CORONARY HEART DISEASE - DIETARY LIPIDS OR REFINED CARBOHYDRATES?

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ABSTRACT

The epidemiological evidence associating dietary lipids, refined carbohydrates and coronary heart disease (CHD) is evaluated. Population studies often show a high correlation between dietary lipid and heart disease mortality. It is argued that most of this association is secondary to the high correlation that refined carbohydrates have with dietary lipids, on the one hand, and with CHD on the other. This becomes apparent only when examples are found of a weak or negative correlation between dietary lipids and refined carbohydrates. Evidence from diet and drug intervention studies support the hypothesis that dietary lipid is only of secondary importance in CHD. It is concluded that refined carbohydrates are of primary importance.

A. INTRODUCTION

The lipid hypothesis of CHD has dominated investigations of this subject for many years. A crucial part of the evidence cited in its support is epidemiological (1,2). This evidence is reinterpreted as supporting an alternate hypothesis, namely that CHD is caused mainly by refined carbohydrates (3). Sugar, being the most refined carbohydrate, is consequently the most harmful, but refined starches (white rice, white flour and related products) also play a crucial role. An excess of dietary cholesterol and fat (particularly with a low ratio of polyunsaturated: saturated fats, P/S) plays a much lesser role.

In both comparative studies of populations and historically within many Westernised populations the consumption of refined carbohydrates closely parallels that of total fat as well as of saturated fat and cholesterol. Consequently, there is a most misleading high correlation between fat consumption and CHD. Merging the intake of refined and unrefined carbo-

hydrates, as is the usual practice, further confuses the picture. However, when examples are found of a dissociation between the intake of fat and refined carbohydrates, then it is the latter that maintains its association with CHD.

Cleave (3) has proposed a 30 year rule, being the approximate incubation period between the exposure of a population to refine carbohydrates and the appearance of CHD. During this period a pattern of other diseases, including diabetes mellitus, emerges (4).

B. COMPARISONS OF POPULATIONS

a) Multi-National Studies

Numerous multi-national studies have compared the average intake of dietary components with CHD mortality (1,5-7). They illustrate the weakness of correlation studies, namely the demonstration of "association not causation". They typically show a high correlation of age - adjusted CHD mortality with fat (and often saturated fat and cholesterol), sucrose and sometimes income. The refined cereals are invariably merged with unrefined ones.

A report by Knox (8) illustrates the limitation of this approach. In 20 advances countries the same pattern of positive and negative correlations for many foods tended to recur over a range of diseases. Thus meat, milk, eggs, butter and sucrose were positively correlated with mortality from CHD, cancer of the colon, breast and leukemia, while cereals, vegetables, pulses, nuts and fish were all negatively correlated with these diseases. These food items showed similar correlations, but with the opposite sign, for another group of diseases which included cancer of the stomach, chronic nephritis and peptic ulcer. Furthermore, refined sugar and total animal fat had a stronger correlation with each other (r=0.90) than with CHD (0.74, 0.69 respectively). Knox (8) draws the following conclusion, "No specific foodstuff, no foodstuff group, and none of the main calorie-providing nutrients can be related on the basis of these data as primary determinant of death rates from ischaemic heart disease, hypertension, or cerebrovascular disease".

In populations where the intakes of fat and sucrose are closely correlated and where figures on the intake of refined starches are unavailable (and even if they were, would probably parallel the intake of fat), it will clearly be impossible to demonstrate which dietary components (if any) are causally linked to CHD. The rest of this paper examines populations which provide a better insight into the relationship between diet and CHD. In particular, examples are given where the intake of fat and refined carbohydrate are not correlated with each other.

b) __Polynesia__

Hunter (9) reported on Polynesian inhabitants of the Cook Islands. The Atiu-Mitiaro are non-Europeanized but consume a fairly fat-rich diet (33 percent of calories). The fat is mainly saturated being 89 percent coconut oil and 5.5 percent animal fat. Among adult males the mean serum cholesterol was 6.10mM (236 mg/dl) (i.e. similar to Western levels) yet no evidence or myocardial infarction could be found (using clinical and ECG evidence).

In a similar study Prior et al (10) described the inhabitants of Tokelau, also in the Cook Islands. Their major foodstuff is coconuts (63 percent of calories) while imported refined carbohydrate are of minor importance (6 percent of calories). Fat supplies 54 percent of calories of which 89 percent is saturated and only 3.7 percent is polyunsaturated. Adult males have a mean serum cholesterol level of about 5.56mM (215mg/dl). Unfortunately, detailed information concerning CHD is not available but the incidence of ECG abnormalities is described as "very low".

c) __St. Helena__

In the case of the two Polynesian populations a prediction of the CHD incidence using the lipid hypothesis would greatly overestimate the actual figure. In the case of St. Helena the situation is the precise opposite.

The inhabitants of St. Helena were studied by Shine (11). They have a lower fat intake than most Westernized people (29 percent of calories), smoke less but exercise more. Nevertheless, CHD is almost as common as in Britain (based on death certificates and ECG findings). Starch is predominantly bread and to a lesser extent rice, both of which are almost certainly refined. This and the high sucrose intake is the apparent explanation.

d) __Natal Indians__

A similar example is provided by the Indians of Natal, South Africa. They have been reported on by Campbell (12) and Cleave (3). Their fat intake is mostly from processed vegetable oils. Nevertheless, CHD is common and this has been linked to their high intake of sucrose and other refined carbohydrates.

e) __Israel__

A third population where CHD is far more prevalent than the lipid hypothesis would predict is provided by Israel. The average Israeli has an adipose tissue P/S ratio of approximately 1.0 (a figure close to the dietary level) and is the highest reported in the world for a

free-living population (13). Despite this the Israeli CHD mortality is similar to that in most Western countries. Comparison with France and Belgium during the years 1960-62 reveals that the per capita consumption of fat was about one third greater and of saturated fat about twice as great as in Israel (6). Nevertheless, Israel had a far larger CHD mortality rate in later years (14-16).

f) Kenyan Indians

Among the Indian communities of Kenya a substantial section are vegetarians and tend to consume non-hydrogenated vegetable oils in place of animal fats. Nevertheless, Charter and Arya (17) reported that when different groups were compared there was no relationship between vegetarianism and male CHD incidence.

g) Trappist and Benedictine Monks

The present example resembles the previous one in that major differences in dietary fat appear to have manifested negligible impact on CHD.

Groen et al (18) compared two groups of monks living in Holland and Belgium. Whereas the Benedictine monks lived on a fairly typical Western diet. The Trappists consumed a vegetarian one which contained considerably less total fat, saturated fat and cholesterol but more carbohydrate. Not surprisingly the Trappists had a significantly lower serum cholesterol level yet their incidence of CHD was no different.

h) Ireland - US Study

Brown et al (19) studied 1994 middle-aged men of Irish origin. This included 575 in Ireland, their 579 brothers who had emigrated to Boston, USA, plus another 840 who were unrelated and were born in Ireland or the USA. Their diets were similar with respect to cholesterol intake and percent of calories as fat although in Boston the P/S was slightly raised. However, there was little difference between the groups in the level of serum cholesterol or blood pressure. Those living in Boston had a much higher CHD incidence. This is explained in part by the fact that they were more obese, less physically active and smoked slightly more cigarettes. It is significant, however, that those living in Ireland ate much more of their diet as starch and consequently consumed about 65 percent more fibre indicating their carbohydrates were generally less refined.

i) India

Malhotra (20) compared two groups of railroad sweepers in India, one from the South (Madras) and the other from

the North (Udaipur). The Southern group had several times more CHD and only diet seems able to explain this as other differences were trivial. The Northern group consumed a far larger proportion of their calories as fat (23 percent, mainly short chain fatty acids of milk and ghee) than in the South (3.5 percent, mainly long chain PUFA of seed oils) but serum lipid levels were similar.

The explanation for the huge difference in CHD lies in the major source of calories, namely unrefined wheat in the North and refined rice in the South (3). Sucrose intake was much greater in the North (though low by Western standards) and this indicates that its influence is out-weighed by un-refined cereals when they are in a large excess.

C. TRENDS IN CHD INCIDENCE

As societies become wealthier there are usually sharp increases in both fat intake and CHD mortality. This has often been cited as evidence supporting the lipid hypothesis. However, it ignores the usual accompanying changes, including a shift from unrefined to refined cereals and from starch to sucrose. During the Second World War these trends were reversed so that the intake of both animal fat and total fat fell in many countries as did CHD. Qualitative information concerning carbohydrates is generally missing but it is highly likely that cereals were less refined (to reduce wastage) while sucrose was less available. This was certainly the case in Britain. Thus, as with geographical trends, interpretation of the evidence is hazardous. Examples are presented where changes in consumption of fat and refined carbohydrates have not altered in parallel.

a) Multi-National Study

Masironi (6) calculated the ratio of dietary intakes for 23 countries for the years 1962 and 1947. This revealed only a weak correlation between changes in dietary intake of sucrose, on the one hand, and fat and saturated fat, on the other. The correlation of these changes with the ratio of atherosclerotic heart disease death rates 1965:1955 (both sexes, all ages) was much stronger for sucrose (r=0.43) than for fat (0.18) and was actually negative for saturated fat (-0.27). Thus changing mortality resembled preceeding changes in the consumption of sucrose but not of saturated fat.

b) Canadian Eskimos

Schaefer (21) reported the dramatic rise in the incidence of atherosclerosis and diabetes among Canadian Eskimos following their urbanization. Their main dietary changes was a massive rise in sucrose consumption at the expese of cereals to over 20 percent of calories and this is the most plausible

explanation. During the same period there was a fall in calories due to a reduced intake of animal protein while fat and carbohydrate levels were stable. It should be noted, however, that the dietary P/S possibly fell sharply on urbanization (22).

c) Yemenite Jews

Cohen (23,24) studied Yemenite Jews who emigrated to Palestine (Israel). Atherosclerosis, CHD and diabetes were extremely rare among those who had arrived within the previous 10 years but were common among those who had been settled for 25 years or longer. The principal dietary shift following immigration was a sharp increase in sucrose (previously rare) and vegetable oil while animal fats remained fairly constant. Clearly, the consumption of sucrose manifested a much greater impact than an increased dietary P/S.

d) United States

The true change in the CHD death rate in the USA since the first decade of this century is not known but is generally acknowledged to have risen greatly. Between 1909 and the mid-1970's per capita consumption of fat increased 26 percent due mainly to increases in vegetable fat (up 181 percent) rather than saturated fat (up 11 percent). Thus the P/S ratio went up from 0.21 to 0.43 (0.31 in 1961). Cholesterol intake rose by less than 10 percent (25-27). Kahn (28) estimates that the rise in the average serum cholesterol (1909-1913 to 1965) was no more than 0.10mM (4mg/dl). Even allowing for the large increase in tobacco consumption, the lipid hypothesis apparently fails to predict the actual CHD change.

During this century the US carbohydrate intake has become far more refined. Since 1909 crude fibre intake has declined by 28 percent (29) and cereal fibre by about half (29,30) while sucrose consumption rose considerably (26,27). These changes in carbohydrate intake readily explain the CHD trend.

e) Switzerland

Guberan (31) reported a marked drop in CHD mortality among Swiss women between 1951 and 1976. During this period hepertension became less common, smoking among women increased while consumption of animal and vegetable fats rose by similar amounts. However, the rise in animal fats represented a 46 percent reduction in milk intake which is not hypercholesterol-aemic (32), more than offset by rises in consumption of pork, eggs and milk products. The diet therefore shifted to the hypercholesterolaemic long chain saturated fatty acids with, apparently, no adverse effect on CHD.

D. DIET AND DRUG INTERVENTION STUDIES

a) Diet Studies

These provide another avenue for directly exploring the relationship between diet and CHD. Numerous studies have been made in which blood cholesterol has been lowered, generally by increasing dietary P/S. In comparison with controls, subjects on a hypocholesterolaemic diet have often shown a significant reduction in new coronary events. However, there is no clear evidence for a significant drop in total mortality (the whole object of the exercise) (33).

More recently three trials have all yielded essentially negative results. These are secondary prevention trials in Sydney (34) and Tutzing, West Germany (35) and a primary trial in Minnesota (36). In each the experimental diet had a similar fat intake to the control one but with a raised P/S and lowered cholesterol content.

b) Clofibrate Studies

Use of the hypocholesterolaemic drug clofibrate has also been of dubious benefit. A secondary prevention trial in the United States failed to significantly reduce fatal or non-fatal CHD (37). A primary prevention trial in Edinburgh, Budapest and Prague achieved a significant reduction in non-fatal myocardial infarcts of 25 percent while fatal CHD was not significantly affected (38). However, after termination of drug treatment followed by an average of 4.3 years of further observation, subjects previously on clofibrate had a 10 percent higher mortality rate for CHD (39).

c) Comments

According to Keys (40), "...a major lesson gained from World War II is the proof that in a very few years the incidence of CHD could drop to a level of the order of one-fourth the preceding rate". Clearly, the efforts of the world's leading cardiologists have been far less successful in reducing CHD than were the chaotically run food distribution services in the last world war. On this basis diet and drug intervention studies suggest the lipid hypothesis is perhaps true but only to a minor extent.

E. CONCLUSIONS

i) While one can readily point to examples of populations having a diet and CHD incidence "supporting" the lipid hypothesis, one can equally well point to numerous exceptions. This brings the whole hypothesis into question.

ii) Most epidemiological studies have made no attempt to distinguish between refined and unrefined cereals. The consumption of refined cereals probably parallels that of sucrose, fat and cholesterol.

iii) Between populations the correlation of fat and cholesterol intake with CHD can be explained as being little more than secondary to the correlation of fat and cholesterol with refined carbohydrates.

iv) It is only in the unusual circumstances of refined carbohydrates and fat not moving in parallel (historically or geographically) that the above statements can be demonstrated.

v) Intervention studies using diet or clofibrate to reduce blood cholesterol levels are consistent with the lipid hypothesis being true to a minor extent.

vi) In summary, on the basis of human studies the lipid hypothesis may be correct to a minor extent. Refined carbohydrates have been far less extensively examined, warrant much greater attention and appear to play a greater etiological role than dietary lipids.

Acknowledgement

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