Journal of Nutrition, Growth and Cancer (1985) 2, 173–179

DIETARY FATS IN HEALTH

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This review examines the relationship between dietary fat intake and health. Meat produced by modern farming methods has a high fat content in comparison with its natural counterpart and, furthermore, the fat is low in polyunsaturated fat. The combined result of this and dairy food is that our modern diet has far more fat than was consumed by stone-age man. A number of diseases are associated with the Western lifestyle and thus might be caused by a high fat intake. With regard to coronary heart disease, there is a better correlation with refined carbohydrates than with dietary fat. Prospective and case-control studies also indicate that dietary fat is of secondary importance. Human and animal studies show that a diet high in fat is a major factor in cancer of the breast and, to a lesser extent, in cancer of the colon. For obesity and diabetes there is contradictory evidence for the involvement of dietary fat. A high fat diet reportedly has a deleterious effect on the immune system, therefore possibly constitutes a factor in autoimmune disease.

INTRODUCTION

During the vast majority of human evolution man was a hunter-gatherer. The advent of agriculture some 8 000–10 000 years ago is too recent for any significant degree of further evolution. It is reasonable to presume, therefore, that modern man is adapted to the diet of his stone-age forebear. This diet contained much plant material (1) which, no doubt, was rich in fiber and therefore resembles modern fruit, vegetables and unrefined cereals. In addition, much meat was eaten from wild animals (2–4).

It is often assumed that all meat is similar and that man is therefore well adapted to a diet rich in saturated fats. Crawford *et al.* (5,6) compared meat from herbivorous mammals in Uganda with typical butcher's shop meat purchased in London. Whereas the Ugandan meat had a fat content of 2–5%, in London the figure is around 20–40% (before removal of visible fat). Other data for mammalian meat sold in Britain indicates a typical fat content of 16–31% (5–11% in major organs) (7). By comparison Ethiopian meat has been reported to contain only 1.3% fat (8) (ie even less than Ugandan meat). In addition to these quantitative differences the fat of African meat is much more rich in polyunsaturated fatty acids (PUFA): 30% in Ugandan wild meat (5,6), while the reported value for London meat is only 2–4% (5,6) or 4.5% (9–15% in major organs) (9). It is apparent therefore that modern farming methods have greatly increased the fat content of meat and that this fat has a low ratio of PUFA to saturated fat (P/S).

It is only since the advent of agriculture that dairy foods have been available in significant quantities. They are, of course, another major source of fat with a low P/S ratio. The combined effect of meat and dairy food clearly means that the average Western individual now consumes far more fat than did his distant ancestor.

Evolutionary considerations tell us that because man evolved on a low fat diet, this is the type of diet for which we are most suited. Thus, our modern-day fat-rich diet might possibly be a cause of disease.

The writings of Cleave (10,11) stand as one of the great pillars, albeit poorly recognized, of twentieth century nutrition. He, however, stresses that man is adapted to a high fat diet as indicated by the availability of fat-rich meat in biblical times. However, we would argue that this is too recent to be of relevance.

CORONARY HEART DISEASE

The proposition has been made elsewhere (12) that the role of a high fat diet in coronary heart disease (CHD) has been much exaggerated. In brief, the cornerstone of the lipid-hypothesis of CHD is the variation, both geographic and temporal, between dietary fat and CHD. However, there are numerous exceptions to this relationship which throw the whole hypothesis into doubt. Coronary heart disease has, in fact, a stronger relationship with refined carbohydrates. Much of the relationship between dietary fat and CHD may well be due to guilt by association—both being closely correlated with refined carbohydrates (12).

Thus, a high fat diet would appear to have a secondary role in CHD. This is consistent with findings from epidemiological diet and drug intervention studies and evolutionary theory.

Reports from six prospective studies support this conclusion. In each study middle-aged men had their diet assessed and this was related to subsequent CHD. While there may be some tendency for persons who subsequently suffer CHD to consume a diet with an above average proportion of calories derived from fat or with a below average ratio of P/S, this finding is weak and is not consistent (13–19).

Case-control studies of CHD are less reliable than prospective studies since fatal cases are excluded and diet assessment is more likely to be biased in the first type of design. A further problem is that data are usually presented as the absolute intake of dietary components rather than as the nutrient:calorie ratio. This can be misleading since CHD cases tend to have a below average calorie intake (13–16, 19).

The results from several case-control studies have indicated minimal differences in fat intake (20–26). Bassett *et al.* (20), in a study in Hawaii, found the P/S ratio about 12% higher among cases of Japanese origin but no different in cases of Hawaiian origin (versus respective controls). Moore *et al.* (27) determined the former diet of 253 deceased men aged 20 to 60 and compared it with the degree of advanced atherosclerosis. Expressed as a nutrient:calorie ratio, total fat was positively related ($P < 0.05$) as were cholesterol and saturated fat (but not at statistically significant levels).

Overall, there may be a tendency for CHD sufferers to have a history of an above average intake of fat or of a low P/S ratio, but if this is so, it is weak. By way of contrast, the results for other dietary variables, notably calorie intake and alcohol use, have been more pronounced and more consistent.

CANCER

Cancer of the breast and colon are the two forms of the disease most associated with a high fat intake.

Epidemiology provides a major component of the evidence incriminating a high fat diet in the etiology of breast cancer. Strong correlations are seen in both international and intra-national studies (28). However, as with CHD, this might easily be a case—to a greater or lesser extent—of guilt by association. On the other hand, case-control investigations support a true role for a high fat diet in breast cancer (29,30).

Studies of chemically induced and spontaneous mammary tumors in animals are consistent with human studies in indicating an important role for dietary fat in the genesis of this disease (28,31). This occurs principally in the promotional rather than in the initiation phase of carcinogenesis.

Colon cancer studies parallel those on breast cancer, although the evidence is less strong. An international correlation between dietary fat intake and cancer of the colon has been demonstrated (28,32) but suffers, as do the CHD correlations, from serious inconsistencies (33,34). In particular, the finding might reflect a primary role for a low intake of dietary fiber (35,36).

Case-control studies tend to support the involvement of a high fat diet in the disease (37,38) but as with the epidemiological data, there is a lack of consistency (39,40). Investigations using animal models of cancer of the colon have mostly (28), but not invariably (41), reported a positive association of the disease with dietary fat.

OBESITY

There is no doubt of the close association between a Western lifestyle and obesity (42). Unlike the situation with CHD and cancer of the breast and colon, the geographical distribution of obesity is seldom cited as evidence indicating a major role for dietary fat. This almost certainly stems from the lack of reliable quantitative data concerning the prevalence of obesity. The association with refined carbohydrates is at least as good as that with dietary fat.

Several studies have compared the dietary intake of the obese with that of their non-obese counterparts, but no consistent trend emerges in relation to fat intake (eg 43–48). Much caution must be observed in interpreting such data since not only is diet assessment particularly prone to bias but it is also difficult to disentangle cause and effect relationships.

Studies of the effects of an altered dietary fat content on calorie intake also argue against obesity being caused by a high fat diet. Altering the dietary fat content for a period of two weeks was found to leave total calorie intake unchanged (49).

A high fat, low carbohydrate diet has been in vogue for some years as a treatment for obesity. More rapid weight loss has often been observed with this approach than with diets containing a more conventional distribution of calories between fat and carbohydrate (50,51). Before this can be cited as evidence against a role for fat in obesity, two areas of doubt must be resolved. First, such diets often reduce carbohydrate intake to an abnormally low level thus inducing ketosis (52). Weight loss under such metabolic conditions may possibly have no bearing on the question of whether dietary fat level is a factor in obesity. Secondly, contradictory results have been reported on the extent to which the extra weight loss is merely a loss of water (51,53).

Experiments during the last 30 years have shown that a high fat diet induces excess weight gain in rats (54–56), mice (55,57,58); (Temple & El-Khatib, unpublished observations) and hamsters (59). This is probably the only area that supports a role for excess fat consumption in obesity.

MATURITY ONSET DIABETES

Diabetes has a close epidemiological relationship to CHD, obesity and a Western lifestyle (42,60), and the association with refined carbohydrates is closer than with dietary fats (11). For instance, Bang *et al.* (61) report that, although Greenland Eskimos eat a high fat, low carbohydrate diet, diabetes is unknown.

Studies on individuals have, in general, not revealed an association between dietary fat intake and an impaired glucose tolerance (44).

Solid evidence shows that the most suitable diet for maturity diabetics (type II) is one restricted in fat with the calories being supplied mainly by foods rich in carbohydrates and dietary fiber (62,63). If, then, such a diet is an effective therapy, it could be logical to argue that a high fat diet is a causative agent. Such a conclusion apparently contradicts the above mentioned data. Thus, the true role of dietary fat in diabetes is unknown.

THE IMMUNE SYSTEM

The level of dietary fat affects the functioning of the immune system (64): a depressed responsiveness after consuming a diet rich in unsaturated fat being the most commonly reported effect (65,66).

It is possible that autoimmune diseases are related to a high fat diet. They are thought to be associated with a Western lifestyle, but the evidence to this effect is meagre (67,68). In an uncontrolled trial, six patients with rheumatoid arthritis experienced greatly reduced symptoms on a diet very low in fat (69). Similarly, a low (1%) fat diet greatly reduced the spontaneous development of one autoimmune disease (immune complex nephritis) in a susceptible strain of mice (70).

CONCLUSION

On balance, a high fat diet has a deleterious effect on health. Although this conclusion is supported not by clear proof that dietary fat is a true cause of specific diseases, there are strong grounds for suspicion in relation to several diseases. Dietary prudence would, therefore, seem appropriate.

NOTE ADDED IN PROOF

In an excellent article Eaton and Konner (71) have attempted to calculate the fat content of the paleolithic (stone-age) diet. Assuming that meat provides 35% (by weight) of food, the % of energy derived from fat is 21 with a P/S of 1.41. Even if the diet is 80% meat, the fat content is still only 25% and the P/S is 0.91.

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