TITLE: Dietary fats and 16-year CHD mortality in a cohort of men and women in Great Britain

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Abstract

Objective The paper aims to investigate the relationships of dietary fats to subsequent CHD mortality in men and women while taking account of other CHD-related behaviours.

Design A cohort of randomly selected men and women was interviewed in 1984-85 and monitored subsequently for 16 years for deaths. The interview covered health, health related behaviours, physical measurements, socio-demographic details and a dietary questionnaire. Appropriate exclusions left 1225 men and 1451 women aged 40-75 with 98 and 57 CHD deaths respectively. Saturated, polyunsaturated and total fat intakes were estimated.

Setting The sample was randomly selected from households in Great Britain. The interviews took place in participants' own homes.

Results Not consuming alcohol, smoking, not exercising and being socially disadvantaged were related to high saturated fat intake and CHD death. Cox survival analyses adjusting for these factors found that a level of saturated fat 100 grams per week higher corresponded to a relative risk for CHD death for men of 1.00 (0.86-1.18) and 1.40 (1.09-1.79) for women. This difference between the effects of saturated fat in men and women was statistically significant (p=0.0190). Results are also reported for total fat and the relative effects of polyunsaturated and saturated fats.

Conclusions Strong evidence was found for the within cohort relationship of dietary fat and CHD death in women while no evidence was found for a relationship in men.

Possible explanations for this are discussed.

Descriptors:

dietary fats, coronary heart disease (CHD), cohort studies, health surveys, sex differences, epidemiology, health behaviour

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Introduction

The relationship between dietary fat intake and coronary heart disease (CHD) remains a subject of debate despite the many years of research devoted to it (Ravnskov, 1998; Hu *et al*, 2001). There is support for the relationship from between-country comparisons (Keys, 1980), migrant studies (Robertson *et al*, 1977), dietary intervention studies (Miettinen *et al*, 1972; Burr *et al*, 1989; Truswell, 1994; Brousseau and Schaefer, 2000) and studies involving serum cholesterol as intermediary between diet and CHD (Hegsted *et al*, 1965; Stamler *et al*, 1986, Millen *et al,* 1996). A prospective cohort study of dietary fats to CHD death in a population of US male health professionals showed a weak relationship (Ascherio *et al*, 1996). Other prospective cohort studies in men, however, have produced inconsistent support for the hypothesis (Morris *et al*, 1977; Gordon, *et al*, 1981; Shekelle *et al*, 1981; Kromhout and de Lezenne Coulander, 1984; Kushi *et al*, 1985; Fehily *et al*, 1993; Pietinen *et al* 1997). Only one large cohort study of diet and CHD in women has been reported (Willett *et al*, 1993; Hu *et al*, 1997; Hu *et al,* 2000). It found a relationship of dietary fat to CHD. Until now there has been no large cohort study including men and women. However, the cross-sectional Scottish Heart Health Study (Boltonsmith *et al*, 1992) reported no relationship in men or women between dietary fats and concurrent undiagnosed CHD.

The inconsistency of the findings from prospective cohort studies may be partly explained in terms of difficulties with the methods. Large samples are required to generate adequate numbers of CHD deaths. This makes for difficulties with the accuracy of measurement of dietary intakes and with obtaining good information on factors known to relate to CHD which are potentially confounding where they may

also relate to diet. This includes smoking (Doll and Peto, 1976; Doll *et al*, 1980), physical activity (Shaper and Wannamethee, 1991; Sesso *et al,* 2000), alcohol consumption (Rimm, *et al*, 1991), obesity and body shape (Donohue *et al*, 1987) and social class (Marmot *et al*, 1991; Boltonsmith *et al*, 1991). A related difficulty is the identification of classes of individuals likely to have changed their diets due to awareness of health risk.

The Health and Lifestyle Survey (Cox et al, 1987; Cox, 1988; Cox, 2001) has a large number of randomly selected male and female participants, includes measures of diet and of factors likely to confound with dietary fat and has 16 years follow-up of deaths. This makes it possible to compare men and women in the nature and strength of the relationship of dietary fat to CHD mortality while dealing with the above methods issues.

Method

THE SAMPLE

The Health and Lifestyle Survey was conducted in 1984-85 through face-to-face interview, physical measurement and self-completion on a stratified random sample of 9003 individuals aged 18 years or older living in private households in Great Britain. The response rate was 73.5% of eligible addresses, which is normal for this type of study. The age, sex and regional distributions of the sample are similar to the 1981 UK Census. Information was obtained on health, lifestyles, dietary habits, social and demographic circumstances, personality and psychiatric status, health attitudes and beliefs and physiological measurements. The Office for National Statistics (formerly OPCS) flagged 97% of the participants for on-going death certificate monitoring. In this paper account has been taken of CHD deaths (ICD

codes 410 to 414) until December 2000. The analysis was limited to those aged 40-75 at the time of the initial interview.

To demonstrate a possible effect of diet on subsequent CHD death it was necessary to exclude individuals whose diet was likely to have been influenced by awareness of a specific illness or condition itself carrying a raised CHD risk. This required exclusion of those reporting heart disease, diabetes, anti-hypertensive treatment or being on a special diet. The importance of these exclusions is made clear by reference to Table 1. Individuals in these groups had high CHD death rates and low fat intakes compared to other men and women. If these groups were not excluded, the relationship of fat intakes to CHD could be masked. After exclusions there remained 1225 men and 1451 women aged 40-75 with 98 and 57 CHD deaths respectively.

[Table 1 here]

THE MEASURES

Information was obtained about general dietary habits through questions about the quantities and frequency of consumption of bread, butter, margarine, milk, coffee, tea, sugar in coffee, sugar in tea, and the frequency of consumption of 30 different food groups according to six categories ranging from *never* to *more than once a day*. The 30 food groups were selected in order to cover all commonly eaten foods and, in particular, all foods that make a significant contribution to dietary fat. A similar food frequency questionnaire was found by Yarnell (1983) to give estimates of total dietary fat which had a correlation of 0.34 with estimates from a 7-day weighed dietary record in 119 men. Distributions of food frequency categories reported by the respondents

for each food were matched to the corresponding distributions from the weighed intake data provided by the 1986-87 Dietary and Nutritional Survey of British Adults (DNSBA) (Gregory *et al*, 1990; Office of Population Censuses and Surveys, 1991). The resultant estimated food weights corresponding to each frequency category, after reference to food composition data, enabled estimates to be made of individual saturated, polyunsaturated and total fat intakes (Tefft and Boniface, 2000). The balance between saturated and polyunsaturated fats intake was represented by {2 (saturated fat)-(polyunsaturated fat)} as suggested by Keys in his formula relating changes in these fats to changes in serum cholesterol (Keys *et al*, 1965).

Measures were obtained for alcohol consumption, smoking, exercise activity, obesity, blood pressure, social class and local area deprivation as they were potentially confounded with fat intakes in their effects on CHD death. Details are in Table 2. Body shape was available on only a random 63% of the study sample. An analysis to explore the relationship between obesity and CHD death in these data has been published elsewhere (Cox, 1996, 1998).

[Table 2 here]

ANALYSES

Analyses were carried out of the pattern of relationships of physical, behavioural and demographic factors with fat intakes and with CHD death and non-CHD death separately for men and women in two groups according to age at interview, 40 to 59 and 60 to 75 years. Cox survival analyses (Cox and Oakes, 1984), explaining the length of survival without CHD death in terms of the amount of fat consumed while adjusting for the effects of age and any confounding factors, were carried out separately for men and women and for the combined sample. Tests of

statistical significance were carried out for trend by linear contrast and by Mantel-Haenszel in comparisons of means and proportions respectively. Group differences were tested by t-test or by chi-squared test and Cox regression coefficients by likelihood ratio test. All tests were non-directional and 95% confidence intervals were used throughout.

Results

UNIVARIATE ANALYSES

Figure 1 shows that sixteen-year CHD death rates for women followed the pattern of the men but lagged by approximately 10 years. Sixteen-year CHD death rates for men and women by age group are shown by fifths of the distributions of saturated, polyunsaturated and total fat intake and the Keys' fat difference (2S - P) in Table 3. The cut-off points for the quintiles of saturated fat in grams per week were 220, 276, 337 and 427 for men and 159, 202, 252 and 319 for women. There was a clear trend to higher CHD death rates associated with higher total and saturated fats and Keys' fat difference in women. Men exhibited similar but weaker trends. None of the trends across the five sets of quintiles were statistically significant in men.

[Figure 1 here]

[Table 3 here]

Table 4 provides details of the relationship of physical, behavioural, and demographic factors to saturated fat intake, CHD death and death from other causes, separately for men and women aged 40 to 59 and 60 to 75. It shows that behaviours, body shape, hypertension, and social disadvantage formed a cluster of factors that related directly to CHD death but not to death from other causes. In men aged 40-59, age, alcohol, smoking, exercise, and social class relate to both saturated fat and CHD death and would be confounding in analyses relating saturated fat to CHD death. Their status as potential confounders is confirmed by a number of similar relationships in men aged 60-75 and in both age groups of women. In light of the aim to compare men and women these factors were used for adjustment in all multivariate analyses.

[Table 4 here]

MULTIVARIATE ANALYSES

A comprehensive adjustment was made for age as it was the most important confounding variable. In the Cox regression models age was included as a continuous variable to account for the linear effect of age and further as a stratification variable with two levels (40-59, 60-75) to account for any non-linear effect. This worked sufficiently well to make it possible to fit models to a single age group (40-75). The results are given in Table 5.

No relationship between dietary fats and CHD death was found in men, but a substantial and statistically significant relationship was found in women. Analysis separately for age groups 40 to 59 and 60 to 75 showed that the relationship appeared to be stronger for the older women. Adjustment for smoking, alcohol consumption, exercise and social class in the age adjusted multivariate models for men and women had a negligible effect on the relative risks for fat intakes. There were statistically significant differences between the effects for men and women (total fat p=0.0533; saturated fat p=0.0190; Keys' fat difference p=0.0160). Noting that the weekly saturated fat intake for a woman which is in proportion to 100g intake for a man is 72g (Gregory *et al*, 1990) and re-scaling women's fat intake by this amount in the multivariate Cox regression model for women reduced the relative risk of CHD death for a woman from 1.40 to 1.27 (p=0.0074). This re-scaled effect was still significantly greater than the men's effect (p=0.0381).

The Cox analyses for the effect of polyunsaturated fat did not reach statistical significance.

[Table 5 here]

Discussion

The results in Table 5 show that, on average, women (age adjusted to 55 years) whose weekly saturated fat intake is 100g higher have a risk of CHD death over the subsequent 16 years which is 38% higher than in otherwise comparable women. The corresponding difference for men is 5%. This difference between men and women is to some extent supported by separate cohort studies of men (e.g. Pietinen *et al*, 1997) and women (e.g. Hu *et al*, 1997; Millen *et al*, 1996). The result for the Keys statistic indicates that a higher level of saturated fat can be compensated by a lower level of polyunsaturated fat, in the ratio 2:1.

Several explanations may account for this apparent difference between men and women. One possibility is the single measure of dietary fat at interview is a less valid estimate of the diet in men than in women. This could either be because the instrument elicits less accurate responses from men or because men's diets vary more over time. We have no results from validation exercises that suggest lower validity for men, however, the proportions of men in full time work in this study are 87% and 23% in age groups 40-59 and 60-75 compared to 32% and 4% in women. Also 29% of women aged 40-59 were occupied by 'keeping house' compared to 0.3% of men. Thus women were more likely to be purchasing and preparing food and men more likely to be eating out while at work. This could well lead to women reporting their diets more accurately than men. Only further research will clarify this issue.

A second possibility is that non-dietary behaviours (i.e. smoking, alcohol, exercise) relate to dietary fat and CHD death in different ways in men than in women. Support for this is found in the fact that their unique contributions in the Cox models

in Table 5 are highly statistically significant in men but not in women. This could lead to confounding effects not sufficiently controlled in the analysis.

A third possibility is the role played by social factors. Social class is known to be linked to lifestyle factors such as diet, alcohol intake, smoking and exercise (Marmot *et al*, 1991; Boltonsmith, 1991) and in the present study's multivariate analyses for women it had a strong relationship with CHD death ($p=0.0037$) which was not mediated through these behaviours. Thus non-behavioural aspects of social class such as access to health-related resources may provide the link with CHD death. This is supported by the strong relationship in women of local area deprivation with CHD death (see Table 4). Social class showed no relationship with CHD death in the multivariate analyses for men. Further research is required to explore the different role of social class in CHD death and diet between men and women.

LIMITATIONS OF THE STUDY

Validity and reliability of the fat intake estimates are an issue in any large cohort study of diet. Sources of error in the fat estimation method in the present study include errors made by respondents in stating the frequency with which foods are usually eaten, errors due to systematic differences in portion sizes according to the amount of food eaten and errors in the matching to the DNSBA weighed intake study. Purely random errors in the estimation of dietary fats are likely to decrease the apparent size of the relationship of fats to CHD death while not introducing bias.

Errors in death certification with respect to CHD are likely to lead to underestimation of the true strength of the relationship of dietary fat to CHD death. Recent research has found no differences between men and women in the accuracy of death certification with respect to CHD. (UK Heart Attack Study Collaborative

Group, 1998). This cannot, therefore explain the differences between men and women.

A potential source of non-random error arises from the lack of an adjustment for total energy intake of the participants (Willett, 1990). Any apparent effect on CHD risk of dietary fat could, in principle, be due to the effect of total energy intake. However, recent cohort studies which have considered this effect (Kromhout and de Lezenne Coulander, 1984; Kushi *et al*, 1985; Fehily *et al*, 1993; Esrey *et al*, 1996) found lower energy intakes in those with CHD compared to those without by amounts ranging from 1.6% to 14.8% lower. Therefore, not adjusting dietary fats for total energy intake could be expected to reduce their apparent effects on CHD leading to the relative risks for fat reported in this paper being underestimates. The analyses reported in this paper do not provide guidance as to whether or how a person reducing his or her fat intake should replace the calories.

CONCLUSIONS

This large cohort study, a random population of Great Britain, shows important and statistically significant relationships of estimated dietary fat and CHD in women but not in men. A number of explanations for this difference are proposed in terms of behavioural and social factors and in terms of methodology. There is no evidence from metabolic studies to support a difference of the size reported in the present study. Further research should be carried based on large cohorts including both men and women.

The differences between men and women found in this study may have implications for public health. To reduce CHD death rates in men, it may be more important to target smoking and exercise than dietary fat; whereas for women, it may

be most important to reduce dietary fat while also paying attention to the consequences of local area deprivation and disadvantaged social class.

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