

however, longer articles might be expected, but we found no supporting evidence. This may suggest that editors are trying to include more contributions in the available space. Alternatively, narrowing the range of material covered or reporting less experimental work in a given article may be responsible, perhaps a reflection of "publish or perish." These factors, together with the rising number of journals, may place increasing pressure on the interested reader, who may not find time for adequate consideration, even of abstracts. Could it be, then, that the longer titles are an attempt to draw attention to the kernel of the material in a sort of mini abstract? This might be valuable in attracting the more marginal attention of staff working

in adjacent but less related disciplines. Computerised searching based on key words, which in some cases includes a search only within the title, may also encourage this trend.

Could there, however, be another explanation? The correlation between the length of title and the number of authors but not with the other variables raises a question. Substantive discussion among the increasing number of authors per article might have been expected to lead to longer articles influenced by a greater diversity of viewpoints. This is not the case. Our evidence questions whether active contribution by some participants is reserved for the title, which alone bears the thumbprint of all.

The haggis tolerance test in Scots and Sassenachs

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Abstract

To find out if the Scottish national dish, haggis, contributes to the high incidence of coronary heart disease in Scotland the lipaemic effect of a meal of 200 g of haggis was measured in six Scottish and 10 Sassenach men. The Scots had higher fasting cholesterol and triglyceride concentrations and a lower proportion of high density lipoprotein cholesterol than the Sassenachs. Four subjects were found to have hyperlipoproteinaemia, which had been unrecognised previously. Serum cholesterol concentrations did not change after haggis was eaten (mean dose 2.6 g/kg body weight). Serum concentrations of triglycerides increased by 51% at 90 minutes in the Sassenachs but were unaltered in the Scots. There were no serious adverse effects.

This study shows that Scots have higher lipid concentrations than Sassenachs but seem to be resistant to the lipaemic effect of haggis. The haggis tolerance test may be useful in Sassenachs.

Introduction

Coronary heart disease is now the main cause of death in Scotland. One important contributing factor is consumption of fat, but, strangely, the influence of the Scottish national dish, haggis, on blood lipid concentrations and heart disease seems to have been neglected as a subject for serious scientific inquiry.

We are aware of only two previous medical investigations of haggis. Single blind histological studies performed by Professor Horst Oertel at McGill University in 1929 confirmed that haggis consists of degenerate liver cells and cereal products within a sac lined by epithelial and muscle tissue (J R Cameron, personal communication). A paper delivered to the Nutrition Society extolled the virtues of haggis, turnips, and potatoes as a healthy and balanced diet.¹ An exhaustive search of publications produced no other studies, and haggis is not cited in *Index Medicus*. We therefore set up the Cardiff heart, alveoli, gut and grain ingestion study (HAGGIS) to investigate the effects of consumption of haggis on blood lipid concentrations.

Subjects and methods

We studied 16 healthy male volunteers (that is, conscripted medical colleagues), including six expatriate Scots and 10 Sassenachs. Their mean age was 36 (95% confidence interval 31 to 41). Nationality was

assigned according to the country of birth of both parents.

Ethical approval was not sought, and informed consent could not be obtained as so little is known about the consequences of eating haggis. There was no control group because we deemed it unethical to withhold haggis from any subject.

After the subjects had fasted overnight and abstained from alcohol blood samples were taken for measurement of fasting lipid concentrations. All subjects then ate 200 g of freshly boiled haggis (T G Willis, Edinburgh) as their breakfast. Their compliance was ensured by all subjects eating together. Apple or orange juice was permitted to help swallowing, but whisky was withheld until the end of the study. Blood samples were taken 30 and 90 minutes after the last mouthful of haggis. Subjects were given diaries in which to record any unwanted symptoms.

The haggis that we used is commercially available and prepared in bulk (0.5 tonnes a week) according to traditional recipes.² Its ingredients are lambs' pluck (heart and lung), pigs' liver, beef suet (perinephric fat), and pinhead (coarse) oatmeal. These are cooked; minced; seasoned with salt, black pepper, coriander, and ginger; and boiled in a sheep's paunch (stomach). A 200 g portion of haggis contains 21.4 g protein, 43.4 g fat, 38.4 g carbohydrate, 5% inedible fibre, and 2584 kJ.³

Quality was checked by performing histological studies on a generous wedge biopsy specimen excised from a randomly selected haggis (fig 1). Electron

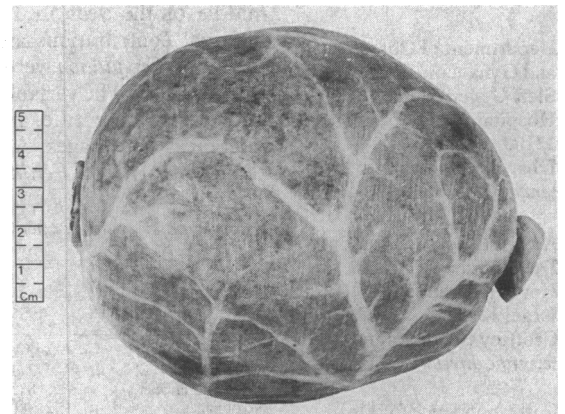


FIG 1—Haggis, cold and before gastrotomy, showing gastric blood vessels

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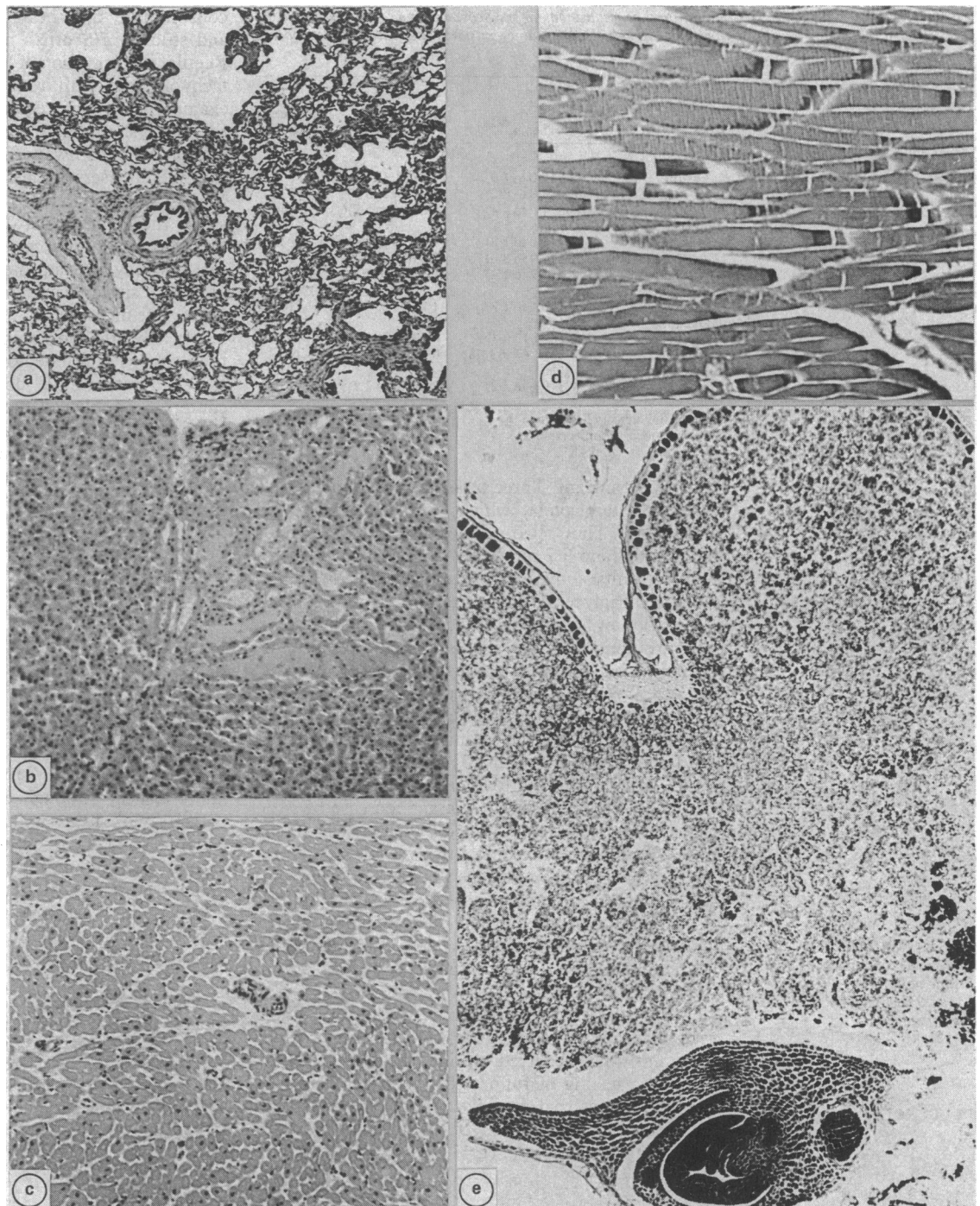


FIG 2—Sections of tissues in haggis stained with haematoxylin and eosin (a) lung; (b) liver; (c) cardiac muscle; (d) skeletal muscle; (e) monocotyledonous endosperm with embryo

microscopy of the specimen was not feasible owing to its particulate nature, but conventional examination confirmed the presence of pulmonary, cardiac, and hepatic tissue as well as seeds of the gramineae family, presumably oatmeal (fig 2). Unexpectedly the sample also contained striated muscle; although this is not a recognised ingredient of haggis, small quantities of diaphragm adjacent to heart, lung, or liver seemed to have become incorporated in the haggises used in this study. Nevertheless, this is unlikely to have seriously influenced the results.

Serum cholesterol concentration was determined by an enzymatic colorimetric method (Boehringer Mannheim). Concentrations of high density lipoprotein cholesterol and subfractions were measured by the method of Gidez *et al.*⁴ Serum concentrations of triglycerides were measured by a Technicon RA1000 analyser with a kit from Technicon Diagnostic. Serum concentrations of apolipoproteins AI and B were

determined by rate nephelometry (Beckman Immunochemistry System).

To conform with current standard practice⁵ we used those statistical tests that produced significant results. Differences in demographic characteristics between the groups were ignored. One Sassenach who had not fasted had high serum triglyceride concentrations and was excluded from the analysis of triglyceride and high density lipoprotein cholesterol concentrations.

Results

The table gives the results of the lipid assays. The Scots had higher fasting total cholesterol and triglyceride concentrations than the Sassenachs and a lower proportion of high density lipoprotein cholesterol. Differences in other variables, including the ratio of apolipoprotein AI to apolipoprotein B, which correlates with the extent of coronary artery disease,⁶

Mean lipid concentrations in six Scots and nine Sassenachs (except where stated otherwise). Values in parentheses are 95% confidence intervals

	Scots	Sassenachs	p Value (unpaired t test)
Cholesterol (mmol/l):			
Fasting	6.5 (5.0 to 7.9)	4.8 (4.0 to 5.6)*	0.037
At 30 minutes	6.6 (5.0 to 8.2)	4.8 (3.9 to 5.6)*	0.037
At 90 minutes	6.6 (4.9 to 8.4)	4.8 (4.0 to 5.7)*	0.049
Triglycerides (mmol/l):			
Fasting	1.4 (1.0 to 1.8)	0.7 (0.4 to 0.9)	0.006
At 30 minutes	1.5 (0.9 to 2.0)	0.8 (0.5 to 1.2)	0.033
At 90 minutes	1.4 (0.7 to 2.1)	1.1 (0.7 to 1.4)	NS
HDL cholesterol (mmol/l)			
HDL: total	1.1 (0.9 to 1.3)	1.2 (1.1 to 1.3)	NS
cholesterol	0.18 (0.13 to 0.22)	0.27 (0.24 to 0.30)	<0.0001
HDL ₂ HDL ₃	0.83 (0.47 to 1.18)	0.69 (0.56 to 0.83)	NS
Apolipoprotein AI:			
apolipoprotein B	1.5 (0.8 to 2.2)	2.0 (1.7 to 2.2)*	NS

HDL=High density lipoprotein.

*Mean value in 10 subjects.

were not significant. Three subjects were found to have hypercholesterolaemia, which had been undetected previously. They had cholesterol concentrations between 6.7 and 9.2 mmol/l, which are above recommended limits.⁷ One of these subjects and one other had raised triglyceride concentrations.

Ingestion of haggis caused no change in cholesterol concentrations in either group (paired *t* test) so the difference between the groups was the same at 90 minutes. Overall, haggis caused a significant increase in triglyceride concentrations, which rose from a fasting value of 1.1 (95% confidence interval 0.7 to 1.4) mmol/l to 1.3 (0.8 to 1.7) mmol/l at 30 minutes ($p=0.012$) and 1.3 (0.9 to 1.7) mmol/l at 90 minutes ($p=0.027$), representing an average increase of 34%. The rise was, however, confined to the Sassenachs (fig 3). At 90 minutes, the Scots' triglyceride concentrations had increased on average by only 6%, compared with an average increase in the Sassenachs of 51% ($p<0.0001$). Increases in the concentration of triglycerides were not related to the dose of haggis, which was comparable in all groups (mean 2.6 (2.5 to 2.8) g/kg body weight).

Only a quarter of the diaries were returned. There were no deaths, and no writs were received. Minor symptoms that occurred during the day of study included a feeling of fullness of the stomach ($n=2$), sneezing, retrosternal chest discomfort during exercise, and "eructations and odour found unacceptable by a largely English company" ($n=1$).

Discussion

This study was ill conceived and badly designed but brilliantly executed. It confirmed the value of screening for hyperlipoproteinaemia in potential haggis eaters, and it showed that the Scots, because of their higher cholesterol concentrations, are at greater risk of developing coronary heart disease than their fellow British citizens.

Most importantly, it showed that haggis has a lipaemic effect, at least in people who eat it sporadically such as the Sassenachs in this study. Intriguingly, the Scots were resistant to this effect, for reasons that are now the subject of intense speculation. We suggest several possible mechanisms, all of which merit further study. The Scots may have evolved a genetic tolerance to haggis, so epidemiological studies of trends in haggis

consumption and heart disease over the centuries and studies of Scottish migrants would be valuable. Regular consumption of haggis may produce a dietary tachyphylaxis with reduced chylomicronaemia—for example, by autoregulation of intestinal haggis receptors (should they exist) or derepression of alternative catabolic pathways for haggis. Sassenachs may be slow metabolisers of haggis. The Scot who showed the greatest postprandial fall in triglyceride concentration spent the interval between the 30 and 90 minute samples performing cardiac surgery, which suggests that this may have a protective effect, but the practice of cardiac surgery could hardly be advocated as a mass preventive measure.

For Scots the clear implication of this study is that they should continue to eat haggis, especially if it is baked rather than boiled as this reduces its fat content (unpublished personal observations). Haggis is a healthy food—for example, sheep's lung contains only 20% of the fat and more protein weight for weight than a leg of lamb.⁸ Haggis has been recognised for centuries as the greatest sausage⁹ and as "real food that nourishes both through feeling and fact,"¹⁰ even if it has on occasion to be disguised as mince in order to deceive and delight the average southerner.¹¹ Queen Victoria attested to its beneficial effects when she ate "the celebrated haggis" at Blair Castle on Wednesday 11 October 1865 and "really liked it very much."¹² Perhaps the Scots should export it to countries with a high incidence of coronary heart disease and campaign for the establishment of haggis subsidies and haggis mountains throughout the European Community.

Finally, we suggest that the haggis tolerance test as described in this study offers a novel test for fat tolerance, although caution may be required in patients with heart failure because 200 g of haggis contains 1 540 mg of sodium.³ The haggis tolerance test is more gastronomically pleasing than previous alternatives consisting of butter, double cream, and ice cream, and it is safer and more physiological than tests with intravenous infusions of lipid that do not assess digestion and absorption.

We thank our colleagues for participating in this study; Dr Mark S Fraser for his help with historical research; our secretary for typing the manuscript; the medical photographers who produced the illustrations; and Dr R G Newcombe for his expert statistical advice, which as usual we partly followed.

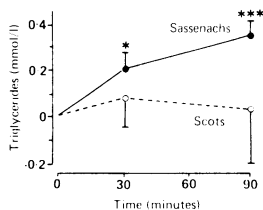


FIG 3—Mean (SE) changes in triglyceride concentrations from fasting values. * $p<0.05$, *** $p<0.01$ compared with fasting values

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