Diet and incident ischaemic heart disease: the Caerphilly Study

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The Caerphilly Prospective Ischaemic Heart Disease (IHD) Study is based on a sample of 2512 men aged 45–59 years when first seen. Nutrient intakes, estimated using a self-administered semi-quantitative food frequency questionnaire, are available for 2423 men (96%). Amongst these, 148 major IHD events occurred during the first 5 years of follow-up. Associations were examined between these events and baseline diet. Incident IHD (new events) was negatively associated with total energy intake: men who went on to experience an IHD event had consumed 560 kJ (134 kcal)/d (6%) less at baseline than men who experienced no event ($P = 0{\cdot}01$). The relative odds of an IHD event was 1·5 among men in the lowest fifth of energy intake, compared with 1·3, 1·2, 0·9 and 1·0 respectively for the other four fifths ($P < 0{\cdot}05$). The difference in energy intake was reflected in lower intakes of every nutrient examined. When expressed as a percentage of total energy, mean intakes of men who experienced an IHD event were virtually identical to those of men who did not. There was some evidence suggesting a positive association between total fat intake and IHD risk, but the trend was not consistent and not statistically significant. There was no association for animal fat. Alcohol consumption was negatively associated with subsequent IHD, but only in men who already had evidence of IHD at baseline ($P < 0{\cdot}05$). Dietary fibre, particularly from fruit and vegetables, was 7% lower in men who had an incident IHD event ($P < 0{\cdot}05$), but the difference was not independent of total energy. There was a trend of increasing IHD risk with decreasing vitamin C intake, the relative odds of an IHD event being 1·6 among men in the lowest one-fifth of the vitamin C distribution, but this was not statistically significant.

Energy intake: Fat intake: Dietary fibre: Ischaemic heart disease: Vitamin C

The Caerphilly Ischaemic Heart Disease (IHD) Study is a longitudinal study of a representative population sample of middle-aged men in a typical town in South Wales (Caerphilly and Speedwell Collaborative Group, 1984). The aims include the assessment of possible risk factors measured at recruitment (haemostatic, lipid, hormone, psycho-social and dietary factors) in relation to incidence of major IHD events (new events).

The recruitment phase was conducted between 1979 and 1983 and complete data on new episodes of IHD are now available for the first 5 years of follow-up.

In the present paper relationships between the incidence of major IHD events and nutrient intakes estimated at the time of the baseline examinations are presented.

SUBJECTS AND METHODS

All men aged between 45 and 59 years inclusive who were resident in the town of Caerphilly or in one of five adjacent villages (total population 41 000), were identified and invited to attend a cardiovascular screening clinic. Of 2818 men found to be eligible, 2512 (89%) attended.

A self-administered, semi-quantitative food-frequency questionnaire (Yarnell *et al.* 1983) was given to every man for completion at home, with the help of his spouse. This contained

questions about most of the foodstuffs in the British diet. Weights of foods were calculated by multiplying the frequency of consumption (number of d per week) by the portion size, and nutrient intakes were then estimated by the use of food composition tables (Paul & Southgate, 1978). Animal fat intake was calculated as the sum of fat from meats, butter, lard, milk, cream, cheese and eggs.

During the initial examinations a 30% sample (665 men) were asked to complete a weighed dietary intake record for 7 d (Fehily *et al.* 1987). Data obtained from these records were used to obtain quantitative measures for food items for which the questionnaires gave only data on frequency of consumption. Comparison of nutrient intakes calculated from the questionnaire with those calculated from 7 d weighed intake records showed that group-mean values were similar by the two methods (Fehily *et al.* 1987). Pearson correlation coefficients were 0·3–0·4 (alcohol 0·75; Yarnell *et al.* 1983). In terms of the questionnaire's ability to classify subjects as high or low consumers of particular nutrients, approximately 50% of subjects were classified in the same third of the nutrient distribution by the two methods and 7–15% classified in opposite thirds (Yarnell *et al.* 1983). In terms of fifths of the nutrient distributions, 24–34% of subjects were classified in the same fifth and only 2–4% classified in opposite fifths. Food composition table data on the vitamin C content of foods do not take into account losses which may occur during cooking. However, Bolton-Smith *et al.* (1991) compared estimates of vitamin C intakes from this questionnaire with serum vitamin C concentrations and reported that 80% of subjects were classified correctly into the upper or lower plus adjacent tertiles of the serum values. The validity of the questionnaire is, thus, very similar to that of other semi-quantitative food-frequency questionnaires (Jain *et al.* 1980, 1982; Willett *et al.* 1985; O'Donnell *et al.* 1991).

The men were invited to attend the clinic again 5 years later. It was found that 132 men (5·3%) had died and only eight men (0·3%) could not be traced. Of the 2372 men available for examination, 2324 (98%) completed a repeat chest pain questionnaire (Rose & Blackburn, 1968) and 2241 (94%) had a repeat electrocardiogram (ECG). These, together with notifications of death from the Office of Population Censuses and Surveys (OPCS), were used as a basis for the identification of major incident IHD events, defined as follows: (1) IHD death; deaths coded to ICD 410–414, (2) myocardial infarction (MI); the chest pain questionnaire (Rose & Blackburn, 1968) was extended to include questions about hospitalization for severe chest pain. This information, together with lists from Hospital Activity Analysis (HAA) for all the hospitals in the study area, was used as a basis for a detailed search of hospital notes for events which satisfied the World Health Organization (WHO) criteria for acute MI, and (3) ECG evidence of MI; the WHO criteria for acute MI include a series of categories based entirely on sequential ECG. We have included just the first of these categories, i.e. no Q/QS wave (1-1 to 1-3) on the recruitment-phase ECG with major or moderate Q/QS waves (1-1-1 to 1-2-5 plus 1-2-7) on the follow-up ECG.

A number of confounding factors were included in the analyses. These included age, body mass index (BMI; weight (kg)/height $(m)^2$) and smoking habit. Smokers were grouped as never-smoked, ex-smokers, pipe or cigar smokers, light (less than 15/d), moderate (15–24/d) and heavy cigarette smokers (25 or more/d).

The other confounding factor considered in the analyses was prevalent IHD, that is, evidence of disease which had been detected at the baseline examinations. The manifestations of IHD which we have accepted as relevant are: angina, a past history of possible MI, or 'possible' or 'probable' ischaemia on ECG, or both, all as defined previously (Bainton *et al.* 1988). Of the 2512 men recruited to the study, 631 (25%) were found to have evidence of IHD at baseline according to these criteria. This value is very similar to that reported in the British Regional Heart Study (Shaper *et al.* 1984). Results are presented separately for the whole cohort, the 75% of men with no evidence of IHD

Table 1. *Mean daily nutrient intakes estimated at baseline in the total cohort of men subdivided into those who went on to experience an ischaemic heart disease (IHD) event during the next 5 years and those who did not*†

(Mean values and standard deviations)

	n ...	Men who had no IHD event 2197–2275‡		Men who experienced an IHD event 137–148‡		
Nutrient		Mean	SD	Mean	SD	*t*
Energy:						
MJ		9·7	2·5	9·1	2·3	−2·55**
kcal		2313	600	2179	548	
Protein (g)		73·9	18·1	71·0	19·0	−1·91
Fat (g)		102·5	32·3	98·7	28·7	−1·34
Animal fat (g)		76·1	30·8	72·1	27·2	−1·47
Carbohydrate (g)		248·5	74·1	232·4	66·4	−2·49*
Starch (g)		153·2	57·0	144·2	52·1	−1·87
Sugars (g)		95·3	38·4	89·1	36·0	−1·84
Fibre (g):						
Total		16·1	5·7	15·0	5·3	−2·42*
Cereal		7·7	4·6	7·1	4·2	−1·57
Fruit/vegetable		8·4	2·8	7·8	2·4	−2·37*
Vitamin C (mg)		51·5	20·7	48·4	20·4	−1·77
Alcohol (g)		22·9	28·5	20·0	27·8	−1·75§

* $P < 0{\cdot}05$, ** $P = 0{\cdot}01$.
† For details of subjects and procedures, see pp. 303–305.
‡ Nos. of subjects vary because of missing data in some of the questionnaires.
§ Calculated after log transformation.

at baseline and for the 25% of men with evidence of IHD at baseline. In analyses of the whole cohort adjustment was made for the presence of disease at baseline using multiple logistic regression analysis. Results are presented in this way since attempting to define a 'disease-free' cohort, as is sometimes done, is somewhat arbitrary.

The detailed analyses which follow examine the incidence of major IHD events within fifths of men defined by their intakes of nutrients at baseline. Incidence is summarized as 'relative odds' within these fifths and these have been adjusted, using multiple logistic regression analysis, for the independent effects of age, BMI and smoking habit. Where the relative odds relate to the total cohort they have been adjusted for evidence of IHD at baseline as well as for age, BMI and smoking habit.

RESULTS

Among the total cohort of 2512 men there were 153 major IHD events during the first 5 years of follow-up. The dietary questionnaire was completed by 2423 men (96% of the total cohort) and among these were 148 major IHD events.

Table 1 displays the mean nutrient intakes of men who suffered a major incident IHD event and those of men who did not. Tables 2 and 3 show these data for men who had no evidence of IHD at baseline and for those who had evidence of IHD at baseline respectively. The total energy intake of the men who went on to experience an event had

Table 2. *Mean daily nutrient intakes estimated at baseline in men who had no evidence of ischaemic heart disease (IHD) at baseline, subdivided into those who went on to experience an IHD event during the next 5 years and those who did not**

(Mean values and standard deviations)

	n ...	Men who had no IHD event 1686–1743		Men who experienced an IHD event 70–74		
Nutrient		Mean	SD	Mean	SD	*t*
Energy:						
MJ		9·7	2·4	9·5	2·3	−0·80
kcal		2328	580	2271	577	
Protein (g)		74·5	17·3	73·0	18·7	−0·71
Fat (g)		102·8	30·6	100·7	29·1	−0·57
Animal fat (g)		76·3	29·0	75·3	24·3	−0·30
Carbohydrate (g)		250·5	73·2	240·2	66·3	−1·17
Starch (g)		155·1	57·2	148·8	56·0	−0·93
Sugars (g)		95·3	37·6	92·9	30·3	−0·54
Fibre (g):						
Total		16·4	5·7	15·1	5·7	−1·88
Cereal		7·9	4·6	7·3	4·6	−1·25
Fruit/vegetable		8·4	2·7	7·8	2·3	−1·81
Vitamin C (mg)		51·5	20·3	49·1	20·3	−1·01
Alcohol (g)		23·2	28·5	24·1	28·2	+0·05†

* For details of subjects and procedures, see pp. 303–305.
† Calculated after log transformation.

been less at baseline than that of the men who experienced no event by about 6% (560 kJ (134 kcal)) in the total cohort ($P = 0{\cdot}01$), by 2% (238 kJ (57 kcal)) in the men with no evidence of IHD at baseline ($P > 0{\cdot}05$), and by 8% (757 kJ (181 kcal)) in men who had evidence of IHD at baseline ($P < 0{\cdot}05$). The lower energy intake was accompanied by lower intakes of every nutrient estimated.

Table 4, which displays the data on total energy intake in greater detail, shows that there was a trend between total energy intake and IHD incidence in the total cohort after adjusting for age, BMI, smoking habit and evidence of IHD at baseline ($P < 0{\cdot}05$). The relationship appeared weaker among those who had no evidence of IHD at baseline than among those with evidence of IHD at baseline. However, a formal statistical test for interaction was not significant and, hence, there was no evidence that the relationship was different in those with and those without evidence of IHD at baseline.

Among the total cohort, after standardizing for energy by expressing nutrient intakes as a percentage of energy (Table 5), differences between men who experienced an IHD event and those who did not became trivial and none was statistically significant. Despite this certain nutrients were examined in greater detail.

Table 6 displays the relative odds of a major IHD event in fifths of men defined by the percentage of energy obtained from fat at baseline. There was some evidence suggestive of an increase in risk with increasing intake of fat. However, the trend was not consistent and was not statistically significant, the highest relative odds being in the 20% of men with the next to highest levels of fat consumption.

Table 7 shows the same relationship for fat of animal origin. Here there was no evidence

Table 3. *Mean daily nutrient intakes estimated at baseline in men who had evidence of ischaemic heart disease (IHD) at baseline, subdivided into those who went on to experience on IHD event during the next 5 years and those who did not*†

(Mean values and standard deviations)

	n ...	Men who had no IHD event 511–532		Men who experienced an IHD event 67–74		
Nutrient		Mean	SD	Mean	SD	*t*
Energy:						
MJ		9·5	2·8	8·7	2·2	−2·16*
kcal		2264	659	2083	525	
Protein (g)		72·2	20·6	69·0	19·3	−1·28
Fat (g)		101·3	37·3	96·6	28·4	−0·98
Animal fat (g)		75·2	36·1	68·9	29·7	−1·37
Carbohydrate (g)		242·0	76·8	224·3	66·0	−1·80
Starch (g)		147·1	55·9	139·7	47·9	−1·09
Sugars (g)		95·2	41·1	85·1	41·1	−1·89
Fibre (g):						
Total		15·3	5·6	14·8	4·9	−0·74
Cereal		7·0	4·3	7·0	3·8	−0·06
Fruit/vegetable		8·3	2·9	7·8	2·6	−1·28
Vitamin C (mg)		51·7	21·9	47·8	20·6	−1·43
Alcohol (g)		21·9	28·4	15·7	27·0	−1·92‡

* $P < 0{\cdot}05$.
† For details of subjects and procedures, see pp. 303–305.
‡ Calculated after log transformation.

Table 4. *Energy intake at baseline and ischaemic heart disease (IHD) incidence in the total cohort, in men who had evidence of IHD at baseline and in men who had no such evidence**

		All men in the cohort			Men with no evidence of IHD at baseline			Men with IHD at baseline		
			Incident IHD			Incident IHD			Incident IHD	
'Fifth' of men	Energy intake (MJ/d (kcal/d))	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†
1	⩽ 7·6 (1820)	467	35	1·5	323	12	1·2	144	23	2·1
2	7·6–8·8 (1821–2102)	467	31	1·3	343	18	1·5	124	13	1·3
3	8·8–9·9 (2103–2369)	466	26	1·2	364	16	1·2	102	10	1·1
4	9·9–11·5 (2370–2741)	467	22	0·9	361	10	0·8	106	12	1·3
5	> 11·5 (2741)	467	23	1·0	365	14	1·0	102	9	1·0

* For details of subjects and procedures, see pp. 303–305.
† Adjusted for the effects of age, body mass index, smoking and, in the figures for the total cohort, for evidence of IHD at baseline.

Table 5. *Mean nutrient intakes (% of total energy intakes) for the total cohort of men, subdivided into those who went on to experience an ischaemic heart disease (IHD) event during the next 5 years and those who did not**†*

(Mean values and standard deviations)

	n ...	Men who experienced no IHD event 2197		Men who had an IHD event 137		
Nutrient		Mean	SD	Mean	SD	*t*
Protein (% energy)		13·0	2·0	13·2	2·4	1·02
Fat (% energy)		40·1	7·2	40·9	6·5	1·34
Animal fat (% energy)		29·5	8·0	29·7	8·5	0·31
Carbohydrate (% energy)		40·4	6·6	40·1	6·3	−0·51
Starch (% energy)		24·9	6·9	24·7	6·7	−0·37
Sugars (% energy)		15·5	4·5	15·4	4·9	−0·18
Fibre (g/4·2 MJ (10^3 kcal)):						
Total		7·2	2·6	7·1	2·5	−0·42
Cereal		3·4	2·0	3·3	2·0	−0·44
Fruit and vegetable		3·8	1·4	3·8	1·3	−0·14
Alcohol (% energy)		6·4	6·8	5·7	6·5	−1·45‡

* For details of subjects and procedures, see pp. 303–305.

† In all estimates of percentage contribution to energy, the contribution from alcohol has been included in total energy.

‡ Significance tested after log transformation.

Table 6. *Total fat intake at baseline and ischaemic heart disease (IHD) incidence in the total cohort, in men who had evidence of IHD at baseline and in men who had no such evidence***

		All men in the cohort			Men with no evidence of IHD at baseline			Men with IHD at baseline		
			Incident IHD			Incident IHD			Incident IHD	
'Fifth' of men	% Energy from fat	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†
1	⩽ 34·2	467	20	1·0	351	11	1·0	116	9	1·0
2	34·3–38·1	467	26	1·3	356	15	1·5	111	11	1·1
3	38·2–41·6	467	21	1·1	368	11	1·0	99	10	1·3
4	41·7–45·8	467	41	2·2	341	18	1·8	126	23	2·7
5	> 45·8	466	29	1·3	340	15	1·3	126	14	1·4

* For details of subjects and procedures, see pp. 303–305.

† Adjusted for the effects of age, body mass index, smoking and, in the figures for the total cohort, for evidence of IHD at baseline.

of any increased risk of subsequent IHD in the men who had obtained the greater amounts of their energy from animal fat.

A similar analysis of the data for the intakes of dietary fibre gave no convincing evidence of any trend in IHD risk with increasing intakes. Although the absolute intakes of fibre differed, particularly that obtained from fruit and vegetables ($P < 0·05$; Table 1), this

Table 7. *Animal fat intake at baseline and ischaemic heart disease (IHD) incidence in the total cohort, in men who had evidence of IHD at baseline and in men who had no such evidence**

		All men in the cohort			Men with no evidence of IHD at baseline			Men with IHD at baseline		
			Incident IHD			Incident IHD			Incident IHD	
'Fifth' of men	% Energy from animal fat	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†
1	⩽ 22·3	467	33	1·0	341	14	1·0	126	19	1·0
2	22·4–27·2	466	17	0·5	351	11	0·8	115	6	0·3
3	27·3–31·3	467	25	0·8	371	12	0·9	96	13	0·8
4	31·4–36·2	467	31	1·0	350	18	1·3	117	13	0·7
5	> 36·2	467	31	0·9	343	15	0·9	124	16	0·8

* For details of subjects and procedures, see pp. 303–305.
† Adjusted for the effects of age, body mass index, smoking and, in the figures for the total cohort, for evidence of IHD at baseline.

Table 8. *Vitamin C intake at baseline and ischaemic heart disease (IHD) incidence in the total cohort, in men who had evidence of IHD at baseline and in men who had no such evidence**

		All men in the cohort			Men with no evidence of IHD at baseline			Men with IHD at baseline		
			Incident IHD			Incident IHD			Incident IHD	
'Fifth' of men	Vitamin C intake (mg/d)	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†
1	⩽ 34·7	483	38	1·6	354	17	1·5	129	21	1·8
2	34·8–43·5	483	31	1·3	358	16	1·3	125	15	1·4
3	43·6–52·3	484	30	1·4	365	15	1·4	119	15	1·4
4	52·4–66·4	480	27	1·2	371	15	1·3	109	12	1·1
5	⩾ 66·5	484	22	1·0	362	11	1·0	122	11	1·0

* For details of subjects and procedures, see pp. 303–305.
† Adjusted for the effects of age, body mass index, smoking and, in the figures for the total cohort, for evidence of IHD at baseline.

difference did not appear to be independent of the bulk of the diet, and when standardized for total energy (using this as a surrogate for bulk) the differences disappeared (Table 5).

Vitamin C is of interest because of its antioxidant property. The data in Table 8 showed a consistent gradient within the total cohort, and within the two sub-groups of men, suggesting a negative association with IHD risk, but none of the trends achieved statistical significance.

The relationship between alcohol consumption and incident IHD is presented in Table 9. Among the total cohort there was a tendency for incidence to decrease with increasing consumption, but the trend was not significant. There was little evidence of such a trend in the men who had no evidence of IHD at baseline. However, there was a significant negative trend ($P < 0{\cdot}05$) in those who had evidence of IHD at baseline. It is possible that

Table 9. *Alcohol intake at baseline and ischaemic heart disease (IHD) incidence in the total cohort, in men who had evidence of IHD at baseline and in men who had no such evidence**

		All men in the cohort			Men with no evidence of IHD at baseline			Men with IHD at baseline		
			Incident IHD			Incident IHD			Incident IHD	
'Fifth' of men	% Energy from alcohol	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†	No. of men	No.	Relative odds†
1	Nil	157	12	1·5	108	5	1·3	49	7	2·4
2	0·01–0·99	431	26	1·2	311	12	0·8	120	14	1·8
3	1·00–4·99	662	44	1·3	488	18	0·8	174	26	2·3
4	5·00–9·99	567	28	1·1	454	17	0·8	113	11	1·4
5	⩾ 10·00	517	27	1·0	395	18	1·0	122	9	1·0

* For details of subjects and procedures, see pp. 303–305.

† Adjusted for the effects of age, body mass index, smoking and, in the figures for the total cohort, for evidence of IHD at baseline.

this association may be generated by men who had the most severe IHD at baseline having stopped or reduced their alcohol consumption. However, our data are limited as no enquiries had been made about past drinking habits in the baseline examinations, so that ex-drinkers cannot be distinguished from life-long abstainers.

DISCUSSION

There have been many cross-sectional studies of dietary intakes and the presence of IHD, and we have previously presented evidence from such an examination of the Caerphilly sample of men (Fehily *et al.* 1987). However, the presence of symptoms of IHD may lead to modifications in food consumption and, therefore, the prospective study, in which estimates of dietary intake are made before an IHD event, is very much to be preferred. One of the main limitations of the prospective approach is the difficulty in establishing a cohort of sufficient size to give a reasonable likelihood of detecting clinically important associations. The Caerphilly Study is of value because the numbers are reasonably large and the work focuses on an age-range of men in whom IHD incidence is fairly high. It is also the only major UK dietary study, the only other prospective evidence for this country coming from the small study of 337 selected subjects (Morris *et al.* 1977).

With one exception (in which the weighed inventory method was used (Morris *et al.* 1977)), all the major studies of diet and incident IHD have been based on 24 h recall, questionnaire or a diet history, and these methods represent a compromise. The questionnaire we used was well developed and tested, and having 7 d weighed intake data from a substantial proportion of the cohort meant that appropriate estimates of mean portion sizes could be used in the estimation of quantitative intakes for items for which the questionnaire gave information only on frequency. At the same time it should be recognized that there is no perfect dietary survey method and that questionnaires may have advantages over short-term food-weighing techniques since they are designed to obtain details about 'usual' intakes of nutrients.

The lower total energy intakes of those who went on to have an IHD event is consistent with the results of virtually every other cohort study which has been reported (Paul *et al.*

Table 10. *Relationship between intake of total fat and of saturated fatty acids (or animal fat) and incidence of ischaemic heart disease (IHD) in major prospective studies*

	Total fat (% total energy)			Saturates (% total energy)		
	Mean intake			Mean intake		
	No incident IHD	Incident IHD	Difference: Incident IHD − no incident IHD	No incident IHD	Incident IHD	Difference: Incident IHD − no incident IHD
Framingham*	38·8	40·0	+1·2	14·9	14·8	−0·1
Puerto Rico†						
Urban	36·6	37·7	+1·1	13·5	13·3	−0·2
Rural	32·2	32·0	−0·2	12·6	14·0	+1·4
Honolulu‡	33·3	35·2	+1·9	12·3	13·0	+0·7
London§	40·5	40·0	−0·5	—	—	—
Zutphen‖	41·7	41·8	+0·1	17·6	17·7	+0·1
Ireland–Boston¶	38·5	39·4	+0·9	16·9	17·4	+0·5
Caerphilly	40·1	40·9	+0·8	29·5**	29·7**	+0·2**

* Gordon *et al.* (1981).
† Garcia-Palmieri *et al.* (1980).
‡ McGee *et al.* (1984).
§ Morris *et al.* (1977); Marr & Morris, (1981).
‖ Kromhout & Coulander, (1984).
¶ Kushi *et al.* (1985).
** Animal fat.

1963; Morris *et al.* 1977; Garcia-Palmieri *et al.* 1980; Gordon *et al.* 1981; Kromhout & Coulander, 1984; McGee *et al.* 1984; Kushi *et al.* 1985). Indeed, the consistency of data from cohort studies with regard to energy intake has already been highlighted (Silman & Marr, 1985). The lower energy intake appears to be the result of a generally lower consumption of most food items rather than an effect of any particular nutrient and is probably a reflection of a lower level of physical activity.

The absence of a difference in mean total fat intake between men who went on to have an IHD event and those who did not is consistent with the literature. A summary of the results of several other major cohort studies (Table 10) showed that men who went on to experience IHD had a very similar mean proportion of energy from fat to that of men who did not experience an IHD event (Morris *et al.* 1977; Garcia-Palmieri *et al.* 1980; Gordon *et al.* 1981; Marr & Morris, 1981; Kromhout & Coulander, 1984; McGee *et al.* 1984; Kushi *et al.* 1985). Recent data from the Framingham Study did, however, show a positive association between the percentage of energy from total fat and 16-year incidence of IHD in 420 men aged 45–55 years, but no association in 393 men aged 56–65 years (Posner *et al.* 1991).

It is unfortunate that the version of our questionnaire used in the baseline examination of the Caerphilly cohort did not allow the estimation of saturated and polyunsaturated fatty acid intakes. We were, however, able to estimate the intake of fat from animal sources and this was found to be identical, when expressed as proportionate contribution to energy, among men who went on to have an IHD event and those who did not. This is also in accord with the results of other within-population cohort studies (Table 10) which have reported very small differences in intakes of saturated fatty acids or of animal fat (Morris *et al.* 1977; Garcia-Palmieri *et al.* 1980; Gordon *et al.* 1981; Shekelle *et al.* 1981; Kromhout & Coulander, 1984; McGee *et al.* 1984; Kushi *et al.* 1985; Posner *et al.* 1991). In the study

of London Busmen and Bankers (Morris *et al.* 1977) and the Western Electric Study (Shekelle *et al.* 1981) intakes of animal fat or of saturated fatty acids were estimated but mean intakes not presented. No significant association was found between the percentage of energy obtained from animal fat (London Study) or the percentage of energy obtained from saturated fatty acids (Western Electric Study) and IHD. However, in both the Western Electric Study (Shekelle *et al.* 1981) and the Ireland-Boston Study (Kushi *et al.* 1985) a positive association was reported between subsequent IHD and both the Keys (Keys *et al.* 1965) and Hegsted (Hegsted *et al.* 1965) dietary scores, which take into account the intake of polyunsaturated fatty acids and cholesterol as well as saturated fatty acids.

Fat intakes are probably more difficult to estimate than those of almost any other nutrient since fats are widely distributed throughout the diet and intakes probably vary considerably from day to day. At the same time, the absence of clear differences in mean intakes of saturated fatty acids or of animal fat in all these published studies is unlikely to be due simply to imprecision in the measurements of intakes. An alternative explanation which has been suggested (Rose, 1985) is that, in populations where the majority have a high fat intake, factors other than fat become important in determining why some individuals develop IHD and others do not.

There was no difference in the baseline percentage of energy from total sugars of those who subsequently had an IHD event and those who did not. This is consistent with data from other within-population cohort studies, none of which has provided any evidence of an association between consumption of sugars and subsequent IHD risk (Morris *et al.* 1977; Garcia-Palmieri *et al.* 1980; Gordon *et al.* 1981; Kromhout & Coulander, 1984; McGee *et al.* 1984; Kushi *et al.* 1985).

Dietary fibre intakes were lower among men who subsequently had an IHD event than among those who did not, but the difference appears simply to be a reflection of the difference in the bulk of the diet. In four previous cohort studies (Morris *et al.* 1977; Kromhout & Coulander, 1984; Kushi *et al.* 1985; Khaw & Barrett-Connor, 1987) fibre intakes were found to be lower among those who subsequently developed IHD. The differences were small, but in three studies they were found to be independent of energy intake. Unfortunately only one randomized controlled trial of a modification of dietary fibre (from cereals) and IHD seems to have been reported (Burr *et al.* 1989) and an overall evaluation of dietary fibre in relation in IHD must take into account the negative result of this trial.

There has been much recent interest in the potential protective effect of nutrients with an antioxidant property. Our data from Caerphilly are limited, and while they give no convincing evidence of a protective effect of vitamin C, there are trends which are of interest (Table 8). Published evidence on vitamin C and IHD is scarce. A study of 150 patients who underwent cardiac catheterization (Ramirez & Flowers, 1980) showed that those with abnormal coronary arteries had lower leucocyte ascorbic acid levels than those with normal coronary arteries and that this was independent of smoking habit. However, in an angina case–control study (Riemersma *et al.* 1991) vitamin C was not significantly related to risk of angina once smoking habit had been taken into account. In the Basel Prospective Study (Gey *et al.* 1987), no association was found between plasma vitamin C and IHD.

Our results for alcohol are limited because of an absence of data on life-time abstinence. However, what we present is in general accord with the literature. Mean consumption has generally been found to be lower among men who subsequently develop IHD than among those who do not (Garcia-Palmieri *et al.* 1980; Gordon *et al.* 1981; McGee *et al.* 1984), and a higher cardiovascular mortality has been reported among non-drinkers than among moderate drinkers (Dyer *et al.* 1981; Kagan *et al.* 1981; Marmot *et al.* 1981; Cullen *et al.* 1982; Kozarevic *et al.* 1982; Shaper *et al.* 1988; Klatsky *et al.* 1990). In our data there was

a significant negative association between alcohol consumption and IHD incidence among men with evidence of IHD at baseline, but little or no trend in men without evidence of IHD at baseline. One possible explanation for this is that men with more severe IHD at baseline had selectively cut down or given up alcohol.

In conclusion, the investigation of dietary factors of relevance to IHD is difficult. Each kind of study has its limitations, and most of these are serious. In epidemiology neither the case–control nor the cross-sectional prevalence study is of great value because of likely changes in diet by subjects who have experienced symptoms of IHD. Amongst the limitations of intervention trials is the difficulty in persuading subjects to change their diet and maintain a sufficient change for a prolonged time. These particular difficulties are somewhat less in trials based on patients who have already had a manifestation of IHD, although it can be argued that the underlying disease processes are too advanced for dietary changes to have much effect in secondary prevention. At the same time, the demonstration of a significant reduction in mortality in a 2-year trial of fatty fish (Burr *et al.* 1989) shows that this last is not always the case.

The prospective strategy, such as the one used in the Caerphilly Study, has its own limitations. These include the difficulties in estimating the usual dietary intakes of free-living subjects. However, prospective studies have the great advantage that in a stable population dietary intakes measured at a particular point in time are likely to reflect intakes over a prolonged period, and here questionnaire methods which attempt to ascertain 'usual' intakes may be of value. It seems reasonable to assume that if a nutrient is of relevance to the risk of a disease those subjects with the highest intakes should subsequently have a higher incidence of the disease than those subjects with the lowest intakes.

The absence in all the major prospective studies of clear evidence of relevance of any nutrient to the subsequent risk of IHD is certainly challenging.

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