Department of Health and Social Security

Report on Health and Social Subjects

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Report of the Advisory Panel of the Committee on Medical Aspects of Food Policy (Nutrition) on Diet in relation to Cardiovascular and Cerebrovascular Disease

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Committee on Medical Aspects of Food Policy (Nutrition)

Advisory Panel on Diet in relation to Cardiovascular and Cerebrovascular Disease

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Preface

In June 1970 the Committee on Medical Aspects of Food Policy set up a Panel to advise on the significance of any relation between nutrition and cardiovascular and cerebrovascular disease. In doing so the Committee were aware of the complexities of the subject, of the vast amount of scientific literature which has accumulated and of the interest of all sections of the community.

The Panel have taken three years and ten drafts to produce their Report. It represents a distillation by long and critical discussion of facts that might be relevant to the formulation of food policy in the United Kingdom. Much of the evidence comes from outside the United Kingdom, and geographical differences which have emerged from the study of the statistics of death from coronary heart disease illustrate the potential dangers of assuming that the results of investigations made in one country necessarily apply in another. Not all members of the Panel have been able to subscribe to all the conclusions. This is noted in the text and the "Note of Reservation" by Professor Yudkin expresses his inability to agree completely with the other members of the Panel in some important respects.

The Report may be considered by many to be unexciting and may well attract less attention because it does not suggest that prevention of coronary disease can certainly or easily be effected by dietary means. It does, however, give advice which, if taken, would be beneficial to the health of our people. The advice cannot be better expressed than in the words of St. Paul 'Let your moderation be made known to all men' (Philippians 4.5). In addition the Report suggests areas in which further research is particularly needed, although no attempt is made to set out a co-ordinated plan for research.

The Chairman, Professor Sir Frank Young F.R.S., has described the members of the Panel as 'intelligent, eloquent, critical, strong-minded protagonists of various views' and we owe it to his skill and perseverance that such a team persisted in their task and without acrimony arrived at a conclusion. The Committee is indeed grateful to the Chairman and to the Panel members for their expert knowledge and for the time and energy which they have expended in producing a Report which will surely be described as 'level-headed' and moderate—attributes of special significance in this subject at the time. Perhaps the most important contribution they have made to the problem of the prevention of coronary heart disease is to eschew the temptation to suggest that simple dietary changes can alone solve a problem of such complexity.

G E GODBER
Chairman,
Committee on Medical Aspects
of Food Policy (Nutrition)
October 1973

Contents

1. Introduction

1.1 Terms of reference

To advise the Committee on Medical Aspects of Food Policy on the significance of any relation between nutrition and cardiovascular and cerebrovascular disease, and on any indication for future action.

1.2 Meetings

The Panel has met twelve times and has had the benefit of discussion with a number of experienced investigators.

1.3 Form of the Report

1.3.1 This Report is almost wholly confined to an interpretation of the evidence concerning the influence of the composition and amount of the diet on the death rate from cardiovascular and cerebrovascular disease in man. Although atherosclerotic thickening of the arterial wall has been described in non-human species, thrombotic obstruction of vessels is found with significant frequency only in man. The results of investigations on animals other than man have therefore been considered only when they appear to be directly applicable to the human condition.

1.3.2 As far as possible the evidence considered has related to dietary and other conditions in the United Kingdom of Great Britain and Northern Ireland.

1.3.3 Our Report refers to the population as a whole, but the conclusions and recommendations do not necessarily extend to people with gross metabolic abnormalities.

1.3.4 Although the clinical consequences of cardiovascular and cerebrovascular disease rarely appear until adult life, changes in the arterial wall may begin in childhood. We have therefore not neglected to consider the paediatric aspects of our terms of reference.

1.3.5 The Report aims to interpret much complex evidence in such a way that the conclusions reached may be clear to, for example: (1) organizations and individuals responsible for guidance of the public in relation to health and diet; (2) doctors who need to give advice to patients, and (3) scientific advisers of food manufacturers. In addition, we are aware of the wish of the Food

Standards Committee of the Ministry of Agriculture, Fisheries and Food, who recommend legislative measures for the control of the composition and labelling of food, to base their decisions upon informed medical opinion (para 2.4).

1.3.6 In our deliberations we have discussed a large number of published scientific papers together with many working papers prepared by individual members of the Panel. We have tried in this way to ensure that a wide range of evidence relevant to any relationship between diet and cardiovascular and cerebrovascular disease has been considered. The Report summarizes the findings and conclusions of the Panel without specific reference to source material, but a list of the publications consulted is appended (p.34). The latest publications consulted appeared in mid-1973.

1.3.7 Because of the complex nature of the evidence and because conflicting interpretations of it are possible, we have not always been able to achieve agreed conclusions. Where unanimity has not been reached this fact is indicated in the text.

1.3.8 The Members of the Panel record their appreciation and thanks for the expert assistance provided by the Assessors to the Panel, and by the Secretariat. The Members have agreed to record their indebtedness to Dr. S. J. Darke, an Assessor from the Department of Health and Social Security, whose help in the preparation of the many drafts of the Report has been specially valuable.

2. General Considerations

2.1 Interpretation of the Terms of Reference

2.1.1 The term cardiovascular disease can be interpreted to include almost all diseases that affect the circulatory system. Coronary obstruction leading to ischaemic heart disease is a more frequent cause of death in the United Kingdom than any other disease of the circulatory system. This Report is mainly concerned with ischaemic heart disease (I.H.D.), which corresponds with what is popularly called coronary heart disease*. Nevertheless we have given some attention to cerebrovascular disease* and to peripheral vascular disease*.

2.1.2 An examination of any relationship between diet and disease does not necessitate a consideration of the intimate mechanisms involved. We have therefore agreed to consider the evidence linking the diet with the development of cardiovascular disease without an examination of the pathways which may be implicated. Nevertheless we agree that research into the underlying metabolic mechanisms is important, and that the investigation of dietary influences on, for example, the mechanisms by which arterial thrombi are produced and on the integrity of the vascular system, should be pursued with vigour.

2.2 The death rate from cardiovascular disease in the United Kingdom

2.2.1 References occur in the medical literature to the "modern epidemic" of coronary heart disease. The evidence about the death rate from a disease is likely to be more reliable than that about its incidence. While suitable statistical adjustments can allow for the rising proportion of older people in the population, the effect on the recorded death rate of better diagnosis and treatment, and of changing fashions in the classification of disease, is much more difficult to assess.

22.2 A major revision (the sixth) of the International Classification of Diseases (I.C.D.) was adopted by all member states of the World Health Organization for use from 1950. The seventh (1955) and eighth (1968) revisions of the I.C.D. introduced substantial changes, some of which affected the classification of cardiovascular disease. From 1950 to 1967, in both the sixth and seventh revisions, the condition that we have referred to as I.H.D. is the major component of the categories 420-422, while from 1968 onwards categories 410-414 of the eighth revision are those which correspond most closely to I.H.D.

*See Glossary, p 24.

2.2.3 Because of these past changes in classification we restricted our preliminary study of death rates from the various forms of cardiovascular and cerebrovascular disease to those from 1950 onwards. Only with respect to I.H.D. was a striking increase in death rate observed, and we therefore confined our attention largely to this condition.

2.2.4 The information summarized in Table 1 (p 25) and in Figures 1, 2 and 3 (pp 29, 30 and 31) reveals that in England and Wales, Scotland and Northern Ireland between 1950 and about 1965 there was a steady rise in the death rate from I.H.D. of men in the three ten-year age groups from 35 to 64 years. During the same period the death rate of the younger men (aged 35-44 years) increased more rapidly than that of older men. But since about 1965 among each of the three age groups a steady rise in the death rate in men is no longer evident.

2.2.5 With one exception there has been little or no change in the death rate from I.H.D. of the women in all the three age groups, throughout the component populations of the United Kingdom (Table 1 (p 25) and Figures 1, 2 and 3 (pp 29, 30 and 31)). The exception is the youngest age group of women (35-44 years) in England and Wales, among whom there has been a significant rise in the death rate from I.H.D. since about 1960.

2.2.6 For men and for women of all the three age groups the death rate from I.H.D. is higher in Scotland and in Northern Ireland than it is in England and Wales. These facts are illustrated by the information given in Table 2 (p 26).

2.2.7 The death rate from I.H.D. is less for women than for men in all three populations among all the three age groups; in women it is very approximately that for men 10 years their junior, and for men in age group 55-64 years the death rate is at least 2-3 times that of women of the same age.

2.2.8 The available evidence about the death rate from I.H.D. necessarily cannot be more reliable than the information upon which the death certificate is based. Understanding of the disease might be made more effective if statistics were more reliable and there were additional information about topographical differences. The possible advantage of designating I.H.D. a notifiable disease, and of more frequent confirmation of the diagnosis by post-mortem examination, should be seriously considered.

2.2.9 The fact that the difference in death rate from I.H.D. between women and men disappears in diabetic patients is a subject for further research.

2.3 Conditions which predispose to the development of ischaemic heart disease

2.3.1 Paragraphs 2.2.6 and 2.2.7 indicate that susceptibility to fatal I.H.D. is influenced by the age and the sex of an individual and differs according to the area of the United Kingdom in which a person lives. In addition to the possible

influence of the nature of the diet, the most important factors which have been cited as predisposing to the development of I.H.D. include (a) a raised serum cholesterol* concentration; (b) a raised serum triglyceride* concentration; (c) hypertension; (d) excessive cigarette smoking; (e) insufficient physical activity; (f) the presence of diabetes mellitus; (g) being overweight from any cause; (h) an inherited predisposition, and (i) emotional stress†.

2.3.2 Discussion of the aetiology of a disease can involve the use of the concept of "risk factors" rather than "causes". For example, the tubercle bacillus is accepted as the cause of tuberculosis because the disease does not occur in the absence of the organism. There are, however, other conditions such as poverty and malnutrition which are frequently associated with tuberculosis but which cannot be regarded as causes of it. Such conditions may be referred to as risk factors, since their presence involves a greater likelihood of the development of the disease, without the implication of a primary causal relationship.

2.3.3 A raised serum cholesterol concentration, hypertension and excessive cigarette smoking can all be regarded as risk factors for I.H.D. In two comparable groups of men living in the United Kingdom, one consisting of those who smoke cigarettes and the other of non-smokers, more cases of I.H.D. will be observed over a period of time in the first group than in the second. But not all the heavy smokers will have heart attacks while some of those who do suffer such attacks will be non-smokers. The assertion therefore cannot be made that excessive smoking is an essential cause of coronary disease: nevertheless it is a risk factor in the sense that among smokers the probability of developing the disease is greater than it is among non-smokers. Similar considerations apply to a high blood pressure and to a raised serum cholesterol concentration.

2.3.4 The relative importance of risk factors differs among the individuals who constitute a population, and between populations. Any given individual may have more than one risk factor, for example, a raised serum cholesterol concentration, a high blood pressure, and excessive cigarette smoking, and the combined effect of two or more risk factors may increase the individual's susceptibility for I.H.D. either in an additive way or to an extent greater than the sum of the effects of the separate factors. Some measure of the degree of susceptibility of an individual or a population to I.H.D. can be assessed by a consideration of how many and which risk factors are involved, but this cannot be done with precision.

2.3.5 The preceding discussion indicates the complex background of the problems involved in the design of investigations, on groups of human beings, which are undertaken with the object of elucidating the influence of any factor, including dietary ones, on susceptibility to I.H.D. (See also paras 6.5 and 6.6).

*See Glossary, p 24.

†In the opinion of the Panel there is not at present enough firm evidence to warrant discussion in this Report of emotional stress as a risk factor for I.H.D.

2.4 Possible legislative implications of the present Report within the United Kingdom

In its Report on Claims and Misleading Descriptions, the Food Standards Committee of the Ministry of Agriculture, Fisheries and Food (1966) recommended (in para 20 of that Report) that no claim be permitted that any type of dietary fat affords protection against heart disease, or is of benefit to sufferers from this condition, until informed medical opinion has come to an agreed conclusion on the subject. The recommendations in the Bulletins of the Code of Advertising Practice Committee have been in accord with this view. In drafting our Report we have kept this situation in mind.

2.5 Public health recommendations about diet and cardiovascular and cerebrovascular disease in other countries

2.5.1 In a number of countries medical and other bodies have recommended dietary changes, on a national scale, which aim to reduce the incidence of cardiovascular and cerebrovascular disease throughout the population. So far as we are aware attempts have not been made to enforce any such recommendations by legislative action.

2.5.2 These recommendations differ among themselves, but involve some or all of the following proposals: (a) a reduction of food intake; (b) a reduction in the total amount of fat in the diet; (c) a reduction in the amount of saturated fatty acids in the diet; (d) an increase in the proportion of polyunsaturated fatty acids in the fat of the food; (e) a reduction in dietary cholesterol; (f) a reduction in the intake of sucrose, and (g) an increase in the proportion of cereal foods, fruit and vegetables in the diet.

3. Special Dietary Considerations

3.1 Diet and ischaemic heart disease

3.1.1 The dietary characteristics which we consider as possible risk factors for I.H.D. are (a) overconsumption of food; (b) an excess of dietary fat*; (c) a low ratio of polyunsaturated to saturated fatty acids* (P/S ratio) in the diet; (d) an excess of dietary cholesterol; (e) an excess of dietary sucrose; (f) a high consumption of common salt; (g) a deficiency of dietary fibre, and (h) softness of the water supply.

3.1.2 Other dietary factors which have been suggested as relevant include (a) an excess or deficiency of certain vitamins—vitamin B_{12}, thiamin and ascorbic acid; (b) an excess of cobalt in the food; (c) a deficiency of chromium in the food; (d) an imbalance of certain other trace elements; (e) the presence of heated milk protein in the food; (f) an excessive intake of coffee, and (g) an excessive consumption of alcohol. We have considered the results of research on a possible relationship between these dietary factors and susceptibility to I.H.D., and conclude that the evidence is at present insufficient to justify further discussion in this Report.

3.2 Diet and cerebrovascular and peripheral vascular disease

3.2.1 For cerebrovascular disease hypertension appears to be of greater significance than other risk factors, and obese persons are more likely to have a high blood pressure than those who are not obese. A constituent of the diet which may be significant in the pathogenesis of hypertension and cerebrovascular disease is common salt (sodium chloride).

3.2.2 We find that there is insufficient evidence on which to base a statement about any specific relationship between the composition of the diet and susceptibility to cerebrovascular or to peripheral vascular disease.

*See Glossary, p 24.

4. The United Kingdom Diet

4.1 In para 2.2.4 we conclude that the death rate from I.H.D. in men in the United Kingdom rose steadily between 1950 and about 1965. We have therefore reviewed the evidence for alterations in the composition of the food consumed by the inhabitants of the United Kingdom before and during that period, and also subsequently, in order to reveal possible relationships between dietary changes and alterations in the death rate from I.H.D. We are uncertain of the time scale within which such relationships would operate, and have therefore considered information relating to the consumption of food both in the longer term—since the beginning of this century, and in the shorter term—since 1950.

4.2 The interpretation of information about secular alterations in the consumption of food in the United Kingdom is complicated by the fact that since the beginning of the century the proportion of children in the population has fallen and that of the elderly has risen. We are unable to quantify the effect on the average consumption of food in the United Kingdom of these changes in the age-structure of the population. Nevertheless, any effect of the changes may not be large (see para 4.12).

4.3 There are two major sources of information about the consumption of food in this country. The first—Food Consumption Level Estimates (C.L.E.)—relates to total food supplies in the United Kingdom, and is derived from records of the import, export and production of food. The second is the National Food Survey of Great Britain (N.F.S.), in which the food purchased for consumption within a household is recorded. Meals consumed and paid for outside the household are not included nor is the consumption of alcohol, either inside or outside the household, taken into account.

4.4 Since the early years of the century the amount of protein available for consumption, derived from C.L.E. and expressed as the average weight per person per day (Table 3, p 26), has altered very little. From 1909 until 1945 there was a small rise of about 10%, while from about 1950 the protein available was almost constant (Table 3, p 26). The information on trends in protein consumption derived from N.F.S. for 1950 to 1971 (Table 4, p 27) agrees broadly with that from C.L.E.

4.5 The consumption of total carbohydrate in the United Kingdom rose somewhat during and after the Second World War (Table 3). During the decade 1950-1960 there was first a small rise followed by a slight fall, and this fall has continued during the 1960's and until 1971 (Tables 3 and 4).

4.6 The consumption of sucrose, as opposed to that of total carbohydrate, has shown a different trend. The available figures, from C.L.E. only, indicate that, except for a fall during both World Wars and in the early 1920s when prices were high, the amount of sucrose available for consumption rose until 1958 (Figure 4, p 32). Since then there has been a decline, although in 1971 sucrose still provided about 16% of the total available food energy.

4.7 Between the beginning of the century and the early 1960s the total amount of fat available for consumption rose (Table 3). This trend continued after 1960 but was less obvious (Tables 3 and 4).

4.8 When the total amount of food energy available is considered the amount per person is seen to rise between 1909 and the post-Second World War years (Table 3) and since then to have varied very little (Tables 3 and 4).

4.9 Tables 3 and 4 also indicate the percentage of food energy derived on the average from protein, fat and carbohydrate. Since the early years of the century the figures for protein from all sources have varied little. Over the last decade about 11.0% of the food energy has been available from protein. For carbohydrate and fat the picture is different. Since 1950 the proportion of food energy available from carbohydrate has steadily declined while that from fat has risen (Figure 5, p 33). In 1971, on average, 46-47% of the food energy was supplied by carbohydrate and about 42% by fat (Tables 3 and 4 (pp 26 and 27) Figure 5 (p 33)).

4.10 The amounts of the various types of fatty acid in the diet of the average household have been calculated from the National Food Survey information. In 1972 saturated fatty acids accounted for about 21% of the total energy value of the diet, which is about one half the total energy derived from fat. The estimated P/S ratio (see para 3.1.1) for the fatty acids in the food has risen from 0.17: 1.0 in 1959, to 0.22: 1.0 in 1972 (Table 5, p 27).

4.11 The figures in Table 6 (p 28) show that there has been little change in the total amount of indigestible fibre in the diet during the past 60 years, though there was a transient rise during the second World War. But the main sources of indigestible fibre have changed: cereals contributed about one quarter of the total fibre in the diet in 1909-13; by 1970 the proportion was only 13%.

4.12 The information available from dietary studies of children within the last decade indicates that the percentages of the total food energy derived from protein, fat and carbohydrate are similar to those for the population as a whole.

4.13 Between 1913 and 1971 the daily consumption of calcium, iron, vitamin A, thiamin, riboflavin, nicotinic acid and vitamin C have all increased.

4.14 Changes in food consumption in Great Britain during the past decade can be summarized as follows. Total dietary energy and the total intake of protein have not significantly altered; the intake of carbohydrate as a whole

has fallen, and the consumption of fat has risen. Over the same period there has been a slight fall in sucrose consumption (Figure 4, p 32). During this decade the rise in the death rate from I.H.D., which was conspicuous in the previous one, has apparently either been interrupted or has ceased (Table 1 (p 25) and Figures 1, 2 and 3 (pp 29, 30, and 31)). As we have pointed out (para 4.1), we are uncertain of the time scale in which any influence of alterations of dietary pattern is likely, if at all, to operate with respect to the death rate from I.H.D., and we cannot at present infer with confidence any causal relationship between the observed dietary changes and alterations in mortality from I.H.D.

4.15 The evidence that is available from the National Food Survey for England and Wales and for Scotland suggests that regional differences in the intake of food energy and nutrients are small and are unlikely to explain the observed regional differences in death rate from I.H.D. (para 2.2.6).

4.16 We know of no evidence that there are any differences in the diet of men and of women which could explain the difference in death rate between the sexes (para 2.2.7).

5. Overconsumption of Food and the Risk of Ischaemic Heart Disease

5.1 There is a widespread belief that obesity is becoming more prevalent in the United Kingdom among both adults and children. There is some evidence for this belief, but there have been no large-scale studies in which the prevalence of obesity has been assessed at intervals of time. The excess of fat in obese individuals is difficult to measure. The simplest, although least accurate, index of obesity is that of weight in relation to height, but this does not allow for variations in body build, and overweight may result from an increase in muscle mass or of body fluid alone, as well as from a greater deposition of fat. Nevertheless, the main source of information about obesity for this Report has been measurement of body weight in relation to stature.

5.2 There is no evidence that the average food energy intake by the population of the United Kingdom is rising (Tables 3 and 4, pp 26 and 27). Obesity results from a consumption of food in excess of energy expenditure, and with the greater availability of labour-saving devices in the home, in the factory and in the field, the expenditure of physical energy of many people has significantly diminished. As life becomes more sedentary people can become overweight even though they consume no more, or even less, food than before.

5.3 The death rate of obese people is higher than that of people of the same age who are not obese and the greater mortality is in part due to death from I.H.D. Obesity is associated with physical under activity, a high blood pressure and diabetes mellitus, each of which is considered to be an important risk factor for I.H.D.; studies suggest that, in the absence of these associated factors, obesity alone may not add much to the total risk of I.H.D. in an individual.

5.4 On the other hand there is evidence that a substantial reduction in the body weight of overweight persons, however induced, is alone sufficient to diminish the high death rate associated with obesity. Such a reduction in body weight can be brought about by a diminution in food consumption or, less easily, by an increase in regular exercise without a rise in the intake of food.

5.5 A proportion of obese children become obese adults and obesity in childhood which continues into adult life may be an important risk factor for I.H.D.

5.6 The need for more accurate information about obesity is clear from the foregoing paragraphs 5.1-5.5. Methods for its routine assessment, knowledge about its prevalence among children and adults, and about its possible control, are all matters to which we think research can usefully be directed at the present time.

6. Dietary Fat and the Risk of Ischaemic Heart Disease

6.1 The results of comparative studies of the population of various countries agree in general that the death rate from I.H.D. correlates positively with the average proportion of the food energy which is derived from fat, and that the correlation is better with the proportion of food energy derived from saturated fatty acids than with the proportion derived from the total amount of fat in the food. Nevertheless some exceptional populations, for example the Masai and the Somali nomads in East Africa whose food includes a high proportion of saturated fatty acids, have a low death rate from I.H.D. Such people are physically active. It is in the well-nourished and physically relatively inactive populations of the type found in the United States and Western Europe that the weight of evidence supports the existence of a positive correlation between the national death rates from I.H.D. and the average proportions of the food energy derived from saturated fatty acids.

6.2 As was stated in para 4.9, the percentage of food energy derived from fat in the United Kingdom has risen and continues to rise, and about one half of the energy derived from fat is provided by saturated fatty acids (para 4.10).

6.3 The interpretation of the evidence which conforms with the view that a diet rich in fat predisposes to I.H.D. is complicated by the fact that people who consume a high fat diet are often heavy cigarette smokers and tend to have a high sucrose intake (para 7.4).

6.4 Trials have been carried out on groups of adults to evaluate any effect of alterations in the fatty acid content of the diet on the incidence of I.H.D. These trials have been of two main types: those on people who have no clinical evidence of I.H.D. to see whether the possible occurrence of I.H.D. can be prevented; and those on patients who have survived one or more attacks to ascertain whether recurrence can be avoided.

6.5 In most of these trials, few of which have been carried out in the United Kingdom, the composition of the food eaten by those in the experimental group was altered by the substitution of part of the saturated fatty acids by poly-unsaturated fatty acids. The results have been interpreted as evidence that the incidence of I.H.D. may be lowered by such a dietary adjustment, but in none of these trials was the evidence clear-cut. In one of the trials a possible disadvantage of the dietary alteration was observed in a rise in the prevalence of gall-stones found at autopsy.

6.6 In all these trials the difficulties inherent in any long-term dietary experiment were clear. Unlike a drug, nutrients are necessary for survival and cannot

easily be tested against an inert placebo. People choose food and not nutrients, and the eating habits of individuals cannot readily be changed. Eating forms part of the social culture and a change by one person of the pattern of food consumed may involve a whole family. Since the time which might be required for the appearance of overt I.H.D. as a possible result of dietary imbalance is not known, the subjects of dietary trials should preferably be young, and adherence to a specified diet by a high proportion of such subjects for a period of perhaps 10 to 20 years, during which they continue their everyday life, is intrinsically improbable. The technical problems involved in ensuring adherence to a dietary regimen, and in detecting failure to adhere, are great. Subjects in whom clinical signs of I.H.D. have already appeared are more strongly motivated than others to adhere to a dietary regimen, but dietary changes may be unable to reverse pathological processes in the heart which have been developing over a period of time. Despite these difficulties we regard dietary trials as of value, although the more rigorously the design and control of studies of this sort are scrutinized, the less convincing do the results appear to be.

6.7 The unexplained existence of regional differences in the death rate from I.H.D. within the United Kingdom (para 2.2.6) behoves one to be cautious about the application to Great Britain as a whole of the results of any dietary trials, and particularly of those made in other countries.

7. Dietary Carbohydrate and the Risk of Ischaemic Heart Disease

7.1 In the United Kingdom the total consumption of all types of carbohydrate is now less than at the beginning of the present century, and has fallen steadily over the past decade (Tables 3 and 4, pp 26 and 27). Except for the periods of the two World Wars and the early 1920s the consumption of sucrose rose substantially from the beginning of the century, with a particularly steep rise after the end of the second World War, to a maximum in 1958. Since then consumption has fallen to about that reached in 1953.

7.2 Comparative studies have revealed that for the populations of different countries there is a positive correlation between the national sucrose consumption and the death rate from I.H.D., and a negative correlation between the amount of starch in the diet and I.H.D. mortality. In many of the countries studied the population consumed a relatively high energy diet which was rich both in sucrose and in saturated fatty acids.

7.3 Epidemiological studies afford some indication that a population with a high sucrose intake has a high death rate from I.H.D., the correlation being better when the diet is also rich in saturated fatty acids. But the correlation between the death rate from I.H.D. and the amount of saturated fatty acids in the diet is better than that between death rate from I.H.D. and the amount of dietary sucrose. There are exceptions; for example in Costa Rica, Cuba, Jamaica, Ecuador and Colombia—countries in which the diet is of low energy content with a small proportion of saturated fatty acids and a high sucrose content. In these countries there is no relationship between the sucrose content of the diet and the death rate from I.H.D. When, because of migration, as in the movement of Yemenite Jews to Israel, the diet of a community has changed from a low to a high sucrose content without much change in total fat, there is evidence that the death rate from I.H.D. has increased.

7.4 In some studies of groups of people in the United Kingdom a statistically significant relationship has been observed between the amount of sucrose consumed and the number of cigarettes smoked daily. The situation is therefore complex. Those who consume more than an average amount of sucrose are also likely to derive a greater proportion of their dietary energy from saturated fatty acids and to smoke more (para 6.3).

7.5 There have been no dietary trials with low and high sucrose diets comparable with those made to study the effect of dietary fat on the death rate from I.H.D., and we think that the feasibility of long-term trials of this sort, with sucrose, should be considered (para 10.5.1).

7.6 Further evidence is needed to establish any effect on the death rate from I.H.D. of replacing part of the fat or of the sucrose in the diet by bread or other starchy foods (para 7.2).

8. Some other Constituents of the Diet and the Risk of Ischaemic Heart Disease

8.1 Cholesterol

In certain animal species a diet rich in cholesterol induces the appearance of arterial lesions which have some similarity to those seen in human subjects, and comparative studies of different human populations show that those which have a diet rich in cholesterol have a higher death rate from I.H.D. However, a diet rich in cholesterol is usually one which is rich also in saturated fatty acids. Most of the dietary cholesterol in western communities is derived from eggs, but we have found no evidence which relates the numbers of eggs consumed to a risk of I.H.D.

8.2 Sodium chloride

There is evidence that some communities which have a high average salt intake have a high average blood pressure. For example the more rural northern Japanese and some Polynesians in the Cook Islands eat a diet with a much higher salt content, and have a higher blood pressure, than people in the industrial south of Japan and certain other Polynesians who eat a diet with a lower salt content. A large reduction in salt intake can, in some individuals, result in a lowering of blood pressure, but we have no evidence that a reduction in the salt consumption of the whole population of the United Kingdom would widely lower blood pressure, and hence reduce the death rate from I.H.D.

8.3 Dietary fibre

At present, the evidence which may relate a rise in the death rate from I.H.D. to a reduction in the intake of dietary fibre is far from complete. The possible importance of dietary fibre in this respect is a subject for further research.

8.4 Trace elements

We know of no convincing evidence to relate susceptibility to I.H.D. with the presence or absence from the diet of trace elements but we think that the possible importance of trace elements should be further investigated, especially in relation to the influence of the hardness of the water supply on the death rate from I.H.D. (section 9 below), and to the possible influence of the composition of the diet on the availability for absorption from the gut of trace elements.

8.5 Other constituents

The influence of alterations in the composition of the diet on the absorption from the gut of a number of materials derived from food may be relevant to our problem, and we think that further research is needed.

9. Hardness of Water and the Risk of Ischaemic Heart Disease

9.1 Water is an essential constituent of the diet, used both in the preparation of food and as a beverage. Differences in the concentration of dissolved substances in the water supply therefore affect the amount in the diet of these substances, some of which are nutrients. Individuals who live in an area where the water supply is soft may receive from food cooked in or otherwise treated with water, and from drinking water, appreciably less calcium (and less of several other metals but not of lead) than those who live in a hard water area.

9.2 During the past 15 years investigators in several countries have observed a negative association between the hardness of the domestic water supply and the local death rate from cardiovascular disease (cerebrovascular, coronary or other heart disease)—that is to say, the harder the water supply the lower the death rate from cardiovascular disease. This association also holds for areas within the United Kingdom.

9.3 With respect to known substances which may be present in different amounts in the water supply, the strongest negative association of the death rate from cardiovascular disease is with the content of calcium and temporary hardness (the bicarbonate fraction of total hardness); the association with the sodium content is small, and that with the content of magnesium negligible. In areas where, in the past, the water supply had been softened the introduction of softening was usually found to be followed by a greater increase in the death rate from cardiovascular disease than occurred in areas where the water supply had been left unchanged. Conversely, where the water supply had become harder any rise in the death rate was in general less than that seen in areas where the hardness of the water supply remained unaltered. The risk factor could be a substance which is more soluble in soft water than in hard water, but the nature of any such substance is not at present known. There may be some substance in hard water that protects from the disease.

9.4 The possibility must be considered that the regional differences in death rate from I.H.D. referred to in para 2.2.6 may at least in part be associated with differences in the hardness of the water supply. Information about the association of the death rate from I.H.D. and the hardness of the water supply and other local environmental factors should be acquired on a much larger scale and in greater detail.

10. Diet and Indicators of the Risk of Ischaemic Heart Disease

10.1 General considerations

A high concentration of serum cholesterol, and in addition or alternatively a high concentration of serum triglyceride, are two of many possible indicators of an enhanced risk of developing I.H.D. which have been studied, and of these two a high concentration of serum cholesterol has been far more frequently employed as an indicator of risk.

10.2 Serum cholesterol concentration as an indicator of the risk of ischaemic heart disease

The mean serum cholesterol concentration of populations is strongly positively correlated with the death rate from I.H.D. in these populations. In healthy individuals the serum cholesterol concentration can be useful for the assessment of susceptibility to I.H.D. By contrast, there is evidence that the concentration of serum cholesterol, except when it is very high, is of little value for the assessment of the likelihood of any recurrence in those who have already developed overt I.H.D.

10.3 Serum triglyceride concentration as an indicator of the risk of ischaemic heart disease

In those population studies in which serum triglyceride concentration has been measured there is a positive correlation between it and the death rate from I.H.D. The results of recent studies have been interpreted to suggest that in an individual a high concentration of serum triglyceride alone may indicate an increased risk of I.H.D. Further research on this subject is needed.

10.4 Effect of alteration of the diet on the concentration of cholesterol in the serum

10.4.1 The serum cholesterol concentration of healthy people can be altered by variations in the composition of the diet. The characteristics of the diet which have been most studied in man in this connexion are the fat content—both total fat and the ratio of polyunsaturated to saturated fatty acids—and the amount of cholesterol, of sucrose and of fibre. Although there is evidence that such dietary variations can alter the concentrations of serum cholesterol, corresponding differences in susceptibility to I.H.D. have not been unequivocally established.

10.4.2 When a group of people leave one country in order to make their home in another the nature of their food and the death rate from I.H.D. may both change. If there is a rise in their consumption of saturated fatty acids, or of dietary cholesterol, or of sucrose, or (less well documented) if there is a fall in their consumption of dietary fibre, the mean serum cholesterol concentration and the death rate from I.H.D. usually both rise. The possible rôle of the dietary changes in the alteration of the death rate from I.H.D. in such circumstances is difficult to assess because of the accompanying social and other environmental changes.

10.4.3 Long-term experimental trials have provided evidence that both in individuals and in groups of people a fall of the serum cholesterol concentration may occur when the amount of dietary fat is reduced or when part of the saturated fatty acid (from either animal or vegetable sources) is replaced by polyunsaturated fatty acid. Reduction of total dietary saturated fatty acid alone has a greater effect on serum cholesterol concentration than the addition of polyunsaturated fatty acid to the usual diet without a reduction of the intake of saturated fatty acids.

10.4.4 Most populations whose food is relatively rich in cholesterol, including that of the United Kingdom, have a high concentration of cholesterol in the serum and a high mortality from I.H.D. Although diets rich in cholesterol are usually also rich in saturated fatty acids (para 8.1), short-term experiments in small groups of individuals have suggested that an increase in the amount of dietary cholesterol may result in a rise in the concentration of cholesterol in the serum even though the intake of saturated fatty acids remains unchanged.

10.4.5 Trials in which the fat content of the diet has not been altered but the proportion of starch or of sucrose has been varied are few in number, and have usually been short-term. When, in such trials, bread has been substituted for sucrose in the diet a fall in serum cholesterol concentration has resulted, and conversely when the amount of sucrose in the diet has been increased at the expense of bread a rise in serum cholesterol concentration has been observed.

10.4.6 Populations who eat a diet rich in fibre (particularly fibre from cereals and legumes) usually have a lower serum cholesterol concentration and a lower mortality from I.H.D. than those who eat a western type diet relatively low in this kind of fibre. There is also some evidence that the serum concentration of cholesterol in healthy persons can be reduced by increasing the consumption of food rich in fibre.

10.4.7 The serum cholesterol concentration can be raised by non-dietary influences, including the inhalation of carbon monoxide as a result of smoking. The interaction of such influences and dietary ones is a matter for further research.

10.5 Effect of alteration of the diet on the concentration of triglyceride in the serum

10.5.1 In general any change in the diet which leads to an alteration in the serum cholesterol concentration has a qualitatively similar effect on the concentration of serum triglycerides. Nevertheless there is evidence that an alteration in the amount of dietary sucrose has a greater effect on the concentration of serum triglyceride than on that of serum cholesterol. When some of the starch in the diet of a healthy individual is replaced by sucrose the concentration of serum triglyceride rises, but the effect may be only short-lived.

10.5.2 The results of some experiments in which part of the starch in food was replaced by maltose, glucose or fructose, suggested that it is the fructose part of the sucrose molecule which induces a rise in serum triglyceride concentration and enhances fat deposition. Although there is little experimental evidence directly linking the intake of fructose with susceptibility to I.H.D. or other vascular disease, the substitution of sucrose by fructose or by sorbitol (which can be converted to fructose in the body) should not, in the light of present knowledge, be encouraged.

10.6 Other possible indicators of the risk of ischaemic heart disease

10.6.1 In addition to the concentration of serum cholesterol and serum triglyceride, serum insulin concentration and glucose tolerance have been cited as possible indicators of the risk of I.H.D. Since the nature of the diet can influence these possible indicators of risk, their use in the assessment of susceptibility to I.H.D. in healthy subjects is a matter for further research.

11. Summary

11.1 Ischaemic heart disease (coronary heart disease) appears to have many contributory causes, and more than one is likely to be involved in determining the incidence of the disease in a population, or its occurrence in an individual. There is at present no evidence that any one cause has the essential importance for ischaemic heart disease which, for example, the tubercle bacillus has for tuberculosis (para 2.3.2).

11.2 Populations which have a high death rate from ischaemic heart disease have certain environmental features in common which appear to predispose to the development of the disease. In any individual the liability to develop ischaemic heart disease is the result of an interaction between external risk factors and internal ones (paras 2.3.1 and 2.3.4).

11.3 There are many risk factors for ischaemic heart disease only some of which are dietary in nature (para 2.3.1). No single dietary factor can be regarded as predominant in determining susceptibility to the disease, and any claim to the contrary is not acceptable in the context of the United Kingdom diet. This is as true for an individual as it is for the population as a whole, though our recommendations are not necessarily applicable to people with gross metabolic abnormalities (para 1.3.3).

11.4 Recognizable overweight, including obesity, increases the risk both of ischaemic heart disease and of other morbid conditions (para 5.3). There is evidence that a substantial reduction of the body weight of overweight people is alone sufficient to diminish the greater death rate usually associated with obesity (para 5.4).

11.5 Changes in the composition of the diet of an individual can reduce the concentration of cholesterol in the blood serum, but there is no certainty that such a reduction diminishes the susceptibility to ischaemic heart disease (para 10.4.1).

11.6 The members of the Panel accept the evidence derived from international comparisons that the death rate from ischaemic heart disease in a population correlates positively with the proportion of the food energy derived from fat, and even better with the proportion of the food energy derived from the saturated fatty acids in the diet (para 6.1). Although the members of the Panel recognize that such statistical correlations do not of themselves establish causal relationships, nevertheless in the judgement of the majority of the members the fact that in the United Kingdom the consumption of dietary fat and the percentage

of the food energy which is derived from fat (animal and vegetable) have both been steadily rising should be viewed with concern. These members therefore recommend that the amount of fat in the diet should be reduced.

11.7 A minority of the members of the Panel consider the evidence that a general nutritional disadvantage would ensue from a continuing rise in the proportion of food energy derived from fat in the United Kingdom diet to be insufficient to justify a recommendation designed to limit the consumption of fat by the population as a whole.

11.8 A rise in the ratio of polyunsaturated to saturated fatty acids in the diet of an individual or of a group of people may be followed by a reduction in the mean serum cholesterol concentration (para 10.4.3). Nevertheless the Panel are unanimous in remaining unconvinced by the available evidence that the incidence of ischaemic heart disease in the United Kingdom, or the death rate from it, would be reduced in consequence of a rise in the ratio of polyunsaturated to saturated fatty acids in the national diet (paras 6.5 and 6.6). In the present state of knowledge any suggestion or claim to that effect, with respect to the nation or to an individual, would be unjustified (para 2.4).

11.9 Over the past 100 years the consumption of sucrose (cane sugar or beet sugar) in the United Kingdom has risen, although this trend was interrupted during the two World Wars and has tended to reverse since 1958 (Figure 4, p 32). Sucrose is widely available, and a cheap, palatable and concentrated source of food energy; it can easily be consumed beyond satiety in a way that is unlikely to occur with other sources of food energy. The Panel believe a reduction in the incidence of obesity to be desirable (paras 5.3 and 5.4), and that a continued fall in the intake of sucrose would assist in achieving this aim.

11.10 A high blood pressure is a risk factor for ischaemic heart disease and cerebrovascular disease, and in some individuals a large reduction in the intake of common salt results in a lowering of blood pressure. Nevertheless there is insufficient information upon which to base a recommendation for a reduction in the salt consumption of the whole population in the expectation that such a reduction would widely lower blood pressure, and hence reduce the death rate from ischaemic heart disease or from cerebrovascular disease (paras 3.2.1 and 8.2).

11.11 There is at present too little evidence on which to assess the possible importance of indigestible material (fibre) in the food in relation to ischaemic heart disease (paras 8.3 and 10.4.6).

11.12 Any changes in dietary habits during adult years usually affect families, and eating patterns which are established during childhood may determine the diet subsequently chosen in adult life (para 4.12). The Panel believe, therefore, that any dietary modifications to be recommended should be applicable during childhood, and we think that the avoidance of obesity and of an excessive sucrose consumption would benefit babies and children (paras 5.5 and 11.9).

11.13 Within the United Kingdom as well as in other countries a negative association has been observed between the death rate from cardiovascular disease and the hardness of the local water supply: the softer the local water supply, the higher the death rate from cardiovascular disease (paras 9.2 and 9.3). The reason for this relationship has yet to be elucidated.

12. Recommendations

12.1 The conclusions of the Panel have been embodied in Recommendations which aim to diminish the risk of ischaemic heart disease (coronary heart disease) and, in some instances, of cerebrovascular disease as well. The order in which the Recommendations follow does not denote our assessment of the ranking of their importance. In agreement with our Terms of Reference our Recommendations relate only to nutritional matters, but we recognize that other conditions (for example, a reduction of cigarette smoking, and an increase in regular physical activity) can under some circumstances be more important in the prevention of ischaemic heart disease than modifications in the quality or quantity of the food consumed. Any dietary changes adopted by an adult are likely to affect a family, and our Recommendations may safely apply to children.

12.2 Obesity should be avoided both in the child and the adult (paras 3.2.1, 5.3 and 11.4). The Panel recommend that those individuals who are already obese should so reduce their food intake in relation to their physical activity that they are no longer obese.

12.3 The majority of the members of the Panel recommend that the amount of fat in the United Kingdom diet, especially saturated fat from both animal and plant sources, should be reduced (paras 10.4.3 and 11.6).

12.4 The Panel unanimously agree that they cannot recommend an increase in the intake of polyunsaturated fatty acids in the diet as a measure intended to reduce the risk of the development of ischaemic heart disease. In their opinion the available evidence that such a dietary alteration would reduce that risk in the United Kingdom at the present time is not convincing (para 11.8).

12.5 The Panel recommend that the consumption of sucrose, as such or in foods and drinks, should be reduced, if only to diminish the risk of obesity and its possible sequelae (para 11.9).

12.6 The Panel recommend that any proposals for softening the water supply in any part of the country should be considered in the light of knowledge about the observed positive relationship between the death rate from ischaemic heart disease and the softness of the local water supply (paras 9.2 and 9.3).

13. Glossary

1. ISCHAEMIC HEART DISEASE (coronary heart disease) is a cardiac disability (either acute or chronic) which arises from a reduction or an arrest of the blood supply to part of the heart muscle either by a narrowing of a blood vessel or by complete obstruction of it. The condition is usually associated with disease of the coronary arteries which supply blood to the heart muscle. The most common disease processes that involve these arteries are (a) changes in the arterial wall—atherosclerosis, and (b) changes in the blood—thrombosis. The processes which lead to atherosclerosis and thrombosis usually occur simultaneously, although they may arise from different causes.

2. CEREBROVASCULAR DISEASE arises from a disturbance in the blood supply to some part of the brain. This disturbance may be caused by the narrowing of an artery, rupture of the arterial wall or the blocking of a blood vessel by a thrombus.

3. PERIPHERAL VASCULAR DISEASE results from a disturbance in the blood supply to the limbs.

4. DIETARY FAT consists largely of a complex mixture of triglycerides, each of which contains three fatty acids esterified with one molecule of glycerol. Naturally occurring fatty acids contain about 4 to 24 carbon atoms, the most abundant having 16 or 18 in a straight chain. They may be saturated (no ethylenic double bond), mono-unsaturated (one double bond), or polyunsaturated (two or more double bonds). For the purpose of this Report, no distinction is made between saturated and mono-unsaturated fatty acids. Animal fats usually occur in the food associated with cholesterol and sometimes with vitamins A and D. Vegetable fats are often associated with vitamins A and E, but their sterols do not include cholesterol.

5. BLOOD FAT In the blood serum fats, including cholesterol, are almost entirely bound to protein in the form of lipoproteins. Triglycerides may also exist in the form of chylomicrons, the number of which rises rapidly after a fat-containing meal. Lipoproteins differ in density according to the proportion of triglyceride, cholesterol (both free and esterified), phospholipid, free fatty acid, and protein. Lipoproteins may therefore be separated by physical means.

Table 1: *Annual death rate per 100,000 population of males and females aged 35-44, 45-54 and 55-64 years in England and Wales, Scotland and Northern Ireland from Ischaemic Heart Disease for the years 1950-1971*.*

	Year	England and Wales						Scotland						Northern Ireland					
		35–44		45–54		55–64		35–44		45–54		55–64		35–44		45–54		55–64	
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
I.C.D.(7) 420-422	1950	33	8	164	43	566	218	52	19	248	74	710	337	52	18	200	77	643	256
	1951	33	9	168	43	673	216	57	16	263	75	771	337	45	19	254	86	604	361
	1952	34	7	170	41	559	200	50	18	232	72	783	329	53	18	235	73	654	286
	1953	33	6	164	39	543	196	62	16	235	68	736	270	36	18	215	68	630	296
	1954	38	8	179	36	566	190	63	19	257	73	813	293	43	19	194	90	678	332
	1955	41	7	181	40	581	189	64	15	277	70	737	315	58	14	234	65	635	300
	1956	41	7	189	36	590	190	60	15	268	72	801	317	53	14	239	70	719	276
	1957	42	6	191	36	594	185	60	15	284	65	818	301	53	20	240	63	728	283
	1958	45	7	201	38	617	189	67	18	273	68	808	311	61	20	269	74	786	315
	1959	46	7	197	36	609	186	71	19	282	62	834	303	70	9	265	76	781	298
	1960	51	8	208	38	635	192	72	15	309	70	868	326	59	16	267	81	813	298
	1961	50	8	216	38	648	191	71	13	309	75	915	319	86	9	265	55	807	288
	1962	57	9	226	41	683	200	82	15	321	74	889	336	71	16	285	87	809	276
	1963	60	9	242	42	707	203	81	18	348	84	959	322	85	23	318	75	942	314
	1964	63	10	245	42	694	193	91	18	355	86	953	327	81	16	324	74	803	274
	1965	68	11	253	45	719	201	92	17	360	92	965	330	94	18	275	78	857	289
	1966	66	10	249	45	709	197	90	17	343	84	910	333	87	16	278	69	851	269
	1967	64	10	244	42	695	195	89	20	331	79	881	298	68	17	325	51	799	257
I.C.D.(8) 410-414	1968	63	11	245	42	698	185	81	15	333	75	890	309	73	11	257	73	801	274
	1969	60	11	255	44	708	189	87	13	327	79	899	309	85	14	319	49	830	278
	1970	64	10	259	42	705	189	81	18	341	81	898	313	73	15	317	78	896	321
	1971	67	9	272	46	708	189	87	20	365	85	884	308	96	14	307	63	882	249

*1950-1967: I.C.D.(7) 420-422. 1968-1971: I.C.D.(8) 410-414

SOURCE: Office of Population Censuses and Surveys.

Table 2: *Comparison of death rates from ischaemic heart disease in England and Wales, Scotland and Northern Ireland. The figures for Scotland and Northern Ireland are expressed as a percentage of those for England and Wales in the corresponding year.* *

Year	Males (age group) 35–44	45–54	55–64	Females (age group) 35–44	45–54	55–64
Scotland						
1951	173	157	133	178	174	151
1961	142	143	141	163	197	162
1971	130	134	125	222	185	158
Northern Ireland						
1951	137	151	105	211	200	167
1961	172	123	125	113	145	151
1971	143	113	125	156	137	132

*Information derived from Table 1. The numbers of female deaths in the younger age groups are small and the territorial comparisons are therefore subject to large fluctuations.

Table 3: *The mean energy, protein, fat and carbohydrate content of the food moving into consumption from 1909 to 1971 in the United Kingdom (Food Consumption Level Estimates) *, expressed as the amount per person per day, and the percentage of the total food energy supplied by protein, fat and carbohydrate.*

Year	Energy kcal	MJ	Protein g	% energy	Fat g	% energy	Carbohydrate† g	% energy
‡ 1909-1913	2760	11.5	81	11.7	98	32.0	415	56.4
‡ 1924-1928	2810	11.8	79	11.2	107	34.3	408	54.5
1934-1939	3050	12.8	79	10.4	131	38.6	414	50.9
1940	2890	12.1	80	11.1	123	38.3	390	50.7
1941	2900	12.1	83	11.5	115	35.6	409	52.8
1942	2930	12.3	87	11.8	119	36.5	403	51.5
1943	2920	12.2	85	11.6	116	35.7	411	52.7
1944	3060	12.8	86	11.2	124	36.4	429	52.5
1945	3010	12.6	90	12.0	116	34.7	427	53.3
1946	2940	12.3	88	12.0	113	34.6	421	53.6
1947	2940	12.3	89	12.1	107	32.7	433	55.2
1948	3000	12.5	88	11.7	111	33.3	438	54.8
1949	3120	13.1	86	11.1	122	35.2	446	53.6
1950	3120	13.0	87	11.2	133	38.4	419	50.4
1951	3080	12.9	83	10.8	127	37.1	429	52.2
1952	3030	12.7	82	10.8	124	36.8	424	52.4
1953	3100	13.0	82	10.6	130	37.8	425	51.5
1954	3190	13.3	82	10.3	138	38.9	431	50.7
1955	3170	13.3	82	10.4	139	39.5	425	50.3
1956	3170	13.3	83	10.5	140	39.7	422	49.9
1957	3180	13.3	83	10.5	141	39.9	422	49.8
1958	3180	13.3	83	10.5	141	39.9	421	49.6
1959	3130	13.1	84	10.7	138	39.6	415	49.7
1960	3130	13.1	85	10.9	138	39.7	414	49.6
1961	3160	13.2	86	10.9	140	39.9	413	49.1
1962	3170	13.3	87	11.0	144	40.9	407	48.1
1963	3180	13.3	87	10.9	143	40.5	412	48.6
1964	3150	13.2	87	11.1	144	41.2	403	48.0
1965	3140	13.1	87	11.1	142	40.8	403	48.3
1966	3150	13.2	87	11.0	144	41.1	402	47.9
1967	3070	12.8	85	11.1	143	41.9	385	47.0
1968	3080	12.9	85	11.0	144	42.0	386	47.0
1969	3100	13.0	85	11.0	145	42.0	387	47.0
1970	3090	12.9	86	11.0	144	42.0	388	47.0
1971	3055	12.8	84	11.0	143	42.0	382	47.0

*SOURCE: 1909-1928 Greaves and Hollingsworth, 1966
1934-1965, 1966-1967, 1968-1971 Ministry of Agriculture Fisheries and Food 1968, 1970, 1972a.

†Available carbohydrate expressed as monosaccharide.

‡Approximate values—not strictly comparable with the figures for 1934 onwards.

Table 4: *The mean energy, protein, fat and carbohydrate content of the diet of the average household in Great Britain 1950-1971* expressed as the amount per person per day, and the percentage of the total food energy supplied by protein, fat and carbohydrate.*

Year	Energy		Protein		Fat		Carbohydrate	
	kcal	MJ	g	% energy	g	% energy	g	% energy
1950	2470	10.3	78	12.5	102	36.8	315	50.6
1951	2470	10.3	76	12.3	100	36.4	318	51.4
1952	2450	10.2	77	12.6	94	34.5	324	52.9
1953	2520	10.5	78	12.4	101	36.0	325	51.6
1954	2630	11.0	77	11.7	107	36.5	340	51.8
1955	2640	11.0	77	11.6	107	36.6	342	51.7
1956	2620	11.0	76	11.5	108	37.1	337	51.4
1957	2590	10.8	75	11.6	110	38.1	325	50.3
1958	2600	10.9	75	11.5	111	38.3	325	50.2
1959	2580	10.8	74	11.5	110	38.3	324	50.3
1960	2590	10.8	76	11.7	112	38.9	320	49.4
1960	2630	11.0	75	11.4	115	39.3	345	49.3
1961	2630	11.0	75	11.4	116	39.6	343	49.0
1962	2640	11.0	75	11.4	117	40.0	342	48.6
1963	2650	11.1	76	11.5	118	39.8	343	48.5
1964	2600	10.9	75	11.6	116	40.3	333	48.0
1965	2590	10.9	75	11.6	116	40.4	332	47.9
1966	2560	10.7	76	11.8	117	41.0	321	47.0
1967	2590	10.8	76	11.7	119	41.3	324	47.0
1968	2560	10.7	75	11.8	118	41.5	318	46.6
1969	2570	10.8	74	11.6	120	42.0	317	46.3
1970	2600	10.9	75	11.5	121	41.8	322	46.5
1971	2530	10.6	74	11.6	119	42.3	310	46.0

*SOURCE: National Food Survey (Ministry of Agriculture, Fisheries and Food, 1952-66, 1967-73).

In 1960, changes in the Food Composition Table resulted in two values for each of the three nutrients.

Table 5: *Estimated fatty acid content* of the average household diet in 1959, 1969 and 1972 (expressed as g/person/day)*

Fat	1959	1969	1972
Total fat	109.6	120.1	112.1
Saturated fatty acids	53.0	56.7	52.1
Mono-unsaturated fatty acids	43.0	46.5	43.0
Polyunsaturated fatty acids	9.2	11.0	11.5
Ratio polyunsaturated to saturated fatty acids	0.17:1.0	0.19:1.0	0.22:1.0

*Based on food consumption figures obtained by the National Food Survey (unpublished results).

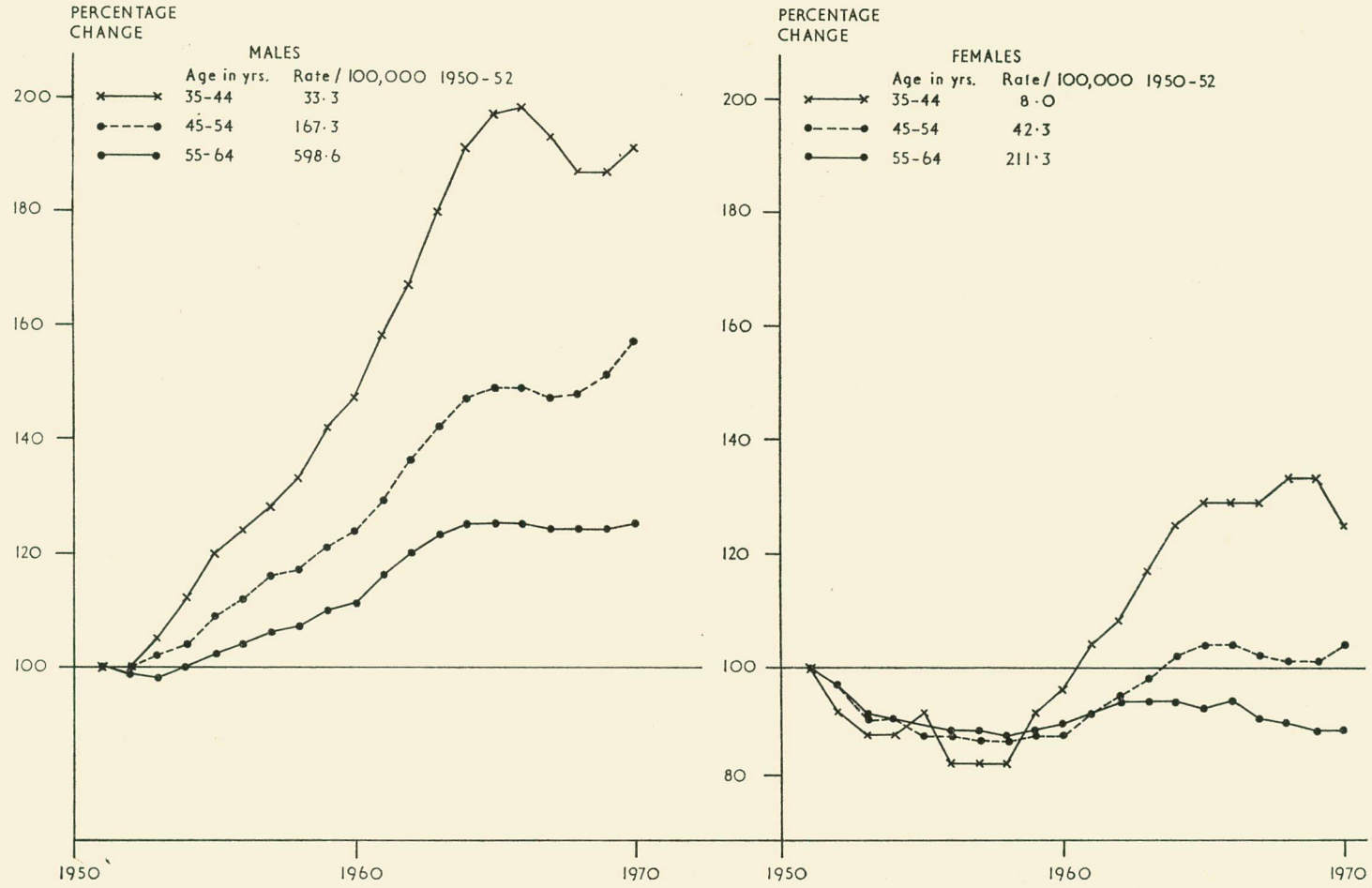
The difference between the total fat and the sum of the component fatty acids is chiefly accounted for by the glycerol component of fats.

Table 6: *Fibre content of the British diet between 1880 and 1970.*

	1880	1909-13	Pre-World War II	1942	1944	1957	1970
(*a*) *Total fibre consumed* (*g per head per day*)	NA	3.6	3.8	5.3	5.0	4.2	4.2
(*b*) *Source of fibre* (*g per head per day*)							
Potatoes	1.25	1.03	0.80	0.95	1.16	0.94	0.94
Other vegetables (including tomatoes)	NA	1.11	1.40	1.45	1.48	1.58	1.62
Fruit and nuts	NA	0.62	1.10	0.84	0.81	1.03	1.10
Wheat flour	0.85	0.58	0.46	1.92	1.30	0.46	0.39
Grain products	NA	0.30	0.06	0.19	0.20	0.14	0.15
(*c*) *Source of fibre* (*percentage of total consumed*)							
Vegetables	NA	59	57	45	53	61	61
Fruit and nuts	NA	17	29	16	17	25	26
Cereals	NA	24	14	39	30	14	13

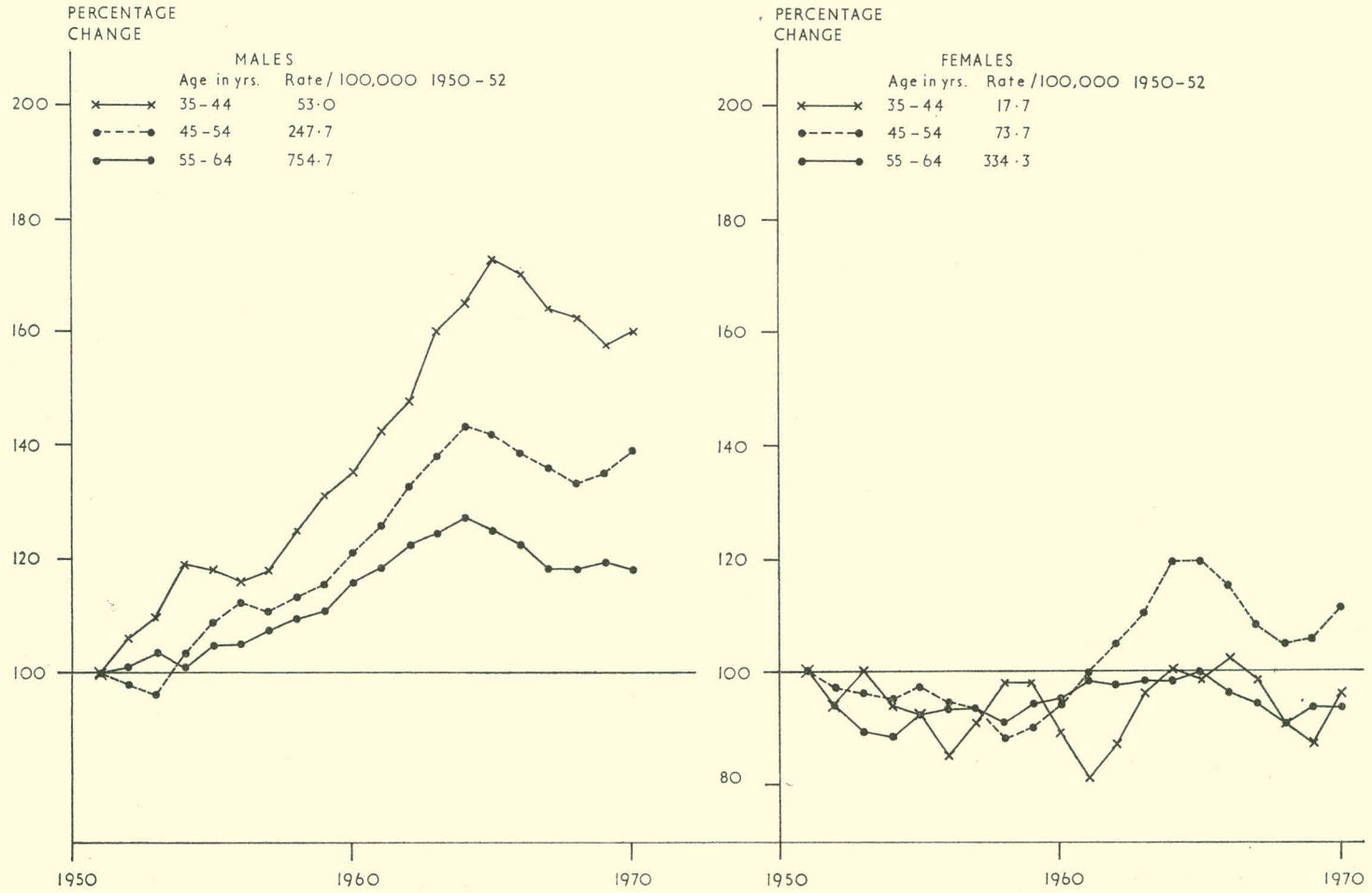
SOURCE: Robertson, 1972.
NA = not available.

Figure 1: *Percentage change in death rates of males and females in three age groups (from 35 to 64 years) from ischaemic heart disease* in England and Wales,* 1950-71. (*Three-year moving averages with* 1950-52=100)



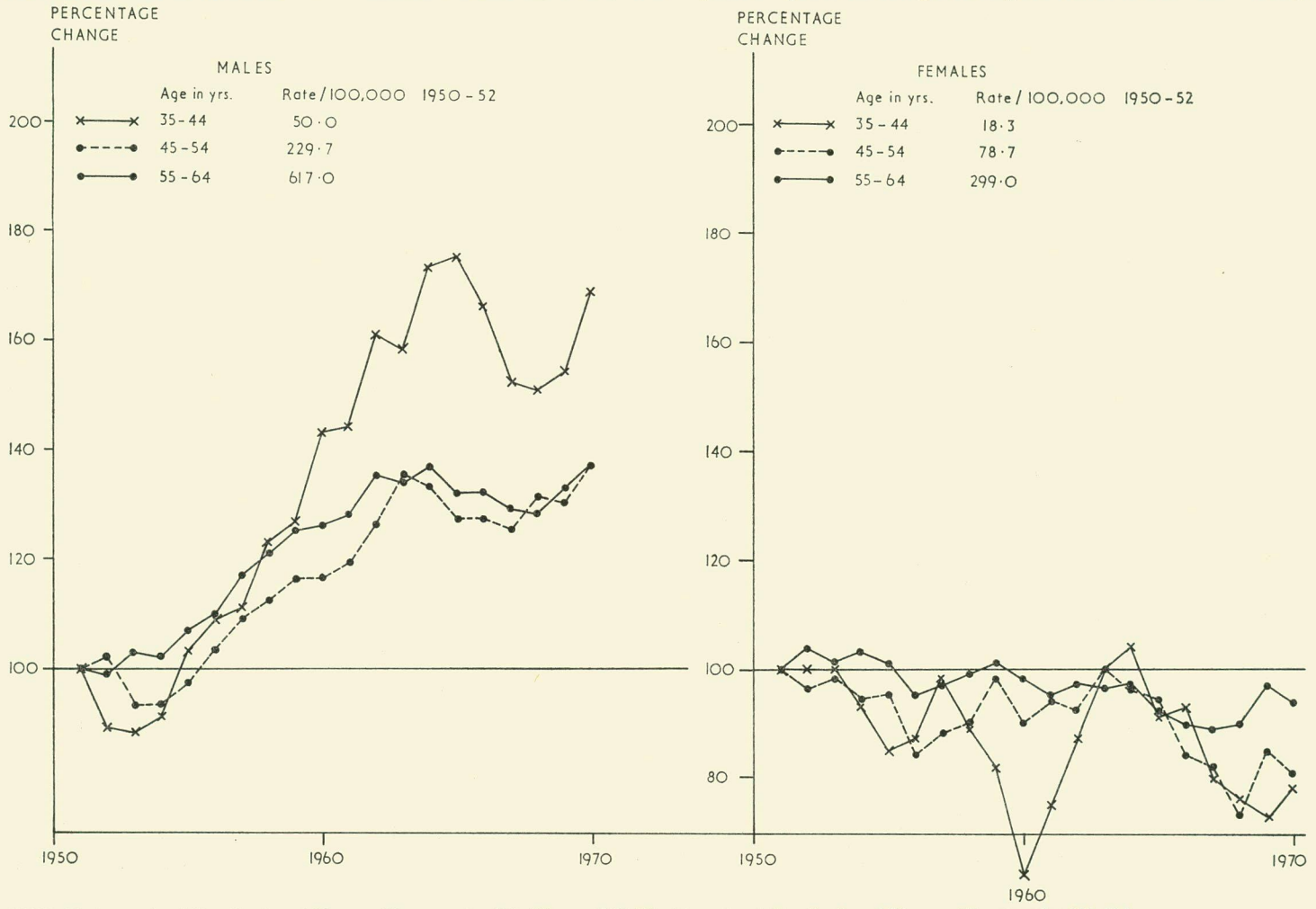
*1950-67—International Classification of Diseases (7), categories 420-422. 1968-71—International Classification of Diseases (8), categories 410-414.

Figure 2: *Percentage change in death rates of males and females in three age groups (from 35 to 64 years) from ischaemic heart disease* in Scotland,* 1950-71. (*Three-year moving averages with* 1950-52=100)



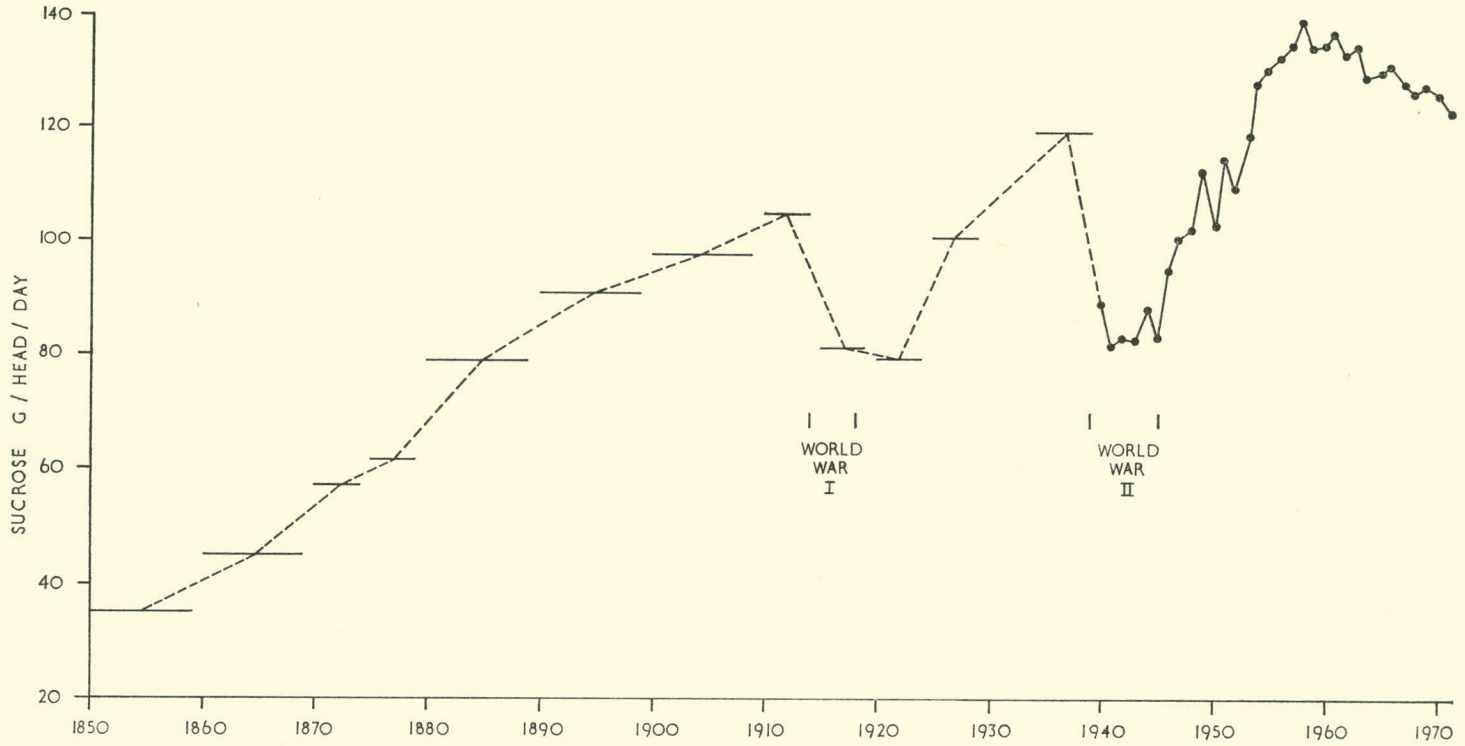
*1950-67—International Classification of Diseases (7), categories 420-422. 1968-71—International Classification of Diseases (8), categories 410-414.

Figure 3: *Percentage change in death rates of males and females in three age groups (from 35 to 64 years) from ischaemic heart disease* in Northern Ireland,* 1950-71. *(Three-year moving averages with* 1950-52=100).



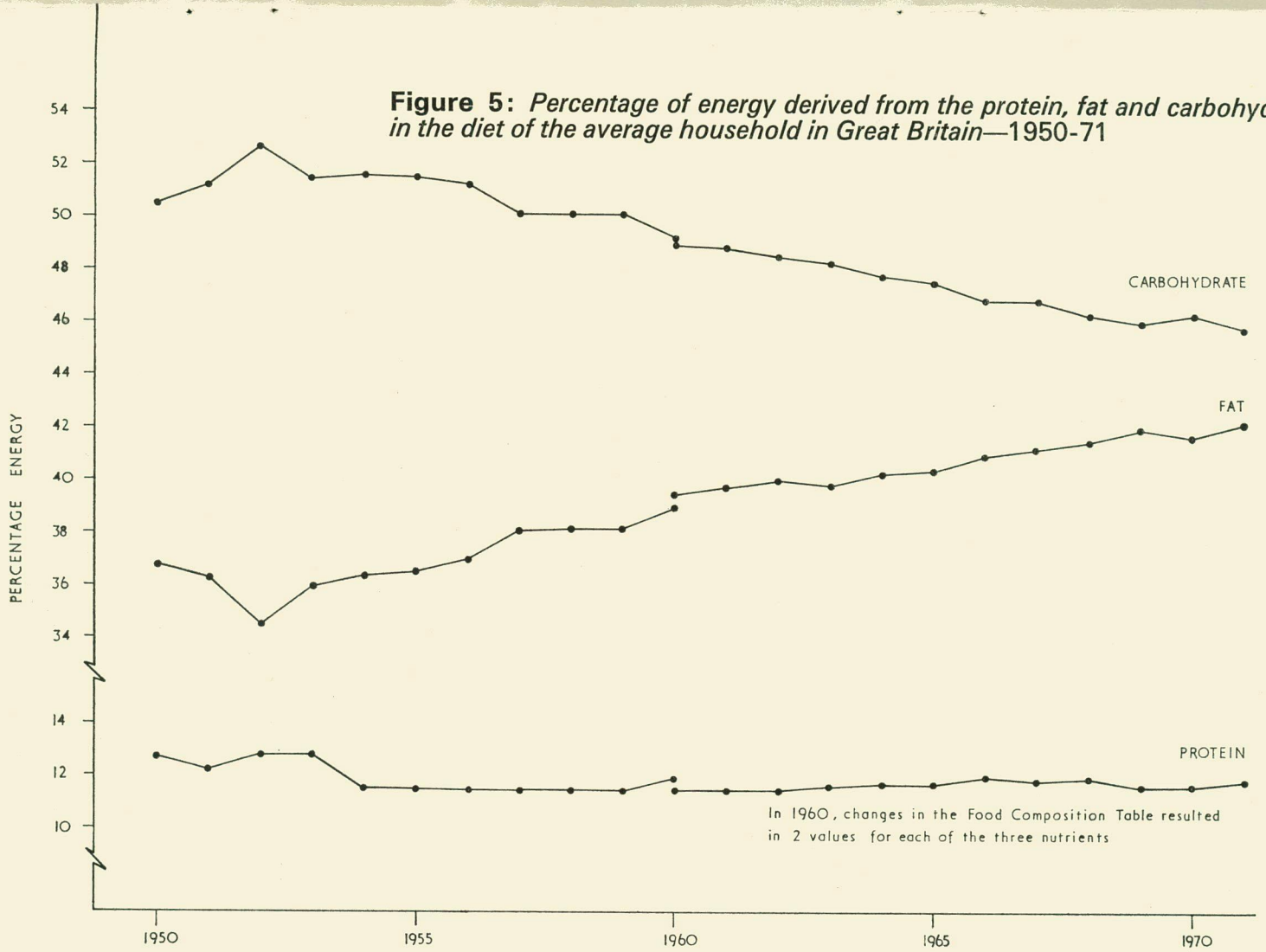
*1950-67—International Classification of Diseases (7), categories 420-422. 1968-71—International Classification of Diseases (8), categories 410-414.

Figure 4: *Total amount of refined sucrose available for human consumption in the United Kingdom (C.L.E.) expressed as g per day/per head of the population*



SOURCE: Until 1929—Deerr (1950) on the basis that 100 parts raw sugar=93 parts refined sugar.
1934-71 —Ministry of Agriculture, Fisheries and Food, 1968, 1969, 1971, 1972a.

Figure 5: *Percentage of energy derived from the protein, fat and carbohydrate in the diet of the average household in Great Britain*—1950-71



SOURCE: National Food Survey (Ministry of Agriculture, Fisheries and Food, 1952-66; 1967-73).

Note of Reservation by Professor J. Yudkin

The role of sucrose

There are three characteristics of I.H.D. that throw light on its possible aetiology. These are the multiplicity of the disturbances that accompany I.H.D., the other diseases that are associated with I.H.D., and the indication that causative factors already identified appear to have at least one feature in common.

With varying frequency, I.H.D. is accompanied by many abnormalities, including an increased blood concentration of cholesterol, triglyceride, insulin and uric acid; an increased adhesiveness and abnormal electrophoretic behaviour of the blood platelets, and an impaired glucose tolerance. It is possible but unlikely that the only one of these several abnormalities that has been extensively studied, namely the raised concentration of blood cholesterol, will also prove to be the one that has the greatest predictive value. Indeed, there is already evidence that a raised concentration of triglyceride, and perhaps also an impaired glucose tolerance, are better predictors of the disease.

The many disturbances found in I.H.D. support the suggestion that the underlying cause of the disease is hormonal in nature; the view has already been put forward that this might be an increased blood concentration of insulin. However, in view of the complex interrelationships of hormone secretion and activity, it may well be that the primary change is with a hormone other than insulin. In any event, there is now a hint that the relative immunity of premenopausal women is linked to a general hormonal involvement in the disease process.

The association of I.H.D. with other diseases is especially evident in regard to diabetes. I.H.D. is the commonest cause of death in diabetics; diabetes is frequently found in patients presenting with I.H.D. Other diseases associated with I.H.D., although in varying and uncertain degree, include obesity, gout and duodenal ulceration.

There is no evidence that alterations in dietary fat can produce all the abnormalities seen in I.H.D.; it is also difficult to see how dietary fat can explain the association of I.H.D. with other diseases. On the other hand, dietary sucrose has been found to produce disturbances in fat, protein and carbohydrate metabolism that are strongly suggestive of disturbances in hormonal balance. It has in fact been shown to produce an increased blood concentration of insulin and of 11-hydroxy corticosterone in a sizeable proportion of men; to increase the concentration in the blood of uric acid, cholesterol and, especially, triglyceride; to reduce glucose tolerance; and to alter the stickiness and electrophoretic behaviour of blood platelets.

The effects on blood uric acid and glucose tolerance relate to the possible rôle of dietary sucrose in gout and in diabetes. As to obesity, sucrose is more readily converted into fat than is starch. As to duodenal ulceration, it has been shown that diets low in carbohydrate, especially in sucrose, are effective in relieving symptoms; on the other hand, a diet high in sucrose leads to an increased gastric secretion of acid and especially of pepsin.

Finally, even though the rôle of insulin may be secondary to some other hormonal change in causing I.H.D., it is relevant to point out that increased physical activity, which reduces the risk of I.H.D., reduces the blood concentration of insulin. On the other hand, obesity, diabetes, peripheral vascular disease and cigarette smoking, all of which are associated with I.H.D., are found to produce increased concentrations of blood insulin. This effect they share with dietary sucrose.

These comments provide some of the reasons for my belief that the Report has exaggerated the possible rôle of dietary fat in causing I.H.D., and has minimized the possible rôle of dietary sucrose.

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