have a specific beneficial effect.[60] Fish consumption does not directly affect serum cholesterol levels significantly.[61] However, the long carbon chain fatty acids present in fish have been found to reduce platelet aggregation in response to contact with the physiological stimulants collagen and adenosine diphosphate. They have therefore a mild anticoagulant effect of potential value in protecting against clot formation in coronary arteries.[62] Such fatty acids are important constituents of fish oil and this may be a basis for beneficial effects of fish consumption on CHD incidence. Lastly, fish oil has been found to improve endothelium dependent coronary arterial relaxation in patients with established coronary heart disease (page 72), thus facilitating an increase in blood flow to the heart in response to need.[63]

In conclusion, eighteenth-century changes in eating habits with a decline in religious observance of dietary constraints and a fall into abeyance of legal restrictions on meat consumption may have resulted in an appreciable fall in fish consumption, possibly to below a suggested critical threshold level. Reduced intake of fish with loss of its various beneficial effects could have contributed to the rising eighteenth-century coronary heart disease incidence, with its concurrent replacement by fatty animal foods constituting an inter-related additional risk factor.

Fibre

Some of the greatest changes in farming in Georgian England related to the growing of cereals and, in particular, to a shift in emphasis from coarse grains to wheat; the relative importance of oats and rye declining while that of wheat rose. With abandonment of the strip system and other improvements in methods of cultivation during the eighteenth century, yields of wheat and oats, as measured in bushels per acre, increased in roughly similar proportions, by about one-eighth in the case of the former and one-sixth in the latter. However, the acreage under wheat rose between 1695 and 1750 from about one and a third million to over 2 million acres, but the increase under oats was much less, from 1.22 to 1.44 million acres. As a result, wheat production increased by 73.5 per cent, almost exactly double the 37.5 per cent rise in the yield of oats. During the last half of the eighteenth century the rate of increase in the amount of wheat harvested rose sharply while the rate of rise in oat production lessened somewhat (Table V.7).[64] As a result oat production fell considerably as a proportion of total cereal output while that of wheat rose. The absolute amount of oats available for human consumption actually declined during this time. In 1696 the horse population of England was estimated by Gregory King

[60] M L Burr *et al.*, 'Effect of changes in fat, fish and fibre intakes on death and myocardial reinfarction: diet and reinfarction trial', *Lancet*, 1989, **ii**: 757–61, p. 759.

[61] Kromhout, Bosschieter, Coulander, op. cit., note 58 above, p. 1206.

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

[63] Vekshtein *et al.*, op. cit., note 32 above, p. 434.

[64] B A Holderness, 'Prices, production and output', in G E Mingay (ed.), *The agrarian history of England and Wales, Volume VI: 1750–1850*, Cambridge University Press, 1989, p. 145.

Table V.7
English arable cereal yields, 1750–1800

Crop	Area cultivated (acres × 10^6)		Yield/acre (quarters)		Total yield (quarters × 10^6)		% Change 1750–1800
	1750	1800	1750	1800	1750	1800	
Wheat	1.8	2.5	3.6	6.1	6.5	15.3	+135
Rye	0.5	0.3	1.0	0.9	0.5	0.27	−48
Barley	1.4	1.3	3.8	4.5	5.3	5.9	+10
Oats	2.0	2.0	6.0	7.8	12.0	15.6	+30

Source: B A Holderness, 'Prices, production and output', in G E Mingay (ed.), *The agrarian history of England and Wales, Volume VI: 1750–1850*, Cambridge University Press, 1989, p. 145 (with permission).

at 1.2 million.[65] A corresponding estimate for 1750 was close to 1.5 million.[66] More importantly, as they replaced oxen at the plough, the number of draught horses with their high energy needs doubled, with consequent diversion of oats from human consumption to animal feed.[67] During this time the production of rye, a coarse grain, declined by some 48 per cent.[68] As described below, these changes were reflected, notably among the affluent, in increasing consumption of the more expensive white bread and diminishing intake of high fibre staples.

As with other risk factors, any contribution of these changes to eighteenth-century emergence of overt clinical manifestations of coronary heart disease cannot be measured directly. They must be inferred from relevant twentieth-century studies, which have taken two forms. One is based on observation of the effect of a deliberately varied fibre intake on the lipid profile. The other involves epidemiological studies that relate differing levels of fibre consumption to the incidence of CHD morbidity and mortality. Studies of the effects of experimentally varying fibre intake on lipid profiles involve fewer problems than do the epidemiological studies. Randomizing is possible, and fairly precise regulation of intake can be maintained during the short time needed to detect any biochemical changes. Compliance for a limited time is the more easily assured and regulation of the type of fibre to be consumed is not difficult. Other risk factors, notably those that involve diet, can be held constant throughout the study. Epidemiological studies do not have these advantages, but can use the incidence of clinical coronary heart disease events or deaths as the gold standard by which to gauge the possible effectiveness of high fibre consumption as a preventive measure. Furthermore, epidemiological studies have the potential for determining whether the effect of fibre on the lipid profile is or is not the only mechanism of any

[65] Gregory King, *Natural and political observations and conclusions upon the state and condition of England 1696*, ed. George E Barnett, Baltimore, Johns Hopkins Press, 1936, p. 37.
[66] J A Chartres, 'The marketing of agricultural produce', Joan Thirsk (ed.) *The agrarian history of England and Wales, Volume V: 1640–1750. II. Agrarian change*, Cambridge University Press, 1985, p. 446.
[67] Jonathan Brown and H A Beecham, 'Farming practices', in G E Mingay (ed.), *The agrarian history of England and Wales, Volume VI: 1750–1850*, Cambridge University Press, 1989, p. 289.
[68] Holderness, op. cit., note 64 above, p. 145.

benefit. These investigations too have taken two forms. One is prospective observation of the incidence of coronary heart disease in populations whose fibre consumption has been varied deliberately. The other is a retrospective dietary survey of fibre intake by patients who had suffered manifestations of CHD and who are compared with apparently healthy control subjects.

Observations of the effect of varying fibre intake on the lipid profile are now numerous and on the whole consistent. K Storch and colleagues studied twelve healthy college students in a cross-over investigation. Their diet was supplemented by either oat or wheat bran during a six-week period. The reduction in total serum cholesterol with oat bran was about double the modest lowering achieved with the wheat equivalent.[69] Kurt Gold and Dennis Davidson reviewed six investigations apart from their own (Table V.8). All showed falls in serum total cholesterol with experimental diets in which oats were the source of the designated high fibre. The reductions ranged from 3 to 26 per cent.[70]

The mechanisms by which fibre produces a favourable effect on the serum lipid profile have been reviewed by R C Spiller who has pointed out that the changes are more than can be explained by coincident reduction in total energy and saturated fat intake. They can also be dissociated from the laxative action and the increase in stool bulk associated with high bran diets. It was concluded that in the case of oat bran, the effects are probably related to its breakdown products trapping water in a gel and thereby reducing mixing movements. This has a slight negative effect on cholesterol absorption by lessening its contact with the walls of the small intestine. In addition, fibre derived from oats results indirectly in bile acid sequestration by bacteria normally present in the colon. This reduces the enterohepatic cycle, a process by which bile acids pass from the liver to the small intestine where they facilitate fat absorption and are themselves subsequently re-absorbed into the bloodstream.[71]

There are many studies of the relationship between fibre intake and coronary heart disease incidence. These include the Ireland–Boston diet-heart study in which, as part of their investigation, L H Kushi and colleagues recorded the diet history of 390 men resident in Ireland, 386 Irish immigrants to the Boston area and 225 first generation immigrants born in the United States to Irish immigrant parents. At entry into the study they were aged from thirty to sixty-nine years. A dietician recorded each man's initial diet history. When the study population was classified by tertiles, an inverse relationship between fibre intake and CHD mortality was demonstrated, the differences actually becoming greater after adjustment for other coronary risk factors (Table V.9) and just reaching statistical significance. Even the highest mean fibre intake of the three groups was low by current recommendations and during the follow-up period the overall risk factor profile was changing, so that in these circumstances the significance of the differences is especially noteworthy. In this study it was not possible to adjust for an observed inverse relationship between

[69] K Storch, J W Anderson and V R Young, 'Oat bran muffins lower serum cholesterol of healthy young people', *Clin Res*, 1984, **32**: 740A (abstract).

[70] Kurt V Gold and Dennis M Davidson, 'Oat bran as a cholesterol-reducing dietary adjunct in a young, healthy population', *West J Med*, 1988, **148**: 299–302, p. 301.

[71] R C Spiller, 'Cholesterol, fibre and bile acids', *Lancet*, 1996, **347**: 415–16, p. 415.

Table V.8
Human studies evaluating oat products and lipid effects

Studies and grain(s)	Subjects No.	Amount g/day	No. of Days on Diet	Prestudy S. Cholesterol (mg/dl)	Cholesterol Reduction (%)
De Groot *et al.* 1963[a]					
Rolled Oats	21	140	21	251	11
Judd & Truswell, 1981[b]					
Rolled Oats	10	125	21	203	8
Kirby *et al.* 1981[c]					
Oat Bran	8	94	10	269	13
Anderson *et al.* 1984[d]					
Oat Bran	10	100	21	257	19
Chen & Anderson, 1986[e]					
Oat Bran/Beans	10	41/145	182	257	26
Oat Bran/Beans	4	41/145	693	257	22
Van Horn *et al.* 1986[f]					
Oat Bran	69	39	28	196	3
Rolled Oats	69	35	28	195	3

[a] A P De Groot, R Luyken and N A Pikaar, 'Cholesterol lowering effect of rolled oats', *Lancet*, 1963, **ii**: 303–4.
[b] P A Judd and A S Truswell, 'The effect of rolled oats on blood lipids and fecal steroid excretion in man', *Am J Clin Nutr*, 1981, **34**: 2061–7.
[c] R W Kirby *et al.*, 'Oat-bran intake selectively lowers low-density lipoprotein cholesterol concentrations of hypercholesterolemic men', *Am J Clin Nutr*, 1981, **34**: 824–9.
[d] J W Anderson *et al.*, 'Hypocholesterolemic effects of oat bran or bean intake for hypercholesterolemic men', *Am J Clin Nutr*, 1984, **40**: 1146–55.
[e] W J L Chen and J W Anderson, 'Hypocholesterolemic effects of soluble fibres', in G V Vahouny and D Kritchevsky (eds), *Washington symposium on dietary fibre*, New York, Plenum Press, 1986, pp. 275–86.
[f] L V Van Horn *et al.*, 'Serum lipid response to oat product intake with a fat modified diet', *J Am Diet Assoc*, 1986, **86**: 759–64.

Reproduced from Kurt V Gold and Dennis M Davidson, 'Oat bran as a cholesterol-reducing dietary adjunct in a young, healthy population', *West J Med*, 1988, **148**: 299–302, p. 301. (With permission from the BMJ Publishing Group.)

fibre and fat consumption, but any such tendency constitutes an additional if indirect cardioprotective benefit of high fibre intake.[72]

M G Marmot and colleagues showed that between 1951 and 1971 there was a slight increase in heart disease mortality in the two highest British social classes, but a more rapid rate of rise in the lowest two. In 1931 heart disease mortality was greater among men in social classes I and II when compared with classes IV and V.

[72] L H Kushi *et al.*, 'Diet and 20-year mortality from coronary heart disease. The Ireland–Boston diet-heart study', *N Engl J Med*, 1985, **312**: 811–18, pp. 813–14.

Table V.9

Relationship between fibre intake tertiles and male CHD mortality expressed as relative risk (RR) males. 17–23 year follow-up.

		CHD mortality		
Fibre intake tertile		Lowest 3rd	Middle 3rd	Highest 3rd
RR	Crude *	1.0	0.73	0.46
	Adjusted **	1.0	0.88	0.57[1]

* Adjusted for cohort and age

** Adjusted additionally for systolic BP, total serum cholesterol, EKG abnormalities, cigarette smoking and alcohol intake

[1] Difference from reference RR significant ($P<0.05$).

Adapted from data published by L H Kushi *et al.*, 'Diet and 20-year mortality from coronary heart disease. The Ireland–Boston diet-heart study', in *New Engl J Med*, 1985, **312:** 811–18, p. 816.

By 1961 the situation had become reversed. The fibre consumption of the highest income groups relative to that of the lowest increased steadily during this period. When compared to the lowest two social classes the highest two with the *higher* overall fibre intake emerged with a *lower* incidence of CHD over the twenty years.[73] Finally, in a prospective secondary prevention study, Daan Kromhout and his colleagues obtained a dietary history with respect to fibre intake in a group of 871 men who were followed for six to twelve months. The twenty-seven men who died from CHD during the period under review had an average daily fibre intake of 27.2 $\pm$ 8.1 g per day, appreciably less than the 30.8 $\pm$ 9.7 g per day of the survivors. When corrected for other factors by multivariate analysis, the difference approached conventional levels of significance, the P value being 0.06.[74]

In conclusion, the fibre intake of the English middle and upper classes declined during the Georgian era. Recent studies have shown that a low fibre intake affects the lipid profile and incidence of coronary heart disease adversely. The decline in oat fibre intake, in particular, during the eighteenth century could therefore be considered a contributory cause for angina pectoris then becoming manifest in England as a disease of the affluent and increasingly common thereafter.

Sugar

Wild cane sugar plants probably grew originally on the island of New Guinea, which was possibly the site of its first cultivation.[75] By the end of the fifteenth century

[73] M G Marmot *et al.*, 'Changing social-class distribution of heart disease', *Br Med J*, 1978, **ii**: 1109–12, p. 1110.

[74] D Kromhout, E B Bosschieter and C de Lezenne Coulander, 'Dietary fibre and 10-year mortality from coronary heart disease, cancer, and all causes. The Zutphen study', *Lancet*, 1982, **ii**: 518–22, p. 519.

[75] N Deerr, *History of sugar*, 2 vols, London, Chapman and Hall, 1949–50, vol. 1, p. 44.