Dietary fat and adult diseases and the implications for childhood nutrition: an epidemiologic approach[1–3]

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ABSTRACT Reducing dietary saturated fat by 7% of energy, a realistic target, would reduce serum cholesterol by 10% and mortality from ischemic heart disease by 25–30%. Randomized trials show that this mortality reduction is attained rapidly, usually by the third year after initial reduction of dietary saturated fat intake. Dietary change in adulthood may therefore reverse the adverse health effects of a high-fat diet in childhood. In the absence of such change, however, dietary fat in childhood may increase the risk of cardiovascular disease in adult life because of a longer duration of exposure to a high-fat diet. Assessing the effects of diet on cancer risk is more difficult. The intermediary markers of risk that are analogous to serum cholesterol are less satisfactory and there are negligible trial data. Cohort studies of diet and cancer, although subject to bias, do not favor a direct causal relation between dietary fat and cancer. But a reduction in risk is likely when dietary fat is reduced as part of a general change toward a healthier diet. The trend toward increased energy intake and body size in childhood and relatively low dietary fiber contribute to the decreasing age at menarche, which is associated with an increased risk of breast cancer. Low dietary fiber, low fruit and vegetable consumption, and high red meat consumption are associated with colon cancer and other cancers, and important causal effects of diet on cancer are likely. As with cardiovascular disease, this dietary trend that is commenced in childhood is likely to increase age-specific rates of colon cancer in adult life, but the risk may be reversed with later dietary change. *Am J Clin Nutr* 2000;72(suppl):1291S–6S.

KEY WORDS Diet, dietary fat, ischemic heart disease, stroke, cancer, childhood nutrition

INTRODUCTION

Diet is associated with several common adult diseases in Western countries, including cardiovascular diseases and several cancers. This short review concentrates on the epidemiologic evidence of an association between dietary fat and adult diseases, but considers also the health associations of a healthful or prudent diet in general, which is of a Mediterranean dietary pattern and consists of a high intake of fruit, vegetables, vegetable oils, and cereals, and a low intake of sugar, animal fat, and energy-dense foods.

We know more about the associations of dietary fat with cardiovascular disease than with cancer or other diseases for 2 reasons. First, serum cholesterol, and to a lesser extent clotting factor VII, is useful in dietary studies as an intermediate marker of cardiovascular disease risk; it is easier to show the effect of a dietary change on serum cholesterol than on death from cardiovascular disease. The available markers for cancer are less satisfactory. Second, the effect of dietary fat reduction on risk of ischemic heart disease is directly demonstrable in randomized trials because ischemic heart disease is common and its risk is rapidly reversible. Clinical trials record only a small number of cancers of any specific site and, even if more events were available, risk may be less rapidly reversible so that no such reduction can be shown during the few years of a trial.

The relation between dietary fat and cancer must therefore be judged mainly from prospective epidemiologic studies, or cohort studies, that relate cancer incidence to an initial dietary assessment. Such studies are limited by the difficulty in accurately measuring individual dietary consumption of saturated fat and other nutrients. The error in the dietary assessment of an individual is likely to be large in comparison with the variation in true saturated fat consumption between individuals, which is relatively small. This may introduce considerable attenuation of any association with disease (1). Moreover, the correlation between nutrients may be high, thus making it difficult to specify disease associations with any one nutrient. Even if a person's diet is measured accurately over a few days (ie, by weighed dietary inventory or preparation of duplicate meals), dietary fat on any one day, or even when measured over a few days, may poorly reflect an individual's long-term average value. It was shown 20 y ago that these errors may introduce substantial "dilution bias" such that the failure of a cohort study to show an association with disease may not negate an important relation (1). These problems of underestimation of fat consumption by some individuals and overestimation by others, and the relatively small variation between individuals in true dietary fat consumption, is

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TABLE 1
Estimates (from cohort studies) of the percentage decrease in risk of ischemic heart disease according to the extent of serum cholesterol reduction at age 60 y[1]

	Risk reduction
	%
Serum cholesterol reduction [mmol/L (%)]	
0.3 (5)	15
0.6 (10)	27
1.2 (20)	47
1.8 (30)	61

[1] Data from reference 2. The reduction in risk according to cholesterol reduction follows an exponential relation. The 15% reduction in risk with a 5% serum cholesterol reduction is equivalent to a relative risk of 0.85 (100% − 15% = 85%). Cholesterol reductions 2, 4, and 6 times as great as the relative risk are 0.85^2 (ie, 0.73 or a 27% reduction), 0.85^4 (ie, 0.53 or a 47% reduction), and 0.85^6 (ie, 0.39 or a 61% reduction).

a much smaller problem in dietary trials because the average fat consumption of a group is measured.

CARDIOVASCULAR DISEASES

The relation between dietary fat and cardiovascular diseases has been assessed directly in cohort studies, but as indicated above, dilution of the association can occur. The relation is best assessed by using serum cholesterol as an intermediary marker and separately determining the effect of dietary fat on serum cholesterol and the effect of serum cholesterol on risk of disease.

Ischemic heart disease

Serum cholesterol and ischemic heart disease

Cohort studies provide the best estimate of the size of the association between serum cholesterol and ischemic heart disease because they cover a wide age range and have a high statistical power. In addition, serum cholesterol differences between individuals recorded on entry to a cohort study will have been present for decades beforehand, so cohort studies can show long-term associations (2). Clinical trials, on the other hand, show the effect of short-term differences—how rapidly risk is reversed and to what extent the long term relation (shown in cohort studies) is reversed over the duration of a randomized trial (typically ≈5 y).

In an analysis of the 10 largest cohort studies conducted to date, it was shown that the relation between serum cholesterol and ischemic heart disease mortality is continuous and that a serum cholesterol reduction of 0.6 mmol/L (ie, ≈10%) is associated with a decrease in the risk of ischemic heart disease of ≈27% at 60 y of age (after correction for the regression dilution bias) (2). The effect of serum cholesterol reduction on decreased risk of ischemic heart disease, according to the extent of serum cholesterol reduction, is shown in **Table 1**. This proportional reduction in risk decreases with age, although the absolute benefit increases because the disease becomes more common with age.

To assess speed of reversal, data were analyzed from the "old generation" of 28 randomized trials in which the average serum cholesterol reduction was ≈0.6 mmol/L (ie, 10%) (2). The trial endpoints were ischemic heart disease death and nonfatal myocardial infarction. In the first 2 y there was little reduction in disease risk, with no reduction in the first year. From 2 to 5 y, the average risk reduction was 22% and after 5 y the reduction was 25%. The ischemic heart disease events in these trials mostly occurred at an average age of ≈60 y. The estimates of risk reduction in the short term can therefore be compared with the estimate of the long-term effect at 60 y of age from the cohort studies, which is 27% (Table 1). The similarity of the estimates of effect from the cohort studies and from the trial data from the third year onward therefore indicate that the reversal of risk is near maximal after 2 y—a surprisingly rapid effect. The reversal of risk according to trial duration was similar in trials that used diet or drugs to lower serum cholesterol (2); an impression that the reduction of risk was lower in the dietary trials arose mainly because the duration of some dietary trials was short. The large trials of statin drugs attained larger serum cholesterol reductions of 0.9–1.8 mmol/L (3–6), and these too showed a relatively small reduction in heart disease in the first 2 y, but a reduction after 2 y that was close to the maximum indicated by cohort studies (Table 1). All the randomized trial data indicate that the expected reduction in risk of ischemic heart disease, following small or large reductions in serum cholesterol, is largely attained after 2 y. The large statin trials (3–6) have confirmed the conclusion from an analysis of data from cohort studies and the earlier trials (2) that reduction in dietary fat or serum cholesterol does not cause an increase in mortality from noncirculatory diseases.

Dietary fat and serum cholesterol

The difference between dietary saturated fat and serum cholesterol in Japan and Britain is shown in **Table 2**. This comparison is a useful one because the differences in serum cholesterol between the 2 countries can be attributed to the differences in dietary saturated fat, because dietary polyunsaturated fat and dietary cholesterol are similar in the 2 countries. The dietary fat intake difference, which varies little with age, produces serum cholesterol differences that increase greatly with age. The tendency to generalize the results of dietary trials conducted in younger age groups to older age groups is therefore inappropriate (most of the trials were conducted in people <30 y of age and only a few trials were conducted in people >50 y of age). The comparison indicates that in older people, a reduction in dietary saturated fat equivalent to 10% of energy will lower serum cholesterol by ≈1 mmol/L and that randomized trials in older persons produce similar estimates of the same effect (2). The serum cholesterol reduction of 1 mmol/L will in turn reduce ischemic heart disease mortality in the long term by ≈40%.

TABLE 2
Serum cholesterol and dietary saturated fat in Japan and Britain[1]

	Japan	Britain	Difference
			%
Dietary saturated fat (% of energy)			
All ages	6	16	10
Serum cholesterol (mmol/L)			
20–9 y	4.5	5.0	0.5
30–9 y	5.0	5.6	0.6
40–9 y	5.1	6.0	0.9
50–9 y	5.2	6.2	1.0
60–9 y	5.0	6.2	1.2

[1] Data from reference 7.

trans-Unsaturated fatty acids illustrate the importance of serum cholesterol as an intermediate marker. Small randomized trials are sufficient to show that these fatty acids increase serum total and LDL cholesterol by about as much as the longer-chain saturated fatty acids (8, 9). It can therefore be concluded that their effect on cardiovascular mortality is similar to that of saturated fats, whereas showing this directly in a randomized trial would be a formidable undertaking. t*rans*-Unsaturated fatty acids constitute 6–8% of dietary fat (ie, 2% of energy) in Western diets; about one-third occurs naturally in animal fat and the remainder is generated by the hydrogenation of vegetable oils for use as hardening agents in manufactured foods.

Naturally occurring *cis*-unsaturated fatty acids reduce serum cholesterol by approximately half as much as longer-chain saturated fatty acids increase serum cholesterol, and monounsaturated fatty acids reduce cholesterol less than do polyunsaturated fatty acids. A reduction in dietary cholesterol has a small effect on blood cholesterol concentration, so that a substitution of *cis*-unsaturated fats for saturated fats in the Western diet would be the most appropriate change for lowering the high concentrations of blood cholesterol in Western populations.

A reduction in dietary saturated fat of ≈7% of energy, a realistic target for a population with a high intake of fat, would lower serum cholesterol by 0.6 mmol/L (≈10%), which in turn would reduce mortality from ischemic heart disease at age 60 y by 25–30% (2). The reduction in serum cholesterol that can easily be attained by an individual trying to alter his or her diet in isolation from family, friends, and workmates is relatively small (≈0.3 mmol/L or 5%). A larger serum cholesterol reduction, ≈0.6 mmol/L (10%), is realistic on a community basis, because the availability of palatable low-fat food increases when other family members or the community alter their diet, and the dietary change is perceived more positively. Reductions in serum cholesterol of ≥0.6 mmol/L through dietary change in Western communities have occurred in the United States and other countries. Implementing national and international policies on food subsidies that are linked to health priorities is important in achieving dietary change.

Stroke

Of the large cohort studies, the Multiple Risk Factor Intervention Trial, which screened 330 000 men for participation in the study, was particularly useful because hemorrhagic and nonhemorrhagic stroke were distinguished (10). Mortality from thromboembolic stroke and ischemic heart disease increased continuously in accordance with increases in serum cholesterol. The older cholesterol-lowering trials were uninformative on stroke because they recorded relatively few deaths and attained relatively small reductions in cholesterol. In the large statin trials, which are more numerous and are informative because they recorded nonfatal events and attained larger cholesterol reductions (3–6), the overall reduction in incidence over 5 y was 27% (95% CI: 0.11%, 0.4%; $P = 0.001$) (11), supporting the association shown in the cohort study. As in the heart disease trials, this 27% overall reduction in incidence is likely to reflect a composite of a smaller reduction in the first 1–2 y and a greater reduction in the longer term.

Peripheral arterial disease

In a large case-control study, the association between peripheral arterial disease and serum cholesterol was equivalent in magnitude to an increased risk of intermittent claudication of ≈24% for a 0.6 mmol/L increase in serum cholesterol (12). In the Scandinavian Simvastatin Survival Study trial (serum cholesterol reduction of 1.8 mmol/L), the incidence of intermittent claudication was reduced by 38% (13). Again, it is likely that a greater long-term reduction was diluted by the absence of an effect in the first 1–2 y.

Dietary fat and coagulation

Dietary fat increases blood concentrations of coagulation factor VII and increases the risk of thrombosis and hence myocardial infarction and cerebral thrombosis. Saturated and unsaturated fat increase factor VII to a similar extent; the increase appears directly related to the extent of postprandial lipemia (14, 15). The effect on risk is difficult to quantify, but the greater part of the effect of dietary fat on cardiovascular disease is likely to be mediated through serum cholesterol (2). This effect of dietary fat on coagulation is reversed in weeks.

Childhood nutrition and cardiovascular disease in adults

As discussed above, randomized trials show that serum cholesterol reduction by diet or drugs reverses the risk of ischemic heart disease, stroke, and peripheral arterial disease over a relatively short period of time. It might be argued, therefore, that a high-fat diet in childhood is of little consequence because its adverse effects are rapidly reversed if people reduce their fat consumption in adulthood. The same is argued in relation to teenage smoking. However, starting smoking at a young age increases risk in those who do not quit in adulthood. In carcinogenesis, the duration of exposure to a carcinogen, more so than dose, is of crucial importance; risk increases with the fourth or fifth power of duration, but only the first or second power of dose (16). Thus, age at first exposure is an important determinant of the risk of cancer—as has been shown, for example, in relation to smoking with lung cancer, asbestos with mesothelioma, and sexual activity with cervical cancer. Bringing forward the age of first exposure by, say, 10 y, will bring forward the age at which a person develops a cancer by about the same amount.

We recently suggested that similar considerations apply to serum cholesterol and ischemic heart disease (17). Across countries, current mortality from ischemic heart disease was more strongly correlated with dietary fat consumption and serum cholesterol concentrations 20–30 y ago than with present-day measurements of serum cholesterol (17). In Britain, national animal fat consumption increased and reached its peak ≈30 y ahead of the corresponding increase and peak of ischemic heart disease mortality. This was analogous with the increase in smoking in a population, which was followed by an increase in lung cancer mortality ≈30 y later (17). The process of atheroma, like cancer, begins at a young age and decades elapse before atheromatous lesions are sufficiently large or extensive to induce coronary or cerebral thrombosis. On a "duration of exposure" basis, dietary fat and serum cholesterol concentrations in childhood may directly influence the risk of cardiovascular disease later in adult life. As with smoking, exposure at younger ages may be of relatively minor importance if the exposure is reversed at older ages, but it the exposure is not reversed, a longer duration of exposure may substantially increase disease risk.

CANCER

The incidence of most cancers varies greatly in different populations, confirming the general importance of environmental

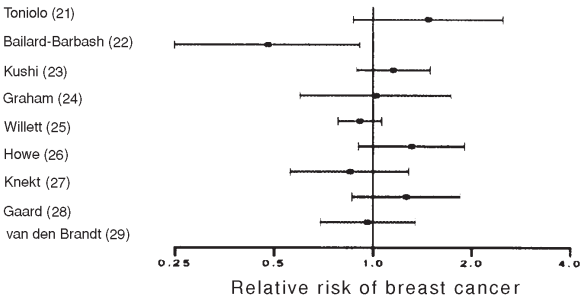

FIGURE 1. Relative risk (95% CI) of breast cancer in women at the 90th percentile of dietary fat consumption compared with those at the 10th percentile in 9 cohort studies (21–29). A relative risk > 1.0 suggests a higher risk of cancer with greater fat consumption.

factors in carcinogenesis (16). It is estimated that about one-third of cancer risk in Western countries is attributable to diet (16). As discussed above, it is more difficult to specify and quantify the relations of diet with cardiovascular disease than with cancer. The randomized trials of dietary fat reduction did not record sufficient cases of site-specific cancers to be informative and in any event, the duration of these trials was probably too short to show a reduction. Epidemiologic studies provide the main evidence. Case-control studies are subject to recall bias, as discussed below. Prospective, or cohort studies, in which diet is assessed in a cohort of healthy persons who are then followed up for the development of cancer, provide the best evidence, albeit subject to dilution of any association because of inaccurate dietary measurement and confounding by correlations between nutrients, as discussed above. The best guide to an important relation between dietary fat and cancer is therefore an association shown consistently in cohort studies, supported by evidence of a plausible mechanism of effect. In addition, although there is no intermediate marker for cancer risk that is as useful as serum cholesterol for ischemic heart disease, circulating estrogens provide some intermediate measure of risk for breast cancer as large bowel polyps do for colorectal cancer.

The relation strength between dietary factors and cancer was expressed in epidemiologic studies by dividing the cohort, or the control subjects in a case-control study, into subgroups of generally 4 or 5 according to ranked measurements of nutrient intake. The regression coefficient would then be expressed across the subgroups as the incidence in the highest level relative to that in the lowest level of nutrient intake (approximately the risk at the 90th percentile relative to the risk at the 10th percentile of intake) and adjusted for differences in the consumption of other nutrients.

Breast cancer

Case-control studies show an association between dietary fat (ie, essentially animal fat) and breast cancer (18–20). In a meta-analysis published in 1993, of 16 studies with 6831 breast cancer cases, the summary odds ratio was 1.21 (95% CI: 1.10, 1.34) (19). This suggests that the risk of breast cancer was ≈21% greater at the 90th percentile than at the 10th percentile of fat consumption. An earlier analysis produced a similar result (20). However, in cohort studies, in which the measurement of dietary fat consumption was obtained before the onset of illness, there was no association between fat intake and risk of breast cancer (**Figure 1**). Of 9 cohort studies conducted to date, the relative risk estimate was < 1.0 in 4 studies, > 1.0 in 4 studies, and 1.0 in 1 study (21–29). A 1996 meta-analysis of 8 cohort studies (4980 cases) confirmed the absence of an association: the summary relative risk estimate was 1.02 (95% CI: 0.94, 1.11) (30). There was no association with either saturated fat (relative risk: 1.03; 95% CI: 0.95, 1.11), monounsaturated fat (relative risk: 0.99; 95% CI: 0.90, 1.08), or polyunsaturated fat (relative risk: 1.03; 95% CI: 0.95, 1.12) (30). The largest cohort study, the American Nurses Health Study with 1439 breast cancer cases, showed no association with total fat (relative risk: 0.90; 95% CI: 0.77, 1.07) or with saturated fat (31). Moreover a large case-control study of 820 cases showed no association (32).

The difference in results between the cohort studies and most of the case-control studies suggest that the association in the case-control studies may have been due to recall bias (ie, women with breast cancer tending to exaggerate their dietary fat intake to account for their illness) (18). This was confirmed by an analysis of data from the Nurse's Health Study. A dietary questionnaire was initially completed by the entire cohort on entry to the study and then later, a dietary questionnaire was sent to a subset of 300 women after they developed breast cancer and to 600 control subjects (33). The assessment of dietary fat intake was greater in the retrospective questionnaire that was completed by women after the diagnosis of breast cancer than in the questionnaire completed by the same women at the beginning of the study. This was particularly evident in the cases, which suggests that the retrospective data show an association of dietary fat intake with the incidence of breast cancer (odds ratio 1.4), but that the prospective analysis did not (odds ratio 0.87). Individual dietary assessment is subjective and is affected by knowledge of illness.

Blood concentrations of estrogens, estradiol in particular, are associated with risk of breast cancer. Cohort and case-control studies consistently show an association between free and albumin-bound estrogens that are available for tissue uptake and breast cancer (18). There is, however, no association with estrogen bound to sex hormone binding globulin. After menopause, circulating estrogens are derived from the aromatization of androstendione produced by the adrenals and the ovary; this takes place in peripheral tissues and particularly in adipose tissue. There is consistent evidence in epidemiologic studies of an association between body mass index and postmenopausal breast cancer (18), which may reflect this synthesis of estrogen in adipose tissue in postmenopausal women. On the other hand, intervention studies that examine the effect of low-fat diets on circulating estrogen concentrations in postmenopausal women do not collectively show any material change in estrogen concentrations (18). Hence, the evidence of estrogen does not support a specific, direct effect of dietary fat and increased risk of breast cancer, but does show an effect of high energy intake, body fat, and obesity on increased breast cancer risk.

Younger age at menarche is associated with a higher risk of breast cancer and increases lifetime exposure to estrogens. There has been a decrease in the average age at menarche in Western countries; this together with the increase in body size and body fat has been attributed in part to nutritional factors. It has been argued that overnutrition in early life causes rapid growth resulting in early menarche and in turn an increased risk of breast cancer (34). There is evidence from cohort studies, after control for body size and energy intake, that higher consumption of grains, nuts, and legumes is associated with later menarche and higher consumption of meat with earlier menarche (18).

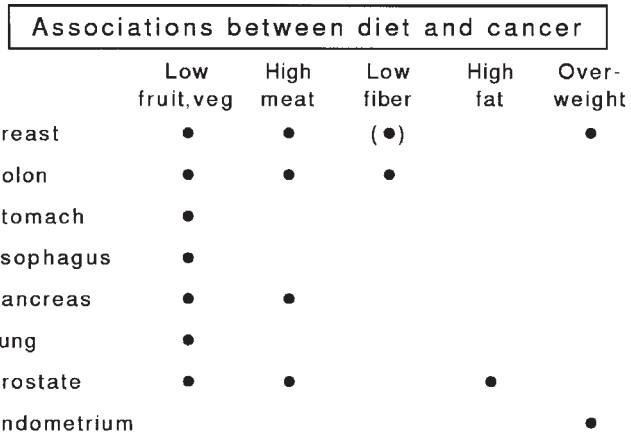

FIGURE 2. A summary of the associations between diet and cancers indicated by the results of cohort studies after adjustment for correlations between nutrients and other confounding factors. Veg, vegetable.

Colon cancer

The 8 published prospective (cohort) studies on a possible relation between total dietary fat intake and colon cancer show no association between the 2 (18). The Nurse's Health Study suggests an association, as the risk in the highest one-fifth of animal fat intake was almost double that in the lowest (35, 36), but the association disappeared when controlled for red meat consumption (37). Six studies showed relative risk estimates close to 1 (18), and one study found a negative association; risk was low in those persons whose dietary fat intake was high (38). Most case-control studies report an increased risk of colon cancer in individuals with high dietary fat intake, but this risk often disappeared after adjustment for energy intake (36). Adenomatous polyps were examined as a marker for risk of colon cancer: one cohort study suggested an association with saturated fat, while another study did not (18). In a randomized trial, a reduction in dietary fat showed no detectable reduction in the number of patients with polyps or in the number of polyps with dysplastic change (39). One must conclude that there is no strong relation between dietary fat and colon cancer. A weak association cannot be excluded. The association in case-control studies when present, as with breast cancer, is likely to be due to recall bias.

Cohort and case-control studies show an association between high dietary intake of fiber (ie, nonstarch polysaccharides) and a low risk of colon cancer (18, 40). Mechanisms of effect include the faster bowel transit time and the dilution of carcinogens by increased fecal bulk. Also, colonic bacterial flora metabolize bile acids to the secondary bile acid deoxycholic acid, which may promote bowel cancer in rodents; this conversion of primary to secondary bile acids is decreased with diets high in starch and fiber (18). A randomized trial suggested a reduction in the number of large-bowel polyps showing dysplastic change in patients consuming bran (6 in subjects taking bran compared with 10 in control subjects) but the difference was not statistically significant (39).

Cohort studies indicate that vegetable consumption is directly associated with a lower risk of bowel cancer and that dietary fiber alone does not account for this association (18, 37). Meat consumption is also directly associated with risk of bowel cancer in cohort studies (18, 37), but the interpretation of the association is uncertain. Comparisons of American Seventh-day Adventists who do and do not eat meat and of orders of British nuns who do and do not eat meat suggest no reduction in the incidence of colon cancer in the vegetarian groups (40); however, those who ate meat consumed only moderate amounts and the studies could not exclude an excess risk of the order of 50%.

Prostate cancer

Ten case-control studies show an association between prostate cancer and dietary fat, with the association being statistically significant in 5 of those studies (18). Four prospective (cohort) studies show an association of prostate cancer and animal fat consumption (18); the summary relative risk estimate is 1.31 (95% CI: 1.04, 1.65; $P = 0.02$). No plausible mechanism to explain a cause and effect relation has been proposed and the interpretation of such results is uncertain.

Summary of cohort studies of diet and cancers

A summary of the relations between foods or nutrients and breast, colon, prostate, and other specific cancers that can be supported by the results of cohort studies is shown in **Figure 2**. Some of the associations are supported by plausible mechanisms of effect; as discussed above, these include the relation between body mass index and breast and endometrial cancer mediated by estrogens, and the relation between dietary fiber and bowel cancer. In other cases, the interpretation is more uncertain. For example, the association between low fruit and vegetable consumption and stomach cancer may be due to confounding by socioeconomic status because poorer people eat less fruit and vegetables and their prevalence of *Helicobacter pylori* infection is high.

Polyunsaturated fat

There is a focus on polyunsaturated fats because they are prone to oxidation, which generates free radicals, and because feeding experiments in animals have suggested that polyunsaturated fats increase the incidence of mammary and intestinal tumors (41). However, there is no evidence that polyunsaturated fatty acids cause cancer in humans (41). Cohort studies showed no excess risk of breast, colon, or other cancers with a higher consumption of polyunsaturated, monounsaturated, or saturated fats (18, 19, 30). A large case-control study of 1953 patients with colorectal cancer suggested, if anything, a protective effect of polyunsaturated vegetable oils and olive oil against certain cancers and excluded more than a trivial excess risk (42).

Childhood nutrition and cancer in adults

The evidence does not favor a direct cause and effect relation between dietary fat and cancer. It is likely that the risk of cancer is reduced when dietary fat is reduced as part of a general change toward a diet high in dietary fiber, high in fruit and vegetables, and low in red meat, but less likely with a diet based on modern processed foods that may be low in fat but also low in fiber, fruit, and vegetable extracts. The dietary trend in Western countries, when commenced in childhood, is likely to increase age-specific rates of cancer in adult life because of the importance of duration of exposure, as discussed above (16); however this increased risk might be reversed if diet is changed in adult life. The trends toward increased energy intake and body size, and relatively low dietary fiber intake during childhood, contribute to the decreasing age at menarche, which is associated with an increased risk of breast cancer.

CHRONIC CONDITIONS OTHER THAN CIRCULATORY DISEASE OR CANCER

Various chronic conditions, although not necessarily linked to high consumption of dietary fat, are linked to the general dietary trend in Western countries, ie, increased consumption of animal fat and sugars, reduced consumption of dietary fiber, and increasing body weight and obesity. These include diabetes, gall bladder disease and gall stones, constipation, and osteoarthritis. Although specific evidence is limited, it is reasonable to expect, as with cardiovascular disease and cancer, that the duration of exposure to a diet high in fat is important in determining the age-specific prevalence of complications, eg, diabetes or obesity.

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