Table V.2

Effect of varying PUFA as percentage of constant total fat intake. Lipid profile at beginning and end of trial

PUFA %	LDL Cholesterol (mmol/L)		VLDL Cholesterol (mmol/L)		Apo B (mg/L)		HDL/Total	
	Initial	Final	Initial	Final	Initial	Final	Initial	Final
3	2.07±0.08	2.33±0.59*	0.22±0.05	0.27±0.12	664±202	718±175	0.36±0.06	0.35±0.05
19	1.91±0.40	1.98±0.60	0.27±0.13	0.23±0.12	580±103	574±120*	0.35±0.06	0.38±0.06

* P<0.05: change from initial values.

Source: Jantine A Brussaard *et al.*, 'Effects of amount and type of dietary fat on serum lipids, lipoproteins and apolipoproteins in man. A controlled 8-week trial', *Atherosclerosis*, 1980, **36**: 515–27, 515. (With permission from Elsevier Science.)

scant source of fats prior to the Agricultural Revolution, but the changes that it brought about resulted in a very great increase in the availability of the animal fats associated with meat, poultry, eggs and dairy produce. There is indirect evidence to suggest that there was an accompanying increase in predominance of saturated as opposed to unsaturated fatty acids. It remains to consider the consequences.

Dietary Fats and Coronary Heart Disease

In the present study it is thought adequate for the most part to provide evidence for a considerable eighteenth-century increase in the availability of animal fats in general and saturated fats in particular. It is not considered necessary to marshall in any more than summary form the now well-established reasons for associating high animal fat intake with a lipid profile conducive to development of coronary heart disease. Reference to a limited number of late-twentieth-century landmark studies should suffice.

A direct relationship between high saturated animal fat consumption and raised serum cholesterol levels has long been established, dietary changes altering the lipid profile significantly in little over a month. For example, as part of a wider study, Jantine H Brussaard and his colleagues studied forty healthy young subjects of both sexes. Following a run-in period on identical diets, two groups took 40 per cent of their energy requirements as fat. For one-half of the subjects, 3 per cent of the fats were polyunsaturated. Saturated and monounsaturated animal fats constituted the remainder. For the second group, the polyunsaturated fats, mainly of vegetable origin, were raised to 19 per cent and the saturated animal fats correspondingly reduced. Initially the lipid profiles of the two groups were almost identical. After five weeks the mean serum total cholesterol, triglycerides, apolipoprotein-B and low density lipoprotein (LDL) cholesterol levels were all significantly higher in the group with the higher saturated fat intake. The HDL/cholesterol ratios were minimally lower (Table V.2).[18] *Mann ist was mann isst.* The Seven Countries Investigation

[18] Jantine H Brussaard *et al.*, 'Effects of amount and type of dietary fat on serum lipids, lipoproteins and apolipoproteins in man. A controlled 8-week trial', *Atherosclerosis*, 1980, **36**: 515–27, pp. 520–3.

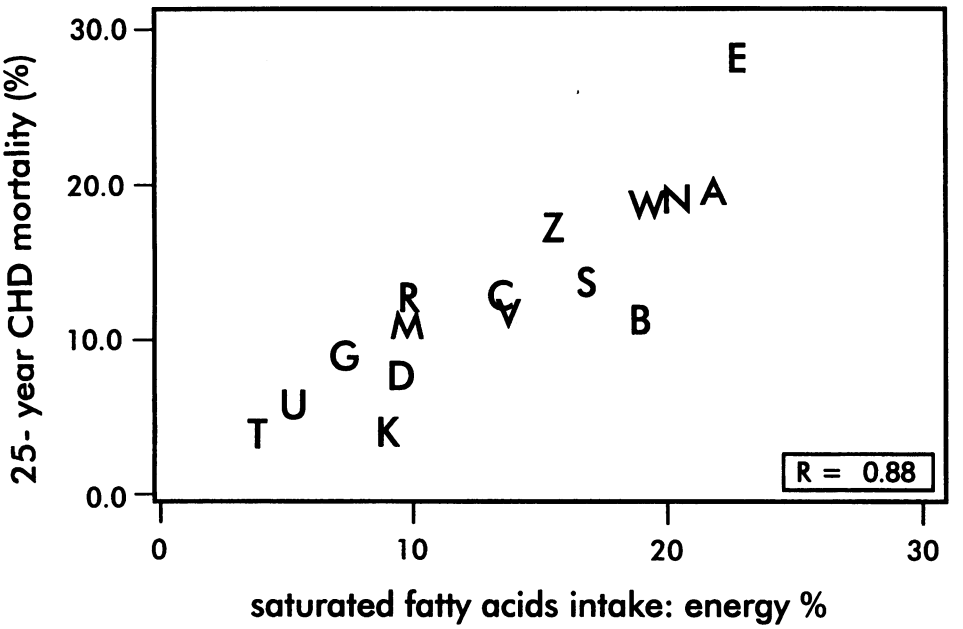

Figure V.2: Correlation between average intake of saturated fats and 25-year mortality from coronary heart disease (%) in the Seven Countries study. The letters relate to location (not listed). Source: Daan Kromhout *et al.*, 'Dietary saturated and trans-fatty acids and cholesterol and 25-year mortality from coronary heart disease. The Seven Countries Study', *Prev Med*, 1995, **24**, 308–15, p. 311. (With permission by Academic Press.)

involved 12,763 middle-aged men living in Greece, the Netherlands, Yugoslavia, Italy, Japan, USA and Finland. The follow-up, which lasted some twenty-five years, showed that the higher the animal fat consumption was in any one location, the higher were the overall serum cholesterol levels and the incidence of coronary heart disease (CHD) (Figure V.2).[19] In the multiple risk factor intervention trial (MRFIT) study over one-third of a million men aged from thirty-five to thirty-seven years at entry were followed for twelve years. At entry, risk factors were present in some instances, but none had a history of heart disease. During the follow-up there was an aged-adjusted CHD mortality that was positively and continuously related to the total serum cholesterol concentrations.[20]

In contrast, there is evidence to indicate that reductions in total fat intake in general, and saturated fats in particular, are followed by lessening risk of myocardial infarction, both fatal and non-fatal. As an example, Thomas Lyon and his colleagues compared survivors of a myocardial infarction whose fat intake was restricted to 50 g daily with a control group of survivors among whom no such limits were

[19] Daan Kromhout *et al.*, 'Dietary saturated and trans-fatty acids and cholesterol and 25-year mortality from coronary heart disease. The Seven Countries Study', *Prev Med*, 1995, **24**: 308–15, pp. 310ff.

[20] J M Martin *et al.*, 'Serum cholesterol, blood pressure, and mortality: implications from a cohort of 361,662 men', *Lancet*, 1986, **ii**: 933–6, p. 934.

Table V.3
Relation of CHD deaths to diet and serum cholesterol levels (mg/dl), Helsinki heart study

Hospital Designation		1st Period	Number of deaths	2nd Period	Number of deaths
N	Males	L 217	20	H 266	52
K		H 268	24	L 234	14
N	Females	L 245	52	H 278	107
K		H 270	22	L 236	21

L: Low Cholesterol Diet H: High Cholesterol Diet.

Source: Matti Miettinen *et al.*, 'Effect of cholesterol-lowering diet on mortality from coronary heart-disease and other causes', *Lancet*, 1972, **ii**: 835–8, p. 836. (Permission granted by The *Lancet* Ltd.)

imposed. During a 3.8 year follow-up of the 155 subjects on the low fat diet, there were a total of 15 recurrent myocardial infarctions, of which 4 were fatal. In contrast, the 125 unrestricted subjects, albeit during a slightly longer 4.2 year follow-up, suffered 51 recurrences, of which 13 were fatal.[21] In the Helsinki heart study polyunsaturated fats were substituted for saturated in the diet of the participants in a cross-over study involving two institutionalized populations, some of whom had already had CHD. In both diets the percentage of energy requirements supplied by one or the other form of fat was higher than is currently considered optimal. Despite this, the polyunsaturated fat regime was associated with a reduction in male and female coronary heart disease mortality of about one-half among the men and one-third in the women (Table V.3).[22] In a prevention study of a free living population without any evidence of CHD at entry, J N Morris, Jean W Marr and D G Clayton examined the relationship of diet to the incidence of coronary heart disease among 337 middle-aged men who lived in either London or other parts of south-east England and who participated in a dietary survey. The subjects were stratified into lowest, middle and highest ranges of polyunsaturated/saturated fat (P/S) ratios. The differences in the ratios were not great and even among the highest third the proportion of polyunsaturated fat was suboptimal by standards generally adopted later. The study began in 1956 and by 1976 forty-five men had developed clinical coronary heart disease. Although the dietary differences between the groups was modest, the incidence of coronary heart disease during the first five years was significantly lower among the men with the highest P/S ratios. Although no longer

[21] Thomas P Lyon *et al.*, 'Lipoproteins and diet in coronary heart disease: a five-year study', *Calif Med*, 1956, **84**: 325–8, p. 328.
[22] Matti Miettinen *et al.*, 'Effect of cholesterol-lowering diet on mortality from coronary heart-disease and other causes', *Lancet*, 1972, **ii**: 835–8, p. 836.

statistically significant, benefits were still present at ten and twenty years. By the end of the twenty-year follow-up, deaths from coronary heart disease totalled 11, 10 and 5 in the lowest, middle and highest tertiles of P/S ratio respectively, i.e. an inverse relationship between polyunsaturated fatty acid intake and coronary heart disease incidence was demonstrated.[23] Whether these benefits were due to the increase in polyunsaturated or the decrease in saturated fat intake is not readily determinable, but the benefits of substituting the former for the latter are evident. Conversely these studies demonstrate that diets low in polyunsaturates but unrestricted in saturated fatty acids are associated with an *increased* incidence of coronary heart disease.

Thus far, emphasis has been placed on the relationship between diets high in animal fats and the incidence of major coronary events, notably myocardial infarction and cardiac deaths. Stress has been placed on the connection between liberal consumption of saturated fats and the adverse cardiac consequences of *structural* coronary arterial disease, notably plaque formation and coronary arterial narrowing and blockage. However, of late a clear-cut and significant relationship has been established between elevation of serum cholesterol levels and deleterious changes, notably loss of coronary arterial and arteriolar capacity to dilate (see Table V.4). The endothelial lining of the coronary arteries has active metabolic properties and *inter alia* produces an endothelial derived relaxing factor that makes possible a response to the increased demands of exercise by blood flow increases of up to four times the basal requirements of the heart. Demonstration of the ability of normal arterial walls to produce nitric oxide and its role as a simple inorganic vasodilator earned Robert Furchgott, Louis Ignarro and Ferid Murad the 1998 Nobel Prize in physiology or medicine.

In the presence of a raised serum total cholesterol, the coronary arterial capacity for vasodilatation is impaired and the possibility of increasing coronary blood flow in response to demand is impeded. This functional impairment has been demonstrated even in coronary arteries with no macroscopic abnormalities or nothing other than fatty streaks in the vessel wall. The consequences of failure of normal parts of the arterial wall to relax are necessarily more profound if structural narrowing is present as well.[24] Andreas M Zeiher and his colleagues used quantitative coronary angiography (a means of outlining a blood vessel radiologically after injection with radiopaque dye) to compare the responses of subjects with either a normal or a raised serum cholesterol to intra-arterial acetylcholine infusion.[25] The latter is a naturally occurring chemical secreted at certain nerve endings and linked with activation of the parasympathetic nervous system. Zeiher and his colleagues found that the coronary arteries of subjects with a normal total serum cholesterol had a larger pre-infusion cross-sectional area on average and they responded to acetylcholine by dilatation. On the other hand, the coronary arteries of the subjects with a raised serum cholesterol had a smaller initial cross-sectional area on average and they

[23] J N Morris, Jean W Marr and D G Clayton, 'Diet and heart: a postscript', *Br Med J*, 1977, **ii**: 1307–14, pp. 1311–12.

[24] Andreas M Zeiher *et al.*, 'Endothelium mediated coronary flow modulation in humans: effects of age, atherosclerosis, hypercholesterolemia and hypertension', *J Clin Invest*, 1993, **92**: 652–62, p. 657.

[25] Ibid., p. 652.

Table V.4
Serum cholesterol status and coronary artery responsiveness

Comparison of angiographic responses of left anterior descending coronary artery (on which the left ventricle is largely dependent for its blood supply) to **1** 72 g/mm acetylcholine (AC) infusion, and **2** papaverine induced maximal coronary flow. Subjects with normal or elevated serum cholesterol.

			Cholesterol	
			Normal	Elevated
	No. of subjects		18	20
	Age (years)		50.2 ± 6.7	52.7 ± 11.7
1	LAD Status at angiography		Normal	13 normal 7 lumen irregular
	Cross-sectional area mm^2	Pre AC infusion	10.6 ± 7.3	7.0 ± 4.0
		During AC infusion	12.8 ± 7.9	4.9 ± 3.9
2	Maximal coronary blood flow increase (%)		476.1 ± 127.7	356.9 ± 150.6

Source: Andreas M Zeiher *et al.*, 'Endothelium mediated coronary flow modulation in humans: effect of age, atherosclerosis, hypercholesterolemia and hypertension', *J Clin Invest*, 1993, **92**: 652–62, pp. 654, 656 (by permission of the American Society for Clinical Investigation).

responded to the infusion by constriction of the artery (Figure V.3).[26] There was a direct relationship between elevation of serum cholesterol levels and the degree of impairment of the vasodilator response. In addition, the maximal inducible increase in coronary artery flow, as established by intra-arterial infusion of the pharmacological agent papaverine, was the less in the group with a raised serum cholesterol.[27]

These effects have also been demonstrated experimentally in pigs in which diet-induced elevation of serum cholesterol resulted in increases in concentration of plasma endothelin. This substance too is generated by the endothelium (the innermost lining of the artery) and has a vasoconstrictor action. In these pigs direct infusion of acetylcholine into coronary arteries resulted in vasoconstriction, in contrast to the absence of coronary artery diameter change in animals with normal serum cholesterol levels.[28]

In the Regress investigation, the effects of administering the cholesterol-lowering medication pravastatin on serum cholesterol levels and the clinical course were

[26] Ibid., p. 652.
[27] Ibid., pp. 654–8.
[28] A Lerman *et al.*, 'Circulating and tissue endothelin immunoreactivity in hypercholesterolaemic pigs', *Circulation*, 1993, **88**: 2923–8, p. 2925.

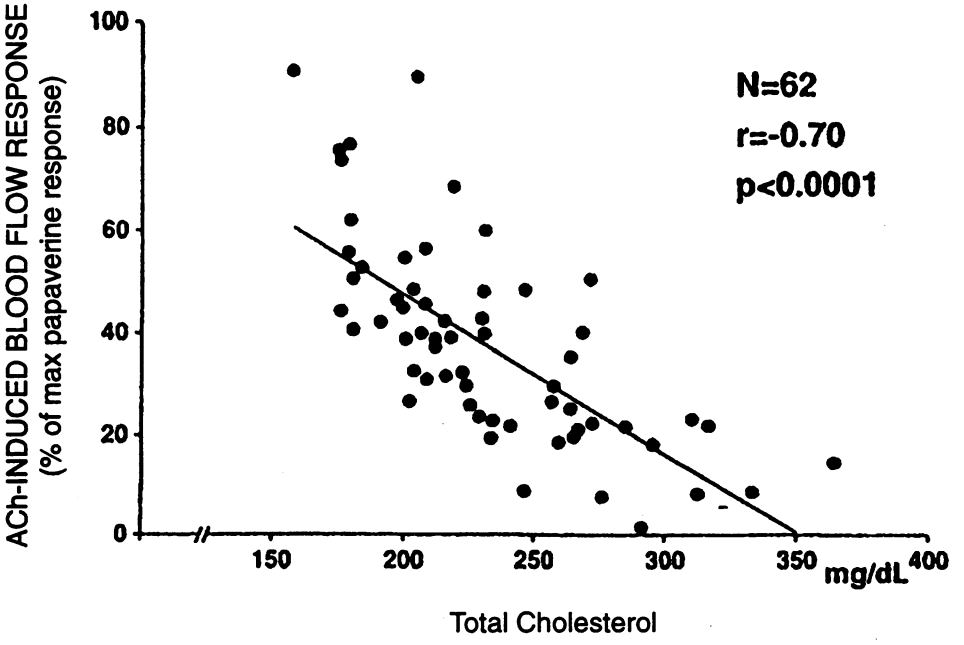

Figure V.3: Correlation between total serum cholesterol level and acetylcholine induced dilator capacity of the coronary system. Source: Andreas M Zeiher *et al.*, 'Endothelium mediated coronary flow modulation in humans: effects of age, atherosclerosis, hypercholesterolaemia and hypertension', *J Clin Invest*, 1993, **92**: 652–62, p. 657. (By permission of the American Society for Clinical Investigation.)

studied. It was found that when compared with the controls who were given a placebo, the patients in the active treatment arm and whose serum total cholesterol was lowered significantly had a correspondingly significant reduction in the total ischaemic burden. This was defined electrocardiographically as the product of the duration of S-T segment depression (an indicator of deficient heart muscle blood flow) in minutes and its extent in millimetres. These changes could be either asymptomatic or accompanied by angina pectoris. The total ischaemic burden decreased from an average of 34.6 millimetre-minutes at baseline to 26.4 in the placebo arm, but from 41.5 to 22.5 millimetre-minutes in the pravastatin treated group, a 23 per cent lowering in the former as opposed to 46 per cent in the latter. The Regress investigators found that after some five months a functional and cholesterol-related vasoconstrictor basis for periodic spontaneous reductions in myocardial blood flow was countered by cholesterol lowering, a concept now widely accepted.[29] Lessening of serum total cholesterol levels has also been shown by coronary angiography to be followed by a beneficial effect on coronary arterial responsiveness to the effects of acetylcholine, constriction being replaced by moderate

[29] Ad J van Boven *et al.*, 'Reduction of transient myocardial ischemia with pravastatin in addition to the conventional treatment in patients with angina pectoris. REGRESS Study Group', *Circulation*, 1996, **94**: 1503–5.

https://doi.org/10.1017/S0025727300073750 Published online by Cambridge University Press

dilatation.[30] Together these findings suggest that in a population with a low saturated fat intake and resulting low serum total cholesterol levels, the incidence of functionally induced transient ischaemic events with their possible symptomatic associations would be relatively low. By implication, these conclusions suggest the converse: the eighteenth-century English increase in saturated fat consumption, with its now known association with serum cholesterol elevation, had the potential to result in functionally induced imbalances between myocardial blood supply and cardiac muscle requirements. These changes, occurring independently of any structural coronary arterial disease, could precipitate angina pectoris in a patient otherwise asymptomatic or aggravate it in a person already susceptible. They thereby constitute a possible additional link between dietary changes during the Georgian era and the concurrent emergence of angina pectoris.

Finally, note must be made of the nearly complete absence of the very long carbon chain PUFAs (C20:5 N-3 and C20:6 N-3) in the lipids of cattle farmed in the twentieth century, although constituting up to 8 per cent of the fatty acids of free living bovids.[31] This difference suggests that changes in cattle management as initiated during the Agricultural Revolution could account for very considerable falls in the concentrations of these naturally occurring long chain fatty acids. They are known to improve endothelial dependent coronary arterial and microvasculature relaxation and thereby facilitate increase in blood flow to the heart in response to need.[32] They also have mildly anticoagulant properties[33] and are, incidentally, plentiful in fatty fish and the blubber of marine mammals. They may therefore contribute to a lowered incidence of coronary heart disease among peoples who eat large amounts of these foods (see pages 64, 182). Conversely, the disappearance of these beneficial long chain PUFAs from the fats of land animals with changing eighteenth-century feeding practices could have been one more factor contributing to development of coronary heart disease in that period.

During the 1960s saturated fat consumption in the United States started to fall and coronary heart disease rates to decline. The Framingham study compared CHD incidence and mortality in a ten-year follow-up of 1950 and 1970 cohorts of men who were aged fifty to fifty-nine and free of heart disease at entry into the study. The end points included myocardial infarction, whether fatal or otherwise, stable or unstable angina pectoris, sudden death, congestive heart failure and any other clinical presentations. The authors were able to separate the fall in CHD incidence from the effects of recently introduced treatment on survival after the disease had become established. They were thereby able to show that the *incidence* of coronary heart disease with its first manifestations was lower in the 1970 than in the 1950 cohort (Table I.1).[34] Lee Goldman and E Francis Cook came to similar conclusions. They

[30] Charles B Treasure *et al.*, 'Beneficial effects of cholesterol-lowering therapy on the coronary endothelium in patients with coronary artery disease', *N Engl J Med*, 1995, **332**: 481–7, p. 484.

[31] Crawford *et al.*, op. cit., note 8 above, p. 297.

[32] Vladimir I Vekshtein *et al.*, 'Fish oil improves endothelial dependent relaxation in patients with coronary arterial disease (abstract 1727)', *Circulation*, 1989, Suppl II, p. 434.

[33] Schacky and Weber, op. cit., note 12 above, pp. 2447–8.

[34] Pamela A Sytkowski, William B Kannel and Ralph B D'Agostino, 'Changes in risk factors and the decline in mortality from cardiovascular disease. The Framingham Heart Study', *N Engl J Med*, 1990, **322**: 1635–41, pp. 1637–8.

studied the decline in CHD death rates between 1968 and 1976. They first calculated the contribution to falling mortality made by intervening changes in the clinical management of established disease. These included the introduction of intensive care units and resuscitation procedures, prevention of serious heart rhythm disturbances, widespread use of beta adrenergic receptor blocking drugs to counter excessive sympathetic nervous system activity, treatment of high blood pressure and introduction of surgical coronary artery bypass grafting.[35] The authors then reviewed epidemiological studies reporting the effect of various primary preventive measures, such as those aimed at lowering serum cholesterol and reduction in smoking.[36] In conclusion, Goldman and Cook calculated that newer lifestyles, including low fat diets, accounted for about 54 per cent of the reduction in mortality observed during the eight-year study.[37] It would be equally reasonable to conclude that the reverse is true and that the substantially higher previous CHD incidence was associated in large measure with the earlier lifestyles that included higher consumption of animal fats.

During the late twentieth century the death rate from coronary heart disease began to fall not only in the United States[38] but in countries as disparate as Finland,[39] New Zealand,[40] Iceland,[41] Australia and, albeit to a lesser extent, the United Kingdom.[42] Although improved management of established disease played an important part, there was, as noted in the case of the USA, a reduction in incidence as well as in mortality. Concurrently, there were changes in lifestyle risk factors in all of these five countries, among them a fall in saturated fat consumption that was taking place in the developed world generally. Thus in Finland, selected as the country whose eastern regions once had the world's highest CHD death rate, there was a reduction in mortality of over 50 per cent. During the 25-year period in which this decline occurred, the average total fat content of the Finnish diet fell from 38 per cent of energy intake to 34, and saturated fat from 21 to 16 per cent.[43] The Finnish investigators applied previously established epidemiological data relating dietary fat to total serum cholesterol levels and the effect of their reduction on coronary heart death rates. On this basis they calculated that almost half of the observed fall in mortality could be attributed to the reduction in total and saturated fat consumption and the concomitant increase in intake of polyunsaturated fats, as

[35] Lee Goldman and E Francis Cook, 'The decline in ischemic heart disease mortality rates. An analysis of the comparative effects of medical interventions and changes in lifestyle', *Ann Intern Med*, 1984, **101**: 825–36, pp. 825–31.

[36] Ibid., pp. 831–2.

[37] Ibid., pp. 832–4.

[38] Sytkowski, Kannel, D'Agostino, op. cit. note 34 above, p. 1638.

[39] Pirjo Pietinen *et al.*, 'Changes in diet in Finland from 1972 to 1992: impact on coronary heart disease risk', *Prev Med*, 1996, **25**: 243–50, p. 243.

[40] Rodney Jackson and Robert Beaglehole, 'Trends in dietary fat and cigarette smoking and the decline in coronary heart disease in New Zealand', *Int J Epidemiol*, 1987, **16**: 377–82, p. 378.

[41] Nikulas Sigfusson *et al.*, 'Decline in ischaemic heart disease in Iceland and changes in risk factor levels', *Br Med J*, 1991, **302**: 1371–5, p. 1373.

[42] Terry Dwyer and Basil S Hetzel, 'A comparison of trends in coronary heart disease mortality in Australia, USA and England and Wales with reference to three major risk factors—hypertension, cigarette smoking and diet', *Int J Epidemiol*, 1980, **9**: 65–71, p. 66.

[43] Pietinen *et al.*, op. cit., note 39, pp. 244–6.

in soft margarines. Their findings thus conformed closely to those of Goldman and Cook.[44] The conclusions from the comparable studies conducted in the United Kingdom, Australia, New Zealand and Iceland have been similar.[45]

In eighteenth-century England the dietary changes were in the reverse direction to the twentieth as animal fats then became available in greatly increased quantities. In so far as twentieth-century findings can be applied to that time, it is probable, as pointed out earlier, that these fats were for the most part saturated. It therefore seems probable that the eighteenth-century dietary changes, which were in the opposite direction to recent trends, had correspondingly opposite and therefore adverse effects on the lipid profile in general and on the serum total cholesterol levels in particular. The richer diet could have thereby contributed significantly to the eighteenth-century development of clinically recognizable coronary heart disease. As part of the present postulate, the changes in fat consumption are considered the most conclusive and the most important of all the eighteenth-century developments examined either in this or later sections where the role of changes in ancillary risk factors is analysed. The evidence cited indicates that excessive fat consumption, as characterized the eating habits of the affluent during the late eighteenth century, results in serum cholesterol abnormalities that are conducive to development of coronary heart disease, even in the absence of other risk factors. The Seven Countries study results showed that over 40 per cent of the variance in the ten-year CHD death rate could be accounted for by differences in serum total cholesterol levels.[46] The authors of the MRFIT study concluded that by themselves serum total cholesterol levels in excess of 4.68 mmol/L could alone account for about half of the observed coronary heart disease mortality.[47] As discussed later under their respective headings, the effects of other dietary factors, protective in the case of fish and fibre, deleterious in the case of sugar and coffee, are associated with corresponding changes, harmful or beneficial, in the lipid profile. This is one important way in which their effects are produced. Conversely, the effects of non-dietary risk factors, notably smoking, high blood pressure and stress, are greatly reduced or even nullified when fat intake and serum cholesterol are low. A striking example of this is provided by early post-Second World War Japan. It was and is a developed society where in the 1950s the fat intake was very low and what there was came from fish and was therefore very high in unsaturated fatty acid content. During this time the annual male death rate from heart disease was 363/100,000 in England and Wales, but only 131 in Japan. The corresponding numbers for females were 158 and 81 respectively. Heart disease was then more likely to be reported in Japan than in England and Wales and about a quarter of the Japanese cardiac deaths were due to conditions other than CHD. The incidence of the latter was low despite heavy smoking, severe stresses, aggravated by the course and outcome of the Second World War and its aftermath, and a severe

[44] Ibid., p. 246; Goldman and Cook, op. cit., note 35 above, p. 832.

[45] Dwyer and Hetzel, op. cit., note 42 above, p. 65; Jackson and Beaglehole, op. cit., note 40 above, p. 378; Sigfusson *et al.*, op. cit., note 41 above, p. 1374.

[46] Kromhout *et al.*, op. cit., note 19 above, p. 319.

[47] Martin *et al.*, op. cit, note 20 above, p. 935.

Table V.5
Average systolic blood pressures: Japanese* and American** applicants for life insurance. Age groups 45–65 (mmHg)

	United States		Japan	
Age Group	Systolic BP	% ≥150	Systolic BP	% ≥150
45+	130.0	13.4	130.6	17.3
50+	134.5	20.2	137.2	26.3
55+	137.8	26.3	138.2	29.7
60+	141.8	34.9	144.0	39.8

* 75,000 (including females; not separately tabulated).
** 269,583 males (86% of applicants).

Reprinted from Henry A Schroeder, 'Degenerative cardiovascular disease in the Orient. II hypertension', *J Chron Dis*, 1958, **8**: 312–33, p. 315. (With permission from Elsevier Science.)

housing shortage.[48] Salt intake was high in Japan, estimated at 10 to 15 g daily per person, and may have been a factor contributing to the proportion of middle-aged men with elevated systolic blood pressures being higher in Japan than in the United States (Table V.5), possibly a cause for the then very high Japanese incidence of cerebral haemorrhage.[49] In the balance, the role of animal fat intake, harmful when excessive but beneficial when restricted, overrides other lifestyle risk factors in importance.

Faith and Fish

Some further developments in eighteenth-century English dietary practices may have had their origins in changes in religious observance. The Reformation in England resulted initially in establishment of a national church that retained many features of Catholicism, and even after the Elizabethan modifications, it resulted in a form of Protestantism less extreme than was usual on the continent.[50] There was consequently a tendency to retain some Roman Catholic dietary laws, particularly periodic abstinence from meat. These restrictions were reinforced by sixteenth-century civil laws designed to protect the fishing fleet, a potential source of men for the navy. Eating meat was prohibited during Lent, on all Fridays and on occasion on Wednesday. These were designated fish days. The laws were civil, but the choice of days and times Roman Catholic in origin.[51] All of this resulted in over a quarter

[48] Michael G Marmot and George Davey Smith, 'Why are the Japanese living longer', *Br Med J*, 1989, **299**: 1547–51, p. 1547.
[49] Henry A Schroeder, 'Degenerative cardiovascular disease in the Orient. II Hypertension', *J Chron Dis*, 1958, **8**: 312–33, pp. 315, 320.
[50] G R Elton, *England under the Tudors*, London, Methuen, 1974, pp. 156, 271–6.
[51] George Macaulay Trevelyan, *English social history*, London, Longman, 1978, p. 189.