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European Code against Cancer 4th Edition: Diet and cancer[☆]



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ABSTRACT

Lifestyle factors, including diet, have long been recognised as potentially important determinants of cancer risk. In addition to the significant role diet plays in affecting body fatness, a risk factor for several cancers, experimental studies have indicated that diet may influence the cancer process in several ways. Prospective studies have shown that dietary patterns characterised by higher intakes of fruits, vegetables, and whole-grain foods, and lower intakes of red and processed meats and salt, are related to reduced risks of death and cancer, and that a healthy diet can improve overall survival after diagnosis of breast and colorectal cancers. There is evidence that high intakes of fruit and vegetables may reduce the risk of cancers of the aerodigestive tract, and the evidence that dietary fibre protects against colorectal cancer is convincing. Red and processed meats increase the risk of colorectal cancer. Diets rich in high-calorie foods, such as fatty and sugary foods, may lead to increased calorie intake, thereby promoting obesity and leading to an increased risk of cancer. There is some evidence that sugary drinks are related to an increased risk of pancreatic cancer.

Taking this evidence into account, the 4th edition of the European Code against Cancer recommends that people have a healthy diet to reduce their risk of cancer: they should eat plenty of whole grains, pulses, vegetables and fruits; limit high-calorie foods (foods high in sugar or fat); avoid sugary drinks and processed meat; and limit red meat and foods high in salt.

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Abbreviations: BMI, body mass index; ECAC, European Code against Cancer; EPIC, European prospective study; WCRF/AICR, World Cancer Research Fund/American Institute for Cancer Research; WHEL, Women's Healthy Eating and Living Trial; WINS, Women's Intervention Nutrition Study.

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1. Introduction

Lifestyle factors, including diet, have long been recognised as potentially important determinants of cancer risk. In 1981, Doll and Peto estimated that 35% of cancer deaths might be avoidable by changes in diet and tackling obesity [1]. Ecological studies showing significant correlations between dietary habits and cancer incidence and mortality [2], and the large global variations in cancer incidence and mortality rates, along with rapid changes in cancer rates among migrant populations [3], stimulated further research on the role of diet in cancer prevention.

The most extensive review of the existing evidence on diet and cancer is the 2007 World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) [4] report and its subsequent

updates [5]. Lifestyle recommendations for cancer prevention were drawn up on the basis of nutrition-related factors judged to be convincingly or probably causally related to cancer, according to predefined criteria for judging the strength of the evidence regarding causality [6] (Table 1). According to the recommendations, a healthy diet for cancer prevention is a diet (1) that allows a person to be as lean as possible without being underweight; (2) is rich in fruits, vegetables, whole grains and pulses; (3) contains low amounts of red meat; (4) does not contain processed meats; and (5) limits salt intake. In addition, a healthy diet is characterised by the avoidance of sugary drinks and limited intake of calorie-rich foods, thereby contributing to achieving and maintaining a healthy weight. A healthy diet also limits consumption of alcoholic drinks. The preventability of several cancers in the UK through healthy eating, being physically active, and maintaining a healthy weight as recommended by WCRF/AICR was estimated to range from 5% to 34% [7] (Fig. 1).

1.1. Main mechanisms involving nutritional factors in cancer development

Excessive caloric intake results in weight gain and ultimately obesity, which in turn is associated with an increased risk of several cancers. Many experimental studies have shown that calorie restriction suppresses the carcinogenic process [8]. The biological mechanisms linking adiposity and cancer risk include hyperinsulinaemia and insulin resistance, up-regulation of insulin-like growth factors, modification of the metabolism of sex hormones, chronic inflammation, changes in production of adipokines and vascular growth factors by adipose tissue, oxidative stress, and alterations in immune function [9].

In addition to its calorie content, diet may influence cellular processes and lead to the accumulation of the eight hallmarks of cancer cells: self-sufficiency in growth signals, insensitivity to anti-growth signals, limitless replicative potential, evasion of apoptosis, sustained angiogenesis, reprogramming of energy metabolism, evasion of immune destruction, tissue evasion and metastasis [10].

The links between diet and cancer are complex. Thousands of dietary components are consumed each day; a typical diet may provide more than 25,000 bioactive food constituents, and the amounts of bioactive components within a particular food may widely vary [11]. Each bioactive food constituent has the potential to modify multiple aspects of the cancer process, alone or in combination with several micronutrients, and the quantity, timing, and duration of exposure modulate the cell response. Thus, it is not possible to ascribe a causal effect to specific compounds; it is more likely that the effect results from a combination of influences on several pathways involved in carcinogenesis.

A growing body of evidence indicates that lowering the energy density (the amount of energy in a particular weight of food) of diets can reduce caloric intake (reviewed by Rolls [12]). Energy-dense diets contain less fibre-rich foods, and are usually high in fats, processed starch, and added sugars.

Trans fatty acids are used in industrially processed sweet and salty foods, such as chocolate bars, candies, biscuits, cakes, crackers, industrial bread, and packaged snacks. The array of potentially harmful effects of industrial trans fatty acids is wide, and include alterations in metabolic and signalling pathways, higher circulating levels of lipid, systemic inflammation, endothelial dysfunction, and possibly increased visceral adiposity, body weight and insulin resistance [13]. There is evidence that trans fatty acids could be associated with an increased risk of breast cancer [14,15], non-aggressive prostate cancer [16], gastric adenocarcinomas [17] and colorectal adenomas [18]. The World Health Organisation recommends eliminating the use of trans fatty acids in food processing for cardiovascular disease prevention [19].

Table 1

2007 World Cancer Research Fund – American Institute for Cancer Research Personal Recommendations for Cancer Prevention.

Recommendations
Body fatness
Be as lean as possible within the normal range of body weight
<i>Personal recommendations</i>
Ensure that body weight throughout childhood and adolescent growth projects towards the lower end of the normal BMI range at age 21
Maintain body weight within the normal range from age 21
Avoid weight gain and increases in waist circumference throughout adulthood
Physical activity
Be physically active as part of everyday life
<i>Personal recommendations</i>
Be moderately physically active, equivalent to brisk walking, for at least 30 min every day
As fitness improves, aim for ≥60 min of moderate or ≥30 min of vigorous physical activity every day
Limit sedentary habits such as watching television
Foods and drinks that promote weight gain
Limit consumption of energy-dense foods and avoid sugary drinks
<i>Personal recommendations</i>
Consume energy-dense foods sparingly
Avoid sugary drinks
Consume fast foods sparingly, if at all
Plant foods
Eat foods mostly of plant origin
<i>Personal recommendations</i>
Eat at least five portions/servings (at least 400 g or 14 oz) of a variety of non-starchy vegetables and fruit every day
Eat relatively unprocessed cereals (grains) and/or pulses (legumes) with every meal
Limit refined starchy foods
People who consume starchy roots or tubers as staples should also to ensure sufficient intake of non-starchy vegetables, fruit, and pulses
Animal foods
Limit intake of red meat and avoid processed meat
<i>Personal recommendations</i>
People who eat red meat to consume <500 g (18 oz) a week, very little – if any – to be processed
Alcoholic drinks
Limit alcoholic drinks
<i>Personal recommendations</i>
If alcoholic drinks are consumed, limit consumption to no more than two drinks a day for men and one drink a day for women
Preservation, processing, preparation
Limit consumption of salt
Avoid mouldy cereals (grains) or pulses (legumes)
<i>Personal recommendations</i>
Avoid salt-preserved, salted, or salty foods; preserve foods without using salt.
Limit consumption of processed foods with added salt to ensure an intake of <6 g (2.4 g sodium) a day
Do not eat mouldy cereals (grains) or pulses (legumes)
Dietary supplements
Aim to meet nutritional needs through diet alone
<i>Personal recommendations</i>
Dietary supplements are not recommended for cancer prevention
Breastfeeding
Mothers to breastfeed; children to be breastfed
<i>Personal recommendations</i>
Aim to breastfeed infants exclusively up to 6 months and continue with complementary feeding thereafter
Cancer survivors
Follow the recommendations for cancer prevention
<i>Personal recommendations</i>
All cancer survivors should receive nutritional care from an appropriately trained professional.
If able to do so, and unless otherwise advised, aim to follow the recommendations for diet, healthy weight, and physical activity

Source: Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective, 2007 [4].

Notes:

Normal (or healthy) weight correspond to a body mass index between 18.5 and 24.9 kg/m². Examples of light physical activity are walking slowly, light gardening, housework; examples of moderate activities are walking briskly, cycling, dancing, swimming and vigorous activities (running, tennis and football).

Energy-dense foods are defined as those with an energy content of more than about 225–275 kcal/g.

Sugary drinks refer to drinks with added sugars. Fruit juices should also be limited.

“Fast foods” refer to readily available convenience foods that tend to be energy-dense and are frequently consumed in large portions.

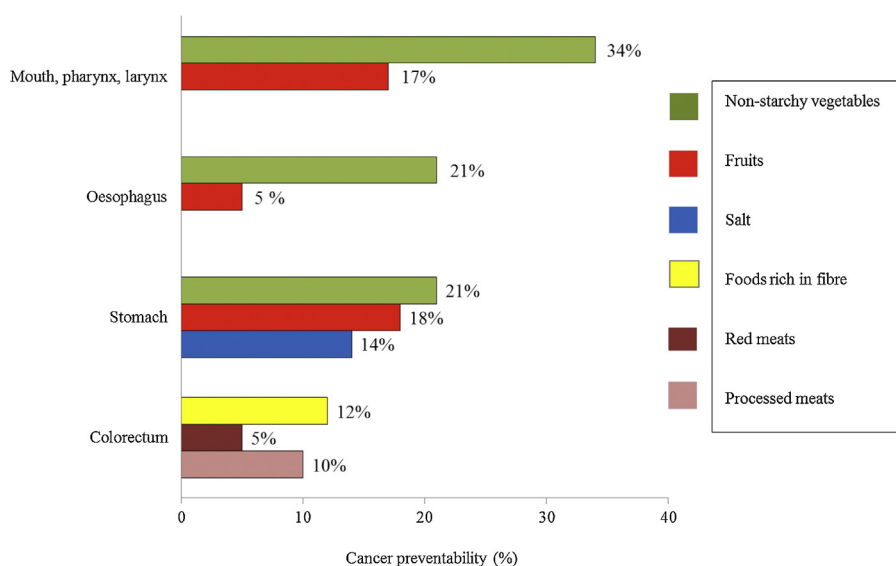


Fig. 1. Cancer preventability estimates for the United Kingdom for dietary factors judged to be convincingly or probably related to the risk of some cancers. The figure shows the percentage of cancer cases preventable by adequate diet in the United Kingdom through adequate intakes of dietary factors considered probably or convincingly related to the risk of some cancers [4,5]. Processed meats and red meats convincingly increase the risk of colorectal cancer; foods rich in fibre convincingly decrease the risk of colorectal cancers; salt, salted and salty foods probably increase the risk of stomach cancer; and fruits and vegetables probably decrease the risk of cancers of the mouth, pharynx and larynx, oesophagus and stomach.

Source: Figure based on data from the Diet and Cancer Report website [7].

Diets high in sugars may promote carcinogenesis by increasing insulin production, increasing oxidative stress [20], or promoting weight gain [21]. In metabolic syndrome, foods rich in sugars are likely to interfere with levels of blood glucose and/or triglycerides, either directly or through insulin and other hormones [22], while sugar-sweetened soft drinks play a role in the development of obesity (reviewed by Hu et al. [21]).

Some food compounds are direct mutagens and carcinogens. Many nitroso compounds are mutagenic and potentially carcinogenic. Haem iron, which is abundant in red meat but not in white meat, promotes colorectal carcinogenesis through its catalytic activity in the formation of nitroso compounds and of lipid oxidation end products such as 4-hydroxynonenal [23]. In processed red meat, haem iron is nitrosylated, because curing salt contains nitrate or nitrite; there is evidence that nitrosylated haem iron promotes carcinogenesis at 5–6 times lower doses than non-nitrosylated haem iron. Experimental studies show that calcium salts, chlorophyll, vitamin C, and several polyphenols may reduce the deleterious effects of haem (reviewed by Bastide et al. [24]). Heterocyclic amines and polycyclic aromatic hydrocarbons are other potential carcinogens that are formed in meats when cooked at high temperatures for a long time, or exposed to a direct flame [25,26]; this is not limited to red and processed meats. Various single-nucleotide polymorphisms involved in the metabolism of these potential carcinogens may modulate the association of carcinogens formed in meat with cancer risk [27–29].

Dietary fibre from plant foods stimulates bacterial anaerobic fermentation in the large bowel, leading to the production of short-chain fatty acids: acetate, propionate, and butyrate. In cell lines, butyrate reduces cell proliferation and induces apoptosis. Dietary fibre may protect against colorectal cancer by reducing the contact between the intestinal content and the mucosa, and may interfere with the enterohepatic circulation of oestrogens [30]. Some studies have shown reductions in circulating oestrogen and androstenedione levels with high fibre intake [31,32], and in experimental studies in mice soluble fibre reduced mammary tumour growth, angiogenesis and metastasis [33]. In addition, fibre-rich foods are important sources of phyto-oestrogens, oestrogen-like plant compounds that may interact with and modulate the activity of

oestrogen receptors [34,35], thereby modulating the risk of hormone-dependent cancers (especially breast cancer) [36]. Polyphenols from plant foods have multiple biological effects, including scavenging of oxidative agents, anti-inflammatory and detoxifying actions, inhibition of platelet aggregation, and antimicrobial activity [37,38].

Vitamins and minerals such as carotenoids, folate, vitamins C, D, E and B6, selenium, and phytochemicals might reduce cancer risk through preventing oxidative damage, inhibiting cell proliferation, inducing cell-cycle arrest, maintaining DNA methylation, and/or modulating steroid hormone concentrations and hormonal metabolism (e.g., via phyto-oestrogen contained in pulses) [39].

Diallyl disulphide from garlic and other allium vegetables, and sulphoraphane, a glucosinolate from cruciferous vegetables, can behave as histone deacetylase inhibitors and act to maintain DNA stability or enhance transcription [40]. Naturally occurring bioactive compounds such as curcumin, resveratrol, and isothiocyanates have potential antioxidant and/or anti-inflammatory and anti-carcinogenic activities (reviewed by Chung et al. [41]).

Sodium chloride is a food preservative used for increasing the safety and shelf life of processed foods. In animal experiments, salt intake facilitates gastric colonisation by *Helicobacter pylori*, one of the main predisposing factors for stomach cancer development, and induces mucosal damage [42], potentially leading to chronic atrophic gastritis. Salt intake may also promote or enhance the effect of nitroso compounds and other carcinogens (reviewed by D'Elia et al. [43]).

1.2. Overview of the main European dietary patterns

As a consequence of the convergence of factors that influence food choices, differences in dietary patterns across European countries are becoming less marked. However, data from food availability at the national and household levels, and dietary surveys on individual food consumption (European Nutrition and Health Report 2009 [44]), show that regional differences still exist (Fig. 2). Similar findings had previously been reported in large population studies that collected data on food and beverage intakes using detailed dietary questionnaires [45]. On average, the

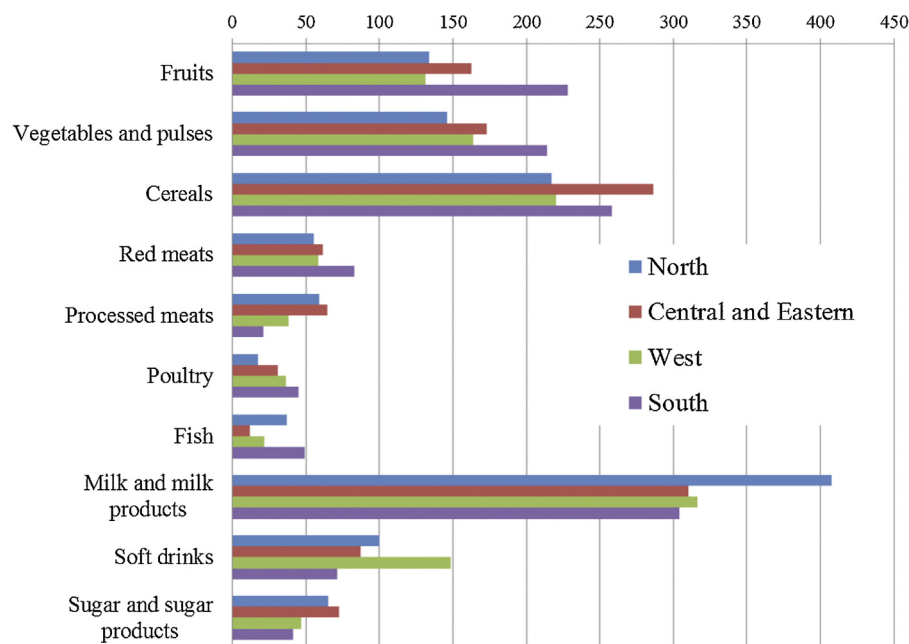


Fig. 2. Mean household availability of selected food and beverages in European regions. The figure shows the mean quantity (g/day) of selected foods available for each person in a household in European regions, assuming an equal distribution of foods across household members. Soft drinks are in ml/person/day. Foods and beverages consumed in restaurants, canteens or similar establishments were not consistently available. The countries included in the survey were Finland, Latvia, Norway, Sweden (North); Austria, Germany, Hungary, Poland, Slovenia (Central and Eastern), Belgium, France, Ireland, Luxembourg, United Kingdom (West); Cyprus, Greece, Italy, Spain, Portugal (South). The year of data collection ranged from 1991 to 2005, depending on the country.

Source: Figure based on data from Elmadfa et al. [44].

most marked difference is the North–South gradient of fruits, vegetables, cereals and pulses availability. Southern Europe is characterised by a lower availability of processed meats. The observed pattern is similar to the “Mediterranean” pattern, which is characterised by high intakes of vegetables, grains, dried beans, olive oil, seafood and fruits, and by moderate wine intake during meals.

The highest availability of milk and dairy-based products is found in Northern Europe, which, as in Central and Eastern Europe, is characterised by a higher household availability of processed meats. Sugary drinks are more available in Western European households [46]. Overall, higher educational level in the household is correlated with higher availability of fish, fruits, and vegetables. Lower socioeconomic status is associated with more frequent and greater availability of soft drinks [47].

2. Evidence of the association of diet and cancer in population studies

The randomised controlled trial is considered the best study design for assessing the effect of an intervention because the processes used to conduct this study type minimise the possibility that confounding factors could influence the results. However, randomised controlled trials are only justified when there is considerable evidence from animal, in vitro and observational studies that the intervention could have a beneficial effect. Testing potentially harmful interventions would not be ethical. Randomised controlled trials are also limited by a number of methodological issues, including the difficulty of incorporating whole-diet interventions into a randomised controlled trial design and in a not-blinded way, the compliance of study participants with dietary changes over a long period of time, and determination of the adequate nutrient dose or dietary intervention for a beneficial effect to occur.

Randomised controlled trials of nutrition and cancer are rare compared with observational studies. For less frequent cancers, the evidence from observational studies comes mainly from case–control studies. In these studies, dietary intake is assessed using questionnaires after cancer diagnosis, and they are more prone to recall and selection biases than are prospective observational studies. For more frequent cancers, most of the existing data on the relationship between diet and cancer risk comes from prospective observational studies, in which dietary assessment takes place before cancer diagnosis. The identification of new cancer cases during follow-up includes record linkage with cancer and mortality registries, self-reporting by study participants with further confirmation through medical records, or a combination of methods to minimise losses to follow-up and increase data reliability. Prospective studies are expensive, and several years are needed before the number of cancer cases required for analyses can be documented.

2.1. Fruits, vegetables, pulses, and whole-grain foods

A potentially protective effect of fruits and vegetables against cancer was supported by evidence from earlier case–control studies [48]. Subsequent data from prospective studies indicate that the association may be restricted to specific cancers and may be weaker than previously observed for some cancers [4,49].

The accumulated evidence supports the idea that low intake of fruits and vegetables is related to the development of cancers of the respiratory and upper digestive tracts (Table 2).

A recently published large consortium of case–control studies confirmed the inverse association between fruits and vegetables and cancers of the mouth, pharynx and larynx [50]. Data from cohort studies, which are less prone to bias than case–control studies, are needed to confirm these findings.

Recent cohort studies are also supportive of an inverse association between fruit intake and oesophageal squamous-cell

Table 2

Estimated associations from meta-analyses, pooled analyses and recent prospective studies for dietary factors judged in the 2007 WCRF/AICR as convincingly or probably causes of or protective against cancer.

Cancer type	Number of studies	Comparison unit	Relative risk	Reference
<i>Dietary fibre</i>				
Colon cancer	16 cohorts	For 10 g/d increase	0.90 (0.86–0.94)	[72]
<i>Vegetables</i>				
Mouth, pharynx, and larynx cancer	22 case–control studies	Highest versus lowest	0.68 (0.51–0.90)	[50]
Oesophageal cancer (squamous cell carcinoma)	5 cohorts	Highest versus lowest	0.80 (0.61–1.07)	[51]
Oesophageal cancer (all)	5 case–control studies	For 50 g/d increase	0.87 (0.72–1.05)	[4]
Stomach cancer	19 cohort studies	Highest versus lowest	0.96 (0.88–1.06)	[56]
Stomach cancer	1 cohort, 7 case-control (Japanese or Korean studies)	Highest versus lowest	0.62 (0.46–0.85)	[134]
	7 cohorts	For 100 g/d increase	0.98 (0.91–1.06)	[4]
<i>Allium vegetables:</i>				
Stomach cancer	2 cohorts, 12 case–control studies	Highest versus lowest allium vegetables intake	0.54 (0.43–0.65)	[63]
<i>Garlic</i>				
Colorectal cancer	2 cohorts	Highest versus lowest	Women: 1.21 (0.94–1.57) Men: 1.00 (0.71–1.42)	[135]
	1 cohort	For each additional clove or “4 shakes” of garlic per week	1.04 (0.99–1.08)	[136]
	1 cohort	Any pills per day previous 10 years versus no use	1.35 (1.01–1.81)	[137]
	2 cohorts	Highest versus lowest	0.72 (0.54–0.96)	[4]
<i>Fruits</i>				
Head and neck cancer	22 case–control	Highest versus lowest	0.52 (0.43–0.62)	[50]
Upper aero-digestive tract cancer	3 cohort studies	Highest versus lowest	0.78 (0.64–0.95)	[138]
Oesophageal cancer (squamous cell carcinoma)	5 cohorts	Highest versus lowest	0.68 (0.55–0.86)	[51]
Oesophageal cancer (all)	8 case-control	For 100 g/d increase	0.56 (0.42–0.74)	[4]
Lung cancer	7 cohorts (European)	Per 1 g/day	0.99 (0.99–0.99)	[139]
	14 cohorts	For one serving/day increase	0.94 (0.90–0.97)	[4]
Stomach cancer	22 cohort studies	Highest versus lowest	0.90 (0.83–0.98)	[56]
Stomach cancer		For 100 g/d increment	0.95 (0.89–1.02)	[4]
<i>Red meat</i>				
Colorectal cancer	10 cohorts	For 100 g/d increment	1.17 (1.02–1.33)	[95]
<i>Processed meat</i>				
Colorectal cancer	9 cohorts	For 50 g/day increment	1.18 (1.10–1.28)	[95]
<i>Salt intake</i>				
Stomach cancer	4 cohorts and 7 case-control studies	Highest versus lowest salt intake	1.22 (1.17–1.27)	[100]
	7 studies	Salt intake from salty food	2.41 (2.08–2.78)	[101]
	7 cohorts	Highest versus lowest salt intake	1.68 (1.17–2.41)	[4]
	2 cohorts	For 1 g/d increment salt intake	1.08 (1.00–1.17)	
<i>Foods rich in salt</i>				
Stomach cancer	10 cohorts	Use versus never or occasional consumption of pickle vegetables	1.32 (1.10–1.59)	[140]
<i>Pickled food</i>				
Stomach cancer	11 cohorts	Highest versus lowest	1.27 (1.09–1.49)	[101]
<i>Salted fish</i>				
Stomach cancer	13 cohorts	Highest versus lowest	1.24 (1.03–1.50)	[101]
<i>Salted and salty foods</i>				
Stomach cancer	4 cohorts	For 1 serving/day	1.32 (0.90–1.95)	[4]
<i>Soft sugary beverages</i>				
Pancreatic cancer	11 cohorts	Highest versus lowest	1.12 (0.99–1.27)	[89]
<i>Fructose</i>				
Pancreatic cancer	6 cohorts	For 25 g/day increment	1.22 (1.08–1.37)	[90]

carcinoma (summarised by Liu et al., 2013 [51]); the evidence of an association with oesophageal adenocarcinoma is weaker. With respect to specific types of fruits, no significant associations between intake of citrus fruits and oesophageal cancer were reported in two Asian cohorts [52,53], and the association between oesophageal cancer and dietary vitamin C was not confirmed by two recent cohort studies [54,55]. Oesophageal cancer was shown

to be inversely associated with intake of vegetables in a meta-analysis of cohort studies [51], although no statistical significance was observed.

The evidence for fruit intake and stomach cancer has been confirmed in a meta-analysis of 22 cohort studies showing that individuals with lower fruit intake are at higher risk of stomach cancer [56]. The association between stomach cancer and citrus

fruit intake was also confirmed in a meta-analysis of 12 case-control studies and two cohort studies [57]. These results were not confirmed in three recent reports from cohort studies [54,58,59]. The possibility that errors in diet measurement have obscured the association between citrus fruit intake and gastric cancer was suggested by a large multinational European prospective study (EPIC). This study reported an inverse association between plasma levels of vitamin C and gastric cancer risk, whereas no association was observed with dietary vitamin C [60]. Moreover, a meta-analysis of three cohort studies showed a 30% lower risk of stomach cancer for the highest compared to the lowest levels of pre-diagnosis plasma vitamin C [61].

For vegetables, no association with stomach cancer was observed in a large European prospective study (EPIC) [58], other cohort studies [54,59,62], and in a meta-analysis of 22 cohort studies [56]. A protective association of allium vegetables was reported in a meta-analysis of 19 case-control studies and two cohort studies [63]. No association was observed in the large EPIC study [58]. No effect on stomach cancer incidence or mortality was also observed in the Shandong Intervention Trial, China: a double-blind 7-year oral supplementation trial with garlic extract and steam-distilled garlic oil in *Helicobacter pylori* seronegative participants [64].

The inverse association between fruit intake and lung cancer observed in numerous case-control and cohort studies was confirmed in the EPIC study [65]. The NIH-AARP North American cohort study [66] did not find a significant association. As for vegetables, evidence of an association with lung cancer was provided by a meta-analysis of prospective cohort studies [67] and a study in Chinese men showing an inverse association with carotenoid-rich vegetables [68]. In a multi-ethnic cohort, circulating carotenoid levels were inversely related to lung cancer risk in men but not in women [69], raising the issue of potential confounding by smoking. However, the interpretation of these findings is complicated because the metabolism of carotenoids is affected by many hormonal, genetic and lifestyle factors.

The evidence of an association between fruits and vegetables and colorectal cancer was judged as “limited-suggestive” in the 2007 WCRF/AICR report. Evidence for an inverse association was reported in the more recent WCRF/AICR update [70], in which a non-linear relationship was observed, indicating that the benefit from an increase in fruit and vegetable consumption would be limited to people with the lowest intake levels, and that no substantial benefit would occur in people who already have a high intake of high fruits and vegetables. Not included in this update is the large NIH-AARP American cohort study which more recently reported an inverse association between vegetable intake and colon cancer, but no association between fruits and colon cancer [71].

The evidence for a protective role of dietary fibre on colorectal cancer in recent publications is stronger [72], and there is some evidence that it could be stronger for fibre from cereals. Whole-grain foods are rich in fibre, and have also been found to be inversely related to colorectal cancer risk [72]. Pulses are high in fibre and provide substantial amounts of protein, vitamins, and minerals with a relatively low amount of calories [73]. Case-control studies support a beneficial effect of pulses [4], but the data from published cohort studies on pulse intake are limited.

For other cancers, data on the potential beneficial effect of fibre has been accumulating, as shown by meta-analyses of cohort studies on breast cancer [74] and of case-control studies on oesophageal adenocarcinoma [75] and stomach cancer [76].

2.2. High-calorie foods and beverages

High-calorie foods are rich in fats and/or sugars, and often have low nutritional value except from providing energy for the functioning of the body.

In ecological studies, fat intake has been found to be related to higher risk of several cancers. Prospective observational studies have not confirmed the association between dietary fat and colorectal cancer [77], and studies on breast cancer have been inconsistent. There is some evidence that the relationship between dietary fat and breast cancer may have been obscured by imprecise measurement of fat intake [78,79], although this was not confirmed in another large study [80]. Inconsistencies may also have emerged if the intake of specific fats, and not of total fat, is related only to some breast cancer types. In a recent meta-analysis of cohort studies, intake of polyunsaturated fatty acids was weakly associated with higher risk of postmenopausal breast cancer [81], and in the EPIC study, saturated fat intake was related to a higher risk of positive-receptor breast cancer only [82]. In a nested case-control study within the French EPIC cohort that analysed membrane phospholipids, trans fatty acids from industrial sources were associated with increased breast cancer risk [14].

The Women's Health Initiative Dietary Modification Randomised Controlled Trial investigated whether a low-fat dietary pattern intervention could reduce the incidence of breast and other cancers. After approximately 8 years of follow-up, no reduction in the incidence of breast, colorectal, or endometrial cancers was observed; there was only a marginal reduction in the incidence of ovarian cancer [83]. However, women in the study only modestly lowered their fat intake, from 38% to 29%, and no change was seen in blood lipid levels. There was a small weight loss in the early years of the trial in women in the low fat group.

In a large prospective study sugars from beverages – specifically, added fructose from fructose-glucose corn syrup, but not sugars from solid foods – were positively associated with overall mortality but not with the risk of major cancers [84]. Sugar-sweetened beverages raise insulin and glucose levels, and its consumption is associated with obesity and diabetes [85], which have been found to be related to pancreatic cancer risk [86,87]. Intake of sugar-sweetened beverages was not related to pancreatic cancer in a meta-analysis of case-control and cohort studies [88], but a positive association between sugar-sweetened beverages and pancreatic cancer was observed in a pooled analysis of cohort studies [89]. In another recent meta-analysis of cohort studies, a higher risk of pancreatic cancer was observed for higher fructose intake [90]. Fructose has been a component of sugary drinks in North America, but – other than as part of sucrose – not in Europe [91]. Sugary drinks have not been found to be directly related to the risk of colon cancer [92] or other cancers [4], independently of body fatness. Greater consumption of foods with a high glycaemic index was significantly related to increased risks of breast and colorectal cancer in a meta-analysis of 11 and nine prospective studies, respectively [93,94].

2.3. Red and processed meats and cancer

Numerous studies have shown an association between high intake of processed meat (such as ham, bacon, sausages, and hot dogs), red meat (mainly beef, pork or lamb) and colorectal cancer [4,95] (Table 2). Overall, the increased risk associated with processed meat intake was higher than that with unprocessed red meat. Processed meat cured with nitrite contains high concentrations of preformed nitroso compounds and nitrosylated haem iron, and these are potential carcinogens. There is experimental evidence that meats cured with nitrite may increase oxidative DNA damage [93]. Studies on carcinogens formed during the cooking of meat have not been conclusive, perhaps because of interactions with genetic polymorphisms, such as the acetylator phenotypes, and because of the difficulties in assessing dietary carcinogen intake.

Red and processed meat intake has also been found to be related to the risk of stomach [96] and pancreatic cancers [97], and with

higher overall cancer mortality [98,99]. In addition to the carcinogenicity of nitroso compounds, the relationship between processed meat and stomach cancer could be due to the high salt content of processed meats.

2.4. Salt intake and cancer

Early ecological studies showed a positive association between salt intake and stomach cancer, and this is consistent with the results of observational studies (Table 2). The evidence is consistent across studies investigating the intakes of salt, pickled vegetables, and other salty foods [4,100,101].

Animal and human studies have provided evidence of a synergistic effect of dietary salt intake and *Helicobacter pylori* infection on gastric carcinogenesis, and there is some evidence that salt intake may promote or enhance the carcinogenic effect of nitroso compounds [43].

2.5. Dietary supplements and cancer

Associations observed in epidemiological studies between dietary intake or biomarkers of exposure to certain micronutrients and cancer risk prompted several randomised controlled trials, most of which did not demonstrate a protective effect. Moreover, an unexpected increased cancer risk was observed in some trials.

Specifically, an increased risk of lung cancer was observed in men receiving beta-carotene supplements in the Alpha-Tocopherol, Beta-Carotene (ATBC) Cancer Prevention Study [102] and in the Beta-Carotene and Retinol Efficacy Trial (CARET) [103]. Beta-carotene was thought to reduce cancer risk by reducing oxidative damage, but it is now recognised that high doses of beta-carotene can result in pro-oxidant and anti-apoptotic effects, especially when cells are simultaneously subjected to tobacco smoke. Subsequent randomised controlled trials have confirmed the lack of a protective effect of beta-carotene supplementation on cancer risk [104]. An increased risk of prostate cancer was also observed in the follow-up of the Selenium and Vitamin E Cancer Prevention Trial (SELECT) in patients receiving high doses of vitamin E [105].

Total calcium intake is consistently associated with reduced risk of colorectal cancer in observational studies. Trials have shown a protective effect of calcium supplementation on colorectal adenomas, a precursor of colorectal cancer, but not against colorectal cancer itself [106]. Randomised controlled trials of folic acid supplementation with colorectal adenoma (reviewed by Ziegler [107]) or colorectal cancer [108] do not support the case that folic acid supplementation could prevent colorectal cancer. Several prospective studies suggest a trend towards a protective effect of folate (folic acid) intake on colon cancer risk [109], but in a recent meta-analysis of case-control studies nested in cohort studies, plasma or serum folate was not related to colorectal cancer risk [110]. It has been proposed that folate may play a dual role, with high concentrations promoting tumour development once premalignant lesions are established [111]. The evidence for an association between folate (folic acid) intake and increased or reduced cancer risk in humans is equivocal and does not support the recommendation of supplementation [112].

The null findings of dietary supplement interventions in randomised controlled trials may reflect lack of effect, or inappropriate type, timing, duration, or dose of the intervention.

The evidence for any benefit of vitamin and mineral supplementation for cancer prevention from observational studies is limited [113]. Dietary supplement use is not currently recommended for cancer prevention [4].

3. Justification for recommendation

3.1. Benefits for cancer prevention

There is evidence that diets characterised by high intake of plant foods (fruits, vegetables, pulses and whole-grain foods), low intake of red and processed meats, low intake of sugary foods, and avoidance of high salt intake are related to a lower risk of several cancers (Table 2). Recent data from the EPIC study show that compliance with a “healthy diet” such as recommended by the WCRF/AICR report (Table 1) is associated with a reduction in overall cancer risk (5% risk reduction for adherence to each additional recommendation), with the largest reductions for stomach, endometrial, oesophageal, colorectal, and mouth, pharynx, and larynx cancers (12–16% risk reductions) [114]. In two North American studies, compliance with the WCRF/AICR recommendations was associated with reductions in aggressive prostate cancer (13%) [115] and cancer mortality (10%) [116]. The Mediterranean dietary pattern is also consistently associated with significant reductions in cancer mortality (10%), and specifically with a lower risk of aerodigestive and colorectal cancers (reviewed by Schwingshackl and Hoffmann [117]).

3.2. Benefits on cancer prognosis

Recent reviews have shown that diet may modify biomarkers of cancer progression in individuals who have been treated for cancer [118,119]. The number of studies investigating the influence of diet after cancer diagnosis and cancer outcomes is limited. Current recommendations for cancer survivors are based on the recommendations to reduce cancer risk and emphasise achieving and maintaining a healthy weight, regular physical activity, and a diet rich in vegetables, fruits and whole grains, and limited in red meat and alcohol [4].

In observational studies, better pre- or post-diagnosis diet quality (in general dietary patterns rich in plant foods) has been found to be associated with reduced risk of death after diagnosis of breast cancer [120,121], colorectal cancer [122] and head and neck cancers [123]. A Western dietary pattern – characterised by high intakes of meat, fat, refined grains, and dessert – was associated with a higher risk of recurrence and mortality among patients with stage III colon cancer treated with surgery and adjuvant chemotherapy [124]. Overall, no benefit for cancer-related death has been observed in studies in cancer survivors, but such studies are difficult to interpret due to high risk of confounding or reverse causation.

Two dietary intervention studies among women diagnosed with breast cancer, the Women’s Healthy Eating and Living Trial (WHEL) and the Women’s Intervention Nutrition Study (WINS), found that dietary interventions among breast cancer survivors without weight loss or increase in physical activity do not improve breast cancer prognosis. WHEL focused on a plant-based dietary pattern that included a reduction in dietary fat, while WINS focused on reduced dietary fat intake. Secondary analyses in WHEL showed that the dietary intervention pattern was associated with a reduced risk of second breast cancer events among women with early-stage breast cancer who reported no hot flashes at baseline, and that higher vegetable intake was associated with reduced breast cancer recurrence in tamoxifen users [125,126].

3.3. Benefits for other diseases

Higher intakes of fruits, vegetables and fibre have been consistently associated with lower risk of coronary heart disease

Box 1. European Code Against Cancer.**EUROPEAN CODE AGAINST CANCER****12 ways to reduce your cancer risk**

1. Do not smoke. Do not use any form of tobacco
2. Make your home smoke free. Support smoke-free policies in your workplace
3. Take action to be a healthy body weight
4. Be physically active in everyday life. Limit the time you spend sitting
5. Have a healthy diet:
 - Eat plenty of whole grains, pulses, vegetables and fruits
 - Limit high-calorie foods (foods high in sugar or fat) and avoid sugary drinks
 - Avoid processed meat; limit red meat and foods high in salt
6. If you drink alcohol of any type, limit your intake. Not drinking alcohol is better for cancer prevention
7. Avoid too much sun, especially for children. Use sun protection. Do not use sunbeds
8. In the workplace, protect yourself against cancer-causing substances by following health and safety instructions
9. Find out if you are exposed to radiation from naturally high radon levels in your home; take action to reduce high radon levels
10. For women:
 - Breastfeeding reduces the mother's cancer risk. If you can, breastfeed your baby
 - Hormone replacement therapy (HRT) increases the risk of certain cancers. Limit use of HRT
11. Ensure your children take part in vaccination programmes for:
 - Hepatitis B (for newborns)
 - Human papillomavirus (HPV) (for girls)
12. Take part in organised cancer screening programmes for:
 - Bowel cancer (men and women)
 - Breast cancer (women)
 - Cervical cancer (women)

The European Code Against Cancer focuses on actions that individual citizens can take to help prevent cancer. Successful cancer prevention requires these individual actions to be supported by governmental policies and actions.

in prospective studies (reviewed by Mente et al. [127]). A dietary pattern characterised by high intake of fruits and vegetables, and low intake of processed meat, sugar-sweetened beverages and refined cereal products, have been associated with lower risk of type 2 diabetes mellitus [128]. Numerous studies have shown that greater adherence to a Mediterranean diet is associated with a reduction in overall and cardiovascular disease mortality [129]. High salt intake increases blood pressure [130], which is a strong risk factor for cardiovascular and renal disease [131]. Processed meats have been found to be associated with higher incidence of coronary heart disease and diabetes mellitus [132].

In the EPIC study, participants with a lifestyle in agreement with the WCRF/AICR recommendations for cancer prevention had a 34% lower risk of death (95%CI: 0.59–0.75) compared with participants within the lowest agreement to the recommendations [133].

The 4th European Code against Cancer (Box 1) Nutrition Working Group has developed the recommendation of having a healthy diet in order to reduce the personal risk of getting cancer: “Have a healthy diet:

- eat plenty of whole grains, pulses, vegetables and fruits;
- limit high-calorie foods (foods high in sugar or fat) and avoid sugary drinks;
- avoid processed meat; limit red meat and foods high in salt.”

Conflict of interest

The authors declare no conflict of interest.

Authorship contribution

All the authors substantially contributed to the conception of the manuscript. Teresa Norat, Chiara Scoccianti and Marie-Christine Boutron-Ruault drafted the manuscript. All authors were involved in the revision of the manuscript.

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