Is low energy intake a risk factor for ischaemic heart disease?

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SUMMARY The relation between total energy intake and the development of ischaemic heart disease was investigated from a review of all available data including the results of 26 years of follow up from a longitudinal study of diet and ischaemic heart disease. A consistent inverse relation was found, whose strength and consistency are similar to those of other established risk factors.

It is concluded that there may be a minimum energy intake below which the risk of ischaemic heart disease is increased.

The association of ischaemic heart disease mortality and dietary factors, between populations, was first identified more than 25 years ago¹ and subsequently confirmed by others.²⁻⁴ Positive associations were found not only with the proportion of energy from both total and saturated fat but also with total energy intake.^{5,6} Indeed it was postulated that excessive energy intake from all sources rather than from any one particular kind of food was associated with ischaemic heart disease.⁷ A prospective study in the United Kingdom of groups of bankers and London busmen, however, suggested that a high energy intake may be protective against ischaemic heart disease.⁸ We have subsequently examined available data from other diet-heart studies together with previously unpublished data from the UK study to determine whether this relation is consistent and to consider the possible explanations for such an association.

Review of the evidence

OBSERVATIONAL STUDIES BETWEEN POPULATIONS

The findings from observational ecological studies (Table 1) showed^{2,4,5} that populations with a high ischaemic heart disease mortality are the affluent countries that also have high fat and energy levels available for consumption. The Seven Country study

by Keys^{9,10} using a diet methodology appropriate for characterising groups,¹¹ found no relation between ischaemic heart disease and energy intake, calculated as energy per kilogram body weight. They did, however, find that there was less disease in the communities with high physical activity, though the relation between physical activity and energy intake was not examined.

COMPARATIVE STUDIES OF RELATED POPULATIONS WITH DIFFERING RISKS

In a study comparing dietary habits in Boston men of Irish origin with those of their brothers remaining in Ireland^{12,13} the Irish brothers had a mean daily energy intake of 500 cal_{th} (2.10 MJ) more than their Bostonian counterparts despite being about 10% smaller in weight and having half the mortality rate due to ischaemic heart disease. The Irish brothers were noted to take more exercise.

In an attempt to explain the lower standardised mortality rate of ischaemic heart disease in Iowa farmers compared with their town dwelling counterparts the ischaemic heart disease risk factors were compared in the two groups.¹⁴ This showed that the farmers smoked less, exercised more frequently, and had a mean energy intake of 430 kcal_{th} (1.81 MJ) greater than the towns people. Conversely, the percentage fat intake was similar and the ratio of polyunsaturated to saturated fatty acids was lower in the farmers.

TIME TRENDS WITHIN POPULATIONS

Kahn observed that despite a fall of 10% in total energy intake in the United States in the first six

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Table 1 Between population comparisons of ischaemic heart disease mortality and dietary fat and energy intake

Study	No of countries	Population (age range in yr)	Disease definition	Energy (r_s)*	% Fat (r_s)*
Yerushalmy and Hilleboe ²	22	Male (55-59)	ICD: B25-28	+0.64	+0.44
Jolliffe and Archer ⁴	20	Male (55-59)	ICD: B26	+0.55	+0.73
Masironi ⁵		Male (55-64)	AHD		
1940s and 1950s	25			+0.75	+0.63
1960s	38			+0.69	+0.74

*Food energy data derived from Food and Agricultural Organisation food balance sheets. r_s , Spearman's rank correlation coefficient. AHD, arteriosclerotic and degenerative heart disease; ICD, international classification of diseases.

decades of this century there has been an increase in ischaemic heart disease rates though little change in serum cholesterol concentrations.¹⁵ Figure 1 shows similar data for the United Kingdom from 1960 to 1978 (that is, the period of the Eighth Revision of the International Classification of Diseases). There was an increase in age adjusted ischaemic heart disease mortality rate between 1960 and 1972 followed by a leveling off in both men and women.^{16,17} During this period there was a steady decline in energy intake, which was particularly pronounced in 1970-74.¹⁸ Statistically, for the 19 years, there are strong inverse correlations between this population estimate of energy intake and mortality in both men ($r = -0.78$) and women ($r = -0.69$).

RETROSPECTIVE STUDIES

In the International Atherosclerosis Project there was a trend of increasing coronary artery atheroma with a decrease in energy intake, though this was not statistically significant.¹⁹ In a similar study of 226 men from the Honolulu Heart Study both energy and starch intake were significantly inversely related to the extent of coronary atheroma at necropsy.²⁰ A detailed case-control study in North Dakota, however, found no association between ischaemic heart disease and mean energy intake.^{21,22}

PROSPECTIVE STUDIES

There have been several prospective studies involving dietary assessment. A part of the Western Collaborative Group study used a seven day diary method of dietary assessment.²³ A two and a half year follow up showed that 25 men who developed electrocardiographic changes of ischaemic heart disease had a lower initial energy intake compared with 80 disease free men (2100 vs 2280 kcal_{th} (8.82 vs 9.58 MJ)).

Not all prospective studies of diet and ischaemic heart disease have reported on total energy intake, though such data must have been collected. Thus there are no dietary data pertinent to this question either from the Albany²⁴ or the Minnesota²⁵ studies. We have found seven prospective population studies from which at least four years of follow up data are available. We grouped the studies into areas of high and low incidence of ischaemic heart disease because of the very different levels of energy intake found. The high incidence areas included Framingham,^{26,27} Zutphen,²⁸ Western Electric (Chicago),^{29,30} and UK bankers and busmen^{8,31} and the low incidence areas Hawaii,³² and the studies of both urban and rural populations in Puerto Rico.³³ The studies varied in their study population, the method of dietary inquiry, the outcome measures published, and the duration of follow up; Table 2 shows these differences.

Fig. 1 Trends of heart disease mortality (ICD 410-414 Eighth Revision) and energy intake 1960-78.

Table 2 Methodological details in prospective studies of diet and ischaemic heart disease

Study	Population (age range in yr)	No of subjects	Dietary survey method	Adverse outcome	Duration of follow up (yr)
Framingham	Male (45–64)	831	24 hour recall	Death (ischaemic heart disease, acute myocardial infarction)	4
Zutphen	Male (40–59)	770	Cross check dietary history	Death (ischaemic heart disease)	10
Western Electric	Male (40–55)	1885	Dietary interview	Death (ischaemic heart disease, acute myocardial infarction, angina)	4–4
UK bankers and busmen	Male (30–67)	337	Seven day weighed survey	Death (ischaemic heart disease, acute myocardial infarction, angina)	10–20
Hawaii	Male, Japanese (45–68)	7590	24 hour recall	Death, (ischaemic heart disease, acute myocardial infarction)	6
Puerto Rico (Urban)	Male (45–64)	5714	24 hour recall	Death (ischaemic heart disease, acute myocardial infarction)	6
Puerto Rico (Rural)	Male (45–64)	2381	24 hour recall	Death (ischaemic heart disease, acute myocardial infarction)	6

Table 3 Mean energy intake and disease status from seven longitudinal studies

Study	Patients with heart disease				Disease free subjects			
	No	Mean energy intake (cal _{th})	SD	SEE	No	Mean energy intake (cal _{th})	SD	SEE
<i>High incidence areas</i>								
Framingham	51	2369	700*	98.0	780	2622	700*	25.1
Zutphen	27	2857	582	112.0	743	3080	662	24.3
Western Electric	88	3082	974*	103.8	1797	3174	974*	23.0
UK bankers and busmen	50	2656	400	56.6	287	2869	505	29.8
<i>Low incidence areas</i>								
Honolulu	179	2125	666	49.8	7411	2290	738	8.6
Puerto Rico (urban)	129	2242	856*	75.4	5585	2413	856*	11.5
Puerto Rico (rural)	34	2113	837*	143.5	2347	2353	837*	17.3

*Pooled standard deviation (SD) from patients and disease free subjects.

SEE, standard error of the estimate

Conversion: traditional to SI units—energy intake: 1 cal_{th} ≈ 4.2 J.

Table 3 shows the mean energy intakes of those who did and did not develop ischaemic heart disease. It was necessary, in the absence of complete data, to use pooled standard deviations from both ischaemic heart disease patients and disease free subjects for the Framingham, Western Electric, and both the Puerto Rico population studies. This assumed that the variances were similar in the ischaemic heart disease patients as in disease free subjects. Though there was no a priori reason that they were different, a higher variance for the patients would have reduced the chances of any observed difference being statistically significant. The studies where separate data were available showed, however, that the variance tended to be lower for the patients (Table 3).

The most striking finding was that in each study the men developing ischaemic heart disease had, initially, a lower energy intake than the disease free men (Fig. 2), the difference varying between 100 and 250 kcal_{th} (0.42 and 1.05 MJ) per day. These differences were significant for the Framingham, UK bankers and busmen, Hawaii, and the Puerto Rico (urban) studies. The data were pooled for both the combined high incidence areas and the combined low incidence areas.

The design of the studies in the low incidence areas was identical, but this was not true for the high incidence areas (Table 2). Though the pooling would increase the chances of a statistically significant result by increasing the sample size, it nevertheless is of use in providing the best estimate of the true difference. Calculated 95% confidence intervals for the deficit in energy intake in ischaemic heart disease cases are for high incidence areas 141.4–278.6 kcal_{th} (0.59 and 1.17 MJ) per day and for low incidence areas 96.6–253.4 kcal_{th} (0.41 and 1.06 MJ) per day. The difference in means represents a shift to the left in the distribution of energy intake in the cases of ischaemic heart disease of 7.0% in the high incidence and 7.5% in the low incidence areas. It should be noted, however, that the difference in intake between high and low risk areas is greater than that between patients and disease free subjects within each of these two areas (Table 3).

Discussion

The evidence reviewed above is surprisingly consistent in suggesting an inverse association between energy intake and ischaemic heart disease risk within

Fig. 2 Difference and standard error of difference of total mean daily energy intake between patients with coronary artery disease and disease free subjects from seven longitudinal studies.

population groups and conflicts with the evidence of the comparisons between countries^{4,5} that countries with high levels of energy available for consumption have higher levels of ischaemic heart disease. The likely explanation for this paradox is that differences in energy intake between countries reflect relative affluence, and it is other variables associated with affluence that are the cause of the increased risk. The apparent protective effect of a high energy intake as suggested by the other studies described could possibly be explained by methodological problems and the possibility of confounding variables.

METHODOLOGICAL PROBLEMS

The association over time (Fig. 1) might be spurious. The National Food Survey data covered only food consumed at home, and during this period there had been an increase of 7% in meals taken away from home.³⁴ Thus apart from all the other hazards in interpreting such associations the data may not truly represent energy intake. Similarly, the retrospective studies cited¹⁹⁻²² have to be interpreted with caution. Dietary inquiry in a retrospective or case-control study is open to error. Firstly, current diet may have altered as a result of disease presence and, secondly, a retrospective diet history is itself prone to error owing to poor recall.

The prospective studies cited used a single estimate of energy intake that might not reflect true intake over the duration of the study. Morris *et al* found a correlation of 0.73 within subjects for total energy intake calculated from two seven days weighed surveys performed one to 12 months apart.³⁵ Although the men in the Zutphen study showed pronounced changes between 1960 and 1965 and 1965 and 1976³⁶ there is no indication that the ranked order also changed. Thus, though diet does change, the originally observed differences are probably sustained. Furthermore, it is unlikely that any change over time is selectively different in direction between the ischaemic heart disease and disease free groups before the clinical onset of the disease. Secondly, the

methods of dietary inquiry varied, and different end points and duration of study were used (Table 2). Estimates of energy intake from the methods used in all these studies probably underestimate true consumption.³⁷ The results were, however, similar and this consistency, despite the differences in method, is positive evidence in favour of the results being real.

Thirdly, it is possible that in the short term follow up those already with incipient ischaemic heart disease at the start of the study may, as a consequence, have had a lower energy intake. The short (two and a half to six years) duration of some of the studies^{23, 26, 27, 29, 32, 33} makes segregation between cause and effect difficult. The Zutphen men were, however, followed for 10 years,²⁸ and 10-20 years data are available from the UK bankers and busmen study⁸ with, at the time of writing, a further six years of follow up analysed (Marr and Morris, unpublished data). These data for the UK men, despite the small numbers in each follow up period, show that there was no evidence that the trend was reversing (Kendall's tau 0.155, NS).

Fourthly, it is possible that obese persons may recall lower intakes. This possibility was examined in the Hawaii men,³² and the authors concluded, "age adjusted mean values of total calories were consistently lower in IHD cases than in non cases in all classes of relative weight."

Finally, it is unlikely that these results are chance findings. The standard deviations are large, but the pooling of the studies together showed that the likelihood of a type I error was less than 1%.

POSSIBLE CONFOUNDING VARIABLES

It is possible that the relation to energy intake is explained by the confounding effect of other known ischaemic heart disease risk factors. The two most likely confounders are that both physical inactivity and cigarette smoking are associated with low energy intake, though the data are more substantive for the former. It was impossible, however, in the absence of direct access to all the raw data to do the necessary

multiple logistic analyses to determine whether energy intake had an independent effect in the studies cited.

ENERGY EXPENDITURE

This is the likeliest confounding variable since in a steady state a high energy intake reflects increased muscular activity,⁸ and the latter is possibly protective against ischaemic heart disease.^{38,39} It has been found that a high energy expenditure is associated with an increased intake both in athletes^{40,41} and office workers.^{42,43}

In the prospective studies cited the patients with ischaemic heart disease in Hawaii³¹ had lower levels of physical activity ($p < 0.05$) compared with the disease free men, though the difference became statistically insignificant in multivariate analysis. The age adjusted mean energy intake was significantly lower in patients with ischaemic heart disease than in disease free subjects, even after controlling for physical activity.

The effect of increase in energy expenditure is, however, complex. Exercise does not, however, consistently increase the appetite.⁴⁴ An increase in daily exercise sufficient to produce weight loss over an eight week period did not increase energy intake,⁴⁵ and 45 minutes of light jogging three days a week also had no effect.⁴⁶ Indeed, in animals exercise may decrease the appetite.^{47,48}

Cigarette smoking

There are no data on whether cigarette smoking is associated with a reduction in energy intake compared with non-smokers. Anecdotally, patients who give up smoking report an increase in energy intake with increase in body weight, but whether this is a phenomenon of all non-smokers (never and ex-smokers) is unknown. Analysis of the UK men, with smokers and non-smokers separately divided into equal high and low energy intake groups, showed that in both smoking groups the high intake group had approximately half the risk for ischaemic heart disease of the low intake group.⁸

BIOLOGICAL PLAUSIBILITY

In the absence of a plausible biological explanation a causal relation between low energy intake and

ischaemic heart disease cannot easily be inferred from these data.⁴⁹ The only likely explanation is that high energy intake is associated with an increase in plasma high density lipoprotein cholesterol concentration, though this might be explained by the confounding effect of energy expenditure.

The results of an intervention study comparing the effect of an exercise programme with sedentary controls on energy intake, body weight, and blood lipid concentrations⁵⁰ showed an identical positive relation between mileage run with both increase in energy intake ($r = 0.48$, $p = 0.006$) and increase in plasma high density lipoprotein cholesterol concentration ($r = 0.48$, $p = 0.006$). The increase in energy intake included an increase in total fat consumption. It has, however, been shown that mild exercise in middle aged sedentary men has a favourable effect only on high density lipoprotein cholesterol concentration when energy intake is maintained; a reduction in intake leads to a reduction in high density lipoprotein concentration.⁵¹ Thus perhaps increased exercise and increased energy intake act synergistically in reducing ischaemic heart disease risk rather than as confounding variables. An alternative hypothesis is to consider the effect that ischaemic heart disease risk is related to trace element deficiency in the diet,^{52,53} and thus those with a high energy intake, assuming that they eat more of everything, might be less likely to show such deficiencies. Such hypotheses are virtually impossible to test without detailed and repeated chemical analyses of food and drink consumed, food tables being inadequate for the purpose. Such a methodology is likely to be unacceptable in free living populations participating in a prospective study.

BIOLOGICAL IMPORTANCE

It is of interest to note, however, that if the inverse association is causal its strength is high enough to be of biological importance. The observed difference in mean energy intakes represents a shift in the distribution, and a comparable shift, in proportionate terms, applied to other known risk factors would be likely to have a major effect on ischaemic heart disease rates.⁵⁴ As an example, the data from the UK bankers and busmen study⁸ are given in Table 4, which shows that the magnitude of the difference observed in energy intake is comparable to the difference observed with

Table 4 Relative difference in risk factors between men with ischaemic heart disease (group 1) and disease free men (group 2)⁸

Variable	Group 1	Group 2	Difference in group 1 as % of group 2
Mean serum cholesterol concentration (mmol/l)	6.46	6.05	+ 6.8
Mean systolic blood pressure (mm Hg)	144	138	+ 4.3
Mean No of cigarettes per day	15.1	9.4	+60.6
Mean energy intake (kcal _{th})	2656	2869	- 7.4

Conversion: traditional to SI units—energy intake: 1 kcal_{th} $\approx$ 0.0042 MJ.

serum cholesterol concentration or systolic blood pressure.

Conclusion

There appears to be paradoxically an inverse association between energy intake and ischaemic heart disease risk within countries whose strength and consistency are similar to those of other established risk factors. The confounding effect of exercise or indeed other risk factors cannot be entirely discounted, though there is evidence that high energy intake and expenditure are synergistic.

There is no evidence that excessive energy intake in the absence of matched output is anything but harmful to health,⁵⁵ but there is support, we believe, for the hypothesis that there is a minimum energy intake—probably higher than that needed to maintain body weight in the sedentary middle aged man—below which the risk of ischaemic heart disease is increased.

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