however, that there was general recognition in the public health community that Europe was engaged in inappropriate crop and food production proprieties created in part by the effects of CAP.[35,36] Linking the current CAP with nutrition and specially to include the aim of preventing NCDs might be possible but the synergies between the web of CAP's effect on dietary intake and NCD development are not straightforward (Figure 28).

Broadly speaking, CAP can define which products are produced and promoted, with big retailers having a major influence over which products are finally going to reach consumers and at what price.[37] Hence, there is debate about the extent to which CAP promotes unhealthy diets and NCDs.

The relative price of a health-promoting diet compared to an unhealthy diet depends on the specific context. In some situations, for example, a health-promoting diet is not more expensive than an unhealthy diet. In many cases, however, the maintenance of high prices and limitation of the availability of certain foods, especially those associated with healthful eating patterns like fruit and vegetables, may promote consumption of foods linked to CVD such as high fat, high sugar processed food.[38] A recent review and meta-analysis by Rao and colleagues revealed that healthier diets cost on average $1.48/day ($1.01- $1.95) more that unhealthy dietary patterns.[39] CAP was shown to promote the production of beef and dairy products – both being sources of saturated fat [38] – and therefore a driver of the huge effort in cereal production (70% of which goes into animal feeding, a use which is grossly inefficient). Yet only after 2008 was fruit and vegetable production subsidised rather than having fruit destroyed to prevent it reaching the market.[40] It was estimated then that approximately 5 000 CHD deaths and 2 000 stroke deaths were directly attributable to an inadequate fruit and vegetable supply.[41] It has been also estimated that 7 000 CHD deaths per year and 2 000 stroke deaths could be prevented if CAP removed subsidies from products rich in saturated fat like beef and milk.[42] On the other hand, the economist Schmidhuber reports that, due to the high food prices caused by CAP, overconsumption of fat is discouraged (Figure 29).[43] This is a common economic argument which has merit but neglects the vast sums of money used in subsidising the beef, sheep, pig and poultry industry as well as butter and milk production to a stage where these sectors have substantial economic and political power in Europe. Thus the consensual strategic transformation of the food chain by government subsidies over the decades has induced a food supply which is geared to selling ever more food thereby inducing obesity and diabetes with heart disease as one facet of this transformed food chain.

Figure 28 Links between agricultural policies, diet and non-communicable diseases (Adapted with kind permission from: Hawkes, 2007 [189])

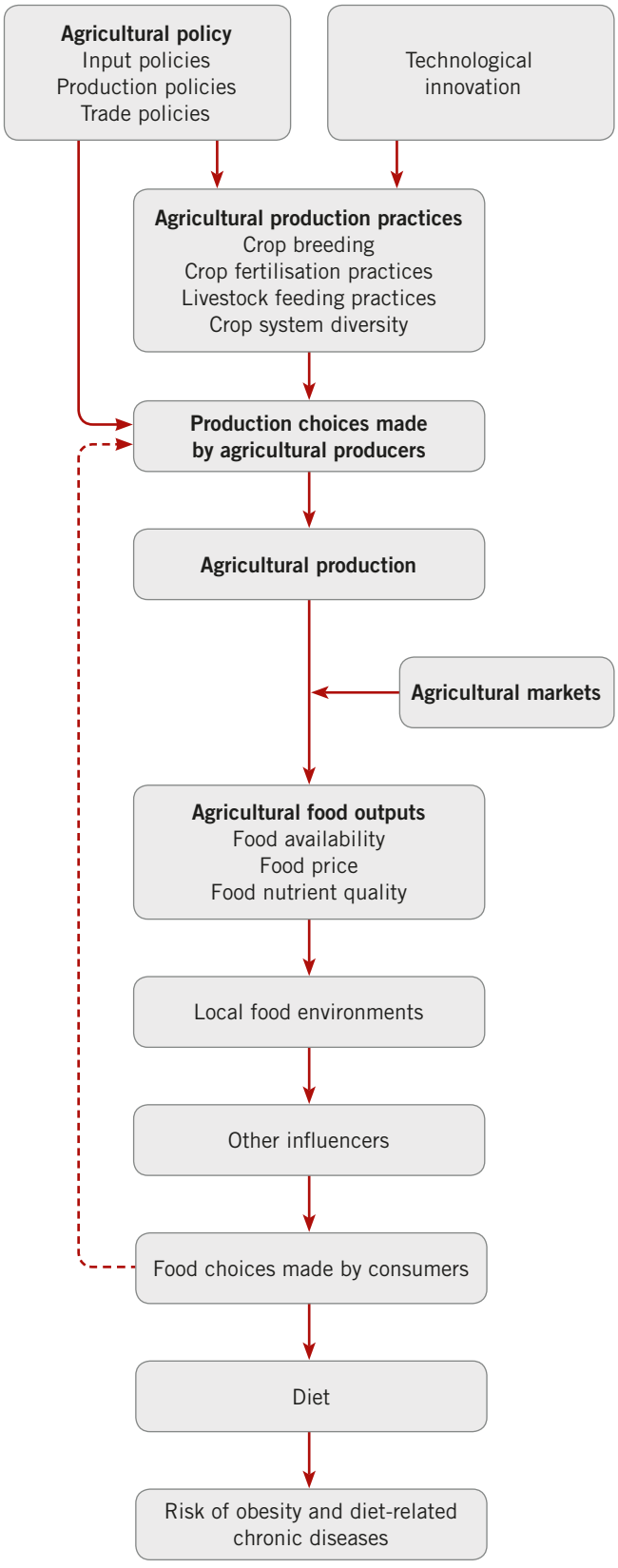
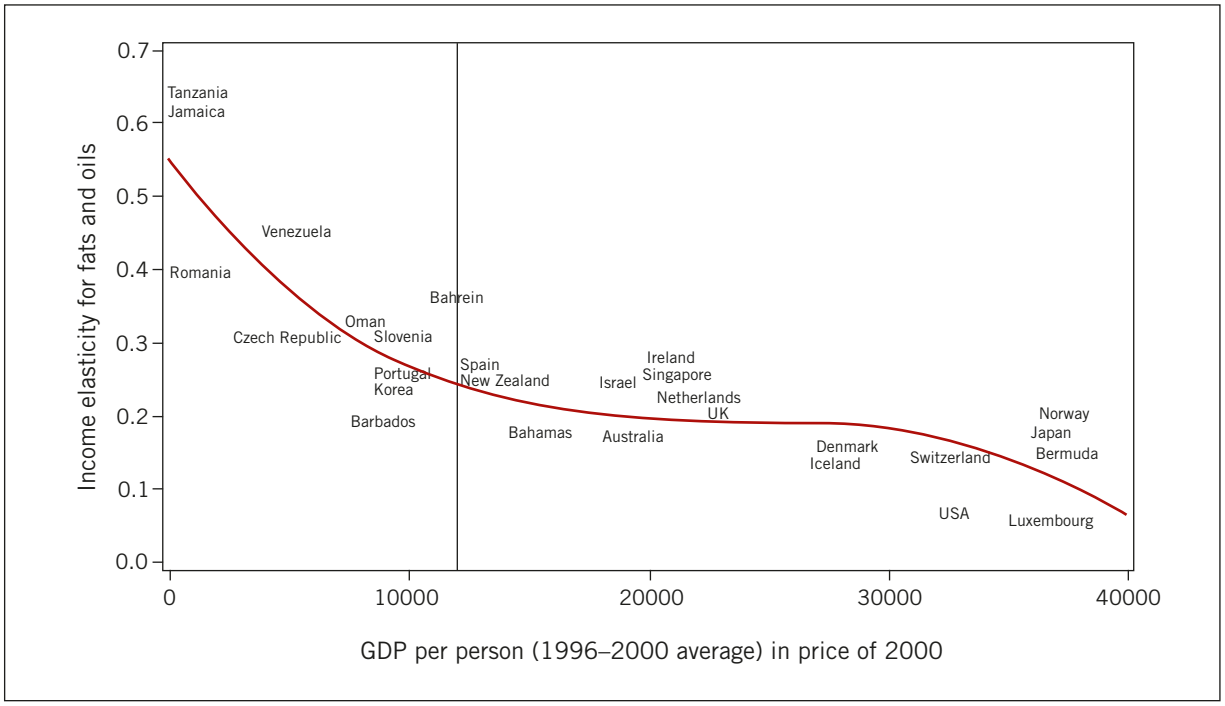


Figure 29 Income elasticity for fats and oils Source: Schmidhuber, 200343

Note: The ordinate indicates the degree to which consumers respond to changes in price of a commodity. So, the higher the elasticity the greater the change in the purchasing of that product. The abscissa specifies the group or community's income and reveals how that the poorer the community the greater the responsiveness to a price change in fats and oils.



The same argument applies to sugar, another product controlled by CAP, and European sugar prices are currently being kept higher than the world sugar prices by CAP, thereby discouraging the intake of sugar or its use by industry in food products. As the Fabian Society noted [44] '… food commodities where the CAP kept consumer prices high is dairy and sugar. For both milk and sugar, the stimulus to production due to high producer prices was controlled by quota limitations. Dairy quotas were removed on 1 April 2015 and sugar quotas will be removed on 1 October 2017. For both commodities, other things being equal (and in the case of both dairy product and sugar markets, there is very high price volatility), the removal of quotas will lead to a fall in prices relative to what they otherwise might have been. In the case of dairy production, academic studies have pointed to a possible fall of up to 10% in raw milk producer prices and of up to 22-23% in the case of sugar beet. The fall in milk prices is mainly due to the impact of increased EU production and exports on world market prices, as EU dairy product prices are now aligned to world market levels.'

Consumers are sensitive to food price changes, as shown in Table 7.[45]

Sugar prices have already started falling from about €700 per tonne in 2012 (before the reform) to about €400 in 2015. In the study on the 'Post-Quotas Sugar sector period' conducted by the European Commission, it is estimated that in the next decade, EU production of sugars will increase by around 15%. This increase will also include an increase in high-fructose corn syrup (HFCS) production.[46] Hence, it is anticipated by the European Commission that Europeans citizens will be consuming more sugar in the coming decade and it will be more economically viable for food companies to incorporate more sugar or HFCS in their products.[47]

Diets are like living organisms that keep evolving over time, being influenced by many factors with complex interactions between household income, food prices (which will affect the availability and affordability of healthy foods), and beliefs, cultural traditions, as well as geographical, environmental, social and economic factors and individual preferences. These all interact in a complex manner to shape individual dietary patterns. Therefore, promoting a healthy food environment, including food systems that promote a diversified, balanced and healthy diet, requires multipronged involvement across multiple sectors and stakeholders. Promotion of meat, milk butter etc. by European governments over many decades, through multiple actions, has proven to be highly effective in changing the whole dietary pattern of the European continent. Many branches of government and the public and private sectors now need, therefore, to become involved to rectify the mistakes of the past.

The availability of low-cost and safe food remains an essential priority for a large share of the population, now that income inequality has increased so markedly in Europe.[48] However, the quality of food, production methods and their impact on the environment, animal welfare, biotechnology, and fair-trade are also now seen as priorities. The UN Sustainable Development Goals (Agenda 2030)[49] require, for example, that forests and national parks are maintained and protected,

*Table 7 Mean percentage change (95% confidence interval) in food demand for 1% increase in food price by country wealth category**
Source: Green et al, 2013 [45]

Food groups	Country wealth category		
	Low income (n=1412)	Middle income (n=827)	High income (n=1124)
Fruit and vegetables	-0.72 (-0.77 to -0.66)	-0.65 (-0.71 to -0.59)	-0.53 (-0.59 to -0.48)
Meat	-0.78 (-0.83 to -0.73)	-0.72 (-0.78 to -0.66)	-0.60 (-0.66 to -0.54)
Fish	-0.80 (-0.85 to -0.74)	-0.73 (-0.79 to -0.67)	-0.61 (-0.67 to -0.55)
Dairy	-0.78 (-0.84 to -0.73)	-0.72 (-0.78 to -0.66)	-0.60 (-0.66 to -0.54)
Eggs	-0.54 (-0.67 to -0.42)	-0.48 (-0.61 to -0.35)	-0.36 (-0.49 to -0.23)
Cereals	-0.61 (-0.66 to -0.56)	-0.55 (-0.61 to -0.49)	-0.43 (-0.48 to -0.36)
Fats and oils	-0.60 (-0.65 to -0.54)	-0.54 (-0.60 to -0.47)	-0.42 (-0.48 to -0.35)
Sweets, confectionery, and sweetened beverages	-0.74 (-0.82 to -0.65)	-0.68 (-0.77 to -0.59)	-0.56 (-0.65 to -0.48)
Other	-0.95 (-1.01 to -0.90)	-0.89 (-0.95 to -0.83)	-0.77 (-0.83 to -0.71)
All food groups combined	-0.74 (-0.79 to -0.69)	-0.68 (-0.73 to -0.62)	-0.56 (-0.61 to -0.50)

*Predictions based on multiple regression model with random effects. Values of all covariates in the model are set to their mean for the purposes of predicting values, with the exception of year of data, which is set to 2008.

biodiversity preserved and the ecological richness of the countryside defended in a sustainable manner. An adequate response to these objectives and concerns is not an easy task as current preferences vary widely and a balance of different options may differ from country to country within Europe. It is also important to bear in mind the considerable changes in climate and the capacity for different food production in various parts of Europe (See section 3.4 later in this chapter).

The current CAP is the outcome of a long historical process and an accumulation of policy devices in response to emerging problems, but focused mostly around markets. Hitherto, despite all the reforms, CAP is still disconnected from nutrition and public health policies and often contradicts other EU policies that deal with competition[50] and consumer protection.[38] CAP will distribute more than €150 billion across the EU countries between 2017 and 2020 to support the production of mostly livestock and industrial crops. This is clearly contrary to the new demands for a sustainable planet and public health.

Governments have a central role in creating a healthy food system that enables people to live in a healthful food environment and adopt and maintain healthful dietary practices. A radical change in food consumption and production in Europe is unavoidable if we are going to conform with the UN Agenda 2030[49] acceptance of the need for agri-food systems to become more sustainable and resilient and now newly aligned with our current understanding of the needs of public health.

3.1.2.1 Governments' policy responses

3.1.2.1.1 Nutrition Taxation

Nutrition taxation has been used as a policy tool for increasing the price of 'unhealthy' products to discourage the purchase and consumption[51] (See Chapter 4). Even though, the political success of nutrition taxation varies, with sugar quotas expected to be removed in 2017, introducing a European-wide taxation on the sugar content as a commodity rather than a retail tax on processed foods and drinks could help prevent the anticipated increase in sugar consumption.

3.1.2.1.2 Investments to improve the food supply

Competitive supply chains require both investment and appropriate government policies. Improving rural infrastructure and market access while developing and conserving natural resources is key to a successful food supply chain. After the latest CAP reform, there has been some consideration about rural development and conservation, with six priorities identified in the policy planned for 2014-2020[52] but there is a lot of room for improvement. Well-functioning input and output markets would also reduce domestic food price volatility and offer more healthy and affordable food to all citizens.

3.1.2.1.3 Aligning of public health policies with agriculture and food policies or other trade negotiations at the EU level – Improving collaboration between the public and private sectors

There has been insufficient consideration both from the public health and agricultural policy areas, to date, of the significance of each other in shaping population health.[50] Public health policymakers need to collaborate with agriculture, food and trade policymakers to ensure that all citizens have access to nutritious and affordable food. More predictability and transparency, both at government and private sector levels, should be pursued through the sharing of market information and various arrangements for public–private partnerships. Preparations of policy interventions

to be introduced in subsequent periods of time should be made public and opportunities for anti-competitive behaviour and corruption in the supply chain should be reduced or eliminated.

3.1.2.2 Farmers' and consumers' responses

3.1.2.2.1 Creation of shorter supply chains/ Food price reductions

The current food system encourages the retail sector to concentrate its bargaining power and primary producers now have only a subordinate economic role. Thus, on a European basis, farmers received on average 31% of the retail price in 1995 but this proportion of retail prices fell to 21% by 2011 although since then it has risen somewhat.[53] By reducing/ eliminating CAP, farmers would be encouraged to create shorter supply chains for their products as that would give them higher prices for local purchases and thereby more bargaining power. This would help with consumers having access to fresh, local, seasonal produce at lower prices because the extra marketing costs etc. of the retailers would be limited and farmers could obtain higher prices for their produce. Food wastage could also be reduced with the use of a shorter supply chain. By avoiding large multi-national retailers, consumers' money spent on local food and local retailers would stay in the community. Overall food prices could be reduced which will have a great impact on poorer consumers that are also mostly affected by NCDs. Society would also benefit from not having to fund food support programmes for the poorer and vulnerable sectors of the population.

3.1.2.2.2 Alignment of sustainable food production with sustainable dietary guidelines across Europe

There is a need to encourage consumers to demand healthful sustainable foods and meals. This could be achieved by; 1) promoting consumer awareness of a healthful sustainable diet through the sustainable dietary guidelines, 2) increasing demand for producers to grow and sell fresh seasonal fruits and vegetables more directly to the consumer.

As has been seen, food systems are highly complex and driven by many different factors; economic, cultural, and environmental. Exploring and understanding these factors better and their interactions help to improve policies related to food systems and food consumption. We need to achieve sustainable and healthful diets for every citizen without compromising the living standards of the farming community, while minimising the environmental impact of food production and consumption, so that they become sustainable. CAP undoubtedly has shaped the current food environment and dietary patterns both in the countries of the European Union and internationally. However, simply eliminating or completely removing CAP will not solve all the problems faced by farmers, consumers and countries when it comes to nutrition. A collective effort from all different stakeholders is required while implementing robust systems for monitoring compliance of any policies.

In conclusion, CVD can be prevented by modifying major CVD risk factors. CAP along with other policies has a strong role in creating an environment conducive of positive dietary changes, one of the major risk factors for developing CVD. There is a need to thoroughly assess the nutrition and health benefits of substantially reforming or abolishing the CAP, compared to the current – or a minimally modified – CAP. A radical rethink of the CAP, for example, could contribute greatly to the promotion of foods such as fruit and vegetables, known to be protective against CVD and other NCDs. In order to maximise the benefits from changes in CAP, those should be accompanied by other relevant public health policies and food industry and retailers' policies.

3.2 Impact of trade and investment agreements on food and nutrition

Food represents a significant component of trade and Gross Domestic Product (GDP), and is thus an economic as well as a health issue. The food supply chain in the EU, for example, generates around 15% of total EU employment and 7% of the EU GDP.[54] New or proposed regional trade and investment agreements, such as the Trans-Atlantic Trade and Investment Partnership (TTIP – currently suspended), thus have substantial implications for the food environment.[55] These agreements govern trade in food and food-related services (in the TTIP, this falls under 'Market Access'), the food-related policy options available to governments ('Regulatory cooperation'), and protection for investors in the food supply ('Rules').[56]

Trade and investment agreements impact upon the food and nutrition environment relevant to cardiovascular disease in two ways:

1. Through direct impacts on the **relative price and availability** of unhealthy foods (foods high in fat, salt and sugar and other energy-dense/nutrient-poor foods, generally falling outside of food-based dietary guidelines regarding healthy diets); and
2. Through **constraining the policy space** available to governments to implement strong public health nutrition policies.

First, trade and investment agreements affect the relative price and availability of unhealthy foods through reducing the costs and barriers to the supply, marketing and retail of highly processed foods. Highly processed foods dominate the food supplies of high income countries and tend to be higher in fat, salt, and/or sugar than unprocessed foods.[57] These foods are also the subject of the majority of food marketing, which creates strong incentives for consumption.[58]

The extent of the impact of a new trade and investment agreement will depend on the level of a country's or an economic region's pre-existing liberalisation. However, further commitments to trade and investment liberalisation through new agreements will continue to reduce barriers to: physical trade in foods, trade in services relevant to food and nutrition

(e.g. fast food franchises, marketing), and food industry investment. Reductions in barriers to trade can increase the availability and affordability of such foods through reducing the costs and barriers to supply. These effects of liberalisation have been shown in other contexts, where availability and affordability of highly processed foods has increased in response to liberalisation.[59–61] In submissions to the negotiations for the Trans-Pacific Partnership Agreement (TPPA, finalised in 2015), the food industry identified significant scope for increased market access for sugar and processed foods.[62] Trade and investment agreements also create incentives for increased investment by multinational food service outlets and retailers, which in turn increase the accessibility and affordability of highly processed foods through increased coverage and efficiencies in supply chains.[63]

Second, trade and investment agreements affect the policy space available to governments through constraining domestic policymaking. Policy space refers to the 'freedom, scope, and mechanisms that governments have to choose, design, and implement public policies to fulfil their aims'.[64] There is evidence that trade and investment agreements can constrain policy space for public health nutrition in three ways: direct constraints on available policy options; increased avenues for stakeholder influence in policymaking; and new avenues for recourse by affected stakeholders.

The direct impact of trade and investment agreements on policy space for public health nutrition is through constraining the policy options available to governments. Although such agreements contain allowances for governments to make and implement policies to protect public health, they also contain provisions that require these to be the 'least trade restrictive' policies. Particular provisions of concern relate to Technical Barriers to Trade, which focus on ensuring that 'technical measures' do not unnecessarily restrict trade. Such provisions can restrict nutrition policy space through requiring stringent justification of measures based on narrowly-defined scientific evidence, and requiring the use of international standards.[65] These requirements undermine the precautionary principle in public health [66] and constrain innovation in nutrition policymaking at a time when the evidence base for effective public health nutrition policy is being built.[6566,67] For example, Weiss reports that 'the USA and other exporting countries have indicated that they consider national policies to …tax sugary drinks and junk foods, require nutritional labelling of foods, … to be unnecessarily trade restrictive'.[66] Other policy options potentially subject to direct constraints include restrictions on cross-border advertising, which may be contrary to efforts to liberalise trade in services,[68] and policies to provide healthy food in public institutions, which may be contrary to provisions on government procurement.[65,68]

Trade and investment agreements also contain provisions that increase the range of stakeholders involved in government policymaking. For example, the provisions on regulatory coherence in the TPPA and those proposed by the European Union in the TTIP, create new avenues for the food industry to participate in domestic/EU policy making.[68] These provisions in the TPPA state that 'Each Party shall allow persons of the other Parties to participate in the development of technical regulations, standards and conformity assessment procedures by its central government bodies…on terms no less favourable than those it accords to its own persons'.

Finally, trade and investment agreements contain provisions for protection of investors that in some cases exceed those afforded to domestic investors. Many recent agreements include a mechanism to resolve disputes between investors and states.[69] This provides an avenue for industry actors to directly sue governments for compensation in certain situations where they have been unduly affected by government action (for a more detailed explanation in relation to nutrition, see Thow & McGrady [70]). Provisions for such Investor State Dispute Settlement (ISDS) mechanisms have been identified as a potential avenue for the food industry to contest government policy intervention that reduces the profitability (e.g. sales) of unhealthy foods, such as nutrition labelling.[67,68,71] A recent analysis of the TPPA identified limitations of the ISDS provisions from a public health perspective, including no exception for public health nutrition, and provisions requiring 'fair and equitable treatment' of investors that favour industry rather than governments.

However, there are opportunities for public health norms and policies to support consideration of public health policy goals – not just economic goals – in arbitration of investor-state disputes. An ISDS mechanism in the Uruguay-Switzerland Bilateral Investment Treaty was recently used by Phillip Morris International to challenge Uruguay's decision to mandate large graphic health warnings on cigarette packaging and a 'single presentation requirement' (a requirement that tobacco manufacturers produce no more than one variant of a single brand family of cigarettes).[72,73] In a positive outcome for public health, the arbitration tribunal dismissed all of Philip Morris' claims against Uruguay's public health policy initiatives. It is notable that the World Health Organization's (WHO) Framework Convention on Tobacco Control (FCTC) was pivotal in establishing a best practice reference point, in support of Uruguay's stringent tobacco control measures.[74] The Tribunal also made use of *amicus curiae* briefs from the WHO\WHO FCTC Secretariat and the Pan American Health Organization (PAHO) in informing their decision to dismiss the claims and support Uruguay's public health measure.

Analyses of the impact of trade and investment agreements on nutrition have identified the need for:

- Stronger consideration of impacts on public health nutrition in trade policymaking;
- Strategic support for regional and global public health norms to support innovation in nutrition policy making; and,
- Further research on nutrition and trade.

Research needs include targeted health impact assessments – ideally mandated within trade policy processes – and policy analyses focused on how nutrition could be considered in trade/investment policymaking.[65,67] Increasing the consideration of nutrition in trade policymaking will require advocacy for transparency and strong counter-arguments

to industry advocacy, that give more prominence to health concerns.[62,67,68] Strong regional and global support for nutrition intervention – in the form, for example, of a global treaty or convention to protect healthy diets – can also provide a counterbalance to regional trade commitments.[75]

3.3 Food promotion as a driver of consumption patterns

The commercial promotion of food and beverage products high in saturated fats, free sugars and salts (hereafter 'HFSS foods') is a significant risk factor for the development of diet-related NCDs. Food promotion, defined as the communication of messages designed to persuade or encourage the purchase or consumption of a product or raise awareness of a brand, has a large impact on the consumption habits of individuals and is a prominent aspect of food environments which are considered 'obesogenic'. Such environments feature an abundance of HFSS foods that are readily available (in schools, leisure centres and elsewhere), accessible (being cheaper, or at least perceived as being cheaper, than healthier foods) and are persistently marketed. This short review summarises the diverse forms of commercial promotion currently used by food and beverage (from hereon referred to as 'food') brands across Europe, including levels of expenditure dedicated to this promotion. Within this arena, three major food promotion trends in Europe are discussed; 1) the emergence of digital food promotion, 2) the rise of personalised marketing within digital food promotion and 3) the increasing calls for food marketing regulation. Finally, a brief summary of the evidence on the impact of promotion on eating patterns in adults and children is given.

Food marketing practices contribute heavily to the current obesogenic food environment in the European region; where children are preferentially targeted by food marketers[76] due to their independent spending power (current and future) and sizable influence over family spending.[77] Marketing to children has an impact on the purchasing choices made by parents and on the food kept in homes,[78] thus affecting the consumption habits of the entire family. This influence that children exert on the decision making process of parents has been labelled as 'pester power',[79] referring to the influence of children's nagging of parents to purchase specific foods, especially in supermarkets and in response to point-of-sale advertising, where it may prove problematic for parents to resist persistent demands.[79,80] Food promotions seek to influence children's immediate dietary preferences and build taste preferences whilst securing brand loyalty early-on in life so that preferences last into adulthood.[81] Moreover, marketing that is seemingly aimed at more mature populations is also influential on children, given that as well as the age group it is intended to target, effects are highly likely to spill over and also appeal to younger children[82] who strive to follow the trends of older peers. Importantly, food marketing directed at adults has escaped the same level of scrutiny awarded to child-directed marketing with a scarcity of literature assessing its impact on food intake behaviours or weight status[83]. This neglect is partly due to industry resistance and partly to policymakers often assuming incorrectly that all purchases by adults are based on informed and appropriate decision making.

3.3.1 The different forms of commercial promotion

Individuals are exposed to an excess of unhealthy food commercial promotion in most traditional platforms (e.g. television, events sponsorship, outdoor advertising, print media, point-of-sale) and, increasingly, via digital avenues (social media, websites). Television advertising is still considered to be highly effective at producing strong brand awareness[84]; this is a critical aspect of advertising, particularly for children and young people. Research from a recent UK survey illustrates that television is the media device that would be most missed by children and adolescents,[85] emphasising its relevance despite the emergence of major digital food marketing forms. Television continues to be a key force in providing children with unhealthy food advert exposure. In response to increasing scrutiny from academics and international health organisations alike, there are evident efforts across Europe towards national and regional policy action with the intention to limit the broadcast of unhealthy food adverts on television (see [86] and Chapter 4 for more on policy efforts in the European region). However, even where statutory frameworks have been implemented (e.g. UK Ofcom regulations), there is cause for concern over their efficacy, and there are worrying practices emerging in the aftermath, such as primarily 'unhealthy' brands continuing to market to children on television by displaying food products with a healthier nutritional profile in the advert itself. In a recent study of a major fast food brand, this marketing approach was demonstrated to promote a liking for fast food in general without any resultant shift towards the selection of healthier products by children.[87]

Children can also be exposed to marketing through sponsorship of prominent and international sporting events. During unrivalled events like the Olympic and Paralympic Games, food brands are keen to exploit these platforms to provide unique brand exposure, most often for HFSS foods, to maximum effect. Lobbyist groups[88] have highlighted the associations consumers explicitly and implicitly draw between food brands (e.g. McDonald's and Coca-Cola in the case of the 2014 World Cup staged in Brazil) and sport, health and physical activity, as a result of such sponsorship.[89] Outdoor advertising,[90] magazines[91] and point-of-sale within retail settings[92,93] all also constitute routes through which individuals are exposed to HFSS food marketing. However, more recently, digital food promotion has come to be incomparable in terms of promotional reach and impact. Marketing in digital media is an evolving concept, but is defined by Tatlow-Golden and colleagues as 'promotional activity, delivered through a digital medium, that seeks to maximise impact through creative and/or analytic methods.'[94] Online marketing now spans digital media such as food company websites, advergames and social media platforms (e.g. Facebook and YouTube) to offer effective avenues of food brand exposure. Although research on digital marketing is much less well established relative to

that for television, emerging trends have been observed in personalised targeted marketing and behavioural tracking techniques (outlined in further detail below).

3.3.2 Promotional expenditure on European food marketing

Data on the financial resources allocated to promotional expenditure on food brands and products in Europe is challenging to obtain, as such information is often withheld from the public domain. However, estimates point towards a decline in television advertising spend and a rise in digital and non-broadcast advertising spend.[95] Spend on internet advertising was expected to rise from 20% of total advertising expenditure to 30% between 2010-2015 in western Europe, and was predicted to be worth US$ 38 billion out of a total of US$ 126 billion by 2015.[96] Across all domains, online advertising has now overtaken television advertising to become the largest advertising medium in Europe. Recent figures on digital expenditure illustrate that online advertising spend grew to a market value of €36.2bn in 2015, surpassing spend on television marketing in Europe (€33.3bn).[97] Data illustrate that manufacturers of consumer goods (which includes food products) spent the most on banner and video display ads in 2015, and were responsible for 18% of the total advertising spend.[97]

In the UK, for example, internet advertising expenditure (including online, mobile and tablet) reached £6.3bn in 2013; an increase of 15.6% compared to 2012. It was forecast to grow 14% in 2014, and a further 12.7% in 2015. Within this, mobile forms of advertising displayed a growth rate of 95.2% in 2013 and have continued growing rapidly. Total advertising expenditure on children's television in 2013 was £142m, out of a total television ad spend of £4 642m.

In terms of the effectiveness of digital media advertising spend, a study into a multi-platform Coca-Cola campaign utilising television and social media (Facebook) found that 27% of Coca-Cola's incremental sales were generated by Facebook, using only 2% of the gross media budget allocated to the promotion. Return on investment data illustrates that €2.74 was generated for every €1 invested on Facebook.[98] However, it is important to consider that in terms of cost comparison, advert spend does not directly equate to exposure; as forms of internet marketing cost relatively less than television advertisements. Thus, less expenditure allocated to digital food advertising does not translate into reduced exposure and compared with television alone, social media marketing campaigns have the capacity to intensify marketing effects via tailored marketing.

3.3.3 Three major trends in food promotion in Europe

This observed shift towards digital marketing, as represented by promotional expenditure data above, is the first major trend in European food promotion discussed in this review. Multinational food companies are dedicating budget spend to online advertising which now constitutes around 50% of total marketing spend.[99] New media marketing varies from traditional forms in numerous respects,[100] one example being the way it facilitates peer endorsement of, and personal relationships with, food and beverage brands.[101] Such qualities are well-established as essential for strengthening brand awareness and encouraging product purchases.[102] Critically, forms of digital marketing are targeted predominantly at children and adolescents, due to increases in their habitual engagement with online media. The major 'EU Kids Online' study (a large survey of 9-16 year olds across 25 European countries) reported that in 2009, children aged 9-16 spent approximately an hour and a half per day (88 minutes) online. Differences in time spent online by age were reported, where 15-16 year olds spend almost two hours per day, on average (118 minutes); twice that of the youngest group (9-10 year olds average 58 minutes per day).[103] UK data from 2016 demonstrates that 7-16 year olds spend 3 hours online daily, with children aged 15-16 reporting 5 hours of online.[104]

Food and beverage companies have recently taken advantage of this trend to expand youth-targeted food marketing into commercial websites, third-party Internet advertising (i.e. placement of banner advertising on other companies websites), online videos, advergames and social media.[105] Vlogging (video blogging) is one example of a relatively recently emerged form of food marketing proliferation on social media, where vloggers are paid to feature HFSS food products (e.g. Oreos) in a game, task or review within an established vlogger's video.[106] Such word-of-mouth effects, delivered via social media, are considered more effective than marketing driven by brands due to the perceived enhanced credibility of friends' recommendations.[107] Indeed, this technique resonates particularly with young adults, as shown by 63% of US adolescents being happy to try a brand suggested by a YouTuber.[108] The impact of such digital marketing campaigns in terms of exposure to food marketing is likely to be substantial.

Further to this, marketers state that digital avenues represent the opportunity to 'deliver media-rich brand campaigns like the ones seen on TV but with more of an opportunity to fine-tune messaging.'[99] Indeed, online marketing forms have facilitated a rise in targeted, personalised marketing. This key shift from dependence on broadcast mediums for message delivery has allowed for contextual advertising (tailoring food adverts to viewers' internet content) and online behavioural advertising (tailoring food adverts to users' individualised characters and online activity). These sophisticated methods are unique to online marketing, and are usually undertaken by installing 'cookies'; allowing for detailed data into consumers' online browsing, personal preferences, and social activities.[109] This approach allows brands access to the individuals they wish to target, where advertisers, advert networks and data providers collect data on individual users across internet locations and use this to deliver target adverts to individuals. Personal data, including an individual's 'likes' on social media, allow marketers to target consumers with more 'relevant' advertising content. Highly personalised digital food marketing derived from data analytics helps brands to engage with consumers for maximum impact. From a regulatory perspective, this is a

challenging development, not previously encountered in traditional marketing forms.

Certainly, HFSS food marketing has amassed recognition from parliamentarians[110,111] and national governments in Europe[112] all concluding that, despite gaps in the evidence base, advertising and the commercial promotion of HFSS foods warrant substantial policy action. The increasing call for regulation in Europe is thus the third food promotion trend discussed in this review. A WHO ministerial conference in the European Region in 2006 cited marketing to children as warranting swift action.[113] Resultantly, a European Network on reducing marketing pressure on children was established in 2008, and around 30 countries in the WHO European region now participate in this network. Its objectives include to 'discuss approaches to control marketing of food and non-alcoholic beverage to children, such as statutory regulation, self-regulation, voluntary measures and co regulation' and to 'develop tools and share experiences to support monitoring of food and beverage marketing to children'.[114] Pressure from the European Network and other bodies (e.g. World Obesity Federation) culminated in arguably the two most crucial documents relating to establishing food marketing regulations: WHO's *Set of recommendations on the marketing of foods and non-alcoholic beverages to children*[115] and *A framework for implementing the set of recommendations on the marketing of foods and non-alcoholic beverages to children*.[116] The purpose of the set of recommendations is to guide countries in designing new and/or strengthening existing policies on food marketing communications to children. Frameworks published by WHO state that governments should apply restriction in this way, thus exerting high-ranking political pressure on nations. WHO asserts that the influence of food marketing related to two components: exposure and power. Exposure relates to the extent of food marketing and is defined as the reach and frequency of the marketing message. Power encompasses the nature of the marketing messages, in terms of the creative content, design and execution of the marketing message.

Some European countries have developed and implemented policies explicitly restricting HFSS food promotion to children (e.g. UK and Ireland), however self-regulatory approaches have been most widely adopted by governments, although these have gained criticism for being narrow in scope[117] and ineffective. For example, the voluntary commitments of the food industry through the EU Pledge[118] were not found to prevent the promotion of HFSS foods to children.[119] Improved transparency and standardisation of commitments from food industries have been posited as necessary to ensure the credibility of this EU pledge.[120]

Notably, the majority of approaches are limited to broadcast media, in Europe and internationally, with minimal progress observed within the realm of digital marketing regulation. Some exceptions are Denmark and Norway, where self-regulatory schemes (government-endorsed) now cover internet advertising targeting children and Portugal has implemented restrictions for HFSS food promotion on websites which are child or adolescent targeted. Crucially, as a result of increasing levels of tailoring and personalisation, digital marketing may be a potentially more powerful medium than broadcast advertising, warranting stricter control. Thus governments must be supported by public health researchers and international health bodies to develop appropriate policy action to limit digital marketing and its substantial effects, especially on children. A significant challenge to the effective regulation of the digital marketing environment includes the internet's borderless nature and the feasibility of controlling cross-border promotion. Notably, regulation at a national level is insufficient to address the international nature of food marketing. This process must be aided by a robust evidence base. It is plausible that the established evidence base on broadcast media may have abetted the implementation of television food advertising policy. Indeed, a brief summary of the existing evidence of the impact of promotion on eating patterns (consumption and consumption related behaviours) in both adults and children follows.

3.3.4 Evidence into the impact of food promotion on consumption and consumption related behaviours

A recent narrative review of studies assessing the impact of food promotion (specific to children; 1970-2013) posits a hypothetical framework of the evidence necessary to demonstrate each of the steps of unhealthy food promotion, spanning awareness of food promotion, attitudes and preferences, purchase intent, purchasing behaviours, consumption and post-consumption effects.[121] Importantly, this model questions the notion of a simple, direct, measurable link between food promotion exposure and obesity. Therefore, although studies demonstrating the impact of television food advertising as a predictor of weight status in children may be the pinnacle in terms of policy action, this effect would be difficult, if not impossible, to show using experimental methods. Therefore, demonstrating effects more downstream may have to suffice. Thus whilst studies have sought to show effects in large samples,[122] research attention has been more focused on demonstrating effects on food intake with this as a proxy for weight gain. This is with the inherent assumption that children (the demographic used in the majority of research studies) do not compensate for excess energy consumed after food advertising exposure and that effects on preference make diets overall more energy dense.

This impact of television food promotion on food intake is readily demonstrated across studies, first scrutinised during the 1980s.[123,124] More recently there has been renewed interest in this area, due to increasing obesity prevalence and associated concern over potential determinants compromising the obesogenic environment. Studies in UK samples use energy intake outcomes (i.e., gram/kilocalorie (kcal)); where food bowls are weighed before and after ad libitum intake session) to measure effect of advert type (food or control). A recent meta-analysis combined the data for all studies that have exposed participants to unhealthy food advertising content, either on television or on the Internet. Results showed that such exposure significantly increased food intake relative to following non-food advertising content or no advertising at all in children, but not in adults.[125] This analysis included a

series of UK studies. The first[126] employed a within-participant, randomised experimental paradigm where children (n=42, aged 9-11) were exposed to food and non-food adverts before a television cartoon. Food advertising increased *ad libitum* food intake across all participants, a finding replicated in a subsequent study[127] with children aged 5-6 (n=93). Total kcal intake was significantly higher after exposure to 10 food adverts (compared to 10 toy adverts in the control condition). As the test foods used in both studies differed from brands displayed in the food adverts, these data demonstrate that exposure to food adverts elicits a 'beyond-brand' effect whereby food consumption *in general* is promoted. A further follow up study found that all children displayed significantly increased consumption of sweet energy-dense snacks in response to the adverts but intake was greatest in obese children. Obese children increased their intake by 155%, overweight by 101% and normal weight by 89% after food ads relative to their intake in the control condition.[128]

Researchers have gone further to assess potential moderators and mediators of effects. For example, a UK-based study categorised children (n=66, aged 5-6) as either high or low on a food neophobia scale (i.e. measuring children's reluctance to eat, or avoidance of, new foods) and exposed them to unhealthy food or healthy food advertisements and toy advertisements in the control condition.[129] Food advert exposure (for unhealthy or healthy items) increased highly neophobic children's intake of foods during an *ad libitum* snack break by 11% (47 kcal). In another study[130] a potential gender effect was described, where food intake in boys was higher when watching food advertising compared to girls. Maternal pressure to control weight gain was subsequently investigated as a factor in children's eating post-exposure to food advertising.[130] Children with high maternal pressure increased intake in response to food advertising compared to neutral adverts.

Studies have also explored the direct influence of television food adverts on children's intake response.[130–132] US research[132] has also demonstrated that food advert promotion prompts greater intake, whereby children consumed 45% more snack food after food advert exposure compared to control adverts. Food advertising has been also found to drive desire to eat and motivation to consume[133] and recent studies investigate further mechanisms underlying this associations. A recent study found that television food adverts increase the accessibility of food-related cognitions and motivation to eat.[134]

In terms of the consumption-related behaviours (e.g. food choice and preference), research shows that children reject unfamiliar foods[135] therefore branding can be used by marketers as a practice to overcome this by fostering a sense of familiarity with an entire product range from the same manufacturer. Children who recognise characters, logos and slogans (branding techniques utilised by marketers) from adverts are more likely to select products and brands.[136] A pivotal study[137] supports this premise but contributed a novel finding to this literature; children did not just choose branded foods, they perceived them to taste better, therefore demonstrating that the value of branding goes beyond conscious choice. Children aged 3-5 years were asked to taste identical foods and beverages in McDonald's or in matched but unbranded packaging. Indeed, although the food and drink samples were identical, children indicated a statistically significant preference for the taste of food and drinks labelled with McDonald's brand logos, typifying how the branding of foods impacts children's preferences. Researchers[138] have sought to investigate this further in a controlled laboratory setting by manipulating brand and packaging cues. It was reported that overweight children displayed a cognitive bias toward some food brand images; although the authors note a small sample of children were tested. Thus, this brief summary outlines some examples of evidence demonstrating the impact of commercial food promotion upon children's consumption behaviours.

For adults, fewer empirical investigations of food marketing impact have taken place and conclusions from studies that have been published are mixed. A systematic review of studies conducted in developed countries explored the effects of televised food advertising on adults food-related behaviour, attitudes and beliefs.[139] The review found a varied impact and inconsistency within subgroups (i.e., relating to gender, weight, and existing food psychology). The authors emphasised the need for longer-term studies (not limited to television food advertising), conducted within countries with differing levels of economic development to further this limited research area. A more recent meta-analysis found that although acute experimental exposure to food advertising did increase food intake in children (as discussed above), in adults, there was no significant overall effect across the seven studies identified.[125] There are several explanations for this. Notably, these studies were primarily conducted in laboratories (rather than the more naturalistic eating settings, such as schools, children were tested in), and therefore participants were potentially more aware of their food intake being monitored and may have consciously regulated their eating behaviour. Moreover, study aims may have been insufficiently disguised in some studies, leaving open the possibility that the adult participants attempted to amend their behaviour in line with what they believed the purpose of the research was (demand characteristics). There is some evidence that in real life price promotions and retail displays increase purchases of high sugar foods.[140–142] In England, for example, 40% of food and drink expenditure is estimated to be on products with price promotions[140], with more promotions on HFSS foods than healthy foods[140], a greater impact of promotions on sales in less healthy categories[141] and an estimated 8.7% of all sugar brought into the home is estimated to be extra sugar bought in response to such promotions.[142] From the relatively limited evidence base on the impact of food promotion in adult populations, it is not appropriate to conclude that food marketing does not affect eating behaviour in adults, especially given the massive scale of marketing budgets that companies allocate to promoting HFSS foods to populations of all ages. Further research in this area is certainly warranted.

3.3.5 Summary

Food environments across Europe exploit individuals' biological, psychological, social, and economic vulnerabilities, making them more likely to consume unhealthy foods[143] and impacting on cardiovascular health outcomes. Commercial

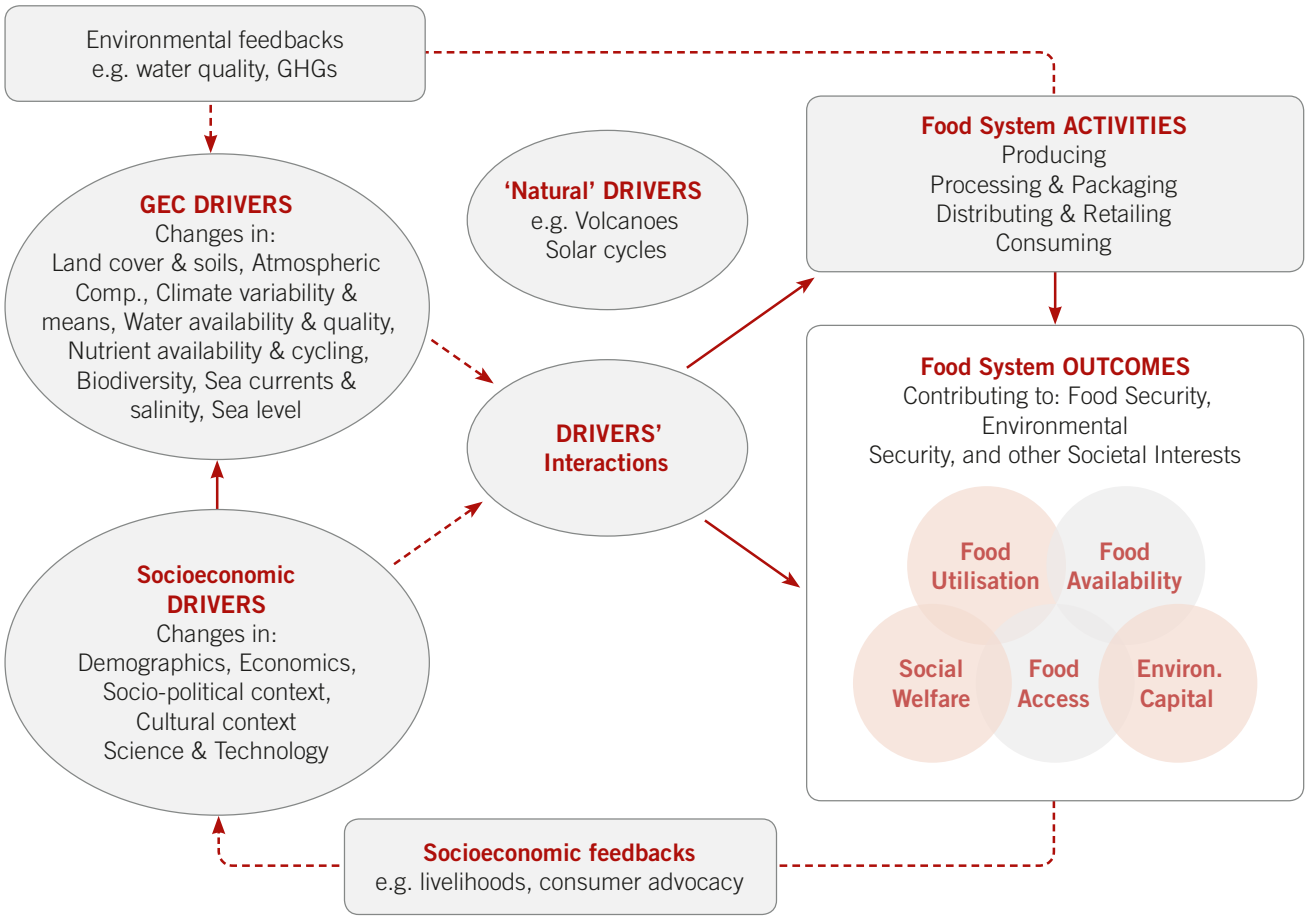
food promotion is one environmental characteristic driving HFSS food consumption. As outlined, promotion now spans both traditional broadcast forms and integrated and targeted digital promotions. Promotional expenditure data discussed in this short review imply shifts from regulated to unregulated media, ensuring improved targeting and personalisation of marketing messages for maximising impact on consumers. Moreover, despite the initiation of voluntary self-regulatory regimes in Europe, as well as statutory regulation in some nations, evidence implies that current approaches are not adequately tackling commercial food promotion where the whole gamut of promotional techniques need to be addressed including digital marketing. This is an active research area, but the balance of evidence currently sits too far in the direction of television, rather than digital, food advertising effects. While more research is warranted on the impact of digital marketing, there is already sufficient evidence of the combined impact of various forms of food marketing to justify decisive policy action to protect consumers from the ubiquitous marketing of unhealthy foods.

3.4 Food-environment links – matching cardiovascular health promotion with a sustainable food system

3.4.1 Introduction

Food systems have multiple outcomes. Food security, diet-related health, and environmental sustainability (with its links into sufficient ecosystem service provision[iv] and renewal) are three focussed on in this sub-chapter. But other important outcomes among which these sit (and sometimes compete) include profit, employment, cultural value and political-economic stability. Figure 30 shows a food systems framework which can be helpful as a way to conceptualise how these outcomes are linked to food system activities and drivers.[144]

Figure 30 Food systems, their drivers and feedbacks Reproduced with kind permission from: Ericksen, 2008.[231]



Note: GEC=Global environmental change; GHG=greenhouse gas.

iv Ecosystem services describe the ways humankind benefit from ecosystems. These are often grouped in to four types: supporting (necessary for the production of other ecosystem services such as nutrient cycling and soi formation), provisioning (products obtained from ecosystems, like food, clean water and genetic resources), regulating (benefits obtained from regulation of ecosystem services, like carbon sequestration and waste decomposition) and cultural (non-material benefits obtained from ecosystems, like spiritual and cultural enrichment).

This framework shows how global environmental change is both a driver and an outcome of food systems. Food systems are seen as comprising 'food system activities' carried out by 'food system actors' which span the entire 'chain' (or cycle) from farm inputs, through to the acts of eating and throwing away food. This framework or approach reminds us that for many food supply chains there are multiple activities and powerful food system actors working between the agricultural stages of production and what is eaten by the final 'consumer'. Along with the consumer, they play an important part in driving food system change and shaping its environmental and health outcomes.

What constitutes a 'sustainable food system' or indeed a sustainable diet in the view of different food system actors will vary, and inevitably involve some trade-offs between different outcomes. This sub-chapter seeks to document the ways in which the food system links into and influences environmental outcomes, and the interactions between environmental and health outcomes. The environment is also changing in ways that affect food systems, with implications for diet and diet-related health. The sub-chapter reviews what we can confidently say about how environmental change may affect diets given the complicated pathways of causation between these elements of the food system. It then goes on to review what we know about policies and practices that are changing what food we consume in ways that promote both sustainability and health outcomes. It ends with some recommendations for next steps.

3.4.2 Impact of food systems on the environment

Food system activities, and particularly agriculture, have considerable environmental impact across a range of areas. These include huge alteration of nitrogen and phosphorous biogeochemical cycling[v] leading to algal blooms and eutrophication[vi] of water courses,[145] the use of agrochemicals to manage pests with detrimental impacts on wildlife and pollinators,[146] climate change impacts, land-use change (with associated impacts on biodiversity), and water use. Here the latter three impacts are focussed on because researchers have been able to quantify them in association with food systems and the production of particular types of food, and in turn have explored the impacts of different dietary mixes.

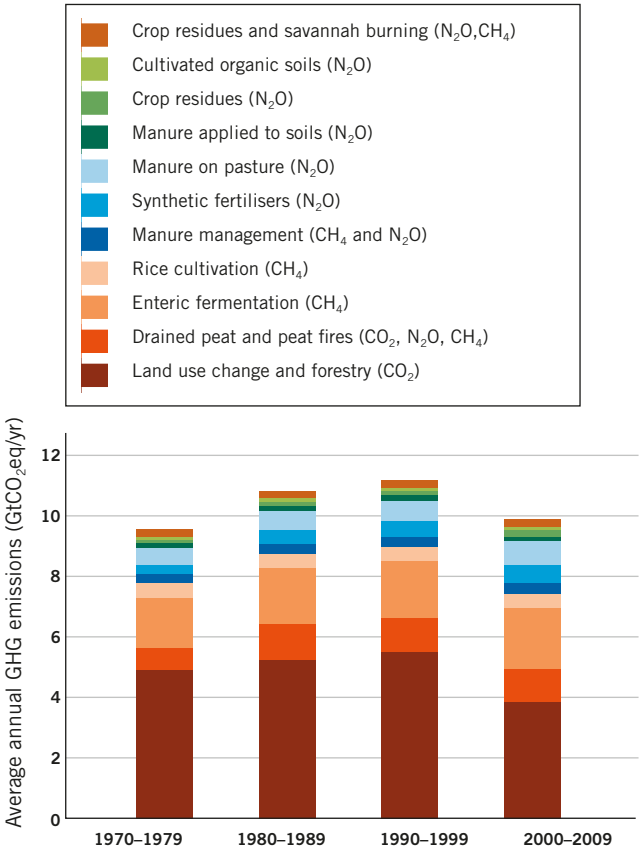
3.4.2.1 Climate change, land use and biodiversity impacts

Globally food production and consumption contributed 19-29% of global greenhouse gas emissions in 2008.[147] Agriculture is responsible for the majority of food system emissions (80-86% according to Vermeulen et al in 2012[147]), although estimates of exact proportions vary according to the boundaries around what is included and excluded in the calculations. Figure 31 shows how this breaks down across different agricultural contributors and over time.[148]

Land use change (LUC) and forestry is a large contributor to the overall greenhouse gas (GHG) footprint of food as agriculture is a major driver of deforestation and LUC. In 2010 the amount of land needed to satisfy the EU's consumption of agricultural goods and services was 43% greater than the land available within its boundaries.[149] The suitability of land for agriculture seems to be a major determinant of the intensity and extent of land use pressures globally.

Tropical deforestation is the single largest threat to biodiversity in land-based ecosystems, in addition to impacts on the livelihoods of around 350 million people who rely on forests, and impacts on ecosystem services.[150] Looking at the possible drivers of deforestation, Defries et al in 2010[151] note the strongly significant correlation between forest loss and both urban growth rates and net agricultural trade per capita between 2000-2005. The latter is a particularly strong correlation in Asia, a major palm oil exporter. They suggest that, 'although these associations do not prove causality, the positive correlations do suggest that the traditional mode of clearing in frontier landscapes for small-scale production to support

Figure 31 Change in Greenhouse Gas emissions over time from the agriculture sector globally Source: IPCC, 2014[148]



v Biogeochemical cycles refer to the movement of chemical elements through the living and non-living parts of the earth system. Biological, geological and chemical aspects to the earth system are all implicated in this cycling.

vi Eutrophication describes the process by which water becomes enriched with excessive amounts of nutrient, causing blooms of plant life, which affect light distribution in the water body and – when they die and decompose – deplete the water of oxygen. This has biological implications through changing the makeup of aquatic plant communities and causing death of oxygen requiring animals in the water body. Some blooms can also be toxic.

subsistence needs or local markets is no longer the dominant driver of deforestation in many places. Rather, our analysis indicates that higher rates of forest loss for 2000-2005 are strongly associated with demands for agricultural products in distant urban and international locations' (p. 178). Expanding oil crop planting was responsible for most agricultural land expansion between 1990-2005.[152] Increasing demand for oil crops – including palm oil – looks set to continue with end uses including direct dietary intake, oilseed cakes for livestock production and non-food uses such as cosmetics, paints, detergents, lubricants and biodiesel.[152]

Biodiversity impacts may be disproportionately large as pressures around LUC are particularly intense, widespread and intensifying in areas with high biodiversity (Venter et al 2016). Because yield increases in major cereal crops are not keeping pace with demand expected to 2050[153] then increases in the area of cropped land seem highly likely. FAO predict increases in cropped areas of land globally by 7% by 2030.[152] Through the development of integrated models, Delzeit et al [154] explored where this might take place globally, finding that while there was some variability in the kinds of areas into which croplands seemed likely to expand, overall, cropland expansion (given climate changes to suitability of land area) risked taking place in many regions that are valuable for biodiversity conservation.

Beyond agriculture other parts of the food system can also contribute significant greenhouse gas (GHG) emissions.

This is particularly the case in countries where high levels of food processing occur, food systems tend to be national/global in nature rather than local, high levels of food waste occur and there is a significant food service sector. In the UK it is estimated that agriculture contributes around 40% of the national 'food GHG footprint' with other contributions including manufacturing (12%), transport (12%), home storage and cooking (9%) and retail and catering (13%).[155]

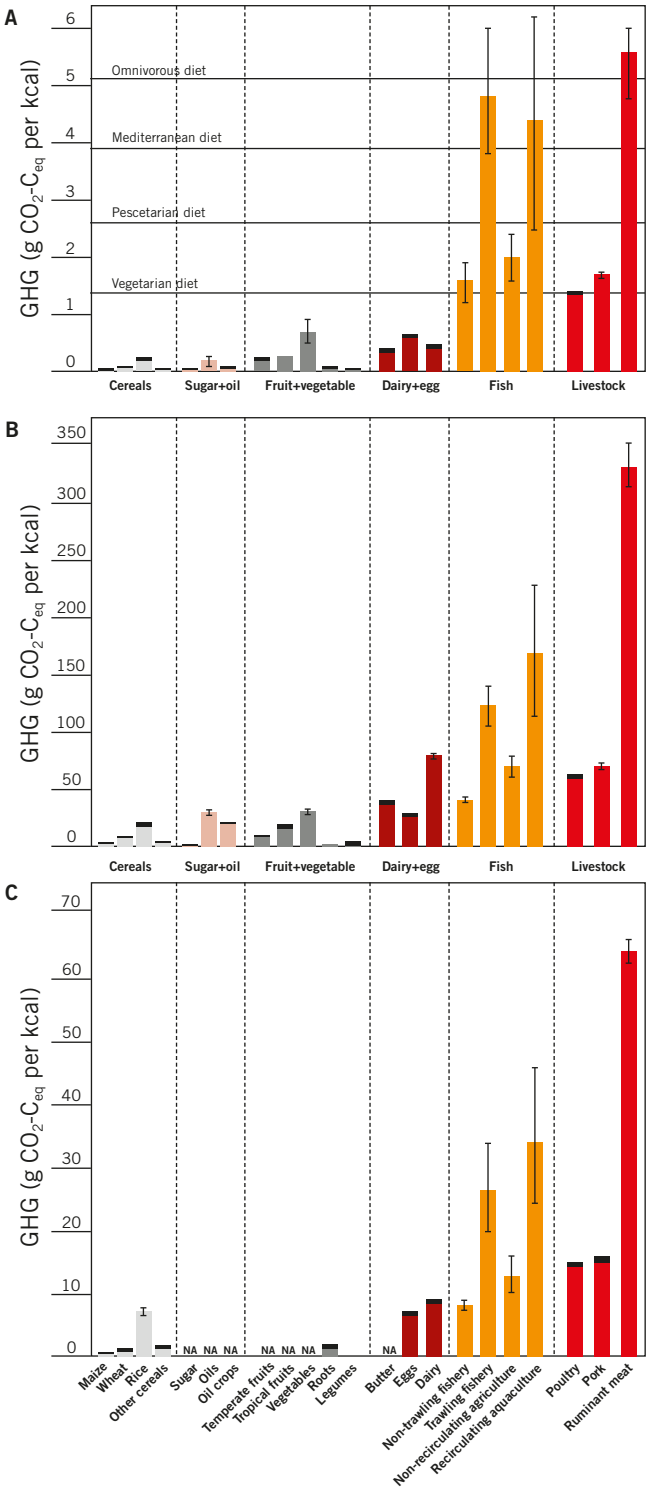
Different food types and individual foodstuffs have different greenhouse gas 'footprints'. This derives from the specificities of their production process – e.g. resource demands for growth, transport mode, growing environment, storage requirements. The following discussion of greenhouse gas impacts and diet tries to draw generalisable lessons, despite the potential for variation more specifically.

Relative to animal-based foods, plant-based foods tend to have lower GHG emissions. This can be seen in Figure 32 comparing the GHG emissions of different food groups from Tilman and Clark.[156]

Figure 32 Lifecycle GHG emissions (CO_2-C_{eq}) for 22 different food types. Source: Tilman and Clark[156]

The data are based on an analysis of 555 food production systems: a) per kilocalorie; b) per United States Department of Agriculture (USDA)-defined serving; c) per gram of protein. The mean and Standard Error from the Mean (error bars) are shown for each case.

NB. Because different food groups play different roles in the diet, it is most useful to look at the comparative GHG emissions per calorie for cereals (and any other food type eaten for calorific benefit), per serving for fruits and vegetables, and per g protein for animal products and legumes.



The generally lower GHG impacts of plant versus animal based foods is because it is more resource efficient to eat from a lower trophic level[vii] (i.e. plants), than have to feed plants to animals and then eat the animals; livestock currently supply 13% of energy and 28% of the protein to the world's diet but consume half the world's production grains to do so (see IAASTD 2009 in Smith et al 2013,[157]). Furthermore, ruminant animals emit methane when feeding, making them additionally powerful emitters of greenhouse gases.

While poultry and pork have lower GHG impacts, they are commonly fed on grains which could otherwise be eaten by humans, while ruminants are able to digest grass and crop-residues therefore using grasslands and – where grazed sustainably – providing some cultural ecosystem services (e.g. maintaining grassland landscapes).[157]

So, environmental impacts depend on a number of factors: what livestock is raised; the conditions under which they are raised; the volume in which they are consumed; the relative importance placed on different environmental impacts they create (e.g. GHG emissions or land-use efficiency); and the opportunity costs and benefits they represent (e.g. how else might the land/grain/water they use be used, but, equally, would more fertiliser inputs be required in place of their waste).[158] If efficient land and resource use is a priority food system outcome in the European setting, it is interesting to note that the Health Council of the Netherlands estimates that 40-50% of existing livestock in Europe could be fed on natural grasslands and food industry waste products.[159]

In some circumstances livestock and ruminants in particular can be important contributors to food security, family asset management, livelihood opportunities and stability. They also help with nutrient cycling (e.g. providing nitrogen for crops) when part of mixed farming systems. So while there are clear environmental impacts of animal-based foods, careful *context-based* assessment is required regarding their 'sustainability'.

Another source of protein important to many diets globally, and in Europe, is fish. Figure 32 above shows that different sources and fishing techniques have different GHG impacts. Trawling fisheries have a high impact, in addition to having high levels of by-catch and, when bottom trawling, being destructive to ocean floor habitats.[160] Fishing in the ocean in general is being done to levels at (60%) or exceeding (30%), the level at which fish stocks have the capacity to recover.[160] Globally about half of all fish consumed are now farmed,[160] although in Europe it is 20%.[161] How and what is farmed also affects the environmental impact of this production method – while recirculating aquaculture has a higher GHG impact, its use of filtered water systems means much less water is used and pollution of this water can be better regulated.[162] In addition, farming of carnivorous or 'fed' fish (the dominant farmed aquatic food producing around 70% of aquaculture output, [160]) currently relies on by-catch/wild caught fish as a source of feed, making their production inextricably linked to sustainability issues in ocean fisheries. Farming of herbivorous fish and molluscs, and development of novel feedstuffs holds promise.

3.4.2.2 Water use impacts

Food production and consumption also relies on considerable water resources, with agriculture being the main water use activity in the food system (although processing and in-home use can be considerable too).[163] Agriculture is responsible for 70% of global freshwater withdrawals and more than 90% of its 'consumptive use'[164] – i.e. use of water that does not return to the land-based water environment for potential downstream use and is instead transpired.

A product's water footprint is defined as the total volume of freshwater used to produce a good. This can then be separated into blue, green and grey water, explained below[viii].

Green water footprint is water from precipitation that is stored in the root zone of the soil and evaporated, transpired or incorporated by plants. It is particularly relevant for agricultural, horticultural and forestry products.

Blue water footprint is water that has been sourced from surface or groundwater resources and is either evaporated, incorporated into a product or taken from one body of water and returned to another, or returned at a different time. Irrigated agriculture, industry and domestic water use can each have a blue water footprint

Grey water footprint is the amount of fresh water required to assimilate pollutants to meet specific water quality standards. The grey water footprint considers point-source pollution discharged to a freshwater resource directly through a pipe or indirectly through runoff or leaching from the soil, impervious surfaces, or other diffuse sources.

Given that using large quantities of water is only an issue if that resource is scarce locally, a further disaggregation of the footprint approach looks at 'blue water scarcity'. This is a more geographically specific measure and links blue water volume available with the human demand on that water in that locale. This 'stress weighted water usage' shows whether products use water in ways that increase scarcity.

The production of meat and dairy products requires a lot of water, again due to the relative inefficiency of converting feed into animal protein. Annex 2, taken from Mekonnen and Hoekstra,[163] illustrates the range of estimated water footprints for different food goods. Given this, a number of studies have

vii Trophic level refers to the position of an organism in the biological food chain. Plants are seen as primary producers and at the first trophic level. Herbivores consume plants and are at the second trophic level. While omnivores/carnivores eat at the second and third trophic levels.

viii http://waterfootprint.org/en/water-footprint/what-is-water-footprint/

looked at the water impacts of reducing or removing meat and dairy from the diet. A review of five studies looking at vegetarian diets (three European and two Californian) in Hess and colleagues in 2015 shows reductions in the overall water footprint of between 33-66% compared to reference diets.[165] However, Meier and Christen in 2013 find that blue water use increased by 85% in their vegetarian diet and 107% in their vegan diet because they assumed a significant increase in consumption of nuts and seeds for these diets.[166] These are grown in areas with low rainfall and high reliance on stored water sources, and hence the high blue water footprint.

While, on the whole, studies comparing 'healthy' (following dietary recommendations) and reference diets show a decreased water footprint for the healthy diet, the extent to which this is the case varies. This is because results are sensitive to what is measured (total water footprint vs. blue water footprint or blue water scarcity), and the assumptions regarding exactly what is eaten in different diets compared to a reference. While Vanham[167] finds a 23% reduction in overall water use when following the German Nutrition Society recommendations compared to the baseline, and Meier and Christen[166] find a ~27% reduction in blue water use following the German Nutrition Society and the Federation for Independent Health Consultation, Hess et al[165] find only a 2.5% reduction in blue water use for a UK diet following the Eatwell plate. The relatively small reduction from Hess's research seems to arise from an increased consumption of rice, fruit and vegetables imported from water scarce areas, and increased milk consumption compared to the reference. This highlights the importance of accounting for where products are grown/ raised, as well as the products themselves.

3.4.2.3 Waste

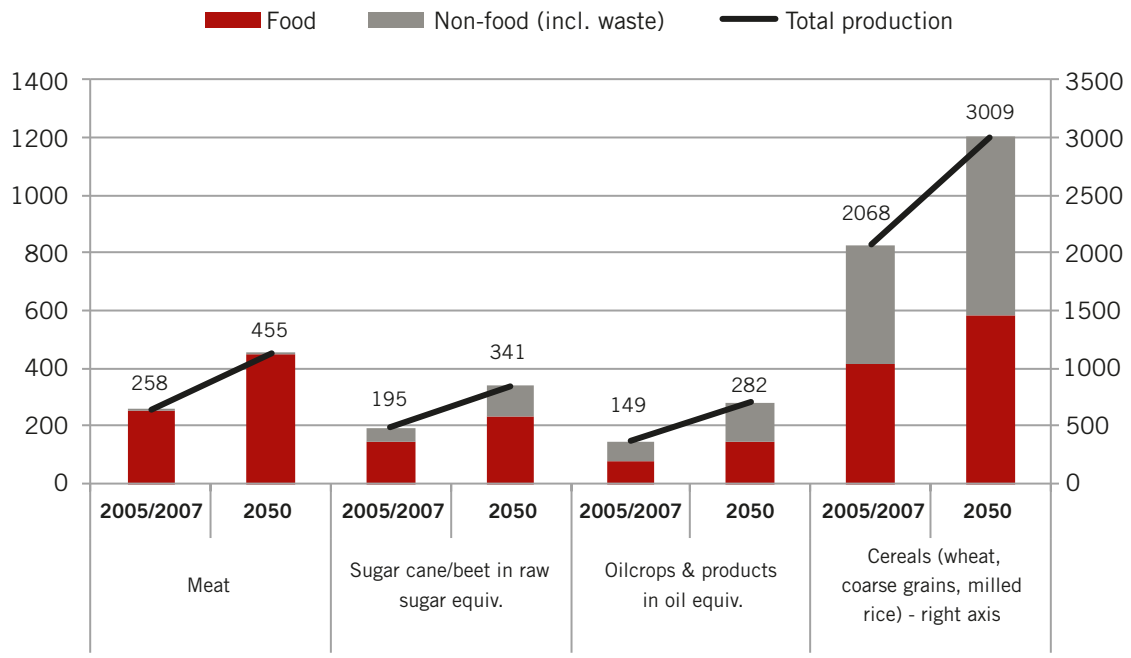
It is estimated that about one third of all food produced for human consumption is wasted, representing not only a lost opportunity for consumption but also unnecessary use of resources. Per capita food losses in Europe and North America are in the region of 280-300 kg/year, while in sub-Saharan Africa and South/Southeast Asia it is 120-170 kg/year.[168] The proportions arising from different stages of the food supply-consumption chain vary from place to place too – more is lost from early and middle stages in low-income countries, while industrialised countries waste more at the consumer and retail stages.

3.4.2.4 Trends

Trends in terms of population increase, and changes to diets globally, suggest that the demand for food, across commodity groups, will increase in the future (see Figure 33). Assumed dietary changes, often encapsulated by the 'nutrition transition' include increased meat consumption, increased consumption of refined sugars, fats, oils, processed foods and alcohol, increased calorie intake and reduced consumption of fruits, vegetables, coarse grains and tubers.[12,169] These trends are linked to increased incomes in low- and middle-income countries, urbanisation, globalisation and cultural homogenisation, and technological diffusion.

From a dietary perspective, Tilman and Clark in 2014 point out that, 'if we look at trends in dietary change with forecasts of per capita income to 2050, relative to 2009, it is predicted that in 2050 global average per capita income-dependent diet would have 15% more total calories and 11% more total protein, 61% more empty calories, 18% fewer servings of fruits and vegetables, 2.7% less plant protein, 23% more

Figure 33 Predicted changes in world production and use of major products (millions of tonnes) Source: Alexandratos and Bruinsma, 2012.152



pork and poultry, 31% more ruminant meat, 58% more dairy and eggs, and 82% more fish and seafood'.[156]

While there has been some decoupling of GHG emissions from food production[ix] at the agricultural stages between 1970 and 2007, with emissions per unit of product declining by 39% and 44% for crop and livestock production respectively, efficiency gains in GHG emissions have not kept pace with the larger increase in demand. Crop and livestock production have increased by 118% and 102% in that time, respectively.[170]

Using Life Cycle Assessment (LCA) emissions data Tilman and Clark calculated annual per capita GHG emissions from food production at the farming stage alone for an average global diet in 2009, and then for the global-average income-dependent diet[x] projected to 2050.[156] Combined with global population projections of 36% increase to 2050, the net effect is an estimated 80% increase in global GHG emissions from food production (from 2.27 to 4.1Gt/Yr of CO2e). Note this may be slightly tempered if the efficiency improvements characterised by Bennetzen et al continue.[170] Nevertheless, global aspirations to prevent greenhouse gas emissions exceeding levels linked to more than 2°C warming require net zero emissions globally by 2100.[171] While it is recognised that the agricultural sector cannot fully decarbonise, this increasing trend presents a serious problem requiring technologically complex net negative emissions from other sectors to compensate. Water and land-use demands will also increase, suggesting even further pressure on the natural resources underpinning our food production systems into the future.

3.4.2.5 Impact of food systems on the environment: conclusions and key messages

- Food based GHG emissions contribute 19-29% of emissions globally. This may increase by around 80% to 2050 given current global trends. This makes the already extremely challenging target of achieving net zero GHG emissions by 2100 – as set out in the Paris Climate Change Agreement – even more difficult.
- Relative to animal-based foods, plant-based foods tend to have far lower GHG emissions. This is because it is generally more resource efficient to eat from a lower trophic level (i.e. plants), than have to feed plants to animals and then eat the animals. On the whole, animal based foods also have higher water footprints than plant-based foods.
- The impact of high water use is determined by local water scarcity. Environmental impacts and other food system outcomes from animal rearing also depend on how animals are raised, and the local economic role of livestock. This suggests that *to a degree* the overall sustainability impact of animal based foods is geographically and practice dependent.
- Trends globally are towards higher levels of meat and animal product consumption.
- It is important that what we eat, particularly in Europe, becomes part of the discourse around achieving our climate commitments.

ix That is a weakening of the strength of the relationship between greenhouse gas emission creation and agricultural food production.

x Because diets globally tend to change quite predictably, for key indicators, as income increases, it was possible to model a future 'income dependent diet' based on assumptions of how incomes would increase into the future.

3.4.3 Impact of predicted environmental changes on diet and diet-related health

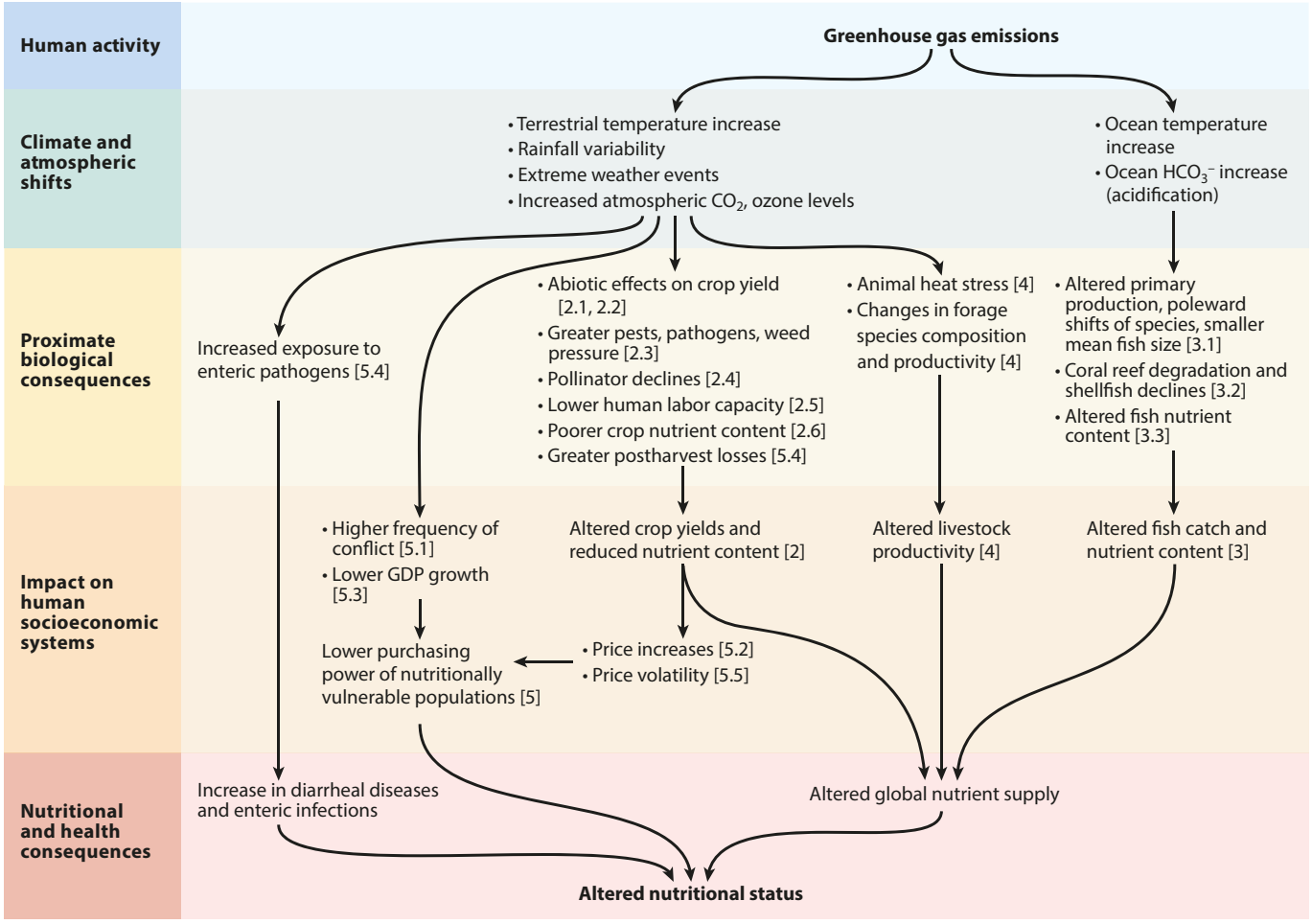
Determining the impact of predicted environmental changes on diet and thus diet-related health involves a pathway of causality which can be quite hard to untangle and attribute. And as Nelson *et al* note in reference to linking climate change and food security outcomes, 'combined biophysical-socioeconomic modelling of this detail and extent is still in its infancy'.[172] Figure 34 below traces out just some of the ways in which climate change in particular links to altered nutritional status.[173] Not included in this diagram are the myriad ways in which climate change may affect diet and diet related health beyond just price impacts – for example around extreme weather events affecting logistics; increased cold storage demands under higher ambient temperatures; and increased food safety concerns with higher ambient temperatures (bacteria and mycotoxins).[174]

The degree of agricultural and food system adaptation will also define the ultimate impacts of environmental change – for example, Challinor et al, in a meta-analysis of studies on climate change-yield links published in 2014, found yields were 7-15% higher with adaptation than without it.[176]

Myers et al, in their 2017 review of studies looking at climate change and food security, also outline potential impacts of climate change on wild fish catch.[175] A study quoted by Cheung et al suggests potential global reductions in catch by 3-13% on average under a high emissions scenario, but with spatial variability; some regions may experience 30-60% reductions.[177] The impacts on aquaculture are less clear, and may even see increases in production in some areas as sea ice recedes and fish potentially grow faster in warmer conditions. Altogether the complexity of marine ecosystems and their interactions with climate change make this a highly uncertain field to model.

But how much changes in production/catch affect the final retail price of food will depend on the proportion of final end-price comprising the cost of the commodity. Other influential factors will include the nature of local markets, the degree of processing and other added value etc. (which may also be affected by environmental change, e.g. cost and availability of water for processing or cleaning) and the ways in which

Figure 34 Pathways for impacts of climate change on food systems, food security, and undernutrition Source: Myers el al, 2017 175



environmental change affects physical access to food and markets (e.g. localised flooding, or food spoilage through heat-related power cuts). Knock-on implications for diet-related health will depend on what foodstuffs make up the diet and how people adapt.

Elevated ambient CO_2 levels also affect the nutritional composition of crops. In field-grown crop experiments by Myers and colleagues some C_3 crops – wheat, rice, field peas and soybeans – were found to have lower concentrations of iron and zinc with elevated CO_2 (around 3-10%).[173] Protein levels were also reduced in wheat and rice (6.3% in wheat and 7.8% in rice).[173] For those relying on these crops as a significant source of dietary zinc, iron and protein in the future, this presents an increased risk of deficiencies in populations already vulnerable to undernutrition (in 2010 about 2.3 billion people were living in countries whose populations received at least 60% of their dietary zinc and/or iron from C_3 grains and legumes).

Different groups of people will also be affected differently – where there is direct reliance on crops, such as in subsistence systems, diet and diet-related health may be directly affected by detrimental changes in the biophysical environment. Price increases may benefit those who sell agricultural products. Where people rely on local and non-integrated markets (i.e. there is little flow of goods between markets), again, changes in food production due to environmental changes will be more strongly reflected in changes to the diet. However, where people rely on food bought in globally integrated markets, the impact of environmental change on diet will be more difficult to predict given the substitutability of products and growing areas. Finally, purchasing power, and how this changes over time and alongside the environment, will mediate the sensitivity of people to changes in food prices.

What research has been done looking at the potential for future environmental change to influence food security has focussed on climate change specifically, and is biased towards impacts at the agricultural stage and towards quantifying impacts on food price and calorie availability (rather than looking at other aspects of what comprises food security such as physical access, safety, suitability, micronutrient availability, etc.). Furthermore, 'substantial differences in projections of price, production, and land-use changes by different models exist, implying a high degree of model uncertainty and impact projections'.[174] In a systematic model comparison, a range of climate, crop and economic models were run with a high emissions climate scenario and a 'middle of the road' shared socioeconomic pathway to 2050. This found global average yield reductions of 11% and price increases of 20% compared to the baseline values for major crops.[178]

In an earlier study and using a single integrated model, Nelson et al determined in 2010 that prices would rise by between 31-106% by 2050 for wheat, rice and maize.[172] This range is caused by variations in assumptions regarding the amount of climate change mitigation, population and economic growth under different future scenarios. In terms of nutritional and human impact, under optimistic scenarios (higher GDP, lower population growth, higher climate change mitigation) there was an average 45% reduction (50% reduction in MICs and 37% reduction in LICs) in the number of malnourished children globally compared to the 2010 baseline. With a pessimistic scenario, the average reduction in the number of malnourished children achieved was 2%, representing a 10% reduction in MICs but an 18% increase in LICs.

For the reasons given above, linking environment or climate changes to *dietary changes* is highly complex. However, Springmann and colleagues have attempted this with a global modelling study focussed on potential changes to fresh fruit, vegetable and red meat production and then consumption under future climate change and socio-economic scenarios.[179] The consequent diet- and weight-related health impacts of this for populations in 155 world regions are also modelled. This study found that climate change (assuming a 'high emissions scenario') could lead to reductions in overall food production globally. Fruit and vegetable consumption was estimated to reduce by 4%, and red meat consumption by 0.7%. Through knock-on implications for undernutrition, dietary change and weight changes, it was estimated this would then lead to 529 000 climate related deaths worldwide by 2050.

The regional impacts are highly uneven however, with higher deaths in the western Pacific region in particular, but also south and central Asia, central Africa and the eastern Mediterranean and eastern Europe. The cause of death also varies – with changes in fruit and vegetable consumption responsible for about 550 000 additional deaths globally, and underweight about 250 000. This is slightly offset by reductions in deaths from red meat consumption, overweight and obesity of about 300 000 deaths. Again, how people are more or less likely to die varies regionally. Additional death from being underweight dominates in the LMICs of Africa, and is responsible for over half of additional deaths in the LMICs of Southeast Asia. While additional deaths from reduced fruit and vegetable consumption dominates in high-income countries and the LMICs of the Americas, eastern Mediterranean region, western Pacific region and Europe. The sensitivity analysis of this study suggests that climate change mitigation would greatly reduce this number of deaths.

3.4.3.1 Impact of predicted environmental changes on diet and diet-related health: conclusions and key messages:

- The 'pathway to impact' between future environmental change and dietary intake is extremely complex and is strongly mediated by future population, economic, trade and cultural change.
- Beyond studies looking at changes to caloric intake with climate change, there is very little research linking a changing climate to dietary impacts. There is virtually no research looking at how environmental change more broadly may impact on diets in the future.
- Modelling research suggests that climate change will have negative implications for diet-related health overall, due to reductions in calorie intake for poorer populations and reductions in fruit and vegetable consumption for wealthier people.

3.4.4 Identification of ideal dietary patterns that satisfy health and sustainability criteria

There is now a reasonable body of evidence exploring dietary patterns that satisfy both health and *some* sustainability criteria – again, with an emphasis on GHG emissions, but also land and water use. Some of the evidence in support of this is reviewed before outlining proposed 'ideal dietary patterns'.

Tilman et al conducted a meta-analysis of research and compared 'emerging global diets' with three 'well studied' diets.[156] Altogether 10 million person-years of observation, across eight study cohorts were amassed to compare disease incidence rates and environmental impacts between these diets.

The diets studied were defined as follows:

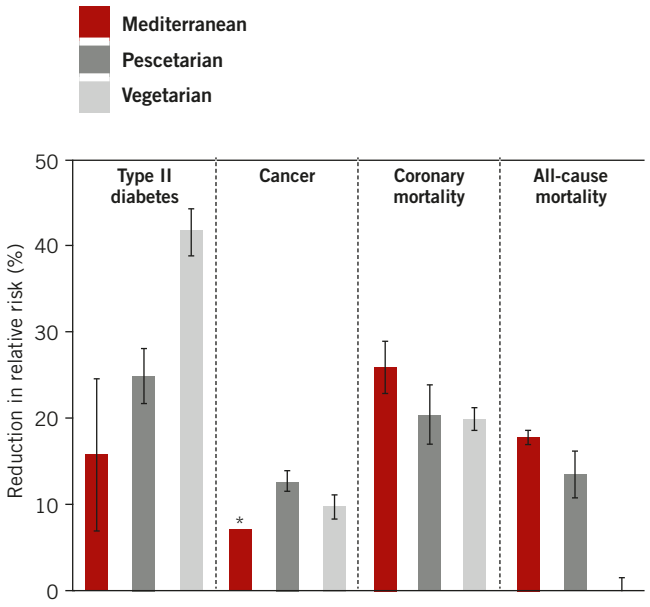
- **'Emerging global diet'** – typical omnivorous diet used in the cohort studies reviewed for comparison with alternative diet.
- **Mediterranean** – rich in vegetables and fruit, seafood and includes grains, sugars, oils, eggs, dairy and moderate amounts of poultry, pork, lamb and beef.
- **Pescetarian** – vegetarian diet and seafood.
- **Vegetarian** – grains, vegetables, fruits, sugars, oils, eggs and dairy, and generally not more than one serving per month of meat or seafood.

Figure 35 shows a reduction in the relative risk of type II diabetes, cancer, coronary mortality and all-cause mortality (except for vegetarianism) in the three alternative diets compared to the 'global diet' baseline.

The GHG emissions and land-use implications of the different diets were also compared, including against a projected, 'income-dependent 2050' diet which assumes an increase

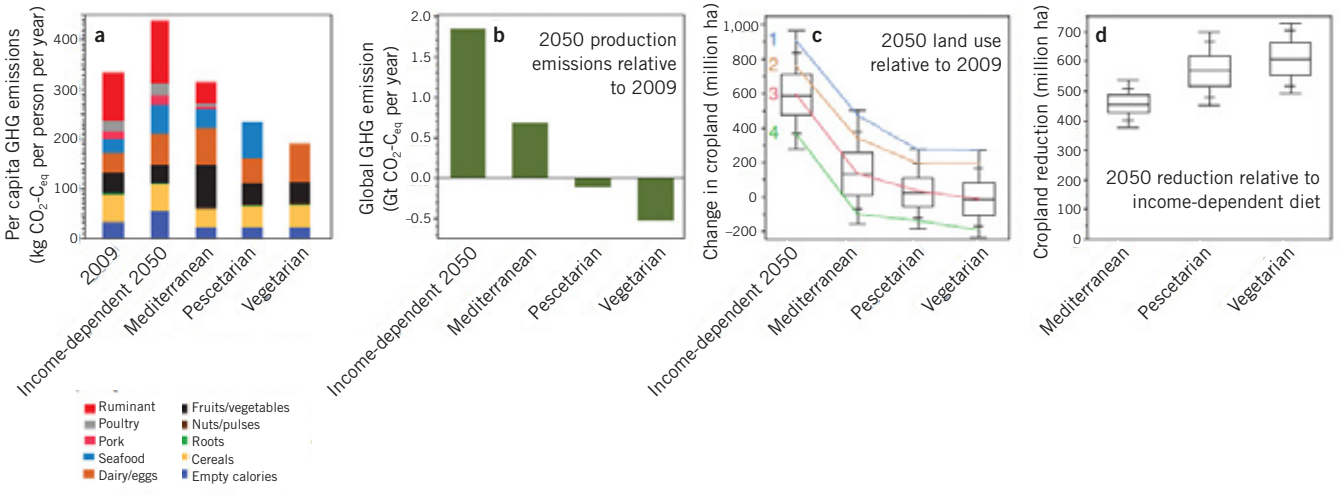
Figure 35 Diet dependent percentage reductions in relative risk of type II diabetes, cancer, coronary heart disease mortality and of all-cause mortality when comparing each alternative diet. * Cancer from Mediterranean diets is from a single study so no s.e.m is shown.

Source: Tilman and Clark, 2014 [156]



in the number of people globally eating more 'westernised' diets. The results, as shown in Figure 36, clearly show the considerable differences between GHG emissions for the different diets, which looking to 2050 would lead to considerable savings in GHG emissions and land use with eating Mediterranean, pescetarian and especially vegetarian diets. For the latter two diets, emissions would be net negative despite large rises in population by 2050.

Figure 36 Effect of diets on GHG emissions and cropland. a) per capita food production GHG emissions for five diets, b) forecasted 2009 to 2050 changes (2009 set to 0) in global food emissions, and c) cropland area used for each diet. d) 2050 global cropland reductions from alternative diets relative to the income dependent diet. The box and whiskers plots show mean and percentiles below (2.5th, 10th, 25th) and above it (75th, 90th, 97.5th) based on 243 scenarios. Source: Tilman and Clark, 2014 [156]



In a systematic review of studies comparing the GHG and land-use impacts of different diets, Hallström et al found the following reductions in land-use across four studies reviewed, in percentage of relative change in land demand compared to reference scenarios.[180]

And the following changes in GHG emissions from 12 articles reviewed – again, in percentage of relative change in GHG emissions compared to the reference scenarios.

Figure 37 Impact of dietary change on current demand of land from the diet, in % of relative change in land demand compared to the reference scenarios. Data presented are from four articles Source: Hallstrom et al 180

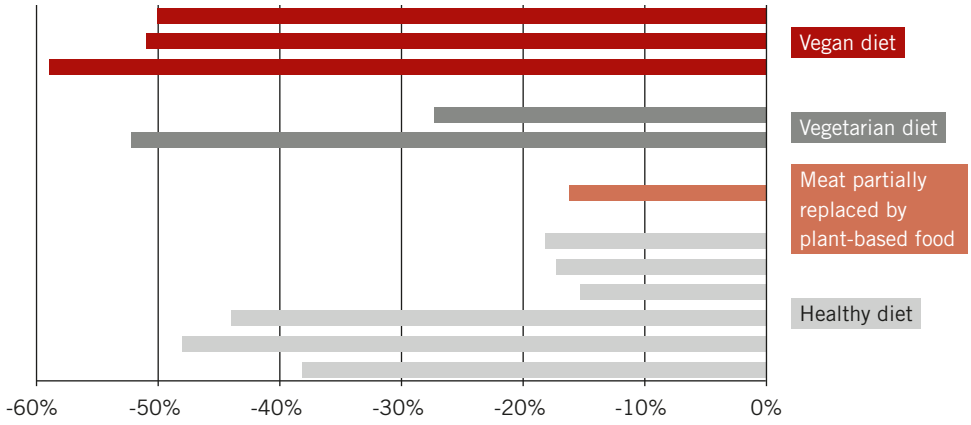


Figure 38 Impact of dietary change on GHG emissions from diet, in % of relative change in GHG emissions compared to the reference scenarios. Results drawn from 12 articles Source: Hallstrom et al 180

It is worth noting that emissions are not always reduced by reducing meat consumption or following healthy guidelines. Some studies – Tom et al[181] and Vieux et al[182] – finding emission increases when following guidelines for a healthy diet. This is a matter of understanding both the nature and 'quality' of baseline existing diets in addition to what 'healthy' means in different countries. In the case of the Tom et al study, the guidelines were the USDA dietary recommendations which advise unusually high dairy consumption compared to many other national dietary guidelines. Equally, very large reductions in the consumption of added sugars is required compared to the baseline American diet, which do not have a large GHG footprint. In part this was then to be replaced by fruit (with considerably higher GHG impacts). To meet these guidelines Americans would need to increase their caloric intake from fruits, vegetables and dairy by 96, 104 and 204 calories daily. The Vieux study similarly had healthy diets that were lower in ruminant meat but higher in dairy, but with similar levels of pork, chicken and egg consumption to 'unhealthy' diets.[182] Here, sugary foods in the unhealthy diets were replaced by high levels of fruit and vegetable consumption. The emissions associated with fruits and vegetables depend on the degree to which they are grown in protected settings, eaten out of season, transported long distances and cold stored etc.

The review of water use implications of food, and of different diets, in section 3.4.2.2, also highlights that while low-meat and 'healthy' diets can have lower water and blue-water footprints, this is not always born out. Substitution of 'unhealthy' foods with high water footprint foods such as rice, fruits and dairy, will lessen (or indeed reverse) any potential savings.

Altogether, this suggests that while following a 'healthy' diet according to national guidelines does not automatically mean reductions in the GHG, land-use and water impacts of that diet, there are considerable reductions possible if a low-meat and healthy diet is consumed via particular types of food. Where there is a potential clash is around the consumption of fish and oily fish given already high levels of fish stock exploitation, and high interdependence between farmed and wild fish stocks. Highly intensive animal rearing practices can also have lower GHG impacts per unit produced, but have poorer animal welfare outcomes.

An internationally accepted definition of a 'sustainable healthy eating pattern' does not exist, but Garnett and colleagues suggest in 2015 that there is increasingly an understanding of what these look like (based on existing research which tends to see sustainability as environmental, and within that emphasising GHG emissions, energy use and to a lesser extent water use.[183] Socio-economic or animal welfare aspects are not included). Broad principles for a healthy and sustainable diet are that they should be:

- Diverse in the energy density of foods (a mixture of some foods that have relatively few calories gram and some that are more energy dense)
- Low in animal products with all parts of the animal eaten
- Fish and fish related products eaten in moderation
- High in minimally processed, robust (i.e., products that have a longer shelf life and do not rely on excessive packaging or energy intensive storage conditions in order to be successfully transported and sold at retail), field-grown vegetables and in fruits
- Rich in whole-grains, tubers and legumes
- Low in processed foods high in fat, sugar and salt

Micronutrient deficiencies are a risk in some contexts, so reducing meat intake needs to be matched with careful increases in quantity and diversity of whole grains, legumes, fruits and vegetables. What this will look like in terms of actual foodstuffs eaten will vary from place to place according to what is able to be grown/ caught locally in an ecologically sound manner.

An example of expert-based dietary guidelines addressing both the healthiness and environmental sustainability of diets is the Swedish dietary guidelines published in 2015 (Livsmedelsverket 2015), which can be summarised as:

- Eat lots of fruit, vegetables and berries - high fibre vegetables such as root vegetables, cabbage, cauliflower, broccoli, beans and onions are an eco-friendly choice with less environmental impact than salad greens
- Eat fish and shellfish two to three times a week – vary the type of fish and look for products with sustainability labels
- Exercise at least 30 minutes every day
- Switch to whole grain for pasta, bread and cereals – all cereals have low carbon footprints and pesticide use is low. Rice is one of the crops with causing the most GHG emissions, so other grains and potatoes are a better choice for the environment
- Choose healthy fats like rape seed oil – rapeseed oil and olive oil generally have less of an impact on the environment than palm oil, while butter has a higher carbon footprint than vegetable oil but can help bring about a rich agricultural landscape and biodiversity
- Choose low-fat, unsweetened dairy products fortified with vitamin D. Methane from cows affect the climate. Therefore do not consume too much cheese and other dairy products; 0.2-0.5 litres of milk (not including cheese) a day is enough for calcium. However, cows can contribute to biodiversity conservation through the grazing of pastures
- Eat less red and processed meat – a maximum of 500 g red and processed meat per week (no limitation on chicken or other white meat) – meat is the food product that affects the climate and the environment the most, and it is therefore important to consume less
- Choose foods with less salt
- Reduce intake of sweets, cake, ice cream and other sugary foods – these unnecessary food cause environmental impact can contain lots of calories but hardly any nutrients
- Try to find your energy balance by eating just enough.[184]

The Dutch dietary guidelines, published in 2015, also seek to take advantage of the synergies between a healthy and sustainable dietary pattern. They have the following key messages, as summarised by the FAO:

- Follow a dietary pattern that involves eating more plant-based and less animal-based food, as recommended in the guidelines
- Eat at least 200 grams of vegetables and at least 200 grams of fruit daily
- Eat at least 90 grams of brown bread, wholemeal bread or other wholegrain products daily
- Eat legumes weekly
- Eat at least 15 grams of unsalted nuts daily
- Take a few portions of dairy produce daily, including milk or yogurt
- Eat one serving of fish weekly, preferably oily fish
- Drink three cups of tea daily
- Replace refined cereal products by whole-grain products
- Replace butter, hard margarines, and cooking fats by soft margarines, liquid cooking fats, and vegetable oils
- Replace unfiltered coffee by filtered coffee
- Limit the consumption of red meat, particularly processed meat
- Minimise consumption of sugar-containing beverages
- Don´t drink alcohol or no more than one glass daily
- Limit salt intake to 6 grams daily

Nutrient supplements are not needed, except for specific groups for which supplementation applies.[185]

The DEFRA publication and Swedish guidelines note the importance of eating seasonal and sustainably caught fish.[185,186] This is an area where there is a potential discordance between healthy eating guidelines and environmental outcomes. Many healthy eating guidelines suggest fish consumption above current levels, and given global fish stocks are already highly vulnerable to exploitation, meeting this healthy eating target has environmental trade-offs. However, it is interesting to note that the Dutch guidelines now recommend only one portion of fish a week, compared to two in its previously published guidelines and two to three portions in the Swedish guidelines.

3.4.4.1 Identification of ideal dietary patterns that satisfy health and sustainability criteria: conclusions and key messages

- Encouragingly there is considerable overlap between consuming 'healthy, lower meat diets' and achieving higher levels of sustainability as defined by GHG emissions, land-use and water use.
- However, clear guidance would be needed to ensure that nutritional demands are met while achieving better sustainability outcomes; there is the potential for poorer sustainability outcomes when some foods are substituted into the diet.
- There is a lack of research looking at healthier, low meat diets and wider indicators of environmental sustainability such as biodiversity impacts, nitrogen and phosphorous use, pollinator impacts, etc. (Although there is no obvious reason to think that results would look significantly different when considering these other impacts. It seems more likely they would provide a more nuanced picture rather than a different one).
- There is a lack of research on the implications of a low meat and healthy diet for the socio-economic aspects of sustainability (such as equity or livelihood impacts).

3.4.5 Existing proposed policy solutions to health-environment issues around food, and evidence to justify approaches

Very few policy solutions bridge the health and environment impacts of food at present. As noted above, some dietary guidelines – including those for Sweden, the Netherlands, Germany, Australia, Brazil and the Nordic countries – have incorporated sustainability criteria to a greater or lesser extent. These, along with an exploration of other interventions that can be used to shift diets towards healthier and more sustainable eating patterns, will be briefly discussed here. This review will heavily draw from Garnett and colleagues who reviewed policies and actions to shift eating patterns towards better health and sustainability outcomes.[183]

Combining health and sustainability in dietary guidelines is a significant development in this field. There is potential to reach a wider audience for dietary guidelines, with different motivations to act on those guidelines, by integrating sustainability issues.

While health professionals may know the content of dietary guidelines well, if they are to effectively and confidently to communicate these, knowledge of the evidence behind such messages is required. However, Murphy suggests this is 'less well established' amongst UK health professionals than knowledge of the guidelines themselves. The complexities associated with health-environment links would need to be understood in the case of effective healthy and sustainable eating guidelines.[187]

In addition, while 100% of participants in a questionnaire conducted by Rooney et al (2013) knew of the 5-a day guideline on fruit and vegetable consumption in the UK,[188] over 60% of adults do not meet this target in the UK.[189] So, there is considerable evidence of the knowledge-action gap when it comes to eating behaviours (as well as environmentally motivated behaviours).

As well as being a pre-cursor for action, knowledge among the general public may be an important basis for governments, public health bodies and companies being able successfully to introduce new policies. While evidence from a six-country European survey[xi] suggests there is quite good knowledge of nutrition such as what should be eaten often, a bit and rarely, knowledge around different types of fat and consumption of red meat was limited.[190] In terms of environmental knowledge on food, an 18 country survey[xii]191 found that while people generally believed significant change is needed to improve the sustainability of the food system, they felt personally alienated and powerless to make changes. There was also little understanding of the links between meat production/consumption and environmental impacts. However, the context may be shifting in some countries, with a 2013 YouGov poll in the UK finding 31% of respondents knowing that there are significant environmental impacts from producing meat, up from 14% in 2007.[192] The most significant change in knowledge was seen among younger respondents.

Research to evaluate the understanding among the general populace of health-environment links, as well as the cultural and identity based roles of particular products in the diet, is in its infancy. As Macdiarmid and colleagues note, 'studies have modelled 'ideal' sustainable diets based on objective criteria for environmental and nutritional goals but as yet few have fully taken account of the social world of eating, with personal and cultural acceptability of dietary choices'.[193] The role of, for example meat, in the diet is likely to be highly culturally specific also, suggesting engagement on the issue needs nuance and sensitivity.

Openness to the idea of reducing meat consumption is limited. In the YouGov survey in the UK mentioned earlier, only one third of respondents said they would be willing to *consider* reducing their meat consumption,[192] while a quarter of the respondents had already cut back on meat consumption (mainly for health and economic reasons). In a focus group study with 87 participants from NE Scotland (one of which was vegetarian and three were ex-vegetarian), three dominant themes appeared: 1. Lack of awareness between meat consumption and climate change; 2. Perceptions that personal meat consumption plays a minimal role in the global context of climate change; 3. Resistance to the idea of reducing personal meat consumption. The latter theme was found to prevail across men and women, socio-economic group, and urban/rural location.[193]

In addition to the need to build the knowledge base, is an acceptance that for systemic change of the scale required, we need to focus beyond the individual and beyond traditional 'rational actor' models as the locus and means of action. This requires engagement with all stages of the supply chain and beyond that with the macroeconomic policies that form the institutional architecture within which companies operate and trade is shaped.

Garnett et al conducted a review of possible policies and actions to shift eating patterns towards more healthy and sustainable outcomes.[183] Some aspects of that review are very briefly summarised in Table 8.

xi UK, Sweden, France, Germany, Poland and Hungary.

xii Britain, Sweden, Canada, Australia, America, Japan, France, Hungary, Germany, Spain, South Africa, South Korea, Argentina, Mexico, Brazil, Russia, China, India.

Table 8 Shifting eating patterns towards more healthy and sustainable outcomes

1 Disincentivise or incentivise choices through fiscal measures. E.g. Taxes, subsidies or trading.
Efficacy in changing production and consumption of sustainable and healthy foods
Although there is an emerging body of evidence on use of health-related food taxes, there are currently no taxes or subsidies that target both environment and health. It is not clear how supply chains would react to the imposition of a tax and there is a lack of research into substitution behaviour, leakage and the rebound effect[xii] on other environment/ health outcomes. The cross-issue impacts of fiscal measures aimed at either environment or health need better researching but some environment-tax studies find the lower GHG impact of sugars can lead to perverse health outcomes.
2 Change the governance of production or consumption E.g. Macroeconomic policies or agreements, national public procurement, planning policies & other regulations
Efficacy in changing production and consumption of sustainable and healthy foods
Macro-economic policies (e.g. trade, liberalisation, foreign direct investment, national R&D strategies) are strongly implicated in the nutrition transition suggesting they are a powerful driver of change. Beyond agricultural subsidies there have been no attempts to date to change macroeconomic policy towards environment or health ends relating to food specifically. We need a much better understanding of what this macro-economic intervention might look like while appreciating that directly linking policies to better outcomes for health and environment may be difficult given the complex ways this scale of policy creates impact. Research exploring planning policies, consumption patterns and environment outcomes has not been done. But there are a number of studies showing how planning can influence more generally healthy and sustainable behaviours.
3 Encourage collaboration and shared agreements E.g. Voluntary industry agreements and certification schemes
Efficacy in changing production and consumption of sustainable and healthy foods
Reviews have found that, if properly implemented and monitored, businesses can aid in achieving policy aims through voluntary agreements. But because they tend to take the place of regulatory alternatives, it is difficult to ascertain their relative effectiveness or strength. Similarly determining whether voluntary agreement actions are additional to what might have happened anyway is difficult. Significant disincentives for non-participation and sanctions for non-compliance are found in some of the most effective voluntary agreements. Proposed actions need to be evidence-based, well defined, measurable and additional. A follow up to the current environmental voluntary agreement in the UK – Courtauld 2025 – seems likely to include some focus on healthy sustainable eating. Certification can shift markets, but evidence of measurable benefits on the environment is more mixed. Market for certified products is fairly weak, but can work with policy to raise standards – e.g. public procurement of good certification scheme products. Policies are needed to ensure certification works well – improve transparency, enable access and ensure robust monitoring. Policy also needs to work beyond certification in setting standards for levels of consumption.
4 Changing the context, defaults and norms of production and consumption. E.g. Changing choice architecture, nudge, store layouts, catering provision, reformulation etc.
Efficacy in changing production and consumption of sustainable and healthy foods
Interventions linked to 'nudge' approaches in supermarkets, schools and workplaces can be somewhat effective when a mixture is used, often with a fiscal component. When a mixture of more active interventions was compared with passive information provision, the former was found to be more effective. In general, nudge interventions have limited robust evidence though. More research is needed, especially on the longevity of effects. Nudge 'cannot be seen as a substitute for regulatory or fiscal interventions' (ditto certification).
5 Inform, educate, promote or empower through community initiatives, labelling and other means. E.g. Labelling, gardening or cooking projects, media or other campaigns, education programmes.
Efficacy in changing production and consumption of sustainable and healthy foods
This politically-acceptable approach has been the backbone of health promotion policy in recent years, but – as discussed elsewhere in this paper – has been of limited effectiveness. The impact of labelling is 'weakly positive', but is not always understood and is used more by more concerned people. However, it may 'soften up' the public to more 'interventionist approaches'. It may also promote a 'race to the top' by companies, especially with benchmarking by NGOs. On the environment, knowledge and desire for it is relatively low among consumers. Community initiatives – evidence for impact is weak, reflecting difficulty of and low levels of evaluation. May have constructive role to play in consumer engagement.

Source: Adapted from Garnett et al[183]

One recent US-UK initiative (launched mid 2016 by the World Resources Institute) is the Better Buying Lab, which is undertaking research in partnership with a range of large companies into how to shift people's purchasing habits towards healthier and sustainable diets (in its first phase this is framed as increasing the purchase of plant-based food). It will be interesting to see what this partnership achieves and finds.

Finally, it is worth noting that at the macro-policy level, the Paris Climate Change Agreement coupled with the Sustainable Development Goals provide an important pair of policy drivers for encouraging a more serious and integrated look at health-environment issues. While agriculture is only indirectly included in the Paris Climate Change Agreement, the huge contribution of agriculture to global GHG emissions and the implications of omitting this sector altogether from mitigation efforts for what other sectors need to achieve, suggests that where 'wins' are available they need to be taken. The range of the 17 SDGs linked to environment, diet, health and hunger (2: zero hunger; 3: good health and wellbeing; 6: clean water and sanitation; 11: sustainable cities and communities; 12: responsible consumption and production; 13: climate action; 14: life below water, 15: life on land) highlight that seeking to achieve these goals individually and without looking at interactions between them would be folly. The question now is, 'how do we move forward with doing this?'

3.4.5.1 Existing proposed policy solutions to health-environment issues: conclusions and key messages

- There are some pioneering examples of integrating health and environmental outcomes in food system interventions, such as around dietary guidelines and engaging public institutions. But examples are still few and far between.
- There have been some important advances around the Paris Climate Change Agreement and the Sustainable Development Goals which provide an opportunity to promote an integrated approach to health-environment interactions.
- There is currently too much focus on passive information based approaches to seeking to shift the health and environmental food consumption behaviours of people.
- A large range of interventions will be needed to shift diets towards healthier and more sustainable outcomes, including more active forms of intervention, and at a range of levels – people, institutions, regions, economies and global political-economic regimes. In particular, more research and action is needed regarding interventions at the macro-economic level.
- There is probably a significant bias towards insights in shifting diets in English speaking and western countries due to volume of research and access.

3.4.6 Recommendations for further action

This section has suggested that in the following areas further research is required:

- Stronger research engagement with the role of food production beyond the farm gate in helping to realise health-environment win-wins.
- A better understanding is required of the geographically specific nature of what a 'healthy and sustainable' diet looks like on a plate, and as a set of practices, and how this varies across place and culture.
- Research into the impact of integrating environmental factors into dietary guidelines is important. Existing healthy eating guidelines that integrate an environmental component (by Germany – published 2013, Brazil - 2014, Sweden - 2015 and Qatar - 2015) have been in place for the last 2-4 years. As the impacts of these guidelines 'play out' and begin to filter through the food system it will become increasingly viable to research and measure them.
- More research is needed to look at the socio-cultural elements of shifting diets towards lower meat, healthier and more sustainable diets.
- Further research should examine what macro-economic architectures that might support healthier and more sustainable food system outcomes look like.
- Cross health-environment interactions of policies aimed at each of these individually need to be better explored to ensure synergies can be maximised and negative trade-offs reduced or avoided.

More broadly, the following are recommended as possible courses of further action:

- Use recent policy developments around the Paris Climate Change Agreement and the SDGs, in addition to the pressing health challenges around food, to argue for an integrated health and environment approach to food systems (not just agriculture and not just the consumer).
- Promotion of health-environment win-wins in dietary guidelines beyond those countries currently doing this.
- Encouraging a move away from a dominant emphasis on action through individual choice in helping to realise health and environment outcomes around food and diet. A large range of interventions will be needed, at multiple levels, and examining what this suite of approaches looks like and how they achieve change will be important.
- Promote ambitious, staged, and robust school food and public institution food provision programmes, such as the Food For Life programme in the UK.
- Testing of promising approaches where evidence is scarce; using experimentation to build the evidence base.

References

1. Global Panel on Agriculture and Food Systems for Nutrition. *Food systems and diets: Facing the challenges of the 21st century.* (2016).
2. Food and Agriculture Organisation of the United Nations. Rome Declaration of Nutrition. *Second Int. Conf. Nutr.* 19–21 (2014). doi:10.1044/leader.PPL.19102014.18
3. FAO & WHO. Framework for action. Outcome document of the Second International Conference on Nutrition (2014).
4. FAO/WHO. *Proceedings of the Preparatory Technical Meeting for the Joint FAO/WHO Second International Conference on Nutrition.* (2013).
5. Fanzo, J., Downs, S., Marshall, Q., de Pee, S. & Bloem, M. in *Nutrition in Developing Countries* 753–770 (2017).
6. The Food Foundation. *Food environment policy index for England. Final version.* (2016).
7. FoodDrinkEurope. *DATA & TRENDS 2016.* (2016).
8. World Health Organization. *Global health risks and burden of disease attributable to selected major risks.* (2009).
9. Arnold, M. *et al.* Global burden of cancer attributable to high body-mass index in 2012: a population-based study. *Lancet Oncol.* 16, 36–46 (2015).
10. Forouzanfar, M. H. *et al.* Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 386, 2287–2323 (2015).
11. Malik, V. S., Willett, W. C. & Hu, F. B. Global obesity: trends, risk factors and policy implications. *Nat. Rev. Endocrinol.* 9, 13–27 (2013).
12. Popkin, B. M., Adair, L. S. & Ng, S. W. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr. Rev.* 70, 3–21 (2012).
13. World Health Organization. *Global action plan for the prevention and control of noncommunicable diseases 2013-2020.* (2013).
14. Darmon, N. & Drewnowski, A. Does social class predict diet quality? *Am. J. Clin. Nutr.* 87, 1107–1117 (2008).
15. Grant, W. *The Common Agricultural Policy. 1997* (Macmillan Press Ltd.).
16. Kay, A. *The reform of the Common Agricultural Policy: the case of the MacSharry reforms. CAB INTERNATIONAL* (CAB INTERNATIONAL, 1998).
17. Mann, H. C. *Diets for boys during the school years.* (HMSO, 1926).
18. Boyd Orr, J. *Food, Health and Income.* (Macmillan and Co Ltd, 1937).
19. Acheson, E. D. Tenth Boyd Orr memorial lecture. Food policy, nutrition and government. *Proc. Nutr. Soc.* 45, 131–138 (1986).
20. Bowers, J. K. British Agricultural Policy since the Second World Vol. *Agric. Hist. Rev.* 33, 66–76 (1985).
21. Moscow CPSU. Kruschev report of the First Secretary of the Central Committee of the Moscow, Russia: the proceedings of the XXii congress of the CPSU gospolitizdat. (1961).
22. Popkin, B. M. Agriculture Policies, Food and Health, Science and Society Series on Food and Science. *EMBO Rep.* 12, 0–11 (2011).
23. Weiss, G. S. Farewell document. Duping the Russians in Intelligence. *See* https://www.cia.gov/library/center-for-the-study-of-intelligence/csi-publications/csi-studies/studies/96unclass/farewell.htm
24. Norum, K. L., Johansson, L., Botten, G., Bjørbeboe, G.-E. A. & Oshaug, A. Nutrition and food policy in Norway effects on reduction in cardiovascular disease. *Nut Rev* 55, S32–S39 (1997).
25. Ritson, C. & Harvey, D. *The Common Agricultural Policy.* (CAB INTERNATIONAL, 1997).
26. Wilkinson G. *Agriculture and the Problem of Food Surpluses. Newsletter on the Common Agricultural Policy.* (European Community, 1980).
27. CIHEAM/FAO. *Mediterranean food consumption patterns: diet, environment, society, economy and health. A White Paper Priority 5 of Feeding Knowledge Programme, expo milan 2015.* (2015).
28. Petit, M. *et al. Agricultural Policy Formation in the European Community: the birth of milk quotas and CAP Reform.* (Elsevier, 2012).
29. Veerman, J. L., Barendregt, J. J. & Mackenbach, J. P. The European Common Agricultural Policy on fruits and vegetables: exploring potential health gain from reform. *Eur. J. Public Health* 16, 31–35 (2006).
30. European Commission. Agriculture 2015. (2016). Available at: http://ec.europa.eu/agriculture/cap-overview/2014_en.pdf.
31. Esposti, R. Regional growth and policies in the European Union: Does the Common Agricultural Policy have a counter-treatment effect?. *Am. J. Agric. Econ.* 89, 116–134 (2007).
32. CAP Reform-an explanation of the main elements. (2013). Available at: http://europa.eu/rapid/press-release_MEMO-13-621_en.htm
33. European Commision. A short history of milk quota.
34. Elinder, L. S. Obesity, hunger, and agriculture: the damaging role of subsidies. *BMJ* 331, 1333–1336 (2005).
35. Lang, T. & Rayner, G. Obesity: a growing issue for European policy? *J. Eur. Soc. Policy* 15, 301–327 (2005).
36. Hawkes, C. Promoting healthy diets and tackling obesity and diet-related chronic diseases: what are the agricultural policy levers? *Food Nutr. Bull.* 28, S312-22 (2007).
37. Consumers International. *The relationship between supermarkets and suppliers: What are the implications for consumers?* (Consumers International, 2012).
38. Birt C. *A CAP on health? The impact of the EU common agricultural policy on public health.* (2007).
39. Rao, M., Afshin, A., Singh, G. & Mozaffarian, D. Do healthier foods and diet patterns cost more than less healthy options? A systematic review and meta-analysis. *BMJ Open* 3, e004277 (2013).
40. DG Agriculture and Rural Development. *The 2007 reform.* (European Commission, 2007).
41. Pomerleau, J., Lock, K. & McKee, M. The burden of cardiovascular disease and cancer attributable to low fruit and vegetable intake in the European Union: differences between old and new Member States. *Public Health Nutr.* 9, 575–583 (2007).
42. Lloyd-Williams, F. *et al.* Estimating the cardiovascular mortality burden attributable to the European Common Agricultural Policy on dietary saturated fats. *Bull. World Health Organ.* 86, 535–541A (2008).
43. Schmidhuber, J. *The EU Diet-Evolution, Evaluation and Impacts of the CAP.* (FAO, 2003).
44. Matthews, A. *Food Prices, poverty and the CAP.* (Fabian Society, 2016).
45. Green, R. *et al.* The effect of rising food prices on food consumption: systematic review with meta-regression. *BMJ* 346, f3703 (2013).
46. European Commission. *Research for Agri-committee-The post-quotas EU sugar sector.* (European Commission, 2016).
47. OXFAM International. *A Europe for the Many, Not the Few.* (2015).
48. European Commission (DG Competition). *Working Paper: The interface between EU competition policy and the Common Agriculture Policy (CAP): Competition rules applicable to cooperation agreements between farmers in the dairy sector.* (2010).
49. UN. *Transforming our world: the 2030 agenda for sustainable development.* (2016).
50. Mytton, O. T., Clarke, D. & Rayner, M. Taxing unhealthy food and drinks to improve health. *BMJ* 344, e2931 (2012).
51. European Parliament. Second Pillar of the CAP. Factsheet on the European Union. (2016).
52. Walls, H. L., Cornelsen, L., Lock, K. & Smith, R. D. How much priority is given to nutrition and health in the Agricultural Policy? *Food Policy.* 59, 12–23 (2016).
53. Matthews, A. Farmers' share of food chain value added. (2015). Available at: http://capreform.eu/farmers-share-of-food-chain-value-added/
54. FoodDrinkEurope. *Promoting an EU industrial policy for food and drink: Competitiveness Report 2013-2014.* (2014).

55. Friel, S. *et al.* A new generation of trade policy: potential risks to diet-related health from the Trans Pacific Partnership agreement. *Global. Health* 9, (2013).
56. European Commission. *Report of the 13th round of negotiations for the Transatlantic Trade and Investment Partnership. New York, 25-29 April 2016.* (2016).
57. Monteiro, C. A., Moubarac, J. C., Cannon, G., Ng, S. W. & Popkin, B. Ultra-processed products are becoming dominant in the global food system. *Obes. Rev.* 14, 21–28 (2013).
58. Cairns, G., Angus, K., Hastings, G. & Caraher, M. Systematic reviews of the evidence on the nature, extent and effects of food marketing to children. A retrospective summary. *Appetite* 62, 209–215 (2013).
59. Baker, P., Friel, S., Schram, A. & Labonte, R. Trade and investment liberalization, food systems change and highly processed food consumption: a natural experiment contrasting the soft-drink markets of Peru and Bolivia. *Global. Health* 12, 1 (2016).
60. Schram, A. *et al.* The role of trade and investment liberalization in the sugar-sweetened carbonated beverages market: a natural experiment contrasting Vietnam and the Philippines. *Global. Health* 11, 1 (2015).
61. Baker, P., Kay, A. & Walls, H. Trade and investment liberalization and Asia's noncommunicable disease epidemic: a synthesis of data and existing literature. *Global. Health* 10, (2014).
62. Friel, S. *et al.* Shaping the discourse: What has the food industry been lobbying for in the Trans Pacific Partnership trade agreement and what are the implications for dietary health? *Crit. Public Health* 1–12 (2016). doi:10.1080/09581596.2016.1139689
63. Reardon, T. & Timmer, C. P. The Economics of the Food System Revolution. *Annu. Rev. Resour. Econ.* 4, 225–264 (2012).
64. Koivusalo, M., Schrecker, T. & Labonté, R. in *Globalization and Health: Pathways, Evidence and Policy* (ed. Ronald Labonte Corinne Packer, Vivien Runnels, T. S.) (Routledge, 2009).
65. Labonté, R., Schram, A. & Ruckert, A. The Trans-Pacific Partnership: Is It Everything We Feared for Health? *Int. J. Heal. Policy Manag.* 5, 487–496 (2016).
66. Weiss, M., Middleton, J. & Schrecker, T. Warning: TTIP could be hazardous to your health. *J. Public Health (Bangkok).* 37, 367–369 (2015).
67. Hirono, K. *et al.* Is health impact assessment useful in the context of trade negotiations? A case study of the Trans Pacific Partnership Agreement. *BMJ Open* 6, (2016).
68. Thow, A. M. *et al.* Will the next generation of preferential trade and investment agreements undermine implementation of the Global Action Plan for Prevention and Control of Noncommunicable Diseases? A prospective policy analysis of the Trans Pacific Partnership Agreement. *Health Policy (New. York).* 119, 88–96 (2015).
69. Kelsey, J. & Wallach, L. 'Investor state' disputes in trade pacts threaten fundamental principles of national judicial systems. (2012).
70. Thow, A. M. & McGrady, B. Protecting policy space for public health nutrition in an era of international investment agreements. *Bull. World Health Organ.* 92, 139–45 (2014).
71. Ruckert, A. *et al.* Policy coherence, health and the sustainable development goals: a health impact assessment of the Trans-Pacific Partnership. *Crit. Public Health* 1–11 (2016).
72. Castaldi, M. & Esposito, A. Phillip Morris loses tough-on-tobacco lawsuit in Uruguay; Fri Jul 8, 2016. *Reuters* (2016).
73. ICSID. *Philip Morris Brand Sàrl (Switzerland), Philip Morris Products S.A. (Switzerland) and Abal Hermanos S.A. (Uruguay) v. Oriental Republic of Uruguay (ICSID Case No. ARB/10/7).* (International Centre for Settlement of Investment Disputes, 2016).
74. McCabe Centre for Law and Cancer. *The Award on the merits in Philip Morris v Uruguay: implications for WHO FCTC implementation.* (2016).
75. Thow, A. M. *et al.* Regional trade and the nutrition transition: opportunities to strengthen NCD prevention policy in the Southern African Development Community. *Glob. Health Action* 8, (2015).
76. Linn, S. E. Food marketing to children in the context of a marketing maelstrom. *J. Public Health Policy* 25, 367–78 (2004).
77. Boyland, E. J. & Halford, J. C. G. Television advertising and branding. Effects on eating behaviour and food preferences in children. *Appetite* 62, 236–41 (2013).
78. Buijzen, M., Schuurman, J. & Bomhof, E. Associations between children's television advertising exposure and their food consumption patterns: a household diary-survey study. *Appetite* 50, 231–9
79. Kelly, J., Turner, J. J. & McKenna, K. What parents think: children and healthy eating. *Br. Food J.* 108, 413–423 (2006).
80. Buijzen, M. & Valkenburg, P. M. The effects of television advertising on materialism, parent–child conflict, and unhappiness: A review of research. *J. Appl. Dev. Psychol.* 24, 437–456 (2003).
81. Lobstein, T. *et al.* Child and adolescent obesity: part of a bigger picture. *Lancet* (2015).
82. Winpenny, E. M. *et al.* Exposure of children and adolescents to alcohol marketing on social media websites. *Alcohol Alcohol* 49, 154–9 (2014).
83. Nelson, M. C., Story, M., Larson, N. I., Neumark-Sztainer, D. & Lytle, L. A. Emerging Adulthood and College-aged Youth: An Overlooked Age for Weight-related Behavior Change. *Obesity* 16, 2205–2211 (2008).
84. Heath, R. Emotional Engagement: How Television Builds Big Brands At Low Attention. *J. Advert. Res.* 49, 62–73 (2009).
85. Ofcom. Children and Parents: media use and attitudes report. (2014).
86. World Cancer Research Fund/American Institute for Cancer Research. WCRF International Nourishing Framework. (2016). Available at: http://www.wcrf.org/int/policy/nourishing-framework.
87. Boyland, E. J., Kavanagh-Safran, M. & Halford, J. C. G. Exposure to 'healthy' fast food meal bundles in television advertisements promotes liking for fast food but not healthier choices in children. *Br. J. Nutr.* 1–7 (2015).
88. Clark, M. & Brownell, R. The inside track on the marketing strategies of Olympic food and soft drink sponsors, and the sponsorship deals behind them.
89. Temple, N. J., Alp, K., Temple, N. J. Marketing of Soft Drinks to Children and Adolescents: Why We Need Government Policies. *Nutr. Health* doi:10.1007/978-3-319-23672-8_18
90. Bhargava, M; Donthu, N. Sales Response to Outdoor Advertising. *J. Advert. Res.* 39, 7–7 (1999).
91. Jones, S. C., Gregory, P. & Kervin, L. Branded food references in children's magazines: 'advertisements' are the tip of the iceberg. *Pediatr. Obes.* 7, 220–9 (2012).
92. Glanz, K., Bader, M. D. M. & Iyer, S. Retail Grocery Store Marketing Strategies and Obesity: An Integrative Review. *Am. J. Prev. Med.* 42, 503–512 (2012).
93. Thornton, L. E. *et al.* The availability of snack food displays that may trigger impulse purchases in Melbourne supermarkets. *BMC Public Health* 12, 194 (2012).
94. WHO Regional Office for Europe. *Tackling food marketing to children in a digital world : trans-disciplinary perspectives.* (2016).
95. WHO. *Marketing of foods high in fat, salt and sugar to children: update 2012-2013.* (2012).
96. eMarketer. Digital Media Spending Thrives in Western Europe. www.emarketer.com (2011).
97. Fennah, A. European Online Advertising surpasses TV to record annual spend of €36.2bn | IAB Europe. www.iabeurope.eu (2016).
98. Coca-Cola sees success with brand marketing on Facebook.
99. Adweek. Mondelez, Parent of Oreos and Chips Ahoy, Will Buy Programmatic Video Through TubeMogul. Adweek.com (2014).
100. Kelly, B., Vandevijvere, S., Freeman, B. & Jenkin, G. New Media but Same Old Tricks: Food Marketing to Children in the Digital Age. *Curr. Obes. Rep.* (2015).
101. Mangold, W. G. & Faulds, D. J. Social media: The new hybrid element of the promotion mix. *Bus. Horiz.* 52, 357–365 (2009).
102. Sprott, D., Czellar, S. & Spangenberg, E. The Importance of a General Measure of Brand Engagement on Market Behavior: Development and Validation of a Scale. *J. Mark. Res.* 46, 92–104 (2009).

103. Livingstone, S., Haddon, L., Görzig, A. & Ólafsson, K. The perspective of European children Risks and safety on the internet. (2009).
104. Childwise. Childhood 2016. (2016).
105. Faber, R. J. Lee, M. Nan, X. Advertising and the Consumer Information Environment Online. *Am. Behav. Sci.* 48, 447–466 (2004).
106. Newsround report. Vloggers paid for promotions 'need clearer labelling'. *BBC news* (2014).
107. McCaskill, A. Recommendations From Friends Remain Most Credible Form of Advertising Among Consumers; Branded Websites Are the Second-Highest-Rated Form. (2015).
108. eMarketer. Do YouTubers Fuel Purchase Intent Among Teens? *emarketer* (2015).
109. Smit, E. G., Van Noort, G. & Voorveld, H. A. M. Understanding online behavioural advertising: User knowledge, privacy concerns and online coping behaviour in Europe. *Comput. Human Behav.* 32, 15–22 (2014).
110. Barnabè, D. *The Effect of Advertising and Marketing Practices on Child Obesity*. (2008).
111. Guittard, Christine; Sjölin, K. *Advertising and marketing practices on child obesity*. (2008).
112. G. Hastings, M. Stead, L. McDermott, A. Forsyth, A.M. MacKintosh, M. Rayner, et al. *Review of Research on the Effects of Food Promotion to Children. Report to the Food Standards Agency prepared by Centre for Social Marketing* (2003).
113. WHO. WHO European Charter on counteracting obesity. (2006).
114. European Network. European Network on reducing marketing pressure on children. (2011).
115. WHO. *Set of recommendations on the marketing of foods and non-alcoholic beverages to children.* (2010).
116. WHO. A framework for implementing the set of recommendations on the marketing of foods and non-alcohol beverages to children. 1–61 (2012).
117. Galbraith-Emami, S. & Lobstein, T. The impact of initiatives to limit the advertising of food and beverage products to children: a systematic review. *Obes. Rev.* 14, 960–974 (2013).
118. EU. EU Pledge. (2016). http://www.eu-pledge.eu/.
119. Huizinga, O. & Kruse, M. Food industry self-regulation scheme 'EU Pledge' cannot prevent the marketing of unhealthy foods to children. *Obes. Med.* 1, 24–28 (2016).
120. Jensen, J. D. & Ronit, K. The EU pledge for responsible marketing of food and beverages to children: implementation in food companies. *Eur. J. Clin. Nutr.* 69, 896–901 (2015).
121. Kelly, B. *et al.* A hierarchy of unhealthy food promotion effects: Identifying methodological approaches and knowledge gaps. *Am. J. Public Health* 105, e86–e95 (2015).
122. McClure, A. C. *et al.* Receptivity to television fast-food restaurant marketing and obesity among U.S. youth. *Am. J. Prev. Med.* 45, 560–8 (2013).
123. Gorn, G. J., Goldberg, M. E. Children's Responses to Repetitive Television Commercials on JSTOR. *J. Consum. Res.* 6, 421–424 (1980).
124. Jeffrey, D. B., McLellarn, R. W. & Fox, D. T. The development of children's eating habits: the role of television commercials. *Health Educ. Q.* 9, 174–89 (1982).
125. Boyland, E. J. *et al.* Advertising as a cue to consume: a systematic review and meta-analysis of the effects of acute exposure to unhealthy food and nonalcoholic beverage advertising on intake in children and adults. *Am. J. Clin. Nutr.* 103, 519–533 (2016).
126. Halford, J. C. G., Gillespie, J., Brown, V., Pontin, E. E. & Dovey, T. M. Effect of television advertisements for foods on food consumption in children. *Appetite* 42, 221–5 (2004).
127. Halford, J. C. G., Boyland, E. J., Hughes, G., Oliveira, L. P. & Dovey, T. M. Beyond-brand effect of television (TV) food advertisements/commercials on caloric intake and food choice of 5-7-year-old children. *Appetite* 49, 263–7 (2007).
128. Halford, J. C. *et al.* Beyond-brand effect of television food advertisements on food choice in children: the effects of weight status. *Public Health Nutr.* 11, 897–904 (2008).
129. Dovey, T. M., Taylor, L., Stow, R., Boyland, E. J. & Halford, J. C. G. Responsiveness to healthy television (TV) food advertisements/commercials is only evident in children under the age of seven with low food neophobia. *Appetite* 56, 440–6 (2011).
130. Anschutz, D. J., Engels, R. C. M. E. & Strien, T. van. Maternal encouragement to be thin moderates the effect of commercials on children's snack food intake. (2010).
131. Anschutz, D. J., Engels, R. C. M. E. & Van Strien, T. Side effects of television food commercials on concurrent nonadvertised sweet snack food intakes in young children. *Am. J. Clin. Nutr.* 89, 1328–33 (2009).
132. Harris, J. L., Bargh, J. A. & Brownell, K. D. Priming effects of television food advertising on eating behavior. *Heal. Psychol.* 28, 404–413 (2009).
133. Cohen, D. A. Obesity and the built environment: changes in environmental cues cause energy imbalances. *Int. J. Obes. (Lond).* 32 Suppl 7, S137-42 (2008).
134. Kemps, E., Tiggemann, M. & Hollitt, S. Exposure to television food advertising primes food-related cognitions and triggers motivation to eat. *Psychol. Health* 29, 1192–205 (2014).
135. Fallon, A. E., Rozin, P. & Pliner, P. The child's conception of food: the development of food rejections with special reference to disgust and contamination sensitivity. *Child Dev.* 55, 566–75 (1984).
136. Batada, A., Seitz, M. D., Wootan, M. G. & Story, M. Nine out of 10 food advertisements shown during Saturday morning children's television programming are for foods high in fat, sodium, or added sugars, or low in nutrients. *J. Am. Diet. Assoc.* 108, 673–8 (2008).
137. Robinson, T. N., Borzekowski, D. L. G., Matheson, D. M. & Kraemer, H. C. Effects of fast food branding on young children's taste preferences. *Arch. Pediatr. Adolesc. Med.* 161, 792–7 (2007).
138. Keller, K. L. *et al.* The impact of food branding on children's eating behavior and obesity. *Physiol. Behav.* 106, 379–86 (2012).
139. Mills, S. D. H., Tanner, L. M. & Adams, J. Systematic literature review of the effects of food and drink advertising on food and drink-related behaviour, attitudes and beliefs in adult populations. *Obes. Rev.* 14, 303–314 (2013).
140. Which? More supermarket promotions on less healthy food. Press release 04/08/2016. (2016).
141. Nakamura, R. *et al.* Price promotions on healthier compared with less healthy foods: A hierarchical regression analysis of the impact on sales and social patterning of responses to promotions in Great Britain. *Am. J. Clin. Nutr.* 101, 808–816 (2015).
142. Public Health England. Sugar Reduction : The evidence for action Annexe 4 : An analysis of the role of price promotions on the household purchases of food and drinks high in sugar (2015).
143. Roberto, C. A. *et al.* Obesity 1 Patchy progress on obesity prevention : emerging examples, entrenched barriers, and new thinking. *Lancet* 6736, (2015).
144. Ericksen, P. J. Conceptualizing food systems for global environmental change research. *Glob. Environ. Chang.* 18, 234–245 (2008).
145. Steffen, W. *et al.* Planetary boundaries: guiding human development on a changing planet. *Science (80-.).* 347, 1259855 (2015).
146. Potts, S. G. *et al.* Global pollinator declines: trends, impacts and drivers. *Trends Ecol. Evol.* 25, 345–353 (2010).
147. Vermeulen, S. J., Campbell, B. M. & Ingram, J. S. I. Climate Change and Food Systems. *Annu Rev Env. Resour* 195–222 (2012).
148. Intergovernmental Panel on Climate Change. *Climate Change 2014: Mitigation of Climate Change. Contribution of Working Group III to the Fifth Assessment. Report of the Intergovernmental Panel on Climate Change.* 2014, (Cambridge University Press, 2014).
149. De Schutter, L. & Lutter, S. *The true cost of consumption: the EU's land footprint.* (Institute for Ecological Economics, Vienna University of Economics and Business and Friends of the Earth International, Belgium., 2016).

150. Millennium Ecosystem Assessment. *Ecosystems and human well-being: Synthesis.* (2005).
151. DeFries, R., Rudel, T., Uriarte, M. & Hansen, M. Deforestation driven by urban population growth and agricultural trade in the twenty-first century. *Nat. Geosci.* (2010).
152. Alexandratos, N. & Bruinsma, J. *World Agriculture towards 2030/2050. The 2012 Revision. ESA Working Paper No. 12-03.* (FAO, 2012).
153. Ray, D. K., Mueller, N. D., West, P. C. & Foley, J. A. Yield Trends Are Insufficient to Double Global Crop Production by 2050. *PLoS One* 8, e66428 (2013).
154. Delzeit, R., Zabel, F., Meyer, C. & Václavík, T. Addressing future trade-offs between biodiversity and cropland expansion to improve food security. *Reg. Environ. Chang.* 0–1 (2016).
155. Garnett, T. *Cooking up a Storm: Food, greenhouse gas emissions and our changing climate.* (2008).
156. Tilman, D. & Clark, M. Global diets link environmental sustainability and human health. *Nature* 515, 518–522 (2014).
157. Smith, J. *et al.* Beyond milk, meat, and eggs: role of livestock in food and nutrition security. *Anim. Front.* 3, 6–13 (2013).
158. Eisler, M. C. *et al.* Agriculture: Steps to sustainable livestock. *Nature* 507, 32–34 (2014).
159. Health Council of the Netherlands. *Guidelines for a healthy diet: the ecological perspective.* (Health Council of the Netherlands, 2011).
160. Food and Agriculture Organization. *The state of world fisheries and aquaculture. Contributing to food security and nutrition for all.* (FAO, 2016).
161. Westhoek, H. *et al. The Protein Puzzle. The consumption and production of meat, dairy and fish in the European Union.* (PBL Netherlands Environmental Assessment Agency, 2011).
162. Bostock, J. *et al.* Aquaculture: global status and trends. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 365, 2897–2912 (2010).
163. Mekonnen, M. M. & Hoekstra, A. Y. A global assessment of the water footprint of farm animal products. *Ecosystems* 15, 401–415 (2012).
164. Food and Agriculture Organization. *Coping with water scarcity. An action framework for agriculture and food security.* (FAO, 2012).
165. Hess, T., Andersson, U., Mena, C. & Williams, A. The impact of healthier dietary scenarios on the global blue water scarcity footprint of food consumption in the UK. *Food Policy* 50, 1–10 (2015).
166. Meier, T. & Christen, O. Environmental impacts of dietary recommendations and dietary styles: Germany as an example. *Environ. Sci. Technol.* 47, 877–888 (2013).
167. Vanham, D., Mekonnen, M. M. & Hoekstra, A. Y. The water footprint of the EU for different diets. *Ecol. Indic.* 1–8 (2013).
168. Food and Agriculture Organisation. *Global food losses and food waste. Extent, causes and prevention.* (FAO, 2011).
169. Hawkes, C. & Popkin, B. M. Can the sustainable development goals reduce the burden of nutrition-related non-communicable diseases without truly addressing major food system reforms? *BMC Med.* 13, 143 (2015).
170. Bennetzen, E. H., Smith, P. & Porter, J. R. Decoupling of greenhouse gas emissions from global agricultural production: 1970-2050. *Glob. Chang. Biol.* 22, 763–781 (2016).
171. Rogelj, J. *et al.* Paris Agreement climate proposals need a boost to keep warming well below 2 °C. *Nature* 534, 631–639 (2016).
172. Nelson, G. C. *et al. Food security, farming, and climate change to 2050: challenges to 2050 and beyond.* (2010).
173. Myers, S. S. *et al.* Increasing CO2 threatens human nutrition. *Nature* 510, 139–142 (2014).
174. Brown, M. E. *et al. Climate Change, Global Food Security, and the U.S. Food System.* (USDA, 2015).
175. Myers, S. S. *et al.* Climate Change and Global Food Systems: Potential Impacts on Food Security and Undernutrition. *Annu. Rev. Public Health* 38, 259–277 (2017).
176. Challinor, A. J. *et al.* A meta-analysis of crop yield under climate change and adaptation. *Nat. Clim. Chang.* 4, 287–291 (2014).
177. Cheung, W. W. *et al.* Structural uncertainty in projecting global fisheries catches under climate change. *Ecol. Model. Vol* 325, 57–66 (2016).
178. Nelson, G. C. *et al.* Climate change effects on agriculture: economic responses to biophysical shocks. *Proc. Natl. Acad. Sci. U. S. A.* 111, 3274–3279 (2014).
179. Springmann, M. *et al.* Global and regional health effects of future food production under climate change: a modelling study. *Lancet* 387, 1937–1946 (2016).
180. Hallström, E., Carlsson-Kanyama, A. & Börjesson, P. Environmental impact of dietary change: a systematic review. *J. Clean. Prod.* 91, 1–11 (2015).
181. Tom, M. S., Fischbeck, P. S. & Hendricksen, C. T. Energy use, blue water footprint, and greenhouse gas emissions for current food consumption patterns and dietary recommendations in the US. *Environ. Syst. Decis.* 36, 92–103 (2016).
182. Vieux, F., Darmon, N., Touazi, D. & Soler, L. G. Greenhouse gas emissions of self-selected individual diets in France: Changing the diet structure or consuming less? *Ecol. Econ.* 75, 91–101 (2012).
183. Garnett, T., Mathewson, S., Angelides, P. & Borthwick, F. *Policies and actions to shift eating patterns: What works? A review of the evidence of the effectiveness of interventions aimed at shifting diets in more sustainable and healthy directions.* (Food Climate Research Network, 2015).
184. Swedish National Food Agency. *Find your way to eat greener, not too much and be active.* (2015).
185. Health Council of the Netherlands. *Dutch dietary guidelines 2015.* (2015).
186. DEFRA. *Sustainable Consumption Report. Follow-up to the Green Food Project.* (Department for Environment Food and Rural Affairs, 2013).
187. Murphy, M. Dietary guidelines: scientific substantiation and public health impact. *Nutr. Bull.* 39, 290–304 (2014).
188. Rooney, C. *et al.* How much is '5-a-day'?: consumer knowledge of fruit and vegetable portion sizes. *Proceedings of the Nutrition Society* 72, (2013).
189. Bates, B., Lennox, A., Prentice, A., Bates, C. & Swan, G. *National Diet and Nutrition Survey Headline results from Years 1, 2 and 3 (combined) of the Rolling Programme (2008/2009 – 2010/11). A survey carried out on behalf of the Department of Health and the Food Standards Agency.* (2012).
190. Grunert, K. G. *et al.* Socio-demographic and attitudinal determinants of nutrition knowledge of food shoppers in six European countries. *Food Qual. Prefer.* 26, 166–177 (2012).
191. National Geographic. *Greendex Summary of food results and analysis of behaviour change.* (2014).
192. Dibb, S. & Fitzpatrick, I. *Let's talk about meat: changing dietary behaviour for the 21st century.* (2014).
193. Macdiarmid, J. I., Douglas, F. & Campbell, J. Eating like there's no tomorrow: Public awareness of the environmental impact of food and reluctance to eat less meat as part of a sustainable diet. *Appetite* 96, 487–493 (2016).

4 Effective policies for promoting healthy dietary patterns

Key points

- Significant global commitments on healthy diet and nutrition in recent years reflect greater international awareness of the importance of tackling non-communicable diseases (NCDs) and of tackling malnutrition in all its forms (including obesity, overweight and diet-related NCDs).
- Despite these important developments, progress on policy to improve European diets has been patchy and inadequate over the last six years.
- At the European Union (EU) level, progress on diet-related policy matters has been much slower than hoped for. There has been a disappointing lack of progress on nutrient profiles (for regulating use of claims) and marketing of foods high in fats, sugars or salt (HFSS) to children. There have been some promising initiatives in relation to trans fatty acids, reformulation and healthy procurement of food served in schools.
- Some European countries have implemented their own initiatives, specifically in relation to food taxes, reformulation, trans fats, labelling, food in schools and marketing to children. Much more widespread implementation of concrete government-led action in these areas is needed.
- EHN calls for rapid and full implementation of a comprehensive package of recommendations – comprising three overarching recommendations and three clusters of specific recommendations – in order to realise the vision of every European being able to live free from avoidable diet-related cardiovascular disease (CVD).
- Three **overarching recommendations** are needed to underpin the specific recommendations:
    - Implement policies to tackle health inequalities in Europe
    - Ensure that robust mechanisms for nutrition governance are in place and fit-for-purpose
    - Develop an integrated health and environment approach to food systems and promote health-environment win-wins in food-based dietary guidelines
- In relation to the **food supply**, EHN recommendations are to:
    - Establish a global food convention
    - Reform agricultural and food policy to align with public health priorities
    - Ensure trade and investment policies protect and promote public health
- On the **food demand side**, EHN recommendations are to:
    - Use taxes and/or subsidies
    - Implement regulatory controls on marketing of unhealthy foods
    - Adoption of nutrient profiles in the context of the EU regulation of health and nutrition claims; and of mandatory simplified front-of-pack nutrition labelling, and menu labelling.
- In relation to **food composition,** EHN recommends**:**
    - Setting legal limits for levels of industrially-produced trans fats
    - Establishing nutrition standards for food in schools, hospitals and other public institutions
    - Implementing wide-reaching ambitious food reformulation programmes

Cardiovascular disease (CVD) is to a large extent preventable and effective prevention policies have played a major role in the considerable progress in reducing the burden of CVD in recent decades. Nonetheless, the persistent massive burden of CVD requires further, urgent action and a population-wide approach to prevention holds most promise. Given the gaps between the population goals set out in Chapter 2 and the current dietary patterns of Europeans, a major focus for all countries and their governments in Europe should be the promotion of a healthy diet by new strategic measures which alter food systems and do not just rely on consumer choice and education.

EHN has long advocated for policy action to improve European diets for cardiovascular health. The interventions proposed in EHN's 2011, *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe*, are revisited in this chapter, with a review of progress in uptake and implementation of these policies, along with other global, regional and national developments since the publication of EHN's 2011 paper.

This chapter then goes on to develop a new set of recommendations for action. In order to identify effective policies and interventions for action, it is necessary first to consider what we mean by effective policies – in other words how do we define what works, what are the different types of evidence to consider and how should such evidence be assessed (See Annex 3). The next step is to identify potential interventions and to assess these according to the criteria identified and to select priority actions in order to identify a core set of key recommendations. (Chapter 4.2)

4.1 The policy landscape

EHN's paper, *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe*, presented a raft of different areas for policy action (see box below) and made a series of detailed recommendations for different actors in each policy area.[i]

Policy areas for action

- Reformulation of food products to reduce the salt, saturated fat, and added sugar content of foods and portion size
- Legislation to eliminate industrially produced trans fatty acids
- 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
- Promotion of healthy options
- Effective rules on nutrition and health claims
- Promotion of breastfeeding and ensuring appropriate marketing of breastmilk substitutes
- Economic tools (taxes and subsidies) and pricing strategies to make healthier foods more affordable and appealing, and to make less healthy foods more expensive
- 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
- Encouraging and facilitating healthy eating and active living in schools and pre-school facilities
- Measures to enable people to make healthier choices when they eat out
- Actions in the workplace to improve diet and physical activity
- Creation of environments that promote active living
- Health service involvement in promoting healthy lifestyles

In the six years since 2011, progress in these policy areas remains patchy and inadequate. There have, however, been some developments – including some far-reaching commitments – in some of these policy areas, at the global and European levels. In addition, a few governments have taken bold steps at the national level. Nonetheless, much more concrete action is needed.

4.1.1 Global developments

There have been a number of significant developments on nutrition and physical activity on the global stage in recent years, reflecting greater international awareness of the importance of tackling non-communicable diseases (NCDs) and of tackling malnutrition in all its forms (including obesity, overweight and diet-related NCDs).

WHO Global action plan for the prevention and control of noncommunicable diseases

In September 2011, just before the last EHN policy paper was published, the United Nations General Assembly issued a ground-breaking *Political Declaration on the Prevention and Control of NCDs* endorsed by all governments, including those of the European region.[1] This Declaration has been followed by a WHO Global NCD Action Plan for the period 2013-2020[2] which, crucially, sets out a series of nine voluntary global targets to be achieved by 2025, including a number that relate specifically to nutrition, physical activity or related risk factors (see Figure 39). Progress towards these targets will be measured by 25 indicators that have been defined as part of the comprehensive global monitoring framework for NCDs.[3] Countries are encouraged to build on the global targets and set their own national targets, taking into account their specific situation. A Global Coordination Mechanism has been established to facilitate and enhance the coordination of activities in line with global NCD action plan.[ii]

Many remaining barriers to further progress, including the lack of national capacity in some countries, were recognised at a further stand-alone meeting of the UN General Assembly which was held in July 2014 to take stock of progress on NCDs since the 2011 political declaration. This meeting also recognised progress achieved at the national level, including the existence of an operational national NCD policy with a budget for implementation in half of all countries.[iii]

These developments represent high-level political commitment to tackle CVD and other NCDs. Crucially, the political declaration in 2011 shows that governments are responsible for the prevention and control of chronic diseases, which have often considered in the past to be matters for individual responsibility. Progress towards the targets was reported in the 2014 global NCD status report.[4]

In May 2017 the World Health Assembly endorsed an updated Appendix 3 to the global action plan.[5] This new Appendix 3 – effectively the 'best buys' for NCD prevention – includes several more options for unhealthy diet than the previous version. The policy options include restricting marketing of unhealthy foods to children, reformulating

i The recommendations from 2011 are set out in the summary version of the 2011 paper, which can be downloaded from www.ehnheart.org

ii See http://www.who.int/global-coordination-mechanism/en/

iii Resolution A/68/L53. Available from: http://www.un.org/en/ga/search/view_doc.asp?symbol=A/RES/68/300.

Figure 39 Set of nine voluntary global NCD targets for 2025 Source: WHO

processed foods, taxing sugary drinks and subsidising fruit and vegetables, legislating to ban the use of industrial trans fats, improving food in public institutions, implementing front-of-pack labelling, promoting breastfeeding, providing nutrition education and counselling and implementing mass media campaigns.

A comprehensive review of progress achieved in the implementation of these targets will take place at a third High-Level Meeting of the UN General Assembly in 2018.

Rome Declaration on Nutrition

In November 2014, at the Second International Conference on Nutrition (ICN2), world leaders made important commitments to prevent all forms of malnutrition worldwide and to reduce the burden of diet-related NCDs in all age groups.[6]

Importantly, the *Rome Declaration on Nutrition*, which emerged from the Conference, includes obesity and overweight, as well as forms of undernutrition.[7] As mentioned in Chapter 3, the ICN2 also adopted a *Framework for Action* that recommends a set of 60 policy options and strategies – many of these are highly relevant to promoting cardiovascular health in Europe.[8] The Rome Declaration and the Framework for Action have been endorsed by the UN General Assembly, WHO and FAO. The UN has since declared a Decade of Action on Nutrition (2016-2025). For the purpose of accountability, the Framework for Action adopts existing **global nutrition targets** to be achieved by 2025 for improving maternal, infant and young child nutrition[iv] and for NCD risk factor reduction.[v]

It is crucial that countries act on their commitments by systematically implementing the strategies of the Framework for Action. At the 68th World Health Assembly in 2015 – which endorsed the Rome Declaration and the Framework for Action – EHN, as part of a wider coalition of NGOs, called on member states to consider developing a Framework Convention to help realise the ICN2 commitments.[9] The World Cancer Research Fund has translated the Framework for Action recommendations into financial and political commitments that are SMART (Specific, Measurable, Achievable, Relevant, and Time-bound) [vi] and WHO has set out 'double duty' interventions that have the potential to address both undernutrition and overweight and obesity.[10]

Sustainable Development Goals

In New York in September 2015, countries officially adopted a new agenda for development that seeks to balance

iv Namely: (1) 40% reduction of the global number of children under five who are stunted; (2) 50% reduction of anaemia in women of reproductive age; (3) 30% reduction of low birth weight; (4) no increase in childhood overweight; (5) increase exclusive breastfeeding rates in the first six months up to at least 50%; and (6) reduce and maintain childhood wasting to less than 5%.

v Namely: (1) to reduce salt intake by 30%; (2) to halt the increase in obesity prevalence in adolescents and adults.

vi See http://www.wcrf.org/int/policy/our-policy-work/ambitious-smart-commitments-address-ncds-overweight-and-obesity

the economic, social and environmental dimensions of sustainable development.[vii] The agenda – which follows on from the Millennium Development Goals – includes 17 globally-agreed Sustainable Development Goals (SDGs), accompanied by 169 specific targets, to stimulate action over the coming 15 years.[11]

Goal 2, on zero hunger, contains reference to improved nutrition and contains a target to end all forms of malnutrition by 2030. Goal 3, to ensure healthy lives and promote well-being for all at all ages, includes a specific target to reduce by one third premature mortality from NCDs by 2030. The SDGs also encompass the global nutrition targets adopted by the World Health Assembly in 2012.

This inclusion, for the first time, of tackling NCDs as a priority for global development reflects the seriousness of the NCD challenge for richer and poorer countries alike. It is also a measure of long overdue progress in raising the profile of NCD prevention on the global agenda since the drafting of the Millennium Development Goals (MDGs), when NCDs were woefully omitted despite the enormous global burden of death and disability they already posed when the MDGs were being formulated.

Commission on Ending Childhood Obesity

In early 2016, WHO's Commission on Ending Childhood Obesity made a series of recommendations to respond to the global crisis in childhood overweight.[13] These include several clear messages relevant to EHN's policy recommendations on, for example, measures to reduce the impact of marketing of unhealthy foods and beverages to children, front-of-pack labelling, standards for healthy foods in schools, promoting breastfeeding, regulating marketing of breastmilk substitutes and taxing sugar-sweetened beverages.

WHO report on fiscal policies for diet and the prevention of noncommunicable diseases (2016)

To address the increasing number of requests from countries for guidance on how to design fiscal policies on diet, WHO convened a technical meeting of global experts in fiscal policies on 5–6 May 2015 in Geneva. The report on *Fiscal policies for diet and the prevention of noncommunicable diseases* reflects the outcome of this meeting.[14]

The report concludes that there is reasonable and increasing evidence that appropriately designed taxes on sugar-sweetened beverages would result in proportional reductions in consumption, especially if aimed at raising the retail price by 20% or more. There is similar strong evidence that subsidies for fresh fruits and vegetables that reduce prices by 10–30% are effective in increasing fruit and vegetable consumption.

4.1.2 European developments

There have also been some important developments at the European level, both in the WHO European region, and in the European Union.

4.1.2.1 WHO European region

There have been a number of relevant and significant developments since 2011, under the leadership of WHO's Regional Office for Europe across the wider European region.

Health 2020 – A European policy framework and strategy for the 21st century, 2013, WHO.[viii] The new health policy framework for the European region, *Health 2020*, adopted at the Regional Committee in September 2012, focuses on improving health for all and reducing health inequalities, through improved leadership and governance for health. Implementing Health 2020 in countries is now the fundamental top-priority challenge for the European region.

Vienna Declaration on Nutrition and Noncommunicable Diseases in the Context of Health 2020 - (July 2013).[ix] At the WHO European Ministerial Conference on Nutrition and Noncommunicable Diseases in the Context of Health 2020, held in Vienna, Austria on 4-5 July 2013, European WHO member states renewed their commitment to take action on obesity and prioritise work on healthy diets for children.

European Food and Nutrition Action Plan 2015–2020.[x] In September 2014, the WHO European Region Health Ministers, at their annual meeting (Regional Committee), formally adopted the third regional food and nutrition action plan. The plan, calls for action through a whole-of-government, health-in-all-policies approach, and its priority actions will contribute to improving food system governance and the overall quality of the European population's diet and therefore nutritional status

Action Plan for Implementation of the European Strategy for the Prevention and Control of Noncommunicable Diseases 2012-2016.[xi] This action plan was adopted by the WHO Regional Committee for Europe in Baku in 2011. Three of its five priority areas for intervention relate very specifically to dietary measures:

- Promoting healthy consumption via fiscal and marketing policies;
- Replacement of trans fats in food with polyunsaturated fats;
- Salt reduction in foods;
- Cardio-metabolic risk assessment and management;
- Early detection of cancer.

vii http://www.un.org/ga/search/view_doc.asp?symbol=A/69/L.85&ampampLang=E.
viii http://www.euro.who.int/__data/assets/pdf_file/0011/199532/Health2020-Long.pdf?ua=1
ix http://www.euro.who.int/en/media-centre/events/events/2013/07/vienna-conference-on-nutrition-and-noncommunicable-diseases/documentation/vienna-declaration-on-nutrition-and-noncommunicable-diseases-in-the-context-of-health-2020
x http://www.euro.who.int/__data/assets/pdf_file/0008/253727/64wd14e_FoodNutAP_140426.pdf
xi http://www.euro.who.int/__data/assets/pdf_file/0019/170155/e96638.pdf?ua=1

While these first three clear diet-related priorities were most welcome, specific measures to address intakes of total or saturated fats and/or sugars were missing.

WHO Regional Office for Europe's Nutrient Profile Model.[xii] In order to support European countries in their efforts to restrict marketing of foods high in fat, sugars or salt (HFSS) to children, the WHO Regional Office for Europe launched a nutrient profile model in early 2015. This tool is designed to help countries identify those foods for which marketing should be restricted. Countries can adapt the model to their specific context and use it to define foods not to be marketed to children and/or to monitor the extent and nature of food marketing

Action Plan for the Prevention and Control of Noncommunicable Diseases in the WHO European Region.[xiii] This new WHO Action plan, adopted in August 2016, continues and updates the *Action Plan for implementation of the European Strategy for the Prevention and Control of Noncommunicable Diseases 2012–2016* (see above). The action plan focuses on priority action areas and interventions for the next 10 years (2016–2025) in order to achieve regional and global targets to reduce premature mortality, reduce the disease burden, improve the quality of life and make healthy life expectancy more equitable. The first three of its five priority interventions at a population level are related to diet:

- Healthy consumption via fiscal and marketing policies: tobacco, alcohol, food
- Product reformulation and improvement: salt, fats and sugars
- Salt reduction
- Promoting active living and mobility
- Promoting clean air

Monitoring Food and Beverage Marketing to Children via Television and the Internet – a Proposed Tool for the WHO European Region. In 2016, the WHO Regional Office for Europe proposed a protocol to provide the basis for monitoring the extent and nature of children's exposure to marketing for HFSS foods via television and the internet. This protocol will enable countries to obtain data on both exposure and the power of marketing to children.

4.1.2.2 Developments in the European Union

Progress at the EU-level on diet-related policy matters has been much slower than hoped for since EHN's 2011 report. Nonetheless there have been some developments, which are summarised below.

4.1.2.2.1 Regulatory Developments in the EU

4.1.2.2.1.1 Health and Nutrition Claims Regulation

Nutrition and health claims are regulated by the *EU Regulation on Nutrition and Health Claims*[xiv] adopted in 2006. The Regulation, which became applicable from 1 July 2007, aims to ensure that any claim made on a food's labelling, presentation or advertising in the EU is clear, accurate and based on scientific evidence. Food bearing claims that could mislead consumers are prohibited on the EU market.

In this Regulation the Commission was asked to develop nutrition profiles by 2009, and in its 2011 Paper EHN urged progress in this area. The application of nutrient profiles is important to ensure that foods that are, in general terms, less healthy are not permitted to mislead consumers by carrying health or nutrition claims.

The Commission has still not developed nutrition profiles and – in its latest REFIT programme – has questioned the need to do so in its EU Roadmap to review the work under the claims regulation.[xv] In December 2016, a consultant was mandated by DG Health and Food Safety of the European Commission to carry out a study to support the evaluation of two elements of this Regulation. One of the elements that the study will evaluate is whether nutrient profiles are fit to determine whether products high in certain nutrients (in particular, salt, sugar and fat) can bear claims. The report is expected by the end of 2017. EHN continues to argue vigorously that setting nutrient profiles is an essential element of the regulation[xvi] and in 2015 published a paper which further sets out the role of nutrient profiles in this context[xvii] and highlighted examples of food products with nutrition claims, but which do not have a healthy nutrition profile.[xviii] In May 2017, EHN sent a joint letter[xix] – along with two health and consumer organisations and five major food companies – to the Commission calling for urgent adoption of nutrient profiles.

4.1.2.2.1.2 Food Information to Consumers Regulation

The *Food Information to Consumers Regulation,*[xx] adopted in 2011, came into force in 2014, requiring mandatory nutrition declarations for energy value, fats, saturated fats, monounsaturated fats, polyunsaturated fats, starch, fibre, carbohydrates, sugars, protein, salt, vitamin and minerals.

Article 35 of this Regulation, allows for 'additional forms of expression and presentation'. It states that 'the energy value and the amount of nutrients referred to in Article 30(1) to (5) may be given by other forms of expression and/or presented using graphical forms or symbols in addition to words or

xii http://www.euro.who.int/__data/assets/pdf_file/0005/270716/Nutrient-Profile-Model_Version-for-Web.pdf?ua=1
xiii http://www.euro.who.int/__data/assets/pdf_file/0011/315398/66wd11e_NCDActionPlan_160522.pdf
xiv http://ec.europa.eu/food/safety/labelling_nutrition/claims/index_en.htm
xv http://ec.europa.eu/smart-regulation/roadmaps/docs/2015_sante_595_evaluation_health_claims_en.pdf
xvi http://www.ehnheart.org/publications/responses-to-consultations.html
xvii European Heart Network. Nutrient profiles and nutrition and health claims – a European Heart Network paper. December 2015. Available from: http://www.ehnheart.org/component/downloads/downloads/2153.html
xviii http://www.ehnheart.org/publications/position-papers/publication/949-good-for-you.html
xix http://www.ehnheart.org/media/news/1310-call-for-urgent-adoption-of-nutrient-profiles.html
xx Regulation (EU) No 1169/2011 of the European Parliament and of the Council of 25 October 2011 on Food Information to consumers

numbers'. Specifics which need to be respected in this case are mentioned in the regulation but it allows member states to recommend to food business operators the use of one or more additional forms of expression. This article is the basis for the UK government to propose its voluntary traffic light labelling scheme in 2013.

4.1.2.2.1.3 Audiovisual Media Services Directive

EHN has been a vigorous advocate of measures to protect children from advertising for HFSS foods and beverages.

In September 2015, EHN, together with Liverpool University, published a paper on marketing of HFSS food to children.[xxi] The paper calls for the Commission to use its regulatory power to limit the exposure of children to HFSS food marketing, rather than relying on voluntary, non-enforceable codes of conduct. It also calls on the EU to implement a comprehensive prohibition on all forms of HFSS food marketing to children.

A new legislative proposal to amend the *EU Audiovisual Media Services Directivexxii* – which sets the framework for television marketing and advertising to children – was adopted by the Commission in May 2016.[xxiii] EHN has repeated its call, from the 2011 paper, for a watershed for all audiovisual commercial communications for HFSS food on television. Based on data from a recent report on peak times of television watching by children[xxiv], EHN consider it wise to extent the period, during which audiovisual commercial communications for HFSS foods cannot be shown between 6 am and 11 pm.

4.1.2.2.1.4 Trans fatty acids

In December 2015, the Commission published a long awaited report on trans fatty acids (TFAs) in Europe,[15] as requested in the *Food Information to Consumers Regulation*.[xxv] The report summarises a preliminary analysis of the potential effectiveness of the measures that could be adopted at EU level. It concludes that 'a legal limit for industrial TFA content would be the most effective measure in terms of public health, consumer protection and compatibility with the internal market.' This is an important statement that should be translated, without delay, into concrete action.

EHN has long advocated for an EU-wide legislative solution to trans fats, with a mandatory upper limit for industrially-produced trans fats (IPTFAs), thereby harmonising standards across the Union and ensuring that foods with harmful levels of IPTFAs are not placed on the market.[1617] EHN welcomes the Commission's paper, but urges speedy action and is concerned about the need to undertake a fully-fledged impact assessment that will, inevitably, lead to delay.[xxvi] In October 2016 the European Parliament adopted a *Resolution on Trans Fatty Acids* which calls upon the European Commission to establish as soon as possible an EU legal limit on industrial TFAs.[xxvii] Also in October 2016, the European Commission launched its inception impact assessment (IIA) on limiting industrial trans fats in products in the EU The impact assessment process is continuing in 2017, after which it is expected the Commission will come forward with a legislative proposal.

4.1.2.2.2 Council Conclusions

4.1.2.2.2.1 Council Conclusions on Nutrition and Physical Activity

Council Conclusions on Nutrition and Physical Activity[xxviii] were adopted in June 2014. In these Council conclusions:

- the Commission and member states were invited to promote action to reduce the exposure of children, to advertising, marketing and promotion of foods high in saturated fats, trans fatty acids, added sugars or salt, and
- the Commission was invited to establish nutrient profiles (as set out in the Claims Regulation).

A *Joint Action on Nutrition and Physical Activity* (JANPA)[xxix,xxx] involving 25 member states, was launched in September 2015. The main objective of JANPA is to stop the rise of overweight and obesity in children and adolescents by 2020, focusing on specific outcomes that strongly contribute to the nutritional and physical activity policies dedicated to childhood.

4.1.2.2.2.2 Council Conclusions on Food Product Improvement

The Netherlands' Presidency of the EU Council in 2016 included reformulation as one of its priorities, resulting in publication of *Council Conclusions on food product improvement* in June 2016.[xxxi] Food production and food marketing have become an international issue within the EU internal market – because separate national initiatives do not facilitate a level playing field for food companies. The Dutch Health Minister, therefore, prioritised this focus on reformulation to encourage handling of reformulation at

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xxi http://www.ehnheart.org/publications/position-papers.html
xxii http://eur-lex.europa.eu/legal-content/EN/ALL/?uri=CELEX:32010L0013
xxiii https://ec.europa.eu/digital-single-market/en/audiovisual-media-services-directive-avmsd
xxiv http://www.ecorys.com/news/european-commission-publishes-ecorys-report-exposure-minors-alcohol-advertising-0
xxv http://eur-lex.europa.eu/legal-content/en/ALL/?uri=CELEX:32011R1169
xxvi European Heart Network. European Commission Report on trans fats in the EU. Press Release. 3 December 2015. http://www.ehnheart.org/media/news/1123-european-commission-report-on-trans-fats.html
xxvii http://www.europarl.europa.eu/sides/getDoc.do?pubRef=-//EP//TEXT+TA+P8-TA-2016-0417+0+DOC+XML+V0//EN&language=EN
xxviii http://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:52014XG0708%2801%29&rid=14
xxix http://ec.europa.eu/chafea/news/news410.html
xxx http://www.janpa.eu/
xxxi http://www.consilium.europa.eu/en/press/press-releases/2016/06/17-epsco-conclusions-food-product-improvement/

the EU, rather than national, level. A Roadmap for product improvement was developed to establish a shared long-term agenda for reducing salt, saturated fats and added sugars (calories) in foods throughout the EU.[xxxii]

At the conference of the Netherlands' Presidency, the EHN director called for governments to be in the driving seat and set targets. She presented the case for regulatory intervention in terms of saving lives and cost.

The Council Conclusions call on member states to have a national plan for food product improvement in place by the end of 2017 and, with the Commission, to report regularly on progress, support research and development, raise awareness and facilitate the involvement of small and medium sized businesses in reformulation. It also sets out a number of actions for the Commission including, among other things, assessing current benchmarks on salt and saturated fats under the existing Frameworks, supporting coordination and cooperation, establishing working groups and facilitating research, data collection, monitoring and reporting.

4.1.2.2.2.3 Council Conclusions on Childhood Obesity

On 16 June 2017, under the Maltese Council Presidency, Health Ministers adopted Council Conclusions to contribute towards halting the rise in Childhood Overweight and Obesity.[xxxiii] The text calls for tackling childhood obesity by addressing both the lack of physical activity and unhealthy diets. Member states are invited to promote physical activity in schools and leisure clubs. They are also asked to reduce the advertisement and sponsorship of sugary and fatty foods which are targeted at children and adolescents.

4.1.2.2.3 High Level Group on Nutrition and Physical Activity

The High Level Group on Nutrition and Physical Activity [xxxiv] (hereafter referred to as 'the High Level Group') is a group of European government representatives dealing with this issue, led by the European Commission.

4.1.2.2.3.1 Reformulation efforts – EU Framework for national salt initiatives

EHN's 2011 paper called for efforts to reduce the saturated fat, sugar and salt content and portion size of mainstream food and drink products to be a key priority in Europe.

The European Commission took the initiative to assist EU member states in reducing salt levels in EU countries on a voluntary basis and developed an EU Framework for national salt initiatives.[xxxv] The High Level Group [xxxvi] is responsible for this area of work.

In 2010 the Council of the EU approved this Framework and adopted *Conclusions on Action to Reduce Population Salt Intake for Better Health*.[xxxvii] An implementation report[xxxviii] was published in 2012 which concluded, amongst other things, that:

- the EU salt framework has been a catalyst for action by a number of member states with new initiatives introduced across the EU;
- those countries that have been working on salt reduction for a number of years were the most likely to report specific actions on salt reduction and reported that the Framework supported their action, strengthened or broadened their approach or helped to increase dialogue with industry;
- many countries have started awareness-raising actions. Work in the coming years should focus on increasing the number of quantifiable commitments to reductions from the food industry and on installing adequate monitoring programmes in member states.

Although EHN has supported the Commission's efforts, there are serious concerns that the largely voluntary approach is inadequate. Member state participation in this Framework is entirely voluntary. The Framework sets a feeble goal of a 16% dietary salt reduction over four years for all processed food products. Member states continue to favour voluntary, rather than mandatory, approaches to salt reduction and there is no regular monitoring of progress made by member states.

4.1.2.2.3.2 Reformulation efforts - EU framework for national initiatives on selected nutrients

In February 2011, the EU member states agreed on an *EU Framework for National Initiatives on Selected Nutrients*[xxxix] (such as energy, total fat, saturated fat, trans fats, added sugars, portion sizes and the frequency of consumption of specific foods). Since June 2012, the High level Group has been focussing on the reduction of saturated fat[xl], with a view to reducing saturated fat by 5% in four years from 2012, and thereafter by an additional 5% by 2020.

At the Informal Meeting of Health Ministers in Riga on 21-22 April 2015, the majority of the Ministers of Health of the European Union expressed clear support for the necessity of having a common framework for action to reduce free sugars in food and called for such a framework to be developed to decrease overweight, obesity and NCD risk factors. It

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xxxii Ministry of Health, Welfare and Sport. Roadmap for Action on Food Product Improvement. EUNL 2016. Available from: https://english.eu2016.nl/documents/press-releases/2016/02/23/european-cooperation-for-healthier-food

xxxiii http://ec.europa.eu/health/sites/health/files/nutrition_physical_activity/docs/201706_childhoodobesity_council_en_1.pdf

xxxiv high level group on nutrition and physical activity

xxxv http://ec.europa.eu/health/archive/ph_determinants/life_style/nutrition/documents/salt_initiative.pdf

xxxvi http://ec.europa.eu/health/nutrition_physical_activity/high_level_group/index_en.htm

xxxvii http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:C:2010:305:0003:0005:EN:PDF

xxxviii http://ec.europa.eu/health/nutrition_physical_activity/docs/salt_report_en.pdf

xxxix http://ec.europa.eu/health/nutrition_physical_activity/docs/euframework_national_nutrients_en.pdf

xl http://ec.europa.eu/health/nutrition_physical_activity/docs/satured_fat_eufnisn_en.pdf

adopted an Annex II on Added Sugars[xli] to the EU framework for national initiatives on selected nutrients. The goal is to achieve a sugar reduction of 10% by 2020 compared to the benchmark year of 2015.

4.1.2.2.3.3 EU Action Plan on Childhood Obesity

In 2014, the High Level Group adopted the *EU Action Plan on Childhood Obesity 2014 – 2020* that aims to halt the rise of childhood obesity by 2020.[xlii] In 2017, under the Maltese Council Presidency, a study on the implementation of that *Action Plan* was published (mid-term review). [xliii] The study gives an overview of the efforts made during the first-half period of the *Action Plan* and its state of implementation at the EU level, in every EU member state as well as in Iceland, Norway, Switzerland, Serbia, and Montenegro.

4.1.2.2.4 EU Platform for Action on Diet, Physical Activity and Health

In 2005, the EU platform for action on diet, physical activity and health[xliv] was created. It is a forum for European-level organisations, ranging from the food industry to consumer and health NGOs, willing to commit to tackling current trends in diet and physical activity. It is one of the Commission's major vehicles for tackling overweight and obesity in the EU.

In November 2016, a new methodology to improve the functioning of the platform was adopted. The new methodology now states that it is the objective of the platform to support the member states in reducing the avoidable and economic burden of unhealthy lifestyles and related chronic diseases. New commitments have to be directly related to the members' core missions and businesses and aim at being followed by as many stakeholders in as many member states as possible. Monitoring of the commitments will be undertaken by the WHO, the Joint Research Centre and the European Commission (DG SANTE). EHN, which is one of the founding members of the Platform, welcomes this new structure.

4.1.2.2.5 Other Initiatives

4.1.2.2.5.1 Food taxation initiatives

EHN highlighted, in its 2011 paper, the potentially important role for taxes and subsidies to make healthier foods more affordable and less healthy foods more expensive. The paper called for exploration of the use of taxes, particularly when combined with subsidies, and urged that the introduction of subsidies on healthy foods become a priority for European action. In recent years the evidence base for the effectiveness of taxes has grown – from both modelling studies and from studies monitoring the impact of the growing number of taxes introduced by governments around the world (See Chapter 4.2). The European Commissioner for health and food safety commented in 2017 that such taxation instruments are 'very powerful' and that the Commission is very positive about member states' use of these instruments.[xlv]

At the EU level, DG Enterprise published a study in September 2014 on *Food taxes and their impact on competitiveness in the agri-food sector*.[xlvi] EHN commented that the study should be welcomed by any country that has introduced, or is considering, food-related taxes because there is evidence that food taxes reduce consumption of the taxed products and may benefit low-income people more. Furthermore, there is little evidence that food taxes have a major impact on net profitability, and no good evidence that food taxes lead to increased cross-border shopping or loss of employment,[xlvii] (the last two issues were the subject of exaggerated scare stories when Denmark introduced a fat tax).

4.1.2.2.5.2 Strategic Research

The *Joint Programming Initiative: A healthy diet for a healthy life – Strategic Research Agenda 2012-2020 and beyond* provides a roadmap for harmonised and structured research efforts in the area of food, nutrition, health and physical activity and offers defined priorities to reach the goals. The aim of is to understand better the factors that determine food choices and physical activity behaviours, and thus human health, and subsequently to translate this knowledge into programmes, products, tools and services that promote healthy food choices.

4.1.2.2.5.3 Public procurement of food for health

From 1 January till 30 June 2017, Malta held the Council presidency of the European Union. During this Council presidency, the Maltese government, together with the European Commission Joint Research Centre and DG SANTE , presented a report on *Public Procurement of Food for Health*.[xlviii] The document focuses on school food as a means of supporting healthy eating as an integral part of learning. All EU member states have school food policies and guidelines in place to define healthy diets and nutritious food offerings. The challenge is in translating these food policies and guidelines into procurement contracts for school food. The purpose of the document is to support member states in translating their school national food standards related to health and nutrition into food procurement specifications. The report includes a range of options and considerations that each member state may wish to use and adapt in respect of its specific context. The report is expected to become a living document that will benefit from updates in the future.

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xli Annex II : Added Sugars – EU framework for national initiatives on selected nutrients
xlii http://ec.europa.eu/health/nutrition_physical_activity/docs/childhoodobesity_actionplan_2014_2020_en.pdf
xliii Study on the implementation of the EU action plan on Childhood Obesity 2014-2020
xliv EU platform for action on Diet, physical activity and health
xlv https://ec.europa.eu/avservices/video/player.cfm?ref=I135714
xlvi http://ec.europa.eu/growth/tools-databases/newsroom/cf/itemdetail.cfm?item_id=7668
xlvii EHN statement on the study Food taxes and their impact on competitiveness in the agri-food sector.
xlviii Public Procurement of food for health

4.1.2.2.5.4 Food companies initiatives

On 8 March 2017, six food companies (The Coca-Cola Company, Mars, Mondelez, Nestlé, PepsiCo and Unilever) acknowledged that their existing reference intake (R.I.) front-of-pack nutrition labelling scheme could be enhanced by integrating traffic-light colours. However, they propose to integrate portion sizes into an 'evolved scheme' with a view to accommodating smaller portion sizes. The companies are inviting stakeholders to join a taskforce to assess the feasibility hereof. EHN welcomes the use of colour coding in front-of-pack labelling, but is concerned about the provision of nutrition information on a per portion basis because it can confuse consumers and is open to manipulation. Currently there is no evidence that the provision of nutrition information on a per portion basis can reduce consumption.

4.1.2.3 National developments

Some European countries have implemented their own initiatives, specifically in relation to food taxes, reformulation, trans fats, labelling, food in schools and other public institutions, and marketing to children. While these initiatives are welcome, and some countries have shown admirable leadership, EHN would have liked to be able to report much more widespread implementation of concrete action in these areas. The lack of progress on tackling Europe's biggest cause of death with government-led interventions that are known to work is extremely disappointing. The following sections summarise some of the recent developments at national level; it does not claim to be an exhaustive list.

4.1.2.3.1 Food taxes

In the last five years, several EU countries have introduced taxes on unhealthy food products – or have obtained evidence on the impact of previously-introduced taxes. These taxes are generally intended to steer consumers to a healthier diet, although in some cases their primary purpose is to raise revenue:

- **Denmark** introduced a saturated fat tax in 2011. Unfortunately, the tax was abolished after just over a year following a change of government and negative media coverage (often erroneous) blaming the tax for rising inflation, people buying products in other countries, and job losses in a time of economic crisis. Research has since shown that the tax reduced consumption of fat (by between 10% and 15%)[18] and saturated fat and that it made a positive contribution to public health.[19] Analysis points at shifts in demand from high price supermarkets towards low-price discount stores – at least for some types of oils and fats – a shift that seems to have been utilised by discount chains to raise the prices of butter and margarine by more than the pure tax increase.[xlix]
- **Finland** imposed a tax on ice cream and soft drinks in late 2010, and raised the rates on some products in 2012 and 2014. In 2015 the tax was levied on confectionery and chocolate, but excluded certain products such as biscuits, baked goods, yoghurt products, puddings, jellies, mousses and granulated sugar.[20] There has not been any formal evaluation, but a reduction in consumption of sweets and soft drinks was reported in 2011 and 2014.[20] This measure is currently being contested by the European Commission. However, the Finnish parliament voted to abolish the tax on confectionery and ice cream as of 1 January 2017 after it was accused by the European Commission of providing state aid to certain companies.
- **France** adopted a tax on sugar- and artificially-sweetened beverages in 2011. Effective from January 2012, the tax covers all non-alcoholic beverages with added sugar or sweeteners and is equivalent to around €0.11 per 1.5 litres.[20] A 2016 review found that the tax had filtered through to the price of drinks and the measure is generally viewed favourably by the public.[21] An increase in the tax and possible extension to sugary foods was recommended in a parliamentary report in 2016.[l]
- **Hungary** introduced a public health product tax in 2011. The tax is aimed at products for which healthier alternatives are available. It is applied to sugar-sweetened beverages, some energy drinks, confectionery, salted snacks and condiments, alcoholic drinks with a high sugar content, fruit jams and ice creams.[20] A first assessment of the tax observed a 20-35% decrease in consumption of taxed products, a significant shift to reformulate products by manufacturers, and heightened public understanding of the health issues.[22]
- **Belgium** levied a health tax on soft drinks and alcohol from 2016. The federal government announced the relatively small tax of €0.03 per litre on soft drinks in October 2015, in line with the National Nutrition Plan which aims to reduce sugar consumption. Developed jointly by the Minister of Social Affairs and Health and the Minister of Finance, the tax is predicted to bring in €50 million annually in tax revenue.[li]
- **United Kingdom:** On 17 March 2016 the UK government announced there will be a sugar tax on some food products from 2018. The tax will be levied on the companies involved, based on the volume and concentration of sugar in the soft drinks they produce or import.
- **Portugal** approved a Special Consumption Tax on beverages with added sugar and other sweeteners, from 2017. The tax will increase the final price by from €0.14 to €0.30 per 1.5 litre bottle.
- **Three other EU countries (Estonia, Spain and Ireland)** have also announced plans for taxes of soft drinks

Food taxes have also been introduced in a number of countries outside Europe. There has been a great deal of attention, for example, on the Mexican tax on sugary drinks which was introduced in 2013 and increases prices by about 10%. Results of an evaluation found that the tax was associated with reductions in purchases of taxed soft drinks by an average of 6% in 2014 with an increasing rate of decline, reaching 12%

xlix http://www.sciencedirect.com/science/article/pii/S0306919213000705
l http://www.assemblee-nationale.fr/14/rap-info/i3868.asp
li http://www.deblock.belgium.be/fr/affaires-sociales-sant%C3%A9-publique-et-budget-r%C3%A9duction-des-charges-patronales-taxe-sant%C3%A9-non-marchand

in December 2014. All socioeconomic groups reduced their purchases of taxed drinks, but the decline in purchases was greatest in lower socio-economic groups – averaging 9% over the year and reaching 17% in December 2014.[23]

Findings from those experiences are bolstering the considerable existing evidence base from studies that taxes can be effective in influencing food consumption – and thus nutrition – with benefits across all socio-economic groups.

4.1.2.3.2 Food labelling

EHN has long advocated for mandatory, legible, back-of-pack and front-of-pack labelling (See Figure 40 for examples). The 2011 paper repeated the call for a simplified front-of-pack scheme containing key elements – energy, fat, saturated fat, sugars and salt – and colour coded with red, yellow and green indicating high, medium and low levels of these elements. This type of labelling scheme is more visible and easier for all groups of consumers to interpret than standard back-of-pack nutrient declarations. Experience from Finland, New Zealand, the US and the Netherlands shows that front-of-pack nutrition labelling also encourages manufacturers to reformulate products to make them healthier.[24–27]

In the United Kingdom, the Government published, in 2013, a recommended voluntary front-of-pack nutrition labelling scheme using green, amber and red traffic lights to indicate low, medium or high levels of energy, fat, saturated fat, salt and sugar. The European Commission, responding to concerns raised by Italy, has questioned whether this constitutes a barrier to trade. In February 2014, EHN issued a statement in support of the UK's hybrid traffic light system and highlighted the lack of evidence that this voluntary scheme will hinder the free movement of goods and the functioning of the EU internal market

In Sweden, Denmark, Norway, Iceland, Lithuania and FYR Macedonia the voluntary Keyhole labelling scheme is in use. The work with the Keyhole label started in Sweden in 1989 and the Swedish National Food Agency owns the Keyhole brand. It involves 33 food groups and contains less sugars and salt, more fibre and wholegrain and healthier or less fat than food products of the type not carrying the symbol. The nutritional criteria have recently been updated and tightened.[lii] All Keyhole-labelled products have had to meet these new requirements by 1 September 2016. The Finnish Heart Symbol scheme indicates which foods in product categories are healthier in terms of quantity and quality of fat and salt, and, in some product groups, sugar and fibre.[liii]

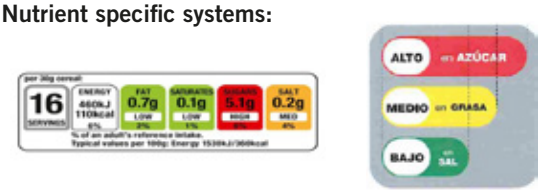
The industry-funded Choices programme, which includes a front-of-pack logo to indicate healthier products that fulfil the Choices criteria, has established national programmes in the Netherlands, Belgium, Poland and the Czech Republic.[liv] It is noteworthy that these systems only carry labels on products that are healthier, and not on those that are less healthy, so are not likely to have the same impact as, for example, traffic light labels. The programme has a green logo, used for basic products, and a blue logo, used for products that it is not necessary to eat, but showing the healthier option within this category. This distinction often led to confusion, making consumers believe that both products were healthy and good for you. The programme was stopped in the Netherlands in October 2016 at the request of the Dutch Minister for Health who said that the two logos led to too much confusion for the consumer. At this stage it is unclear whether the programme will remain in place in the other countries.

From 1 April 2017 onwards the French government proposed to use its Nutri-Score scheme as a voluntary front of pack labelling scheme to be used in France. For the Nutri-Score scheme, points are given based on a per 100g basis. Positive points are allocated for 'good' nutrients (fruits, vegetables and nuts, fibre and protein) and points are deducted for 'negative' nutrients (saturated fat, sugar, sodium (salt) and energy). The Nutri-Score scheme also uses traffic light colours, but rather than providing a colour per nutrient, it provides an overall assessment of the nutritional quality of a product. There are five colours: dark green, light green, yellow, orange and deep orange. The nutrition criteria for the scheme are based on the nutrient profile model developed by the Food Standards Agency in the UK for regulating advertisement on television to children of 'unhealthy foods'. A 2016 trial in real life conditions found that application of the Nutri-Score label improved the nutritional quality of shoppers' baskets, including those shoppers who usually buy the cheapest foods.[28] Three major supermarkets and one food manufacturer have already signed up to use the scheme. However, six EU member states (the Czech Republic, Germany, Hungary, Italy, Poland and Spain) have subsequently objected to the French Nutri-Score system. This means France cannot introduce it until the objections have been addressed.

There are some examples of labels also being used to warn about specific issues. Legislation in Finland, for example, requires 'high salt content' warnings on foods that contain more salt than government-set limits for products such as bread, sausages, cheese, butter, breakfast cereals and crisp bread. In a similar example from outside Europe, chain restaurants in New York City are required to include a salt shaker icon as a warning on menus next to menu items or meals containing 2 300 mg of sodium or more.

lii http://www.norden.org/en/news-and-events/news/keyhole-milestone.
liii http://www.sydanmerkki.fi/en
liv http://www.choicesprogramme.org/government/government.

Figure 40 Examples of front-of-pack labelling schemes

	Underlying nutrient profile models
Nutrient specific systems: 	• Nutrients kept separate • Thresholds set for each nutrient
Summary indicator systems 	• Nutrient levels combined to give overall rating Thresholds for combined score for half star, one star etc.; for green, for yellow etc.
Health/nutrition claims 	• Nutrient levels combined to give overall indication of healthiness • Thresholds for one or more nutrients combined by AND/OR etc.

4.1.2.3.3 Reformulation

Many European countries have introduced salt reduction initiatives and/or other reformulation initiatives, whether on a voluntary or mandatory basis.

Voluntary salt reduction initiatives have been introduced in Austria, Belgium, Croatia, Czech Republic, France, Hungary, Italy, Norway and Spain. Many of these initiatives – often piloted by the Ministry of Health – have prioritised reducing salt levels in bread products. Mandatory salt limits on bread are also in place in Belgium, Bulgaria, Greece, Netherlands and Portugal. National salt reduction strategies, including reformulation, have been shown to work, with at least 10 countries worldwide reporting reductions in population salt intakes following strategies that included reformulation to reduce salt levels.[29] Mandatory approaches, which create a level playing field for all industry, are more effective and cost-effective.[30]

Wider voluntary reformulation efforts are also ongoing. In France, for example, there is a government-led engagement with industry to reduce total, saturated fats, and sugar as well as salt, and to increase fibre contents. Similarly, the UK's voluntary Responsibility Deal with industry targeted reductions in saturated fats, trans fats, calories and portion sizes. In the Netherlands, the Dutch Ministry of Health agreed the Accord Improving Product Composition involving action on food reformulation of processed food for salt, saturated fats and calories (sugars and fats) in cooperation with the sector associations for industry, supermarkets, caterers and restaurants.

4.1.2.3.4 Trans fats

Legislation to limit trans fats in foods was already in place in Austria and Denmark when EHN published *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe* in 2011. Hungary, Iceland, Norway, Serbia and Latvia have since introduced legislation to limit industrially produced trans fats levels in foods. In June 2017, Lithuania notified the European Commission of its draft law to limit trans fat levels. Many other countries have worked with industry to reduce trans fats through voluntary measures. Seven years after WHO called for trans fats to be eliminated from the global food supply and more than 10 years after the first national 'ban' was introduced, and given that there is clear evidence that national or local 'bans' on trans fats are more effective than voluntary measures in eliminating trans fats from the food supply,[31] it is puzzling that more countries have not introduced mandatory limits.

4.1.2.3.5 Food in public institutions

EHN's previous paper made a number of recommendations for improving the quality of food served and/or sold in public institutions. It also made some specific recommendations in relation to food in schools. Namely, that food provided in schools should comply with high nutritional quality standards and that energy dense and high salt snacks, sugary drinks and confectionery should not be available in schools.

In the last five years a number of European countries have taken action to improve food in schools and other publicly funded institutions. Examples include:

- **Bulgaria** – Mandated reductions in salt, fat and sugar content in food served in childcare in 2013, bringing childcare into line with schools. Vending machine restrictions are also in place.
- **Hungary** – Since 2011, products with high salt, sugar or caffeine levels, to which the Public Health Product Tax applies, must not be sold on school premises.
- **Latvia** – In 2012, the government set limits for salt in food served in educational establishments, hospitals and long-term care institutions.
- **Lithuania** – Food and nutrient-based standards for schools, pre-schools and children's institutions were introduced in late 2011. Soft drinks are banned.
- **Netherlands** – A voluntary code for healthier school canteens was introduced in 2011: a minimum of 75% of the products sold are basic foods (as defined by the Netherlands Nutrition Centre) and a maximum of 25% of the products sold are non-basic foods. A Healthy School Approach has also been set up to cover a range of issues, including nutrition.
- **Poland** – An existing regulation on food and nutrition safety was amended in 2014 to set out nutrition standards for canteens in schools and pre-schools.
- **Slovenia** – A new *School Nutrition Law* in 2013 requires school meals to follow specified dietary guidelines. This is complemented by food procurement standards and a 2010 ban on vending machines in schools.
- **Sweden** – Since July 2011 school meals have been required to be nutritious – complying with *Good School Meal Guidelines,* revised in 2013 – and have had to be free of charge since 1946. Sugary drinks, ice cream, pastries and sweets are not to be provided by the school.
- **United Kingdom** – There are now mandatory nutritional standards for all food served or sold in primary schools, following updated legislation in England (in force since January 2015) and Wales (amended 2013).

It appears that few countries are taking action beyond the school/pre-school environment. However, in Latvia, limits were set for salt in food served in hospitals and social care facilities in 2012. In the Netherlands, steps are being taken to implement the healthier school canteens code in sports clubs and workplace canteens. In Finland, amended rules requiring compliance with nutritional criteria as a condition for university catering to qualify for government subsidies came into force in 2013. This is an example of how governments are recognising that the food provided by government-supported establishments should not be helping to promote diets that cause a massive health burden.

Outside Europe, other examples of action to improve the nutritional quality of food in public institutions exist. In Bermuda, food and drinks in vending machines on government premises have to meet specific nutritional criteria. New York City set nutritional standards for all food served (or bought) by city agencies, including prisons, hospitals and social care facilities for older people. The City's Health Code also regulates the drinks that can be served in children's centres or during children's camps.

4.1.2.3.6 Food marketing to children

In the six years since publication of EHN's previous paper, a number of countries have made progress in efforts to limit marketing of HFSS foods to children. In 2013, Ireland prohibited advertising of HFSS foods during children's TV and radio programmes (where over 50% of the audience are aged under 18) and restricted the overall amount of advertising for HFSS foods at any time of day to no more than 25%. Poland and Spain took measures to regulate advertising in schools and pre-schools. In the Netherlands, the *Code of Advertising for Food towards Children* is based on the *EU Pledge* and was updated in January 2015 to include nutritional criteria for children between 7 and 12 years old. In 2013, Norway implemented a government-supervised, yet voluntary, initiative to restrict marketing of HFSS foods to children across a range of media and communication channels. Following an evaluation of the impact of this initiative, the scope was extended and rules tightened up in 2017. In June 2017 the UK amended its rules on marketing to children to, among other things, cover non-broadcast media (including online and digital marketing) and to cover brand advertising that has the effect of promoting a HFSS product.

The nutrient profile model published by the WHO Regional Office for Europe is specifically designed for restricting marketing to children (see above). Since publication of this model some countries have, or are in the process of, adapting it to their national context and this will enable countries to move forward more quickly with regulatory initiatives.

Table 9 Summary of some recent developments in key policy areas in Europe

Labelling	Trans fats	Salt reduction	Other reformulation	Marketing to children	Food in public institutions	Food taxation
EU: The Commission has not established the nutrient profiles required by the claims Directive to prevent health and nutrition claims on less healthy foods. Since 2014 the *Food Information to Consumers Regulation* allows member states (MS) to recommend additional forms of information (e.g. front of pack (FOP) nutrition labelling). UK, 2013: voluntary FOP traffic light labelling scheme recommended by government. Denmark, Iceland, Norway and Sweden: the voluntary Keyhole label-ling scheme is in use. Finland: the Heart Symbol is used to endorse 'better-for-you' choices. Netherlands, 2016: the Dutch Health Minister stopped the industry-funded Choices programme. In March 2017, six multinational food companies came out in favour of a prominent on-pack nutrition colour-coded labelling scheme. France, 2017: Government announced intention to introduce a voluntary Nutri-Score FOP labelling scheme to indicate overall nutritional quality of foods.	EU, 2015: The Commission report concluded that a legal limit for industrially-produced trans fats (IPTFAs) would be the most effective measure. The European Parliament called on the Commission to establish a legal limit on IPTFAs as soon as possible and the Commission launched an impact assessment. Hungary, Iceland, Norway, Serbia, and Latvia have joined Austria and Denmark, introducing legislation to limit IPTFAs. Lithuania has informed the European Commission of a draft law on limiting trans fat levels in foods.	EU, 2010: The Council approved the *EU Framework for National Salt Reduction Initiatives* and adopted *Conclusions on Action to Reduce Population Salt Intake for Better Health*. There has been a disappointing level of participation in the Framework, which sets a relatively feeble goal (16%) for salt reduction. Voluntary salt reduction initiatives – often focusing on bread – have been introduced in: • Austria • Belgium • Croatia • Czech Republic • France • Greece • Hungary • Ireland • Italy • Norway • Slovenia • Spain Mandatory salt limits on bread are in place in: • Belgium • Bulgaria • Greece • Netherlands • Portugal	EU, 2011: EU MS agreed on an EU *Framework for National Initiatives on Selected Nutrients* Then in 2016, under the Dutch Council Presidency, *Council Conclusions on food product improvement* and a roadmap for product improvement were developed. MS are requested to have national plans in place by the end of 2017. France: Government-led voluntary initiative aims to reduce total fat, saturated fat, sugar and salt, and to increase fibre. UK: Ongoing government-led voluntary initiative to reduce saturated fats, trans fats, calories and portion sizes. In the Netherlands, an *Accord Improving Product Composition* was agreed with industry to reformulate processed food for salt, saturated fats and calories.	European Region, 2015: Publication of a WHO regional nutrient profile model specifically for restricting HFSS food marketing to children. In 2016, a tool for monitoring food marketing to children was published. EU, 2014: The Council called on the Commission and MS to promote actions to reduce children's exposure to HFSS food marketing. EU: Proposal to amend the *Audiovisual Media Services Directive* was adopted in 2016. EHN has repeated its call for a watershed for all marketing for HFSS food on television between 6am and 11pm. Ireland, 2013: Prohibited advertising of HFSS foods during children's TV and radio programmes and also limited HFSS food advertising to 25% of all advertising. Norway, 2013: Government supervised voluntary restrictions introduced across a wide range of media. Netherlands, 2015: Code updated to include nutritional criteria for 7-12 year old children. Poland and Spain: Regulated advertising in schools and pre-schools. UK, 2017: Existing rules extended to cover digital and online marketing and to cover brand advertising that has the effect of promoting a HFSS product.	EU, 2017: Report on *Public Procurement of Food for Health,* published under Malta's EU Presidency, to help MS to translate school nutrition standards into food procurement specifications for caterers. Legislation or codes to improve nutritional quality of food provided in schools and/or pre-schools introduced or updated in: • Bulgaria • Hungary • Latvia • Lithuania • Netherlands • Poland • Slovenia • Sweden • England • Wales Latvia: Limits introduced for salt in food served in hospitals and social care facilities. Finland: Rules setting out nutritional criteria for university catering to qualify for government subsidies were updated.	EU: A 2014 Commission paper found evidence that taxes reduce consumption of taxed products and found little evidence that they have an impact on profitability. Denmark: Evidence has mounted that the saturated tax fat, repealed in 2012, had brought about a decrease of 10-15% in fat consumption. France, 2012: Introduced taxes on sugar-sweetened and artificially-sweetened drinks. Hungary, 2011: The Public Health Product Tax has led to an estimated 20-35% decrease in consumption of taxed products. Taxes on soft drinks have been introduced or proposed in: • Estonia • Belgium • Ireland • Portugal • Spain • UK Finland, 2010: introduced a tax on ice cream and soft drinks, but, following challenge from the European Commission, later abolished it.

4.2 An analysis of food and drink policies to promote cardiovascular health

Interventions aimed at improving diets are extremely diverse – seeking to influence different aspects of the food chain as a whole, from international trade policy to how individuals choose what to eat in restaurants. There are many different ways of thinking about these varied interventions, in order to be able to identify all that are relevant and classify them in a systematic way.

To help with systematic identification and classification of possible interventions a number of frameworks have been developed (See Annex 3). One such framework is the NOURISHING Framework developed by the World Cancer Research Fund for reporting, categorising and monitoring policy actions.[32lv] The framework is based on the understanding that food policies should aim to improve dietary behaviours by improving the availability, affordability and acceptability of healthy diets and by decreasing the availability, affordability and acceptability of unhealthy diets.

Using NOURISHING as a framework, EHN's Expert Group on Nutrition identified a number of key areas for action on food and drink policies to promote cardiovascular health (Table 10). Far from covering all the possible areas where effective action could be taken, these key areas represent a small number of priority areas, selected by EHN because of their potential to make meaningful changes to the diet of the entire population (or a large sector thereof) and with potential to reduce health inequalities.

There are clearly many other areas for action, as highlighted in Chapter 3 and also set out in the NOURISHING framework, such as actions to improve the food retail environment (e.g. improving access to healthy foods, restricting promotions on less healthy foods, using planning/zoning laws to restrict unhealthy food outlets in the vicinity of schools) and behaviour change communications (e.g. nutrition education and counselling, mass media campaigns). In addition, many important long-term strategies to improve nutrition – such as the protection, promotion and support of breastfeeding and appropriate infant feeding (see Box, below), as well as policies to improve maternal nutrition – are of fundamental importance. Although these are not explored in detail in the current report, such actions should form part of a comprehensive, food systems approach to improving diet for cardiovascular health

lv http://www.wcrf.org/int/policy/nourishing-framework

Table 10 The NOURISHING framework incorporating the EHN key recommendations

DOMAIN		POLICY AREA	EHN KEY RECOMMENDATION
Food environment	N	Nutrition label standards and regulations on the use of claims and implied claims on foods	Appropriate nutrient profiles must be set to underpin nutrition and health claims as required by the EU regulation on nutrition and health claims (EC) No 1924/2006). Mandatory EU-wide simplified front of pack (FOP) nutrition labelling should be adopted. EHN continues to recommend a scheme which uses traffic-light colours to indicate high, medium and low levels of fat, saturated fat, salt and (preferably added) sugar. EHN will follow developments in evidence for most effective FOP nutrition labelling scheme National governments must be able to recommend/endorse meaningful front of pack symbols in their countries in addition to any harmonised EU rules on FOP nutrition labelling. A few countries use symbols to indicate a healthier option and these are generally thought to be helpful.[33,34] National governments are encouraged to adopt mandatory requirements to provide easy-to understand information about the nutritional quality of foods served in chains with more than 10 outlets (menu labelling).
	O	Offer healthy foods and set standards in public institutions and other specific settings	Establish nutrition standards for food served or sold in schools, hospitals and other public institutions and issue guidelines for healthy public procurement
	U	Use economic tools to address food affordability and purchase incentives	Use of taxes and/or subsidies to promote consumption of healthy foods and reduce consumption of unhealthy foods
	R	Restrict food advertising and other forms of commercial promotion	Regulatory controls to reduce the public health impact of marketing (including advertising and other forms of commercial promotion across all media) of foods high in fat, sugar or salt
	I	Improve the quality of the food supply	Wide-ranging, ambitious food reformulation programmes to reduce levels of salt, saturated fat, free sugars, energy density and portion size in processed foods Mandatory maximum levels of industrially-produced trans fatty acids
	S	Set incentives and rules to create a healthy retail environment	
Food system	H	Harness supply chain and actions across sectors to ensure coherence with health	Global food convention to establish global regulatory framework to protect healthy diets Reform agriculture and food policy to align with public health policies and promote sustainable food production Ensure that trade and investment policies take nutrition into consideration with the aim of protecting and promoting public health
Behaviour change communication	I	Inform people about food and nutrition through public awareness	
	N	Nutrition advice and counselling in health care settings	
	G	Give nutrition education and skills	
Overarching EHN Recommendations not covered by NOURISHING			Implement policies to tackle cardiovascular health inequalities in Europe. Ensure that robust mechanisms for nutrition governance are in place and fit-for-purpose. Develop an integrated health and environment approach to food systems and promote health-environment win-wins in food-based dietary guidelines.

The key policy priorities identified above vary considerably in nature. Some, such as legislation on mandatory limits of trans fatty acids or rules on front-of-pack labelling, are very specific and relatively precise. Others – such as the recommendations on global trade, agricultural policy and a global food convention – are much more general in nature. This is because, if a health-promoting diet is to become a reality across Europe, some major forces that influence food systems need also to be both acknowledged and refocused so that there are health benefits as well as benefits for a particular branch of industry.

Policy measures for optimal early nutrition

A supportive policy environment is needed to protect, promote and support breastfeeding and appropriate complementary feeding, essential for the best start in life and later health (see Chapter 2.3.7). This paper does not explore this policy area in detail. An array of policy instruments already exists, however, and EHN fully supports their comprehensive implementation.

The global *International Code of Marketing of Breast-milk Substitutes*, adopted in 1981 in response to concerns about the impact of infant formula marketing on breastfeeding rates and child health, aims to stop the aggressive and inappropriate marketing of breastmilk substitutes, feeding bottles and teats.[35] Over the years the World Health Assembly has adopted a series of resolutions to provide clarification and to ensure that the rules remain relevant as companies change their marketing tactics. In the European Region, 48 countries have translated some or all of the Code measures into national law, although only three countries have fully implemented *all* provisions of the Code and only four have translated *many* of the Code's provisions into law.[36]

Since the Code was adopted in 1981, different types of breastmilk substitutes have been launched. There is now a sizeable market in 'follow-up' or 'follow-on' formula for infants over 6 months and other milks for young children up to the age of three years (sometimes referred to as 'growing-up milks', 'toddler milks' or 'young child formula'). WHO has clarified that these products are covered by the Code[lvii] and should be included in national legislation.[37]

There has been mounting concern about the impact of inappropriate marketing of baby food products, including concerns that some commercial complementary foods are undermining breastfeeding, that potentially misleading claims about a child's health or academic performance are made, and that breastmilk substitutes are being indirectly promoted by use of formula products' branding and logos on foods for babies and young children. To address these concerns WHO has issued *Guidance on ending the inappropriate promotion of foods for infants and young children;*[37] it is now up to countries to translate the guidance into national laws and monitor implementation.[lviii]

The situation is currently somewhat complicated for EU member states because the regulatory environment for infant formula and baby foods within the EU is in a state of flux. Since July 2016 a new Regulation (EU 609/2013, commonly referred to as the 'foods for special groups regulation') covers infant formula, follow-on formula and processed cereal-based food and baby food. New rules on infant formula and follow-on formula will apply from February 2020. In 2016 the European Parliament rejected new rules on processed cereal-based food and baby food because of concerns that the permitted maximum sugar levels were too high and that labelling should make it clear that products are not appropriate for use before six months. The Commission now has to prepare a new version of these rules (a Delegated Regulation) and has commissioned a study to feed into this process.

Marketing rules only cover, however, one aspect of creating a supportive policy environment for early nutrition. These measures need to be seen in the context of the global *Comprehensive Implementation Plan on Maternal, Infant and Young Child Nutrition.*[38] Measures such as supportive health services (facilities that are certified 'baby-friendly'), labour laws that entitle women to paid maternity leave and policies to facilitate breastfeeding outside the home are, for example, all important.

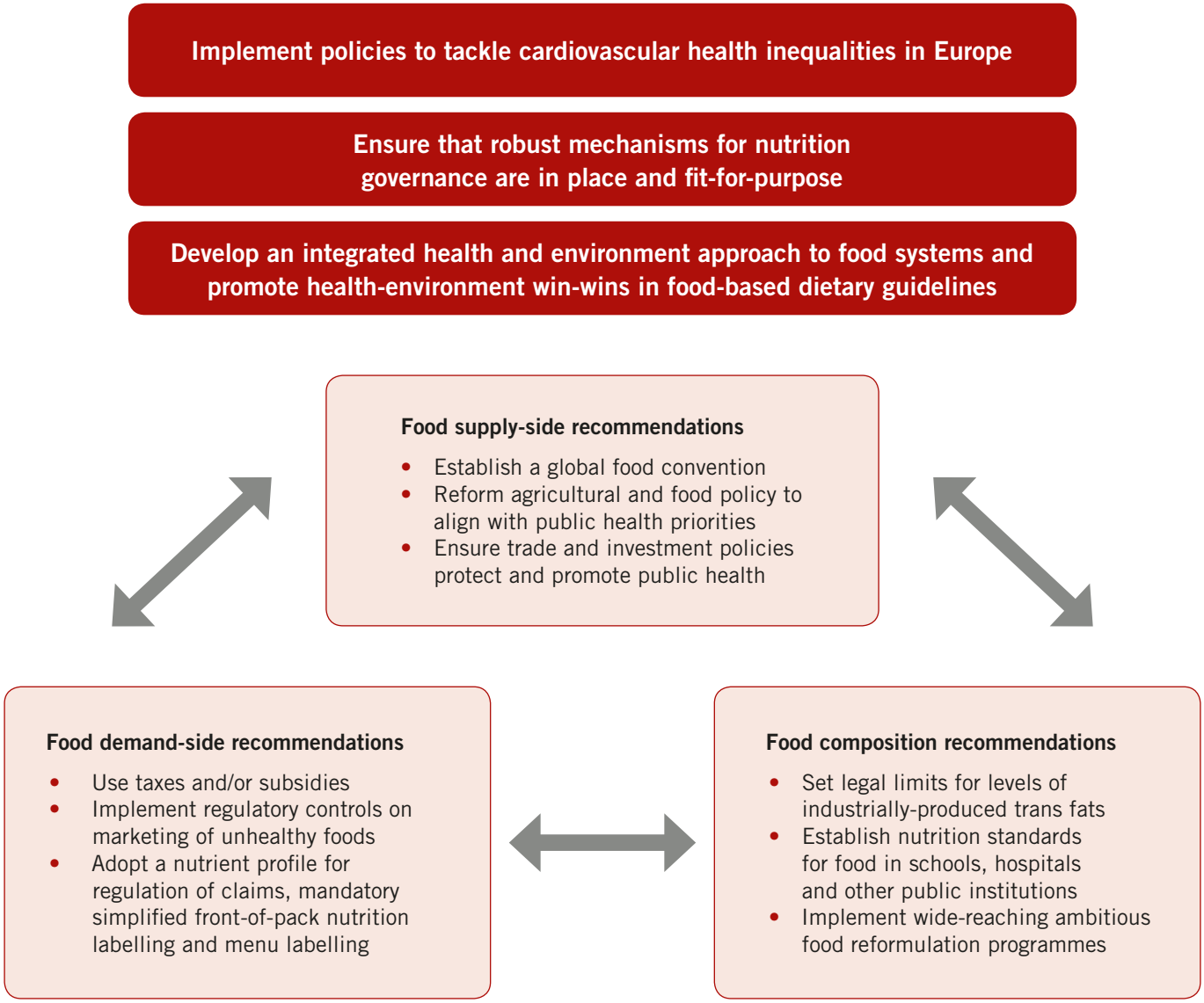
In addition, some fundamental challenges need to be addressed in order to create an environment where these specific recommendations can have an impact. This is why three overarching recommendations – on tackling inequalities, nutrition governance and sustainable food systems – are also set out. These are beyond the scope of the NOURISHING framework.

The recommendations shown in Table 10 are not shown in order of priority and, rather, should be seen as a comprehensive package, comprising three overarching recommendations and three clusters of more-detailed specific recommendations (Figure 41)

lvi The guidance specifically relates to '*any milks (or products that could be used to replace milk, such as fortified soy milk), in either liquid or powdered form, that are specifically marketed for feeding infants and young children up to the age of three years (including follow-up formula and growing-up milks)*'

lvii See http://www.who.int/nutrition/netcode/en/ for a policy brief on the guidance

Figure 41 EHN's recommendations: Overarching recommendations and specific policy recommendation clusters for food and nutrition policies for cardiovascular health



4.2.1 EHN's key recommendations: Three overarching recommendations

To maximise their impact against the backdrop of the 'big picture' drivers highlighted in Chapter 3, the specific recommendations need to be underpinned by three overarching recommendations (as shown in Figure 41):

- Implement policies to tackle health inequalities in Europe
- Ensure that robust mechanisms for nutrition governance are in place and fit-for-purpose
- Develop an integrated health and environment approach to food systems and promote health-environment win-wins in food-based dietary guidelines

4.2.1.1 Implement policies to tackle health inequalities in Europe

Progress in improving cardiovascular health across Europe has not benefited Europe's disadvantaged populations to the same degree as more privileged population groups, as outlined in Chapter 1. There are still alarming differences in cardiovascular health between the continent's wealthiest and poorest groups. Given this backdrop, it is absolutely essential that policy action on dietary factors be underpinned by broader, structural change to tackle health inequalities. Such action is imperative if a heart healthy Europe is ever to become a reality for all.

In its hugely influential global report in 2008, the WHO Commission on Social Determinants of Health issued three over-arching recommendations:

- To improve daily living conditions
- Tackle the inequitable distribution of power, money and resources
- Measure and understand the problem and assess the impact of action.[39]

These recommendations remain as relevant as ever or, rather, are even more relevant since levels of income inequality in developed economies are now higher than ever before.[40] Increasingly, inequality is being described as the key challenge of our time and many economists are now making the case that inequality is harmful to both economic growth and human development.[40–43] Far-reaching political action at global, regional, national and local levels is required. Policies to narrow inequalities across the whole of society (reducing the steepness of the social gradient) and to address the specific needs of particularly vulnerable groups are needed.

In addition, more specific action is also required on food and nutrition policies to address inequalities in cardiovascular health, including by improving availability and affordability of healthy diets. Research in some high- and middle-income countries shows that prices of healthy foods, particularly fruits and vegetables, have risen more than less healthy foods – including oils, fats, sugar and some highly processed foods – over the last 30 years.[44]

There are a number of specific recommendations for policymakers:

- Tackle food poverty and improve the affordability of healthy foods
- Improve the accessibility of healthy foods and address the problem of 'food deserts' where access to fresh foods and healthy options is restricted
- Implement policies to reduce the relative accessibility and affordability of unhealthy foods
- Ensure that an 'inequalities lens' is applied to all other policy initiatives, and ensuring that they are designed to narrow, rather than widen, the gap.

In relation to the last point, the application of an 'inequalities lens' to potential interventions needs to be done by assessing the potential impact on different groups as fully as possible. This should include, crucially, estimates of the potential health impact. This is important to stress because if cursory impact assessments fail to take health outcomes into account they could point to misleading results. For example, while at first glance taxes on unhealthy foods may appear to be regressive because they will have a bigger financial impact on poorest groups, more in-depth analysis points to the potential for greater health gains for disadvantaged groups.[45–47]

4.2.1.2 Ensure that robust mechanisms for nutrition governance are in place and fit-for-purpose

Robust nutrition governance – including high-level political commitment and cross-government, multisectoral coordination mechanisms – is essential to implement policies to improve diets. As clearly demonstrated in Chapter 3, action is needed across many sectors of government and indeed across the whole of society. Very few of the actions recommended in Figure 41 lay solely within the domain of ministries of health. Ministries of finance, trade, education, justice, media and industry are all implicated, as are local authorities.

The ICN2 Framework for Action recommended enhancing 'political commitment and social participation for improving nutrition' and strengthening and/or establishing 'national cross-government, inter-sector, multi-stakeholder mechanisms for food security and nutrition'.[8] WHO's *European Food and Nutrition Action Plan 2015-2020* includes an objective to 'strengthen governance, alliances and networks for a health-in-all-policies approach' and emphasises the importance of support for the development of formal mechanisms to promote cross-government cooperation, including at the local level.[48]

A series of elements or characteristics have been identified as key elements for successful development and implementation of national nutrition policies and strategies and can be used to assess nutrition governance:

- Existence of an intersectoral mechanism to address nutrition;
- Existence of a national nutrition plan or strategy;
- Whether the national nutrition plan or strategy is adopted;
- Whether the national nutrition plan or strategy is part of the national development plan;
- Existence of a national nutrition policy;

- Whether the nutrition policy is adopted;
- Existence of national dietary guidelines;
- Allocation of budget for implementation of the national nutrition plan, strategy or policy;
- Regular nutrition monitoring and surveillance; and
- Existence of a line for nutrition in the health budget.[49]

These elements have been combined into a composite indicator of nutrition governance.[50]

An important factor is the percentage of national budget allocated to nutrition. Public sector investment in improving diets makes economic sense. This is reinforced by the fact that an indicator to measure the overall national government's spending on 'direct nutrition actions' and on 'nutrition-sensitive actions in related sectors' is included as an indicator for the SDGs.[12]

To underpin evidence-based policy action and to be able to report on progress, a strong infrastructure for research and surveillance, monitoring and evaluation is important. Many gaps and needs in research infrastructure for harmonised and standardised data have been identified, particularly in Central and Eastern European countries.[51,52] Those gaps can include food composition databases, food consumption databases, dietary data collection tools, food classification systems, nutrient calculation tools, biomarkers, dietary and nutrition reference values and guidelines.

WHO's *European Food and Nutrition Action Plan* calls on member states to 'continue to strengthen and expand nationally representative diet and nutrition surveys' and 'as a priority, establish national food composition databases'. Further research is needed on the interactions between nutrition and sustainable food systems and how to shift towards healthier and more sustainable diets.

Capacity development in food consumption research has been identified as a key need – with support from the UN University and the UNSCN, Central and Eastern/Balkan countries have developed a capacity development network (www.capnutra.org). As part of the capacity development process, harmonisation of nutrition professional education is an important part of nutrition policy implementation. FAO and WHO are developing a pilot Global Individual Food consumption data Tool (GIFT) for establishing a global database, to collect, harmonise and disseminate data available through an interactive web platform. There is tremendous value in linking different policymakers and academic groups across Europe, and opportunities for such linkages and exchange of experience and good practice should be nurtured.

In order to hold governments to account on their progress towards improving diets, monitoring and surveillance are important. Making information on the evaluation of progress publicly available also helps to raise awareness of the importance of policy initiatives, and thus can help to bolster support for government action. Governments are already working towards targets agreed at the global and regional level, including the relevant targets of the SDGs, the global nutrition targets and the global NCD targets. Furthermore the Rome Declaration on Nutrition contained a number of commitments and European member states should already be working on fulfilling the agreed objectives of the European Food and Nutrition Action Plan 2015-2020.

Against that backdrop, as part of the Decade of Action on Nutrition, FAO and WHO are supporting countries to set country-specific SMART – specific, measurable, achievable, relevant and time-bound – commitments to track their progress in improving nutrition. In May 2017, Brazil and Ecuador became the first countries to make their nutrition commitments for the UN Decade of Action on Nutrition.[lviii] In a European context, EHN urges countries to set SMART commitments, appropriate to their national context, for implementing all of the specific recommendations presented in this section.

4.2.1.3 Develop an integrated health and environment approach to food systems and promote health-environment win-wins in food-based dietary guidelines

Global environmental change is both a driver and an outcome of food systems and associated health outcomes, as described in Chapter 3. There is potential for considerable overlap between consuming healthier diets and achieving higher levels of sustainability in terms of greenhouse gas emissions, land-use and water use. Food systems, therefore, need to be transformed to fulfil their role in promoting environmental sustainability, while also providing healthy diets. To be able to achieve this, further research and analysis will be required to enhance understanding of health-environment interactions and of what interventions are required to shift towards sustainable healthy diets. In the shorter term, governments should promote the health-environment changes that can be considered as 'win-wins' and that are already well understood in national food-based dietary guidelines.

4.2.2 EHN's recommendations – three clusters of specific recommendations

Complementary to the three overarching recommendations, three clusters of specific recommendations are proposed. These have been selected on the basis of a variety of specific criteria, including effectiveness, cost-effectiveness, affordability, impact, acceptability and practicality (See Annex 3 for further explanation).

There is a clear evidence base for the proposed recommendations (see Table 11). It is important to understand the challenges inherent in gathering evidence on effective policies for CVD prevention, and that much of the evidence on the relationships between diet and cardiovascular outcomes and on the effectiveness of action to prevent CVD is indirect rather than direct. It is important for policymakers to understand these challenges and to recognise that it

lviii See http://www.who.int/nutrition/decade-of-action/en/ for details of their commitments.

is not always necessary or desirable to wait for irrefutable conclusive evidence before deciding to implement an action. Government reluctance to take action without definitive proof of effectiveness from national initiatives elsewhere has been a serious impediment to health improvements. In other fields – such as economics – major policy decisions are taken on the basis of logical thinking about the processes involved and theoretical modelling of potential scenarios. So in order to take action sooner rather than later, policymakers need to scrutinise the best possible evidence available and use this as the basis for judgements, taken on the basis of clearly set out criteria and principles.

Given the complexities, there are several different types of evidence for effectiveness of public interventions to prevent CVD. These include:

- **Analogy:** It is possible to draw on evidence of interventions' effectiveness in other areas of public health or public policy (e.g. tobacco or alcohol taxation).
- **Observational data:** From epidemiological studies that do not involve carrying out interventions, but instead make comparisons between different populations or groups.
- **Experimental data:** From experimental studies and/or programme or policy evaluation.
- **Modelling:** Estimations of the effectiveness of an intervention on the basis of data on efficacy, programme uptake and reach.

See Annex 3 for further explanation.

The case for the specific key recommendations in the three clusters (supply-side, demand-side and food composition) is explored further in Tables 11 and 12 and in Annex 4. Table 11 sets out which sector of the population would be reached, a summary of the evidence, examples of national or local experience of similar measures and specific actions for particular actors. Annex 4 gives more detail on the different types of evidence that exist for each recommendation, including the different types of evidence set out in Annex 3, while Table 12 explores considerations of affordability, impact, practicality/feasibility and acceptability. To ensure maximum reach, and therefore impact, many of the recommendations require governments to take a legislative approach and introduce statutory regulation (See box below).

Government regulation or industry voluntary action?

Supermarket shelves full of foods that do not contain any industrially-produced trans fats and have lower levels of fat, sugar and salt. Children no longer exposed to marketing for unhealthy foods through broadcast, print or digital media. Consumers able to tell at a glance which menu choice is healthiest. These are just some of the outcomes that EHN is striving to achieve through the recommendations in this paper.

There are, however, different possible approaches to reach such outcomes. These, and some of the other recommendations in the paper, could be implemented through, for example, statutory government regulation or by voluntary or self-regulatory action by industry. Each approach has both advantages and disadvantages and the choice of approach for implementation depends on the precise problem to be addressed and the regulatory context in the country.

Voluntary or self-regulatory approaches can be relatively quick and inexpensive to develop, implement and enforce. In addition, they can be more easily modified as situations change and can be particularly pertinent where countries have little capacity to regulate cross-border issues. Self-regulatory or voluntary schemes, however, often fail to set sufficiently ambitious targets, have limited impact if they do not include all industry actors and may lack effective monitoring and compliance arrangements. Some essential criteria and proposed standards have been put forward for any self-regulatory scheme – these include setting of appropriate and ambitious targets, objectives and benchmarks, with sufficient scope for wide impact, along with independent monitoring, transparent reporting and oversight by a regulatory or health body.[53,54] WHO has noted that 'experience suggests that self-regulatory, voluntary approaches have loopholes' and recommends government leadership for setting the criteria for policy and for independent monitoring.[55] Moreover, there is an inherent conflict of interest for companies to engage in self-regulation when their primary responsibility is towards their shareholders to increase their profits.[56]

Statutory regulation, while it can take more time to develop and requires political commitment, is more likely to ensure full coverage and to establish a level playing field for all industry players. Specifically in relation to marketing of HFSS foods to children [55,57–59] and salt reduction,[30,60] for example, there is evidence to support statutory regulation as being most likely to be effective and/or cost effective. Legislative approaches, however, can be subject to opposition, including costly and time-consuming legal challenge in the courts. To be able to fend off such challenges it is important for policymakers to involve legal experts from the outset to draw up laws that are evidence-based, well-designed, appropriately targeted, non-discriminatory and proportionate to their objectives.[61]

In light of concerns about the limited effectiveness and impact of self-regulatory or voluntary action, EHN recommends a legislative approach in a number of areas – namely, a global food convention, marketing of unhealthy foods, front-of-pack nutrition labelling, limits for levels of industrially-produced trans fats, nutrition standards for food in public institutions and, where appropriate, widespread food reformulation.

Table 11 EHN recommendations: Evidence base for three clusters of specific recommendations

	EHN KEY SPECIFIC RECOMMENDATIONS	Target groups within the population	Evidence[lx]	Examples of implementation[lxi]	Who to do what
FOOD SUPPLY SIDE CLUSTER	Global food convention to establish global regulatory framework to protect healthy diets.	Everyone	Current regulatory arrangements are clearly inadequate. There is a strong case for a global convention to set a framework for national regulation, ensure that healthy diets are protected across borders and counteract trade agreements.	Global regulatory frameworks exist for infant formula (International Code of Marketing of Breast-milk Substitutes) and tobacco (Framework Convention on Tobacco Control).	• WHO, FAO, Codex, other UN bodies and their member states to negotiate a global convention. • National governments to advocate for a global convention. • Civil society to advocate for a global convention.
	Reform agriculture and food policy to align with public health policies and promote sustainable food production (including by radically reforming the CAP, measures to encourage shorter supply chains and improve the food supply through rural development and conservation).	Everyone	Modelling studies and natural experiments clearly demonstrate that design of agricultural policy (e.g. the CAP) has potential to change population food intakes. A systematic review of studies on agricultural policies that directly affect or could potentially direct food prices (in US, Netherlands and Egypt) found that such policies had an effect on weight and risk of diet-related NCDs.[62]	Brazil's *Fome Zero* (Zero Hunger) strategy included a Food Acquisition Programme. This requires public institutions to spend 30% of federal school feeding budgets on food from family farmers, creating new markets for local farmers, especially those selling fruit and vegetables. Changes to agricultural subsidies in Eastern European countries in the early 1990s changed the relative price of animal fats and vegetable oils, resulting in health benefits.[63]	• EU to review and consider radical reform of the CAP, encourage shorter supply chains and improve the food supply through rural development and conservation. • Non-EU countries to reform agricultural policy, encourage shorter supply chains and improve the food supply through rural development and conservation. • Policymakers in public health and in agriculture/food/environment policy to collaborate on developing effective policies for a sustainable healthy food supply.
	Ensure that trade and investment policies protect and promote public health.	Everyone	Evidence exists to demonstrate that trade and investment policies can impact on the food supply/environment and nutrition value of the food chain and can restrict 'policy space' for governments to take regulatory action.	There are rare examples of nutrition being taken into account in trade discussions. In Samoa, involvement of a nutritionist in the country's discussions on accession to the World Trade Organization (WTO) enabled a development of a strategy to mitigate potentially negative consequences for nutrition.[64] There are also examples of countries, sometimes from countries not bound by WTO rules, (some Pacific Island states, **Ghana**, **Mauritius** and **Iran**) using trade policy instruments to try to reduce imports of particular high fat or high sugar products or to improve their quality.[65,66]	• EU and governments of non-EU member states to mandate targeted health impact assessments as part of trade and investment policy processes, to ensure coherence between nutrition and trade policymaking. • EU and governments of non-EU member states negotiating trade deals to appoint a nutrition focal point to provide advice during trade policy negotiations. • Civil society to advocate for consideration of nutrition and health objectives in trade and investment policies.
FOOD COMPOSITION RELATED CLUSTER	Mandatory maximum levels of industrially-produced trans fatty acids (IPTFAs).	All consumers of affected products.	International experience shows that national 'bans' virtually remove IPTFAs from the food supply.[31,] National and local laws limiting trans fats have been followed, respectively, by a drop in death rates[67] and hospitalisations.[68] Saturated fat in products did not generally increase as trans fats decreased. Local 'bans' have removed trans fats from food outlets.[31] Some voluntary self-regulation has also reduced trans fat intakes.[31,69]	**Austria**, **Denmark**, **Hungary**, **Latvia, Iceland**, **Norway** and **Switzerland** have mandated the removal of IPTFAs in products. Lithuania has notified the European Commission that it intends to legislate.	• EU to implement EU-wide legislation. • National governments in EU member states to legislate pending EU regulation. • Non-EU member states to legislate. • Civil society to advocate for effective comprehensive legislation on use of industrially-produced trans fatty acids.
	Establish nutrition standards for food served or sold in schools, hospitals and other public institutions and issue guidelines for healthy public procurement.	People who eat in public institutions (childcare, schools, other educational establishments, hospitals, prisons, government offices and other publicly-funded facilities).	International evidence, mainly from schools, showing that healthy procurement can improve diet and health outcomes.[70–72] More specifically, there is evidence that school regulation – particularly restricting unhealthy foods – and procurement policies are effective.[24]	In 2008, **New York City** introduced mandatory nutrition standards for all food purchased or served in city entities, covering more than 3 000 sites and 260 million meals/snacks a year.[73] Globally, 17 countries have banned vending machines in schools; **Wales** has standards for vending machines in hospitals/ health facilities. **Bulgaria**, **Estonia**, **Finland**, **France**, **Hungary**, **Latvia**, **Lithuania**, **Poland**, **Romania**, **Sweden** and the **UK** have mandatory nutrition standards for school food. A technical report, produced as part of Malta's EU Presidency, is available as a tool to support member states in ensuring healthy procurement of food served in schools.[74]	• EU to support member states in their efforts to introduce healthy procurement. • National and/or sub-national governments or local authorities, as appropriate, to introduce mandatory nutrition standards for all public institutions. • National and/or sub-national governments to issue guidelines on healthy food procurement. • Civil society to advocate for healthier food in public institution.
	Wide-ranging, ambitious food reformulation programmes to reduce levels of salt, saturated fat, free sugars, energy density and portion size in processed foods.	All consumers of affected products – programmes should first target foods that are widely consumed and make the biggest contribution to diet.	Strong and extensive evidence that salt reformulation programmes can lead to lower salt levels in foods and lower sodium intake.[29] This experience should be transferable to other ingredients/ nutrients. Research suggests that a mandatory approach is best.[30]	Voluntary salt reduction programmes in place in **Austria, Belgium, Croatia, Czech Republic, France, Greece, Hungary, Ireland, Italy, Norway, Slovenia** and **Spain**. Mandatory limits on salt in some foods have been introduced, for example, in **Belgium**, **Bulgaria**, **Greece**, **Hungary, Netherlands** and **Portugal**. Voluntary reformulation programmes in **France**, **Netherlands**, **Switzerland** and the **UK** include other nutrients/ingredients (e.g. fats, sugars) and/or calories.	• European Commission to produce a more ambitious comprehensive framework. • All governments to implement national programmes for reformulation including, where appropriate, mandatory standards. • Civil society to advocate for wide-ranging, ambitious reformulation programmes.

lix See Annex 2 of the full-length version of this paper for more detail on the different types of evidence available for each recommendation.
lx This is not an exhaustive list of all policy implementation examples.

	EHN KEY SPECIFIC RECOMMENDATIONS	Target groups within the population	Evidence[lx]	Examples of implementation[lxi]	Who to do what
FOOD DEMAND SIDE CLUSTER	Use of taxes and/or subsidies to promote consumption of healthy foods and reduce consumption of unhealthy foods.	Consumers of taxed or subsidised products.	There is convincing evidence from country experience and extensive modelling that fiscal policies can promote healthier diets.[14,75] Currently, the evidence is strongest that taxes on sugar-sweetened beverages reduce consumption and raise revenue.[45,75–77] There is also evidence on the effectiveness of fruit and vegetable subsidies[14] and of other targeted taxes (e.g. on foods high in saturated fats, trans fats, free sugars and/or salt) on consumption and as a driver of reformulation.[14,75,78]	• **France**, **Spain**, **Estonia**, **Belgium**, **Hungary** and the **UK** have all introduced, or announced plans to introduce, taxes on sugar-sweetened beverages. • **Hungary**'s public health product tax also applies to other products including confectionery, salted snacks and ice cream.	• National governments to introduce a tax (equivalent to around 20% of price) on sugar-sweetened beverages. • National governments to introduce carefully modelled combination of taxes (e.g. on saturated fat) and/or subsidies (e.g. on fresh fruits and vegetables). • EU to respect member states' right to introduce tax/subsidy schemes (as long as they are not discriminatory). • Civil society to advocate for implementation of effective taxes and subsidies.
	Regulatory controls to reduce the public health impact of marketing (including advertising and other forms of commercial promotion across all media) of HFSS foods.	Children in the short term; Whole population would benefit from wider restrictions applying to the whole society if introduced.	Convincing research that exposure to HFSS food marketing influences what and how much children eat.[79–83] Measures to restrict marketing *can* reduce children's exposure, but overall effectiveness depends on what age group, foods, media channels and marketing techniques are covered.[84–87] Mandatory restrictions are more effective than voluntary or self-regulatory approaches.[57,59] The evidence base to assess the impact of HFSS marketing on adults is less well developed, but there is recent evidence that price promotions influence adults' food purchasing.[83]	• Measures in **Norway** and **Ireland** concern children up to the age of 18. • **Portugal**'s measures relate to audiences with 20% or more of children, compared to the more common 35% or 50% cut-off. • **UK** rules have been extended to cover digital marketing and brand marketing that, in effect, promotes HFSS foods. In other regions, far-reaching restrictions in **Brazil** and **Chile** cover all media (including internet and apps) and a wide range of marketing techniques. • **Ireland** has limited HFSS broadcasting advertising for the whole population (i.e. not specifically targeted at children) to 25% of total advertising time or one in four adverts.	• EU, through the EU *Audiovisual Media Services Directive*, to implement a 6am – 11pm watershed, during which HFSS food marketing cannot be shown. • All governments to introduce national mandatory restrictions to prevent exposure of children (up to 18) to advertising and other forms of commercial promotion of HFSS foods across *all* media. • Civil society to advocate for effective restrictions on HFSS marketing to children. • Public health policymakers and health advocates to develop the evidence base on the impact of HFSS marketing on population intakes (including adults). • The European Union and non-EU member states should adopt the *WHO European Region Nutrient Profile Model* to define HFSS foods.
	Adoption of nutrient profiles in the context of the EU regulation of health and nutrition claims; and of mandatory simplified front-of-pack nutrition labelling and menu labelling	All consumers of pre-packaged foods and those who eat out.	Clear evidence that consumers find front-of-pack labels more noticeable and easier to interpret.[88–91] There is also evidence that front-of-pack nutrition labelling drives product reformulation.[24,26,27,92] Some evidence that menu calorie labelling reduces the calorie content of meals selected,[87] but the evidence to date from real-life settings remains mixed.[57,59]	*Front-of-pack*: The **UK** government published a recommended front of pack nutrition labelling scheme using green, amber and red 'traffic lights' in 2013. **France** intends to implement voluntary front-of-pack labelling with the Nutri-Score symbol. A few countries – including some **Nordic** countries, **Finland** and **Netherlands** – use symbols to indicate a healthier option and these are generally thought to be helpful.[33,34] *Menu labelling*: Many states and cities in the **US** introduced menu-labelling requirements and the Affordable Care Act introduced national requirements for some types of restaurant chain and vending machines. Other examples include **South Korea**, **Taiwan**, **Singapore** and some territories in **Australia**.	• WHO to recommend a unified global scheme. • European Commission to set nutrient profiles to underpin nutrition and health claims as required by the EC regulation on nutrition and health claims (EC) No 1924/2006); EHN recommends a model which favours the 'best in class' products. • EU to adopt mandatory EU-wide simplified front-of-pack nutritional labelling. EHN continues to recommend a scheme which uses traffic light colours to indicate high, medium and low levels of fat, saturated fat, salt and (preferably added) sugar. EHN will follow developments in evidence for the most effective front of pack nutrition labelling scheme. • National governments must be able to recommend/endorse meaningful front of pack symbols in their countries in addition to any harmonised EU rules on front of pack nutrition labelling • Non-EU member states to legislate for mandatory front-of-pack nutrition labelling. • National governments are encouraged to adopt mandatory requirements to provide easy-to-understand information about the nutritional quality of foods served in chains with more than 10 outlets (menu labelling). • Civil society to advocate for clear front-of-pack and menu labelling.

Table 12 EHN proposed policy priorities: Considerations of affordability, impact, practicality/feasibility and acceptability

Action	Affordability	Impact	Practicality/feasibility	Acceptability
Global food convention to establish global regulatory framework to protect healthy diets.	Not assessed.	Enormous potential impact because a convention would establish a legal framework essentially incorporating many of the other recommendations and ensuring their widespread adoption and enforcement, as well as establishing international rules for industry best practice.	Highly feasible, although dependent on international political will.	Growing support for a global convention, given the increasingly global nature of the food supply, the important influence of commercial determinants, and the public health imperative.
Reform agriculture and food policy to align with public health policies and promote sustainable food production (including by radically reforming (or eliminating) the CAP, measures to encourage shorter supply chains and improving the food supply through rural development and conservation).	Will depend on the exact nature of reforms.	Similarly, will depend on the exact nature of the reforms.	Feasible, but will depend on political will throughout the EU.	Public acceptability of reforms is likely to be high, if public health and sustainability objectives are aligned and supported by clear communication.
Ensure that trade and investment policies protect and promote public health.	Not possible to estimate – will depend on the trade and investment policies under negotiation.		Feasible, but will require high-level political support if public health advice is to be listened to seriously as part of trade negotiations.	Public acceptability for protecting public health during trade negotiations likely to be very high.
Mandatory maximum levels of industrially-produced trans fatty acids.	WHO considers legislation to eliminate industrial trans fat costs 0.02 dollars per capita, with an average cost-effectiveness ratio of between 1 000 and 5 000 dollars per DALY averted in high-income countries.[5]	It is estimated that development of legislation to eliminate industrial trans fat could save 21 DALYs per million population annually in higher income countries and 30 in lower income countries. [5]	WHO considers to be 'highly feasible'.[93]	Consumer and industry response from country experience demonstrates that such restrictions are acceptable.
Establish nutrition standards for food served or sold in schools, hospitals and other public institutions and issue guidelines for healthy public procurement.	Costs and, therefore, affordability will depend on the context and funding mechanisms. Strategies to minimise the investment needed include harnessing the collective bargaining/ buying power of public sector institutions and rigorous oversight of contract caterers' charges.[94–96]	No estimates of impact. The potential scale is significant – in the New York City case, for example, the standards cover 260 million meals and snacks per year.[94]	Highly feasible for governments to introduce standards and procurement rules. Inspection and enforcement mechanisms are likely to already exist. Some training for catering and procurement staff, however, is likely to be necessary.	High level of acceptability likely for nutrition standards for all school food (including, increasingly, standards for vending machines, etc.) and hospital food. Advocacy may be needed to build greater support for standards across all public food. New York City experience suggests public acceptability is not a barrier.
Wide-ranging, ambitious food reformulation programmes to reduce levels of salt, saturated fat, free sugars, energy density and portion size in processed foods.	WHO considers salt reduction by reformulation would cost less than 0.01 million dollars per million population to implement. With an average cost-effectiveness ratio of less than 100 dollars per DALY averted, it is included in the updated 2017 core set of 'best buys' for NCDs.[5]	WHO estimates that an effective voluntary salt reformulation process would avert loss of 3 315 DALYs per million population per year. A mandatory approach would potentially have wider reach – covering a broader range of products with more ambitious targets – and thus greater impact.	WHO considers this initiative to be 'highly feasible'.[93]	High level of public acceptance, particularly if reformulation is gradual so that changes to product are not perceived. In general, highly acceptable to policymakers and food industry.
Use of taxes and/or subsidies to promote consumption of healthy foods and reduce consumption of unhealthy foods.	WHO included taxation on sugar-sweetened beverages as one of its 'best buys' for NCDs in 2017.[5]. Implementation of subsidies to increase fruit and vegetable intakes is also a 'best buy'.	WHO analyses estimate that reducing sugar consumption through taxation on sugar sweetened beverages would avert 1 147 DALYs per million population each year.[5]	WHO considers to be 'highly feasible'.[93]	Recent country experience shows that public support for taxes for health purposes is possible.[97,98,14] Political support for taxes is aided by the potential for generating revenue. [99] Nonetheless, clear communication and advocacy emphasising the health bonus is important to build and maintain support.[14]
Regulatory controls to reduce exposure to marketing (including advertising and other forms of commercial promotion across all media) of foods high in fat, sugar or salt.	Implementation of the *WHO Set of Recommendations on marketing of foods and non-alcoholic beverages* is a WHO 'best buy' and is estimated to be 'very low cost'.[93]	Modelling of impact in the UK, for example, estimated 401 000 DALYs would be saved by introducing media restrictions on all high-calorie food advertising on all supports.[100]	WHO considers this to be 'highly feasible'.[93] Measures do need, however, to be carefully designed to ensure full coverage of all media, the target group, all relevant products and cross-border marketing.	Regulatory controls on marketing to children are increasingly common, suggesting a high level of acceptability. Self-regulatory approaches (albeit not as effective as mandatory approaches) also demonstrate industry recognition of need for restrictions.
Adoption of nutrient profiles in the context of the EU regulation of health and nutrition claims; and of mandatory simplified front-of-pack nutrition labelling, and menu labelling	Nutrition labelling more generally, not specifically front of pack (FOP), is a WHO 'best buy' and estimates suggest that it would be low cost to implement and cost-saving in the long term.[46,101]	UK modelling estimated mandated nutrition labelling on packaging and in fast food restaurants would save 575 000 DALYs across UK.[100]	Given that at least 75% of the world's population is already covered by mandatory back-of-pack nutrition information, it is highly feasible to mandate front-of-pack nutrition information. Experience on the ground also shows that mandating menu labelling is very feasible.	High level of acceptability – consumers have shown preference for summary nutrition labelling[89,102] and many authoritative bodies have called for mandatory FOP labelling and menu labelling.[103–106]

References

1. UN General Assembly. Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases. A/RES/66/2. *UN New York* 49777, 1–13 (2012).
2. World Health Organization. *Global action plan for the prevention and control of noncommunicable diseases 2013-2020*. (2013).
3. World Health Organization. *Noncommunicable Diseases Global Monitoring Framework: Indicator Definitions and Specifications*. (2014).
4. World Health Organization. *Global status report on noncommunicable diseases 2014*. (2014).
5. World Health Organization. *Preparation for the third High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases, to be held in 2018. Report by the Director General*. (2017).
6. FAO/WHO. *Second International Conference on Nutrition: Report of the Joint FAO/WHO Secretariat on the Conference, Rome 2015*. (2015).
7. Food and Agriculture Organisation of the United Nations. Rome Declaration of Nutrition. *Second Int. Conf. Nutr.* 19–21 (2014). doi:10.1044/leader.PPL.19102014.18
8. FAO & WHO. Framework for action. Outcome document of the Second International Conference on Nutrition. (2014).
9. World Obesity Federation. *World Obesity Statement to the 68th World Health Assembly on Agenda item 13.1 – Outcome of the Second International Conference on Nutrition*. (2016).
10. World Health Organization. *Double duty actions for nutrition: Policy Brief*. (2017).
11. UN. *Transforming our world: the 2030 agenda for sustainable development*. (2016).
12. UNSCN. *Nutrition Targets and Indicators for the Post-2015 Sustainable Development Goals, Accountability for the Measurement of Results in Nutrition, A Technical Note*. (2015).
13. World Health Organization. *Report of the Commission on Ending Childhood Obesity*. (World Health Organization, 2016).
14. World Health Organization. *Fiscal policies for diet and prevention of noncommunicable diseases: technical meeting report, 5-6 May 2015, Geneva, Switzerland*. (WHO, 2016).
15. European Commission. *Report from the Commission to the European Parliament and the Council regarding trans fats in foods and the overall diet of the Union population. SWD (2015) 268 final*. (2015).
16. European Heart Network. *Diet, physical activity and cardiovascular disease prevention in Europe*. (2011).
17. European Heart Network. *Trans fatty acids and heart disease. Position paper*. (2016).
18. Jensen, J. & Smed, S. *The Danish tax on saturated fat - Short run effects on consumption, substitution patterns and consumer prices of fats*. (2014).
19. Smed, S., Scarborough, P., Rayner, M. & Jensen, J. D. The effects of the Danish saturated fat tax on food and nutrient intake and modelled health outcomes: an econometric and comparative risk assessment evaluation. *Eur. J. Clin. Nutr.* 1–6 (2016).
20. World Health Organisation Regional Office for Europe. Using price policies to promote healthier diets. 50 (2015).
21. Berardi, N., Sevestre, P., Tepaut, M. & Vigneron, A. *The impact of a soda tax on prices. Evidence from French micro data*. (2016).
22. Hungarian National Institute for Health and Development. *The public health impact assessment of taxes on products*. (2013).
23. Colchero, M. A., Popkin, B. M., Rivera, J. A. & Ng, S. W. Beverage purchases from stores in Mexico under the excise tax on sugar sweetened beverages: observational study. *BMJ (British Med. Journal)* h6704 (2016).
24. Afshin, A. *et al*. CVD Prevention Through Policy: a Review of Mass Media, Food/Menu Labeling, Taxation/Subsidies, Built Environment, School Procurement, Worksite Wellness, and Marketing Standards to Improve Diet. *Curr. Cardiol. Rep*. 17, (2015).
25. Vyth, E. L., Steenhuis, I. H., Roodenburg, A. J., Brug, J. & Seidell, J. C. Front-of-pack nutrition label stimulates healthier product development: a quantitative analysis. *Int. J. Behav. Nutr. Phys. Act*. 7, 65 (2010).
26. Young, L. & Swinburn, B. Impact of the Pick the Tick food information programme on the salt content of food in New Zealand. *Health Promot. Int*. 17, 13–19 (2002).
27. Eckel, R. H., Borra, S., Lichtenstein, A. H. & Yin-Piazza, S. Y. Understanding the complexity of trans fatty acid reduction in the American diet: American Heart Association Trans Fat Conference 2006: report of the Trans Fat Conference Planning Group. *Circulation* 115, 2231–2246 (2007).
28. Crosetto, P., Lacroix, A., Muller, L. & Ruffieux, B. Modification des achats alimentaires en réponse à cinq logos nutritionnels. *Cah. Nutr. Diététique* 827, 1 YP-57 (2017).
29. Trieu, K. *et al*. Salt Reduction Initiatives around the World - A Systematic Review of Progress towards the Global Target. *PLoS One* 10, e0130247 (2015).
30. *Effectiveness of food reformulation as a strategy to improve population health*. (National Heart Foundation of Australia, 2012).
31. Downs, S. M., Thow, A. M. & Leeder, S. R. The effectiveness of policies for reducing dietary trans fat: a systematic review of the evidence. *Bull. World Health Organ*. 91, 262–9 (2013).
32. Hawkes, C., Jewell, J. & Allen, K. A food policy package for healthy diets and the prevention of obesity and diet-related non-communicable diseases: the NOURISHING framework. *Obes. Rev*. 12, 159–168 (2013).
33. Raulio, S., Tapanainen, H., Toivola, L. & Sm, V. Potential Effects of Heart Symbol Compliant Foods on Nutrient Intake. *J Nutr. Heal. Food Sci* 5, 1–8 (2017).
34. Roodenburg, A. J. C., Temme, E. H. M., Davies, O. H. & Seidell, J. C. Potential impact of the Choices Programme on nutrient intakes in the Dutch population. *Nutr. Bull*. 34, 318–323 (2009).
35. World Health Organization. *International Code of Marketing of Breast-Milk Substitutes*. (1981).
36. WHO, Unicef & IBFAN. *Marketing of Breast-milk Substitutes: National Implementation of the International Code: Status Report 2016*. (2016).
37. World Health Organization (WHO). Guidance on ending the inappropriate promotion of foods for infants and young children (A69/7 Add.1) (2016).
38. World Health Organization. *Comprehensive Implementation Plan on Maternal, Infant and Young Child Nutrition*. (2014).
39. Marmot, M. Closing the gap in a generation. *Heal. Equity Through Action Soc. Determ. Heal*. 246 (2008). doi:10.1080/17441692.2010.514617
40. Dabla-Norris, E., Kochhar, K., Ricka, F., Suphaphiphat, N. & Tsounta, E. *Causes and Consequences of Income Inequality: A Global Perspective*. (2015).
41. Ostry, J. D., Berg, A. & Tsangarides, C. G. *Redistribution, Inequality, and Growth. IMF Discussion Paper*. (2014).
42. Picketty, T. *Capital in the twenty-first century*. (Harvard University Press, 2014).
43. World Economic Forum. *Outlook on the global agenda*. (2015).
44. Wiggins, S. *et al*. The rising cost of a healthy diet: changing relative prices of foods in high-income and emerging economies. *Overseas Dev. Inst. Report. May* 67, (2015).
45. Thow, A. M., Downs, S. & Jan, S. A systematic review of the effectiveness of food taxes and subsidies to improve diets: Understanding the recent evidence. *Nutr. Rev*. 72, 551–565 (2014).
46. Sassi, F. *Obesity and the Economics of Prevention - Fit not Fat. OECD* 2010, (2010).
47. WHO. Fiscal policies for diet and the prevention of noncommunicable diseases. *WHO* 36 (2016).
48. WHO Regional Office for Europe. *European Food and Nutrition Action Plan 2015-2020*. (2015).
49. Engesveen, K., Nishida, C., Prudhon, C. & Shrimpton, R. Assessing countries' commitment to accelerate nutrition action demonstrated in PRSPs, UNDAFs and through nutrition governance. *SCN News* 37, 10–17 (2009).
50. WHO Department of Nutrition for Health and Development. *WHO Global Database on National Nutrition Policies and Programmes*.

51. Gurinovic, M. *et al.* Improving nutrition surveillance and public health research in Central and Eastern Europe/Balkan Countries using the Balkan Food Platform and dietary tools. *Food Chem.* 193, 173–180 (2016).
52. Brown, K. A. *et al.* Concepts and procedures for mapping food and health research infrastructure: New insights from the EuroDISH project. *Trends Food Sci. Technol.* 63, 113–131 (2017).
53. Buse, K., Tanaka, S. & Hawkes, S. Healthy people and healthy profits? Elaborating a conceptual framework for governing the commercial determinants of non-communicable diseases and identifying options for reducing risk exposure. *Global. Health* 1–12 (2017).
54. Sharma, L. L., Teret, S. P. & Brownell, K. D. The Food Industry and Self-Regulation : Standards to Promote Success and to Avoid Public Health Failures. *Am J Public Heal.* 100, 240–246 (2010).
55. WHO Regional Office for Europe. European food and nutrition action plan 2015 – 2020. *Eur* 24 (2014).
56. Bartlett, O. & Garde, A. Time to Seize the (Red) Bull by the Horns: The European Union's Failure to Protect Children from Alcohol and Unhealthy Food Marketing. *Eur. Law Rev.* 4, (2013).
57. Chambers, S. A., Freeman, R., Anderson, A. S. & MacGillivray, S. Reducing the volume, exposure and negative impacts of advertising for foods high in fat, sugar and salt to children: A systematic review of the evidence from statutory and self-regulatory actions and educational measures. *Prev. Med. (Baltim).* 75, 32–43 (2015).
58. Who. A framework for implementing the set of recommendations on the marketing of foods and non-alcohol beverages to children. 1–61 (2012).
59. Galbraith-Emami, S. & Lobstein, T. The impact of initiatives to limit the advertising of food and beverage products to children: a systematic review. *Obes. Rev.* 14, 960–974 (2013).
60. Cobiac, L. J. *et al.* Which interventions offer best value for money in primary prevention of cardiovascular disease? *PLoS One* 7, (2012).
61. World Health Organization. *Key considerations for the use of law to prevent noncommunicable diseases in the WHO European Region.* (2017).
62. Dangour, A. D. *et al.* Can nutrition be promoted through agriculture-led food price policies? A systematic review. *BMJ Open* 3, e002937- (2013).
63. Zatonski, W., Campos, H. & Willett, W. Rapid declines in coronary heart disease mortality in Eastern Europe are associated with increased consumption of oils rich in alpha-linolenic acid. *Eur. J. Epidemiol.* 23, 3–10 (2008).
64. Thow, A. M. & McGrady, B. Protecting policy space for public health nutrition in an era of international investment agreements. *Bull. World Health Organ.* 92, 139–45 (2014).
65. Thow, A. M., Annan, R., Mensah, L. & Chowdhury, S. N. Development, implementation and outcome of standards to restrict fatty meat in the food supply and prevent NCDs: learning from an innovative trade/food policy in Ghana. *BMC Public Health* 14, 249 (2014).
66. Thow, A. M. *et al.* Trade and food policy: Case studies from three Pacific Island countries. *Food Policy* 35, 556–564 (2010).
67. Restrepo, B. J. & Rieger, M. Denmark's Policy on Artificial Trans Fat and Cardiovascular Disease. *Am. J. Prev. Med.* 50, 69–76 (2016).
68. Brandt, E., Myerson, R., Perraillon, M. & Polonsky, T. Hospital admissions for myocardial infarction and stroke before and after the trans-fatty acid restrictions in new york. *JAMA Cardiol.* 2, 627–634 (2017).
69. Temme, E. H. M., Millenaar, I. L., van Donkersgoed, G. & Westenbrink, S. Impact of fatty acid food reformulations on intake of Dutch young adults. *Acta Cardiol.* 66, 721–728 (2011).
70. Niebylski, M. L. *et al.* Healthy food procurement policies and their impact. *Int. J. Environ. Res. Public Health* 11, 2608–2627 (2014).
71. Chriqui, J. F., Pickel, M. & Story, M. Influence of school competitive food and beverage policies on obesity, consumption, and availability: a systematic review. *JAMA Pediatr.* 168, 279–286 (2014).
72. *Power Up for Health. mpact of healthy food procurement policiesprograms and nutrition standards on sales intake and availability of healthier food and body weight status overview. Evidence synthesis 10.* (2014).
73. Noonan, K., Sell, K., Miller, D. & Rubin, D. Government Purchasing to Improve Public Health : Theory Practice and Evidence Government Purchasing to Improve Public Health : Theory Practice and Evidence By : (2013).
74. Caldeira, S. *et al. Public Procurement of Food for Health: Technical report on the school setting.* (2017).
75. World Health Organisation Regional Office for Europe. *Using price policies to promote healthier diets.* (2015).
76. Powell, L. M., Chriqui, J. F., Khan, T., Wada, R. & Chaloupka, F. J. Assessing the potential effectiveness of food and beverage taxes and subsidies for improving public health: a systematic review of prices, demand and body weight outcomes. *Obes. Rev.* 14, 110–128 (2013).
77. Mozaffarian, D. *et al.* Population approaches to improve diet, physical activity, and smoking habits: a scientific statement from the American Heart Association. *Circulation* 126, 1514–1563 (2012).
78. Biro, A. Did the junk food tax make the Hungarians eat healthier? *Food Policy* 54, 107–115 (2015).
79. Boyland, E. J. *et al.* Advertising as a cue to consume: a systematic review and meta-analysis of the effects of acute exposure to unhealthy food and nonalcoholic beverage advertising on intake in children and adults. *Am. J. Clin. Nutr.* 103, 519–533 (2016).
80. Osei-Assibey, G. *et al.* The influence of the food environment on overweight and obesity in young children: a systematic review. *BMJ Open* 2, (2012).
81. Harris, J. L., Speers, S. E., Schwartz, M. B. & Brownell, K. D. US Food Company Branded Advergames on the Internet: Children's exposure and effects on snack consumption. *J. Child. Media* 6, 51–68 (2012).
82. Scully, M. *et al.* Association between food marketing exposure and adolescents' food choices and eating behaviors. *Appetite* 58, 1–5 (2012).
83. Public Health England. Sugar Reduction : The evidence for action Annexe 4 : An analysis of the role of price promotions on the household purchases of food and drinks high in sugar About Public Health England. 1–43 (2015).
84. Smithers, L. G., Lynch, J. W. & Merlin, T. Industry self-regulation and TV advertising of foods to Australian children. *J. Paediatr. Child Health* 50, 386–392 (2014).
85. Ofcom. *HFSS advertising restrictions: Final Review.* (2010).
86. Scarborough, P. *et al.* How important is the choice of the nutrient profile model used to regulate broadcast advertising of foods to children? A comparison using a targeted data set. *European Journal of Clinical Nutrition* 67, 815–820 (2013).
87. Brinsden, H. & Lobstein, T. Comparison of nutrient profiling schemes for restricting the marketing of food and drink to children. *Pediatr. Obes.* 8, 325–337 (2013).
88. Mandle, J., Tugendhaft, A., Michalow, J. & Hofman, K. Nutrition labelling: a review of research on consumer and industry response in the global South. *Glob. Health Action* 8, 25912 (2015).
89. Hersey, J. C., Wohlgenant, K. C., Arsenault, J. E., Kosa, K. M. & Muth, M. K. Effects of front-of-package and shelf nutrition labeling systems on consumers. *Nutr. Rev.* 71, 1–14 (2013).
90. Hawley, K. L. *et al.* The science on front-of-package food labels. *Public Health Nutr.* 16, 430–439 (2013).
91. Tymms, S. *Design, format and impact of front-of-pack nutrition labelling: an independent review of refereed literature.* 619, (2011).
92. Vyth, E. L., Steenhuis, I. H., Roodenburg, A. J., Brug, J. & Seidell, J. C. Front-of-pack nutrition label stimulates healthier product development: a quantitative analysis. *Int. J. Behav. Nutr. Phys. Act.* 7, 65 (2010).
93. World Health Organization. *Global status report on noncommunicable diseases. (WHO; Geneva)* (2010).
94. New York Academy of Medicine. *Policy Brief : Healthy Food Procurement Policy for New York State.* (2012).

95. Freudenberg, N. Healthy-food procurement: Using the public plate to reduce food insecurity and diet-related diseases. *Lancet Diabetes Endocrinol.* 4, 383–384 (2016).

96. De Schutter, O. The Power of Procurement. 28 (2014). doi:10.1126/science.1255182

97. Julia, C., Mejean, C., Vicari, F., Peneau, S. & Hercberg, S. Public perception and characteristics related to acceptance of the sugar-sweetened beverage taxation launched in France in 2012. *Public Health Nutr.* 18, 2679–2688 (2015).

98. PAHO. *Taxes on Sugar-sweetened Beverages as a Public Health Strategy: The Experience of Mexico.* (2015).

99. Julia, C., Mejean, C., Vicari, F., Peneau, S. & Hercberg, S. Public perception and characteristics related to acceptance of the sugar-sweetened beverage taxation launched in France in 2012. *Public Health Nutr.* 18, 2679–2688 (2015).

100. Dobbs, R. *et al.* Overcoming obesity : An initial economic analysis. *McKinsey Glob. Inst.* 120 (2014).

101. Cecchini, M. *et al.* Tackling of unhealthy diets, physical inactivity, and obesity: Health effects and cost-effectiveness. *Lancet* 376, 1775–1784 (2010).

102. Mandle, J., Tugendhaft, A., Michalow, J. & Hofman, K. Nutrition labelling: A review of research on consumer and industry response in the global South. *Glob. Health Action* 8, 1–10 (2015).

103. WHO. Report of the Commission on Ending Childhood Obesity. *World Heal. Organ. -WHO* (2015). doi:ISBN 978 92 4 151006 6

104. Glickman, D., Parker, L., Sim, L. J., Cook, H. D. V. & Miller, E. A. *Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation. American Journal of Lifestyle Medicine* 6, (Committee on Accelerating Progress in Obesity Food and Nutrition Board; Institute of Medicine, 2012).

105. Piepoli, M. F. *et al.* 2016 European Guidelines on cardiovascular disease prevention in clinical practice. *Eur. Heart J.* 28, 2375–414 (2016).

106. Wartella, E. A., Lichtenstein, A. H., Yaktine, A., Nathan, R. & Sciences, N. A. of. *Front-of-Package Nutrition Rating Systems and Symbols: A Consumer Health Literacy Perspective. Promoting Healthier and Symbols.* (2011).

5 Conclusions

Despite progress in tackling cardiovascular disease (CVD), it remains the leading cause of death and a major cause of illness and disability for men and women in Europe. Dietary risks are responsible for around half of the death and disability caused by CVD at an estimated cost of €102 billion in the EU alone.

Since EHN's last paper on *Diet, Physical Activity and Cardiovascular Disease Prevention in Europe* was published in 2011 there have been multiple and major developments in both the scientific arena and the policy landscape.

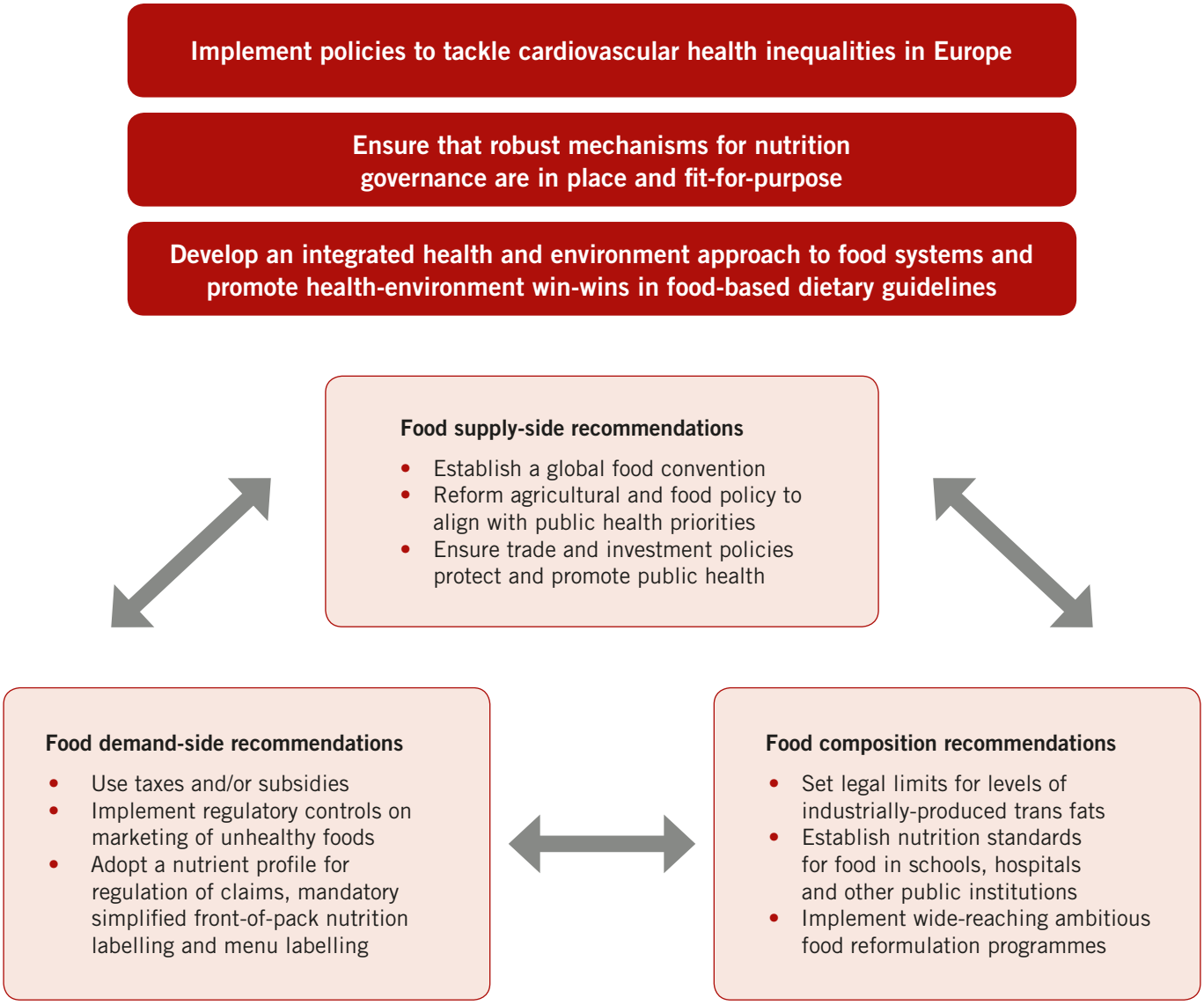
A review of the recent scientific developments and an exploration of some of the recent media reports of 'controversies' on diet and health, reveals that, in general, the evidence on the links between nutrients and CVD has strengthened, rather than weakened, in the last few years. A set of population goals (Tables 4 and 5), revised to take the new evidence into account, provide clear pointers towards a cardiovascular health-promoting diet for Europe. Taken together, these population goals should translate to a diet that has a low energy density, which is important in weight maintenance, and for the prevention of overweight and obesity. A cardiovascular health-promoting diet includes vegetables, fruit and berries in abundance. Whole grain products, nuts and seeds, fish, pulses and low-fat dairy products are important, as are modest amounts of non-tropical vegetable oils. This dietary pattern limits consumption of red meat, processed meat products, and foods or drinks with low content of vitamins, minerals and dietary fibre and/or with a high content of free sugars, saturated/trans fats or salt. A diverse and balanced diet covers the needs for nutrients – food supplements are, therefore, only needed in special cases. It now falls to national authorities to translate these evidence-based population goals into clear food-based dietary guidelines appropriate to the specific context and prevailing dietary patterns.

In a perfect world people would, on the basis of this evidence, change their food choices to reflect the latest advice and markets would respond to the changing demand. In today's complex food systems, however, the 'market' does not function perfectly and there are many other forces – often powerful – driving the food supply in addition to consumer demand. These include the major economic and policy drivers, which determine what food is produced and imported into Europe, along with how foods are marketed. Many of the global and external drivers are well beyond the unilateral control of any particular national or local government. It is also clear, however, that this complex picture presents policymakers – at international, national or local levels – with many different entry points for action within the food system.

In the six years since EHN's last paper was published, international recognition of the need for governments to play an important role in improving the food supply and food environments has continued to grow. While some countries have acted on some of the policy recommendations, by adopting binding or voluntary measures, a great deal more progress is urgently needed to implement effective European and national food and drink policies for preventing diet-related CVD.

To that end, three overarching recommendations and three clusters of specific recommendations are proposed:

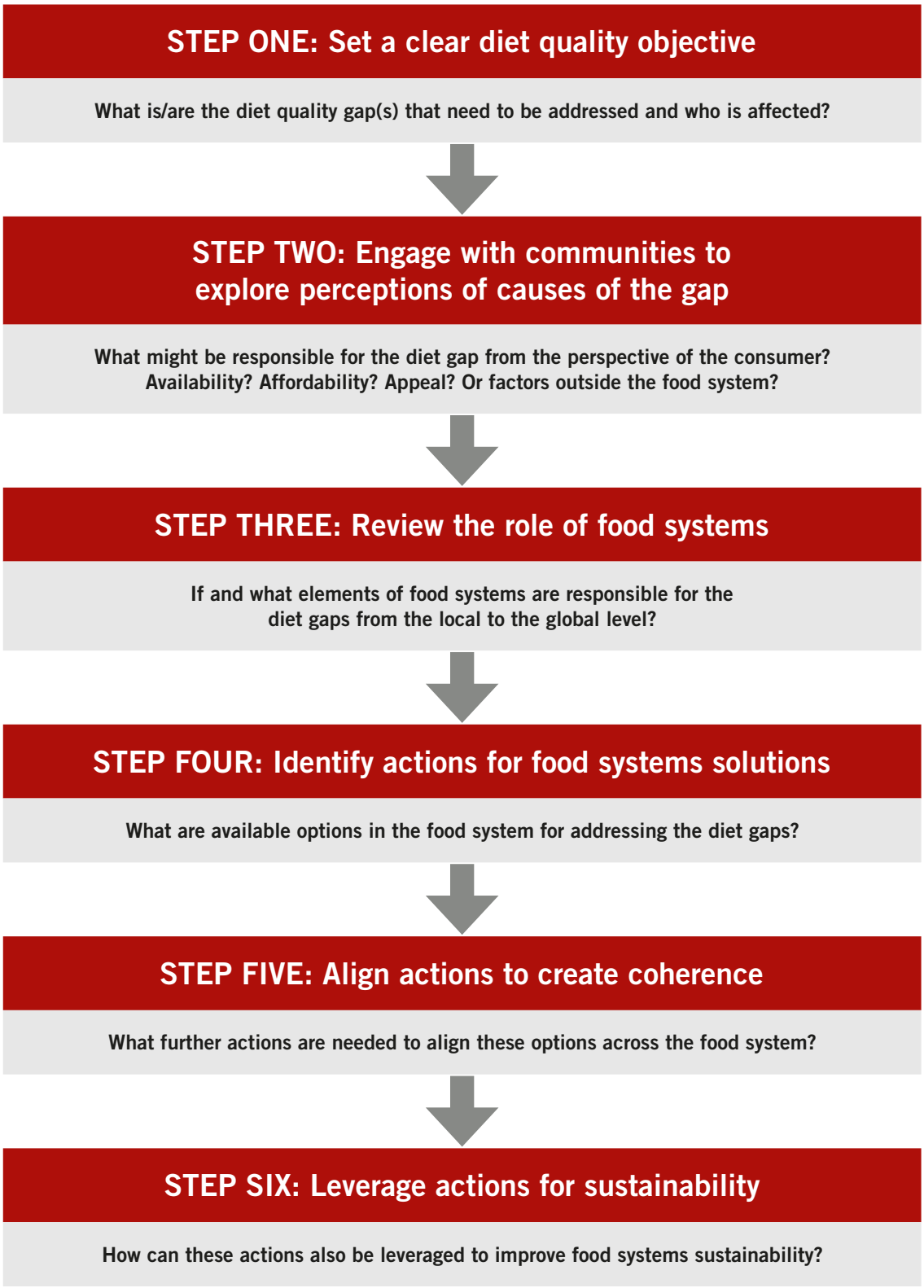
EHN's recommendations: Overarching recommendations and specific policy recommendation clusters for food and nutrition policies for cardiovascular health



This paper has set out the case for each recommendation and, for the specific recommendations, a summary of the different types of evidence is presented, along with considerations of affordability, impact, practicality/feasibility and acceptability.

EHN calls for the rapid and full implementation of this package of recommendations in order to realise the vision of every European – irrespective of the place or socio-economic circumstances into which they are born – being able to live free from avoidable diet-related cardiovascular disease, and thus able to have a productive working life and many years of active retirement free from cardiovascular ill-health or disability.

Annex 1: Six steps to identify policy options to achieve healthy diets



Source: Global Panel on Agriculture and Food Systems for Nutrition (2016). Food systems and diets: Facing the challenges of the 21st Century.

Annex 2: Water footprint of some selected food products from vegetable and animal origin

Table 13. The water footprint of some selected food products from vegetable and animal origin Source: Mekonnen and Hoekstra (2012)[1]

Food item	Water footprint per ton (m/3ton)				Nutritional content			Water footprint per unit of nutritional value		
	Green	Blue	Grey	Total	Calorie (kcal/kg)	Protein (g/kg)	Fat (g/kg)	Calorie (litre/kcal)	Protein (litre/g protein)	Fat (litre/g fat)
Sugar crops	130	52	15	197	285	0.0	0.0	0.69	0.00	0.0
Vegetables	194	43	85	322	240	12	2.1	1.34	26	154
Starchy roots	327	16	43	387	827	13	1.7	0.47	31	226
Fruits	726	147	89	962	460	5.3	2.8	2.09	180	348
Cereals	1.232	228	184	1.644	3.208	80	15	0.51	21	112
Oil crops	2.023	220	121	2.364	2.908	146	209	0.81	16	11
Pulses	3,180	141	734	4.055	3.412	215	23	1.19	19	180
Nuts	7.016	1367	680	9.063	2,500	65	193	3.63	139	47
Milk	863	86	72	1,020	560	33	31	1.82	31	33
Eggs	2.592	244	429	3.265	1.425	111	100	2.29	29	33
Chicken meat	3.545	313	467	4.325	1,440	127	100	3.00	34	43
Butter	4.695	465	393	5.553	7.692	0.0	872	0.72	0.0	6.4
Pig meat	4.907	459	622	5.988	2.786	105	259	2.15	57	23
Sheep/goat meat	8.253	457	53	8.763	2.059	139	163	4.25	63	54
Beef	14.414	550	451	15.415	1.513	138	101	10.19	112	153

Annex 3: Identifying and defining effective policies

To be able to choose effective policy options to improve European diets – and to be able to convince policymakers to take action and the public that such action is warranted – evidence is needed. Gathering evidence on effective policies for cardiovascular disease (CVD) prevention, however, can be challenging. By its very nature, evidence that an action prevents a condition from developing is more difficult to obtain than evidence that treatment improves an existing condition. Randomised controlled trials (RCTs), which are the gold standard for evaluating clinical interventions, are not always possible or necessarily most appropriate for assessing what works in population-based prevention of multi-factorial conditions like heart disease or stroke.

The number of studies on specific interventions for diet-related cardiovascular risk is limited for a number of reasons. First, most interventions have broader goals of improving health outcomes more generally (rather than just cardiovascular outcomes) and they often combine several different actions into a multi-component approach. Secondly, a wide array of societal changes and other inter-related factors can affect diet and disease patterns over time, and short-term simple models are unable to capture all these elements. Unlike for communicable diseases, models of disease prevention for CVD involve predicting outcomes that have a degree of uncertainty because they (a) relate to conditions with highly complex causal webs and (b) depend on predictions of human and organisational behaviour.

Clearly, decisions to take action are much easier when there is a clear evidence base and, ideally, this should be compelling evidence for action. In reality, however, much of the evidence on the relationships between diet and cardiovascular outcomes and on the effectiveness of action to prevent CVD is indirect rather than direct. It is important for policymakers to understand these challenges and to recognise that it is not always necessary – or even desirable – to wait for conclusive evidence before deciding to implement an action. In order to take action sooner rather than later, policymakers need to scrutinise the best possible evidence available and use this as the basis for judgements, taken on the basis of clearly set out criteria and principles.

How do we define what works in public health?

There are various different criteria to consider when assessing what works for cardiovascular disease prevention:

- **Effectiveness:** Best available scientific evidence can be used to estimate an 'effect size' and there should be a clear link to the dietary population goals for preventing CVD (see Chapter 2). Where appropriate and possible, meta-analyses, which pool the results of all relevant published studies, may be used for estimating the effect size.
- **Cost-effectiveness:** Where possible, cost effectiveness analyses can be conducted to provide an estimate (cost-effectiveness ratio) of how much it will cost to save one DALY by implementing the intervention (usually expressed in US dollars). To be able to carry out a cost effectiveness analysis, estimates are needed of the potential health impact and the cost of implementing the intervention.
- **Affordability:** The total costs of implementing an action can be calculated, using estimates of what 'ingredients' are needed and how much each of these costs. The WHO CHOICE database provides price information on price, based on WHO guidelines or other country experience. The total cost is an important feasibility consideration for countries.
- **Impact:** Any decision about potential interventions should clearly consider the scale of the potential impact. The overall health gain can be estimated using estimates of the effect size (see effectiveness) and information on the prevalence of the risk factors/conditions in the population. These elements can then be used to model the potential health impact of the intervention and to compare it with a scenario where there is no intervention – the difference between the two is the overall health gain. It is also important to consider the impact on different socio-economic groups (or particular vulnerable groups) and, therefore, the potential impact on health inequalities. There should also be consideration of other possible effects – including possible harms – induced by the intervention.
- **Acceptability:** In reality, policymakers' decisions are likely to place considerable weight on the acceptability of a particular intervention, both for politicians and the wider public. A lack of political will or widespread public scepticism can result in political inaction despite clear evidence for cost-effectiveness. It is important to consider these issues and to develop strategies to build political and public acceptability.
- **Practicality:** In order for policymakers to have the best possible evidence base for taking decisions, it is also important to consider – and set out clearly – some of the feasibility issues and practical considerations for implementation. Factors such as the capacity of the health system to deliver or the country's capacity to implement, monitor and enforce regulation may be relevant.

Types of evidence for what works

Given the complexities described above in assessing (a) the relationships between dietary risk factors and CVD and (b) the impact of an intervention or action, there are different types of evidence for effectiveness of public interventions to prevent CVD.

- **Analogy:** It is possible to draw on evidence of interventions' effectiveness in other areas of public health (or public policy). Experience with taxation or social marketing in relation to tobacco, for example, can be relevant to efforts to improve diet. Other types of 'indirect' evidence can also be important. Evidence of ongoing high levels of marketing expenditure for HFSS foods during children's television programming, for example, is suggestive that such marketing has an impact.
- **Observational data:** Observational data can come from epidemiological studies that do not involve carrying out interventions, but instead make comparisons between different populations or groups. This type of observational epidemiology includes cross-sectional, case-control and cohort studies. Observational data may also be obtained from monitoring and surveillance – regular data collection at the population level over time. Examples include mortality and morbidity data, birth weights, infant and young child anthropometric data, food supply and census data.
- **Experimental data:** Experimental data can be obtained from experimental studies and/or programme or policy evaluation. Types of studies to assess the impact of particular interventions, in controlled conditions, include randomised controlled trials and non-randomised trials in individuals, settings or communities. Programme or policy evaluation aims to assess whether a programme or policy is meeting or has met its aims and objectives, as well as *how* the outcomes were achieved (process evaluation).
- **Modelling:** Modelling techniques can be used to estimate the effectiveness of an intervention on the basis of data on efficacy, programme uptake and reach. Models built around the relationship between the demand for food products and price, for example, can be used to estimate the overall dietary impact of proposed taxes or subsidies. Where an overall health impact is estimated it is also possible to generate cost-effectiveness analyses by incorporating data on projected costs.

Different frameworks for identifying and classifying interventions aimed at improving diets

Interventions aimed at improving diets are extremely diverse – seeking to influence diverse aspects from international trade policy to how individuals choose what to eat in restaurants. There are many different ways of thinking about these varied interventions, in order to be able to identify all that are relevant and classify them in a systematic way.

The NOURISHING Framework

The World Cancer Research Fund has developed a framework for reporting, categorising and monitoring policy actions.[2i] This framework is also intended to help systematically categorise, update, interpret and communicate the evidence for policy to policymakers. The framework is based on the understanding that food policies should aim to improve dietary behaviours by improving the availability, affordability and acceptability of healthy diets and by decreasing the availability, affordability and acceptability of unhealthy diets.

Table 14 The NOURISHING framework for food policy actions

DOMAIN		POLICY AREA
Food environment	N	Nutrition label standards and regulations on the use of claims and implied claims on foods
	O	Offer healthy foods and set standards in public institutions and other specific settings
	U	Use economic tools to address food affordability and purchase incentives
	R	Restrict food advertising and other forms of commercial promotion
	I	Improve the quality of the food supply
	S	Set incentives and rules to create a healthy retail environment
Food system	H	Harness supply chain and actions across sectors to ensure coherence with health
Behaviour change communication	I	Inform people about food and nutrition through public awareness
	N	Nutrition advice and counselling in health care settings
	G	Give nutrition education and skills

i http://www.wcrf.org/int/policy/nourishing-framework

The 4Ps of marketing theory

Another way to categorise the potentially confusing variety of policies to improve diet is to use the 4Ps of marketing mix theory by categorising policies in four areas: Product, Price, Promotion and Place. This approach – used in the 2011 EHN Paper – was developed for producers and marketers to assess how well products match their targets.[3] Under this framework product could include reformulation, elimination or new healthier products, while price could include taxes, subsidies and other economic incentives. Promotion can include health education, public information and campaigns, advertising controls and food labelling. Place covers interventions in schools, workplaces and other locations.

Interventions by dietary determinants and/or actor

For its 2009 report *Policy and action for cancer prevention*, the World Cancer Research Fund issued a series of organised into actions directed at policymakers and decisionmakers (actors) in nine fields. This categorisation of recommendations by actor followed a process of identifying the most promising interventions in relation to the physical environmental, economic, social and personal determinants of patterns of diet, physical activity, body composition and associated factors.

Frameworks for assessing which interventions work

There are also different methodologies and frameworks for the process of assessing which interventions work.

Updating WHO's 'best buys' for NCD control and prevention

WHO has recently updated its recommended 'best buy' policy options for NCD prevention, as set out in Appendix 3 of the *Global action plan for the prevention and control on noncommunicable diseases 2013-2020*.[4]

The process for producing the update involved analysis of existing interventions if there was new or emerging evidence on their cost-effectiveness, and inclusion of new proposed interventions. To be considered for further analysis, new interventions must have had a demonstrated and quantifiable effect size from at least one published study in a peer reviewed journal and have a clear link to one of the nine voluntary global targets. Interventions meeting those criteria were then considered for various parameters:

- *Cost-effectiveness*: For those interventions where cost-effectiveness analysis is considered possible, analysis using the WHO CHOICE methodology was conducted. Interventions were listed in order of their cost-effectiveness ratio, but no distinct cost-effectiveness threshold was applied.
- *Size of health gain*: The expected size of population health gain was estimated as measured by the DALYs averted in a standardised population of 10 million people. These estimates were calculated using effect sizes drawn from published literature and where feasible meta-analyses were conducted to combine different trial results. The effect size was then used in an epidemiological model of health outcomes to estimate the scale of the health gain for low, lower-middle, upper-middle and high-income countries.
- *Total cost*: The budget required was estimated on the basis of the costs for implementation in a standardised population of 10 million people.
- *Feasibility/implementation constraints:* Non-financial constraints – e.g. health system capacity, regulatory capacity, need for multisectoral action – that could hinder implementation were set out distinctly for policymakers to consider in their own national context.

World Cancer Research Fund Policy and action for cancer prevention report

WCRF's 2009 report on policy interventions relating to food, nutrition and physical activity and cancer prevention systematically assessed a range of policy options. The method employed involved a number of stages:

1. Identify whether, and if so where, intervention is needed (based on authoritative reports on diet and health)
2. Agree programmes and actions that are most likely, from existing evidence, to be efficient and effective
3. Estimate the potential benefit to public health (taking into account effect size and disease prevalence)
4. Agree which determinants are likely to be most powerful and what interventions are likely to be most effective.
5. Analyse costs and benefits of proposed programmes and actions (projected human, financial and other material costs and projected impact and potential adverse effects)
6. Draft the policies, subject them to review and consultation.

The policies then need to be enacted and, finally, evaluated in action.

A number of parameters were assessed for each of the interventions considered:

- Political feasibility and acceptability
- Potential impact (both benefits and harm)
- General acceptability
- Cost
- Timeframe
- Transferability

In addition, an assessment was made of the level of confidence (high, medium or low) in the evidence and whether the intervention was likely to have a high, medium or low impact.

Annex 4: The evidence base for EHN key recommendations

√ = Some evidence of this type exists
x = No evidence of this type identified

EHN KEY RECOMMENDATION	Logic	Analogy	Observational studies of determinants	Small scale experiments	Natural experiments	Modelling
Global food convention to establish global regulatory framework to protect healthy diets	√ An internationally agreed treaty would be the most effective means of tackling many those issues which are cross-border (e.g, marketing and trade) and a food supply in which many food companies are multinationals	√ The global Framework Convention on Tobacco Control established a legal framework for the specific multiple evaluated measures needed to reduce tobacco use and sets a strong precedent	x	x	x	x
Reform agriculture and food policy to align with public health policies and promote sustainable food production (including by radically reforming the CAP, measures to encourage shorter supply chains and improve the food supply through rural development and conservation)	√ The CAP influences the relative prices and availability of different foods, both of which influence consumption	x	√ There is clear evidence that policies which affect price and supply can impact on consumption	x	√ Removal of subsidies on animal fats in the early 1990s in Eastern Europe changed consumption and brought health benefits[5]	√ Different modelling exercises have looked at various aspects of CAP[6–8]
Ensure that trade and investment policies protect and promote public health	√ Since trade policies have been shown to influence the food supply and nutrition,[9] there is a need to ensure that nutrition is protected in trade and investment discussions and a case for exploring the potential use of trade measures to improve the food environment.	√ Similar challenges are faced in relation to other aspects of public health (e.g. food safety, environmental risks, tobacco control).	√ Trade policies affect levels of imports and can influence price and availability.[9–11] They can also encourage inward investment by multinational food companies, thereby potentially increasing availability of HFSS foods.	x	√ Some small countries have shown that nutrition can be protected in trade discussions [12] and that trade measures (import controls, tariffs, standards) can be used to reduce imports of particular high fat foods/oils.[13,14]	x
Mandatory maximum levels of industrially-produced trans fatty acids	√ There is a clear logical case that mandatory limits will reduce amounts of trans fats in foods, and there is strong evidence that these are harmful to cardiovascular health.	√ Legislation to mandate limits on food contaminants has long been part of food control.	√ Observed virtual elimination of trans fats in food supply after national 'bans'.	√ Local 'bans' have effectively reduced trans fats in food from restaurants, fast food chains, etc.	√ International experience shows that national 'bans' virtually remove industrially produced trans fats from the food supply[15] and have led to positive changes in blood lipids.[16] In Denmark this was followed by a drop in cardiovascular death rates.[17] Local legislation has also been followed by a drop in hospitalisation rates for cardiovascular events – as seen in some New York counties but not seen in other counties that had not taken action on trans fats.[18]	√ Removal of trans fats from US food supply estimated to save $1-$2 billion in healthcare costs.[19]
Establish nutrition standards for food served or sold in schools, hospitals and other public institutions and issue guidelines for healthy public procurement	√ Food served in these government institutions make a substantial contribution to national diets and, for some groups, provide the vast majority of their nutrition. Government establishments should not be permitting the sale and promotion of foods that are damaging to health and create a major burden for the state if they pay or subsidise a health service.	x	√ Evidence from widespread experience has shown that school regulation and healthy procurement can improve diet and health outcomes.	√ Emerging results: Nutritional standards across all New York City institutions has reduced amounts of added sugar and solid fats used, the sodium content of dishes and virtually eliminated trans fats and sugar-sweetened beverages.	√ Evidence from widespread experience has shown that school regulation and healthy procurement can improve diet and health outcomes.	√ WHO CHOICE modelling estimates that daily salt intake can be reduced by 7% through establishment of a supportive environment to provide low sodium meals in public institutions.[20]

EHN KEY RECOMMENDATION	Logic	Analogy	Observational studies of determinants	Small scale experiments	Natural experiments	Modelling
Wide-ranging, ambitious food reformulation programmes to reduce levels of salt, saturated fat, free sugars, energy density and portion size in processed foods	√ Yes, provided that reductions are sufficient and coverage of products substantial	√ Reformulation to reduce fats/ sugars modelled on success with salt reformulation	√ Targeted foods make major contribution to dietary intakes; Observed fall in salt levels in food and population sodium intakes after salt reformulation programmes.	x	√ Observed fall in salt levels in food and population sodium intakes after salt reformulation programmes.	√ Cost savings from the UK's salt reduction programme estimated at £300 million/year.[20] WHO CHOICE modelling, based on Argentina's experience, that reformulation can lead to a reduction of 2.2g / day in salt intake.[21]
Use of taxes and/or subsidies to promote consumption of healthy foods and reduce consumption of unhealthy foods	√ Food purchases are known to be price sensitive; Targeted foods for taxation make significant contribution to fat/ sugar content of average diet	√ Taxes on tobacco and alcohol are effective	√ Strong evidence that consumers alter food and beverage purchases in response to price changes	√ Prior to the emerging national experience, there were examples from smaller jurisdictions (cities, states) could introduce local taxes	√ There is a growing body of evidence from country experience (particularly with taxes on SSBs). But EU governments in their policy analyses for promoting selective food consumption have always used price manipulations based on their clear evidence of the price elasticity (purchasing responses to price) of different foods	√ There has been extensive modelling
Regulatory controls to reduce the public health impact of marketing (including advertising and other forms of commercial promotion across all media) of foods high in fat, sugar or salt (HFSS).	√ Children: Scale of marketing activity suggestive of influence on children Adults: The magnitude of HFSS marketing expenditure suggests some impact on adult consumption patterns	√ Regulatory controls on marketing of tobacco and alcohol have been effective;[21] Internet controls on content not suitable for children could also provide lessons	√ Children: Convincing research that exposure to marketing for HFSS foods influences what and how much children eat Adults: Major reviews have not considered or have not found evidence of direct effects of HFSS marketing on adult consumption patterns.	x	√ Children: Marketing restrictions have reduced children's exposure in covered media but loopholes have generally meant that children are still exposed to HFSS marketing.	√ Modelling studies suggest that effective marketing restrictions would have a substantial health impact[22,23]
Adoption of nutrient profiles in the context of the EU regulation of health and nutrition claims; and of mandatory simplified front-of-pack nutrition labelling, and menu labelling	√ Nutrient profiles √ FOP √ Menu There is a strong logical basis for use of nutrient profiles to underpin nutrition labelling and prevent health and nutrition claims on unhealthy products. Clear, prominent, easy-to-understand nutrition information is essential for promoting healthier choices. Food eaten out of the home is an increasingly important part of European diets and consumers tend to overestimate how healthy restaurant foods are.[24]	√ Nutrient profiles √ FOP √ Menu Nutrient profiles have been used effectively in relation to restricting HFSS food marketing to children and in FOP labelling in UK and France.	x Nutrient profiles √ FOP Growing evidence of impact on consumer purchases, and that it drives reformulation √ Menu	√ Nutrient profiles Considerable evidence from studies and field testing confirms that nutrient profile models are a valuable tool for achieving policy goals. √ FOP Studies suggest that consumers prefer front of pack labelling and that such labels can help people make healthier food choices.[25] √ Menu	x Nutrient profiles √ FOP √ Menu Growing evidence that menu calorie labelling results in lower energy meals being ordered	√ Nutrient profiles √ FOP √ Menu Nutrient profiles are developed through use of modelling. Some modelling of FOP and menu combined e.g. UK modelling estimated mandated nutrition labelling on packaging and in fast food restaurants at a cost of $2 000 per DALY[26]

References

1. Mekonnen, M. M. & Hoekstra, A. Y. A global assessment of the water footprint of farm animal products. *Ecosystems* 15, 401–415 (2012).
2. Hawkes, C., Jewell, J. & Allen, K. A food policy package for healthy diets and the prevention of obesity and diet-related non-communicable diseases: the NOURISHING framework. *Obes. Rev.* 12, 159–168 (2013).
3. Lloyd-Williams, F. *et al.* Smorgasbord or symphony? Assessing public health nutrition policies across 30 European countries using a novel framework. *BMC Public Health* 14, 1195 (2014).
4. World Health Organization. *Preparation for the third High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases, to be held in 2018. Report by the Director General.* (2017).
5. Zatonski, W., Campos, H. & Willett, W. Rapid declines in coronary heart disease mortality in Eastern Europe are associated with increased consumption of oils rich in alpha-linolenic acid. *Eur. J. Epidemiol.* 23, 3–10 (2008).
6. Lloyd-Williams, F. *et al.* Estimating the cardiovascular mortality burden attributable to the European Common Agricultural Policy on dietary saturated fats. *Bull. World Health Organ.* 86, 535–541A (2008).
7. Schmidhuber, J. *The EU Diet-Evolution, Evaluation and Impacts of the CAP.* (FAO, 2003).
8. Veerman, J. L., Barendregt, J. J. & Mackenbach, J. P. The European Common Agricultural Policy on fruits and vegetables: exploring potential health gain from reform. *Eur. J. Public Health* 16, 31–35 (2006).
9. Friel, S. *et al.* Monitoring the impacts of trade agreements on food environments. *Obes. Rev.* 14 Suppl 1, 120–134 (2013).
10. Hawkes, C. *Enhancing Coherence between Trade Policy and Nutrition Action: Implementing the Framework for Action of the Second International Conference on Nutrition. UNSCN Discussion Paper.* (2016).
11. Mccorriston, S., Hemming, D. J., Lamontagne-godwin, J. D., Parr, M. J. & Roberts, P. D. *What is the evidence of the impact of agricultural trade liberalisation on food security in developing countries?* (2013).
12. Thow, A. M. & McGrady, B. Protecting policy space for public health nutrition in an era of international investment agreements. *Bull. World Health Organ.* 92, 139–45 (2014).
13. Thow, A. M. *et al.* Trade and food policy: Case studies from three Pacific Island countries. *Food Policy* 35, 556–564 (2010).
14. Thow, A. M., Annan, R., Mensah, L. & Chowdhury, S. N. Development, implementation and outcome of standards to restrict fatty meat in the food supply and prevent NCDs: learning from an innovative trade/food policy in Ghana. *BMC Public Health* 14, 249 (2014).
15. Downs, S. M., Thow, A. M. & Leeder, S. R. The effectiveness of policies for reducing dietary trans fat: a systematic review of the evidence. *Bull. World Health Organ.* 91, 262–9 (2013).
16. Vesper, H. W., Kuiper, H. C., Mirel, L. B., Johnson, C. L. & Pirkle, J. L. Levels of plasma trans-fatty acids in non-Hispanic white adults in the United States in 2000 and 2009. *JAMA* 307, 562–563 (2012).
17. Restrepo, B. J. & Rieger, M. Denmark's Policy on Artificial Trans Fat and Cardiovascular Disease. *Am. J. Prev. Med.* 50, 69–76 (2016).
18. Brandt, E., Myerson, R., Perraillon, M. & Polonsky, T. Hospital admissions for myocardial infarction and stroke before and after the trans-fatty acid restrictions in new york. *JAMA Cardiol.* 2, 627–634 (2017).
19. List, G. Processing and reformulation for nutrition labelling of trans fatty acids. *Lipid Technol.* 16, 173–176 (2004).
20. Barton, P., Andronis, L., Briggs, A., McPherson, K. & Capewell, S. Effectiveness and cost effectiveness of cardiovascular disease prevention in whole populations: modelling study. *BMJ* 343, d4044 (2011).
21. World Health Organization. *Global status report on noncommunicable diseases. (WHO; Geneva)* (2010).
22. Vos, T. *et al. Assessing Cost-Effectiveness in Prevention.* (2010).
23. Cecchini, M. *et al.* Tackling of unhealthy diets, physical inactivity, and obesity: Health effects and cost-effectiveness. *Lancet* 376, 1775–1784 (2010).
24. Wartella, E. A., Lichtenstein, A. H., Yaktine, A., Nathan, R. & Sciences, N. A. of. *Front-of-Package Nutrition Rating Systems and Symbols: A Consumer Health Literacy Perspective. Promoting Healthier and Symbols.* (2011).
25. Cecchini, M. & Warin, L. Impact of food labelling systems on food choices and eating behaviours: a systematic review and meta-analysis of randomized studies. *Obes. Rev.* 17, 201–210 (2016).
26. Dobbs, R. *et al.* Overcoming obesity : An initial economic analysis. *McKinsey Glob. Inst.* 120 (2014).Annex 3: Identifying and defining effective policies

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