In search of causes of ischaemic heart disease

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Sugar intake, dietary fat consumption, especially saturated fats, physical activity at work, serum levels of cholesterol, and smoking have been compared in 44 patients with acute myocardial infarction, 44 healthy controls, and 88 patients suffering from other diseases. All were men between the ages of 35 and 58 years. They were matched for age, social class, and work or trade. No differences were found between the three groups in these parameters. Significant differences were present in the chain length of fed fats between patients with myocardial infarction on the one hand and the 'healthy' and 'patient' controls on the other. The conclusions of this study are consistent with the hypothesis that long-chain fatty acids cause myocardial infarction and the short-chain fatty acids are preventive because of their effect on the dynamic equilibrium between blood coagulation and clot lysis.

The theories linking smoking (*Smoking and Health*, 1964; Doll and Hill, 1956), sugar intake (Yudkin, 1957; Szanto and Yudkin, 1969), decline of physical activity (Morris *et al.*, 1953), and the amount and type of fat consumed (Keys, Anderson, and Grande, 1957; Kinsell, 1964) have aroused considerable interest in the causation of ischaemic heart disease. In our earlier epidemiological studies we found no evidence to support these ideas (Malhotra, 1967a, b) but put forward the suggestion that the relative proportions of long-chain fatty acid triglycerides in the plasma influenced the dynamic equilibrium between blood clotting and clot lysis and therefore the occurrence or immunity from ischaemic heart disease (Malhotra, 1968). The purpose of the present study is to test these hypotheses by the method of 'statistical epidemiology', in which these characteristics are compared between cases of myocardial infarction and matched controls.

Subjects and methods

During the period 1 January 1968 to 31 December 1971, 57 male patients with acute myocardial infarction were admitted to our Railway Hospital at Garden Reach, Calcutta. The diagnosis was accepted only if there was: (1) a history of retrosternal pain at rest, accompanied by Q wave changes in the electrocardiogram, or (2) a history of at least one hour's pain at rest accompanied by ST abnormalities in the electrocardiogram and ancillary evidence of an infarct: viz., one or more of the following, (a) a white blood cell count of over 11,000/mm³, (b) an ESR of over 20 mm/hour, (c) a temperature of 48 hours' duration, rising to at least 37.8°C, or (d) SGOT over

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40 units/100 ml. These criteria are the same as those used by Burns-Cox, Doll, and Ball (1969) to test Yudkin's oft-quoted sugar theory. For comparison with 'Controls' we used two different methods. One, in which two men were chosen to match each infarct patient, as far as possible, with regard to age, socioeconomic status, and body-build (weight in kg/height in cm); men who were admitted to the general medical or surgical wards of the same hospital and who had no evidence of overt coronary artery disease (for example, did not have, or give a history of having had, angina pectoris, myocardial infarction, intermittent claudication, or a cerebrovascular accident). In the second method, we used matched controls who were at work and were free from arterial hypertension, diabetes mellitus, and ischaemic heart disease, as screened by the questionnaire on cardiac pain suggested by Burgess, Fejfar, and Kagan (1963); an 8-lead electrocardiogram showing complete freedom from ischaemic changes (absence of Q wave and ST abnormalities), and absence of a history of angina pectoris, myocardial infarction, intermittent claudication, or cerebrovascular accident. In this second approach we included in our list of controls one man for each case of myocardial infarction of the same age and from the same socioeconomic group, the same trade, and as far as possible the same ponderal index (weight in kg/height in cm).

Fat consumption

This was collected by direct questioning and in the case of the patients with myocardial infarction this related to their habitual fat intake *before* the attack, but in the case of controls the *present* consumption was taken into account. Consumption of milk and fermented milk products, such as yoghurt, curd lassee, and pure ghee (clarified butter) made by the ferment-

FIG. Pathways of lipid absorption.

tation process, or of seed oils, was especially inquired into. The pattern of eating whether 'masticatory' or 'non-masticatory' was deduced from the pattern of diet as well as by direct questioning, since it has been previously suggested that it is not only the diet but how it is eaten that may bear responsibility for ischaemic heart disease (Malhotra, 1967a, 1968), the presence or absence of hypertension (Malhotra, 1970), and diabetes mellitus (Malhotra, 1971).

Smoking

Patients with myocardial infarction and both the control groups were divided into the following categories: (a) non-smoker, (b) ex-smoker, (c) smoker. Smokers were called light smokers if the cigarette consumption was 1-14 g/day, medium 15-24 g/day, and heavy 25+ g/day.

Sugar consumption

The amount of sugar consumed was assessed by the amount taken per cup of hot drink, the total amount taken in hot drinks per day, and the total amount of refined sugar consumed per day in the whole diet. We have relied on the conversion factors used in dietary assessment similar to those applied by Burns-Cox *et al.* (1969) to suit Indian conditions. Bengali Indians eat sweetmeats, called Rasgoolas, Sandesh, and sweetened yoghurt in this geographical area. The amount of sugar needed for each was determined by weighing the ingredients and sucrose in the preparation of the items, and from this standard conversion table, the amount of sucrose consumed was deduced.

Physical activity

The patients as well as controls were classified as: (a) sedentary, and (b) physically active, depending upon the nature of the occupation in their respective trades. For example, clerks are sedentary and firemen and fitters, physically active.

Question of bias

Diet Dietary histories can become biased because patients with myocardial infarction are often told to cut down fat and sucrose consumption. Sometimes they themselves do so as a result of wide publicity of these beliefs in the lay press. To reduce this bias we had inquired into the habitual patterns of diet and eating and of smoking *before* the attack of myocardial infarction. This method also is not entirely free from bias as some patients may have ascribed their attacks to these so-called 'risk factors' and may have, therefore, 'played-up' their answers. As a safeguard against this, the wives of 30 randomly selected patients with myocardial infarction were interviewed by a different interviewer who had no access to the results of our survey. The results of the second interviewer were essentially the same.

There is no reason, therefore, to suspect that our results have been biased.

Patients and controls Out of the 57 patients with myocardial infarction, we excluded 13 for the following reasons: (a) 3 had diabetes mellitus, (b) 7 had hypertension (systolic 160 mmHg and over and diastolic more than 95 mmHg), and (c) 3 had a known history of peptic ulcer with or without (a) or (b) mentioned above. Thus, altogether 44 infarct, 88 'control' patients, and 44 'healthy' controls were studied. The principal conditions for which the control 'patients' were admitted are shown in Table 1. The comparability of the three groups is shown in Table 2 (age distribution), Table 3 (social class distribution), Table 4 (physical activity at work), Table 5 (total dietary sugar), Table 6 (total dietary fat), Table 6a (statistical analysis giving t values for fat consumption), Table 7 (smoking habits), and Table 8 (serum cholesterol levels). The groups are comparable with regard to age, social status, and physical activity at work, as ensured by the design of the study.

TABLE 1 Principal diagnosis of 'patient' controls

Diagnosis	No. of patients
Chronic intestinal amoebiasis	9
Ulcerative colitis	1
Prolapse intervertebral disc	1
Fibroma	2
Chronic bronchitis	5
Pleurisy	1
Anxiety states	2
Pulmonary tuberculosis	4
Diarrhoea	2
Influenza	9
Asthma	9
Arthritis	1
Dyspepsia, spastic colon, etc.	11
Cirrhosis	2
Myasthenia gravis	1
Epilepsy	1
Gout	1
Nephrotic syndrome	1
Vertigo	2
Hypothyroidism	3
Trauma	5
Hydrocephalus	1
Haemorrhoids	1
Hernia	3
Orchitis	1
Corneal ulcer	1
Sinusitis	1
Encephalitis	2
Abscess	2
Paroxysmal tachycardia	1
Mitral stenosis	1
Enlarged prostate	1
Total	88

Results

Age, socioeconomic status, and physical activity

Out of the original 57 patients with myocardial infarction, 31 (54%) were in the age bracket 20 to 45 years and 26 (46%) were above the age of 46 to 58 years (Table 2). 13.6 per cent were in the social class I and II, 75 per cent in social class III, and 11.4 per cent in social class IV (Table 3). Of 'patient' controls and 'healthy' men when combined, 75 per cent were sedentary and 25 per cent physically active at work (Table 4).

Fat consumption

Differences in total fat consumption in the different groups are shown in Table 6. These are further subdivided into saturated fats of animal origin (mainly milk, fermented milk products, such as yoghurt, yoghurt lassee, and clarified butter) and unsaturated fats (mainly seed oils, mustard seeds, or groundnut oil). The total fat consumption in patients with myocardial infarction and the two control groups was more or less identical, the mean figures being 41 g, 37 g, and 46 g for infarct patients, 'patients' in the control group, and the healthy controls, respectively. If statistical analysis is made between saturated versus unsaturated fats, we found no differences, but statistical analysis between long chain ($C_{18:1}$) and short chain C_4 - C_6 fatty acid triglycerides was highly significant (Table 6a - column III).

TABLE 2 Age distribution of patients with myocardial infarction and controls

Group	No.	No. of patients in different age groups				Mean age (yr)
		35	36-44	45-54	55-58	
Myocardial infarction	44	Nil	11 (25%)	25 (56.8%)	8 (18.1%)	47.8
Control 'patients'	88	Nil	22 (25%)	50 (56.8%)	16 (18.1%)	45.4
'Healthy' controls	44	Nil	11 (25%)	25 (56.6%)	8 (18.1%)	45.1
All groups	176	—	44	100	32	—

TABLE 3 Social class distribution of patients with myocardial infarction and controls

Group	No.	No. of patients in social class			
		I and II	III	IV	Total
Myocardial infarction	44	6 13.6%	33 75%	5 11.4%	44
Control patients	88	12 13.6%	66 75%	10 11.4%	88
Healthy controls	44	6 13.6%	33 75%	5 11.4%	44

Yearly income of person in different social classes: Class I and II - £800-2000 per annum; Class III - £100-500 per annum; Class IV - £60-70 per annum.

Sugar consumption

The results showed that the healthy controls took on an average, $32.8 \text{ g} \pm 1.73 \text{ SE}$ of sugar; 'patient' controls $31 \text{ g} \pm 1.1 \text{ SE}$, and myocardial infarction

TABLE 4 *Physical activity at work of patients with myocardial infarction and controls*

Group	Sedentary		Physically active	
	No.	%	No.	%
Myocardial infarction	33	75	11	25
'Patient' controls	66	75	22	25
'Healthy' controls	33	75	11	25

TABLE 5 *Number of patients and controls taking different total amounts of refined sugar per day in g as ascertained from themselves*

Groups	Number of men	Mean daily consumption of sugar (g) $\pm$ SE
(a) Myocardial infarcts	44	34.2 ± 1.58
(b) 'Other' patients	88	31.0 ± 1.1
(c) Controls	44	32.8 ± 1.73

t values - a Vs b = 1.674 NS*; a Vs c = 0.666 NS; b Vs c = 0.621 NS.

* NS, not significant.

patients $34.0 \pm 1.58 \text{ SE}$ (Table 5). These were not statistically different from each other.

Smoking

There were no statistical differences in the amount of tobacco smoked in the infarct and 'healthy' patients; but a slight difference between infarcts and 'healthy' controls was present (Table 7).

Serum cholesterol levels

The data on serum cholesterol levels are shown in Table 8, and there were no significant differences in the three groups.

Discussion

Thomas *et al.* (1960) have emphasized that the blood factor is of greater importance than the atherosclerotic plaque (local factor) in thrombus formation. This is borne out by (a) the observations by Morris (1951) that whereas there has been no increase in the distribution or severity of atheroma in the coronary arteries of the population of the eastern districts of London during the past few decades, there has been a great increase in the prevalence of myocardial infarction in the same population, and (b) the recent work from India, which has shown no differences in the mean atherosclerotic indices in the North Indians and South Indians, though these were considerably less than in the west (Mathur, Patney, and Kumar, 1961; Murthy, Dutta,

TABLE 6 *Mean and $\pm$ SD of total consumption of dietary fat per day*

Group	Seed oil (g) (long chain saturated fatty acids)	Hydrogenated seed oils (g) (long chain fatty acids)	Milk, ghee, and other fermented milk products (g) (short chain saturated fatty acids)
Myocardial infarction	20.0 ± 4.98 (43)	15.3 ± 4.98 (42)	5.73 ± 7.09 (30)
Patient control	18.7 ± 3.70 (72)	11.3 ± 3.95 (70)	16.2 ± 5.42 (74)
Healthy control	20.3 ± 5.81 (44)	11.0 ± 4.88 (41)	14.8 ± 5.10 (37)

Numbers in parentheses show the number of persons.

TABLE 6a *Statistical analysis giving t values for fat consumption in infarcts and controls*

	Myocardial infarction vs. patient controls	Myocardial infarction vs. healthy controls	Patient controls vs healthy controls
Seed oils (unsaturated fatty acids)	1.48 NS	0.26 NS	1.63 NS
Hydrogenated vegetable oils (long-chain unsaturated fatty acids)	2.544 $P < 0.01$	2.632 $P < 0.01$	0.330 NS
Ghee and other fermented milk products (short chain saturated fatty acids)	4.014 $P < 0.01$	3.98 $P < 0.01$	0.831 NS

TABLE 7 *Distribution of smoking in patients immediately preceding illness that caused admission in patients and in normals and control groups*

Group	No.	Non-smokers	Ex-smokers	Current smokers (cigarettes per day)			Other tobacco addicts, individual or in combination with cigarettes
				1-14	15-24	25+	
Myocardial infarction	44	10	16	14	4		5
Other patient controls	88	28	18	23	14	5	15
Healthy controls	44	17	5	15	5	2	4

Comparison between non-smokers and ex-smokers and current smokers.

Between myocardial infarction vs. patient controls, $\chi^2 = 4.08$ (difference not significant).

Between myocardial infarction vs. healthy controls, $\chi^2 = 7.98$; $P = 0.02$.

TABLE 8 *Serum cholesterol levels of infarcts and controls (mg/100 ml) using Sackett's method*

Group	Serum cholesterol (Sackett's method)			P
	Mean	$\pm$ SE	t values	
Myocardial infarction patients (44)	200.3	5.3	A vs. B = 1.833	0.05
Patient controls (88)	189.3	3.2	A vs. C = 0.288	NS
Healthy controls (44)	190.3	3.8	B vs. C = 0.134	NS

TABLE 9 *Mean values for blood coagulation, clot retraction, platelet numbers, and platelet adhesiveness in 28 pairs of age-matched railway sweepers (all men) from Madras and Udaipur*

Groups	Clotting time (sec)	Clot retraction (%)	Platelet count (10^9)	Platelet adhesiveness (%)
<i>Madras</i>				
Mean	292.0	47.63	287.65	21.7
Standard error	± 9.37	± 3.96	± 15.77	± 1.42
<i>Udaipur</i>				
Mean	354	43.87	293.04	22.75
Standard error	± 14.34	± 4.68	± 15.14	± 1.19

and Ramalingaswami, 1962; Subramaniam and Kulangara, 1967), even though ischaemic heart disease was 7 to 15 times more prevalent among South Indians as compared with the North Indians (Malhotra, 1967b). It is reasonable to suppose, therefore, that the occurrence of myocardial infarction is determined more by occlusive thrombogenesis than by an increased severity or extent of coronary atheroma, and therefore atherosclerosis and myocardial infarction are not synonymous. The recent study of Mann *et al.* (1972) confirms this view, because in 50 Masai necropsies, the coronary arteries showed intimal thickening which equalled that of American men, and yet no cases of ischaemic heart disease occurred in the Masai tribes.

Diet may change some aspects of thrombosis and blood coagulation (Billimoria, 1964). Merskey and

Nossel (1957) found their results varied, depending upon the type of meal ingested, and were not directly related to the fat content. In our population studies of some of the haematological factors between North Indians as compared with South Indians (Table 9), we were impressed by (a) mean clotting time being significantly longer in North Indians, as compared with South Indians; (b) the clots were soft and jelly-like in North Indians but hard and dense in South Indians; (c) the North Indians showed the phenomenon of clot lysis to a significant degree, while this was comparatively much less in South Indians (Malhotra, 1968).

These three characteristics depend upon the number, size, and complexity of the chylomicrons (Malhotra, 1967b, 1968, 1970, 1971). When we suggested this explanation we were not aware of

Poole's (1962) important studies. He found that in plasma freed from chylomicrons by high speed centrifugation, its clotting time was increased. The clotting time of such plasma could be returned to normal by returning washed chylomicrons to the plasma. These studies of Poole suggest the crucial importance of the chylomicrons in the haematological differences between our age-matched population studies as reported in earlier articles (Malhotra, 1968, 1971).

As a result of heating fats with turmeric powder (*curcuma domestica*), a practice universally present in the North but absent in the South of India, the fat globules are *completely* broken down, probably because the capsular membrane of the fat globules is disintegrated by boiling (Ling, 1963; Malhotra, 1967a). This also reduces the chain length of the fatty acids, converting the long chain into short chain fatty acids (Malhotra, 1971).

The pathways of lipid absorption are depicted in the Fig. There is good evidence that the chain length of the triglyceride acid composition of plasma depends upon the fatty acid composition of fed fats (Ahrens *et al.*, 1958; Lough, Garton, and Duncan, 1957; Kern and Borgström, 1965). Moreover, the amount of bile salts in the intestinal lumen determines not only the rate and the route (Dawson and Saunders, 1965; Hughes and Wimmer, 1935) of absorption of fats, but also the composition of the plasma triglyceride acids. Thus, ordinarily the food fats, especially the long chain fatty acid triglycerides, are hydrolysed by bile salts into fatty acids which are then resynthesized into complex fatty acid triglycerides, partly characteristic of the species, whereas short chain fatty acid triglycerides are directly absorbed into the blood stream without chylomicron formation. The significantly different rates of coronary artery disease between the South Indians versus the North Indians reported in earlier studies (Malhotra, 1967a, b), appear to be due to the differences in their patterns of diet, cooking, and eating, leading to a relative preponderance of short chain over long chain fatty acid triglycerides in the North Indians as compared with South Indians and the differences in the number and density of the chylomicrons (Malhotra, 1971). The amount and type of fat consumed does not seem to be crucial, and in fact Shaper, Jones, and Kyobe (1961), Shaffer *et al.* (1964), and Mann, Shaffer, and Rich (1965) found higher fat consumption of animal origin and an immunity to ischaemic heart disease in their African tribal studies as compared with studies in the West.

Before accepting this thesis that patterns of diet and eating play a crucial causal role in ischaemic heart disease, other alternative propositions must be examined.

Our results indicating that smoking is a possible causative factor in ischaemic heart disease do not support the observations of others (Doll and Hill, 1956). These results are in agreement with our previous studies (Malhotra, 1967b, 1970) and the studies of Murphy and Mustard (1966), so far as infarcts and 'patient' controls are concerned, but the number of smokers among 'healthy' controls was higher (Table 7). These conflicting results make it unlikely that smoking is a major factor in the causation of ischaemic heart disease.

Our data do not suggest the consumption of refined sugar to be a major and specific factor in the production of myocardial infarction. This not only reinforces our previous conclusions (Malhotra, 1967a, b) but also of others (Burns-Cox *et al.*, 1969; Medical Research Council, 1970; Howell and Wilson, 1969).

Decline of physical activity, though still regarded as a major factor in the causation of ischaemic heart disease, requires reappraisal in the light of our previous findings (Malhotra, 1967a, b) and the results of the present study. If in population groups and in persons subjected to identical physical exercise, there are big differences in the incidence rates of ischaemic heart disease, it is not reasonable to ascribe the cause to decline in physical activity.

The present study is consistent with the hypothesis that dietary factors (mainly the relative proportion of short chain and long chain triglyceride acids in the plasma), absence of roughage and vegetable fibres, and the amount of bile entering the intestinal lumen, play a crucial part in the pathogenesis of ischaemic heart disease. There is, therefore, hope that this disease may be prevented.

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