Carbon monoxide and nicotine yields of nine plain brands and nine non-ventilated filter brands of cigarette. Each CO value is the mean of two to four assays. The crosses represent the means of the two types of cigarette. The plain brands were: Capstan Full Strength, Capstan Medium, Churchmans No. 1, Embassy Plain, Gauloise Caporal Plain, Kensitas Plain, Piccadilly No. 1, Senior Service Plain, and Woodbine Plain. The filter brands were: Consulate Menthol, Dunhill International, Gitanes Caporal Filter, John Player Special, Peter Stuyvesant King Size, Piccadilly King Size, Players No. 6 Filter, Rothmans King Size Filter and Senior Service Tipped.

lower in the filter than in the plain cigarettes ($P < 0.005$). When plain and filter cigarettes were considered separately there was, within each type, a suggestion of a positive correlation between CO and nicotine yields. These complex relationships between CO and nicotine yields were obscured when the type of cigarette was ignored.

Discussion

Ordinary filter cigarettes have unventilated filters, and have higher CO yields than plain cigarettes because the paper surrounding the filter is less porous than that surrounding the tobacco—so that less air can enter through the wall of the cigarette to dilute the smoke. These cigarettes now occupy about 80% of the UK market, whereas in 1955 most cigarettes were of the plain type and filter cigarettes occupied only 2%. Alternatively, cigarettes may have ventilated (perforated) filters, through which air can enter and reduce the concentration of all the constituents of tobacco smoke. Hence it would be expected that the CO and nicotine yields of these cigarettes are highly correlated.¹ These deliver less tar, nicotine, and CO than either plain or unventilated filter cigarettes but occupy only about 5% of the UK market.³

A recent report on the examination of three plain and four filter brands showed that among cigarettes with nicotine yields over 1.0 mg (a level which automatically excludes all those with ventilated filters) there was no apparent relation between nicotine and CO yields, and that the filters of these cigarettes did not appear to reduce CO delivery.¹ Our results suggest they may actually increase it, but data on more brands are needed before the size of this increase can be reliably estimated.

The Government publishes smoking tables to encourage smokers to avoid brands with high tar or nicotine yields. Evidence of toxicity is much less secure for nicotine than for tar, but since the yields of both tend to vary in parallel, this is of little practical importance. The toxicity of CO in tobacco smoke is largely unknown, but present evidence suggests that it may be more harmful than nicotine.⁴ For this reason it has been recommended that CO yields should be included in smoking tables, but this proposal raises a special problem: some brands with "medium" tar yields have relatively high CO yields, and others with lower CO yields produce more tar. Smokers who cannot stop smoking and who wish to base their choice of cigarette on information in tables giving figures for tar, nicotine, and CO may, therefore, find themselves in a dilemma, since the only cigarettes which are low in all three are not generally regarded as satisfying. We believe, therefore, that it would be unwise to include CO yields in official tables until medical advice can be given about the relative risks of exposure to the different components of cigarette smoke.

² Tobacco Research Council. *Research Paper II*, eds K Rothwell and C A Grant. London, 1974.

³ Tobacco Advisory Committee. Unpublished. (Sales figures relate to February and March 1975.)

⁴ Wald, N J, and Howard, S, *Annals of Occupational Hygiene*, 1975, 18, 1.

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Hypertriglyceridaemia and abdominal pain

Hypertriglyceridaemia has long been known to be associated with abdominal pain. Such cases are usually Fredrickson types I and V,¹ with massive rises in the serum triglyceride concentration. We report an example of pain associated, at least between attacks, with modest hypertriglyceridaemia.

Case report

A 33-year-old South African Indian had first been admitted as an emergency in October 1974 complaining of central abdominal pain. He had been well until six weeks previously, when he had started to get bouts of central colicky abdominal pain occurring once a week, lasting 20 minutes accompanied by nausea. Over the previous few days the pain had become continuous. The only abnormal physical sign noted was tenderness in the right iliac fossa. Appendicular or ureteric colic had been diagnosed. The pain had subsided and the patient had been discharged. His attacks had continued and as an outpatient, the results of intravenous pyelography, barium meal and follow-through, and cholecystogram had all been normal. Fasting serum lipids had shown a serum triglyceride concentration of 5.3 mmol/l (469 mg/100 ml) (normal 0.3-2.0 mmol/l (26-178 mg/100 ml)) and a cholesterol of 7.4 mmol/l (286 mg/100 ml) (normal 4.0-7.5 mmol/l (154-290 mg/100 ml)) with the presence of chylomicrons.

On admission for investigation he stated that he was now getting two attacks per week and had lost two jobs as a result. It was noted that there was no family history of similar attacks or of premature vascular disease, and xanthomata or arcus senilis were not present. The results of extensive investigation were all normal. An oral glucose tolerance test gave normal results. Serum uric acid was 441 μ mol/l (7.4 mg/100 ml) (normal 100-400 μ mol/l (1.7-6.7 mg/100 ml)). Fasting serum triglyceride concentration was 3.0 mmol/l (266 mg/100 ml), cholesterol 6.5 mmol/l (251 mg/100 ml), without the presence of chylomicrons. Lipoprotein electrophoretic strip² showed a densely staining pre-beta band indicating type IV hyperlipoproteinaemia. Plasma post-heparin lipolytic activity was normal.³ He was challenged with a high-fat diet and the same evening developed severe abdominal pain. A blood sample taken 10 pm that night showed that the serum was loaded with chylomicrons; the triglyceride concentration was then 14.2 mmol/l (1257 mg/100 ml), and serum amylase 70 Somogyi units/100 ml (normal 0-200).

The pain subsided during the night, and fasting serum triglyceride levels the following morning were 4.3 mmol/l (380 mg/100 ml) and the morning after 4.6 mmol/l (407 mg/100 ml) both samples showing presence of chylomicrons. He was started on a low-fat diet but continued to have attacks of abdominal pain, and six weeks later fasting triglyceride was 4.2 mmol/l (372 mg/100 ml) without chylomicrons. He was then changed to a 120-g carbohydrate diet (previous intake 200 g) with substitution of polyunsaturated for saturated fats. His attacks of pain ceased and two months later the fasting serum triglyceride was 3.4 mmol/l (301 mg/100 ml) without chylomicrons. Though he was free of symptoms we felt it necessary to further reduce his serum triglyceride level, and gave him clofibrate, 1 g twice daily. He was seen again at the end of August 1975, three months after starting his low-carbohydrate/polyunsaturated-fat-substituted diet, and one month after starting clofibrate. He felt well, had had no further attacks of pain, and was holding down an important executive post. Fasting serum triglyceride was 2.0 mmol/l (177 mg/100 ml) with a normal lipoprotein electrophoretic strip. He was last seen on 13 October, when he remained well and symptom free; the fasting serum was clear, with a triglyceride concentration 1.5 mmol/l (133 mg/100 ml).

¹ Russell, M A H, Cole, P V, Idle, M S, and Adams, L, *British Medical Journal*, 1975, 3, 71.

Discussion

Probably this patient's abdominal pain was related to his hyperlipidaemia, though its nature remains obscure. When given a high-fat intake he developed severe pain associated with gross chylomicronaemia; treatment directed at lowering his serum triglyceride rendered him symptom free. This patient is of interest because, atypically, his serum triglyceride concentration was raised only modestly, except during the attack apparently provoked by a high-fat intake. He was classified as having type IV hyperlipoproteinaemia, though he had intermittent fasting chylomicronaemia. Dietary restriction of fat did not prevent his attacks of pain, but carbohydrate restriction did, presumably by reducing his endogenous triglyceride production.

The distinction between types IV and V hyperlipoproteinaemia may well not be absolute, chylomicronaemia in the latter sometimes reflecting the degree of hypertriglyceridaemia rather than a different metabolic fault.⁵ Indeed, possibly the quantity of triglyceride to be transported may partly determine the size and nature of the transporting particles.⁵

This case re-emphasises the need to determine the serum lipid concentration in patients with obscure abdominal pain.

We should like to thank Mr A E Kark for permission to publish this case and Mrs D Bird for performing lipoprotein electrophoresis.

¹ Fredrickson, D S, *et al*, *New England Journal of Medicine*, 1967, **267**, 34.

² Noble, R P, *Journal of Lipid Research*, 1968, **9**, 693.

³ Elkeles, R S, and Hambley, J, *Clinica Chimica Acta*, 1975, **65**, 135.

⁴ Gotto, A, in *Clinics in Endocrinology and Metabolism—Disorders of Lipid Metabolism*. Philadelphia, Saunders, 1973.

⁵ Lewis, B, *et al*, *Atherosclerosis*, 1973, **17**, 455.

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High intestinal lactase concentration in adult Pakistanis

Lactose malabsorption in apparently healthy subjects has been reported as common among Africans, Asians, American Negroes, and American Indians. On the other hand, people of North European stock tend to have a low prevalence, always less than 20% and often about 6%. The difference has been explained on a genetic basis,¹ but is also an adaptation to the lack of consumption of milk among various races.

The present study was designed to estimate the prevalence or otherwise of lactase deficiency among adult Pakistanis according to various ethnic groups.

Patients, methods, and results

After explanation of the procedure to be adopted, 55 healthy Pakistanis agreed to be investigated and were admitted to the medical ward of Jinnah Postgraduate Medical Centre. Blood samples were taken from the finger of each of the fasting patients. Lactose (Analar, Merck) 1 g/kg body weight was administered orally in 400 ml of water. Capillary blood samples were taken 30, 60, 120, and 180 minutes after the administration of lactose. Blood glucose was determined by the Nelson-Somogyi² method. Five patients had a 25-g xylose tolerance test which was normal in each case. A rise in blood glucose of less than 1.1 mmol/l (20 mg/100 ml) above the fasting level was considered to indicate lactose malabsorption. The patients were questioned about the frequency of consumption of milk and other dairy products, and symptoms of intolerance if any. They were observed for symptoms during the tolerance test and were instructed to report any symptoms occurring during the following 24 hours.

The group comprised nine Punjabis, 12 Sindhis, four Baloochis, 15 Pathans, and 15 Mohajirs of both sexes (table). Among the Punjabis, Sindhis and Baloochis there was no malabsorption, the mean maximum rise in blood glucose being 2.4 mmol/l, 2.2 mmol/l, and 2.5 mmol/l (43, 40, 45 mg/100 ml) respectively. All the Pathans had a rise of more than 1.1 mmol/l (20 mg/100 ml), the mean maximum rise being 1.8 mmol/l (32 mg/100 ml). One Pathan, however, developed symptoms of intolerance in the form of diarrhoea. Three of the Mohajirs had a blood glucose rise of less than 1.1 mmol/l. These three, and one other patient with a normal lactose tolerance test (LTT) result, developed diarrhoea or colic, or both.

The three subjects with a rise of less than 1.1 mmol/l, and the Mohajir and Pathan who showed intolerance had a normal absorption after the xylose tolerance test.

Discussion

Our findings indicate that most adult Pakistanis have a high intestinal lactase activity. They therefore seem to be distinct from other Asian, Eastern Mediterranean and African races. Racially, Pakistanis are grouped as true Mediterraneans (Punjab); Oriental Mediterranean (Punjab and Sind); Pashtuns (North-West Frontier Province); and Brachycephalic Baloochis of Iranian stock (Aryans).³ The Mohajirs come from all over India and are a mixture of those shown above and Dravidians.

Environmental adaptation to milk consumption through the centuries may also have contributed. In the plains of Punjab milk consumption is fairly high. Even in the mountains of the north and deserts of the south, goats' milk and camels' milk form a substantial element of the diet. In contrast, the highest incidence of intolerance was found amongst the Mohajirs, in whom milk consumption was the lowest. The high lactase in the Pakistani may have helped him to survive epidemics of cholera⁴ in contrast to those in eastern parts of India. As has already been suggested, it may have caused an increased calcium absorption⁵ and protection from osteomalacia, which we rarely see in our hospitals.

We thank Miss Nabat Khanoo, H Karimi, and Miss Nisfat Khalil for technical help.

¹ *British Medical Journal*, 1975, **2**, 351.

² Nelson, N, *Journal of Biological Chemistry*, 1944, **153**, 375.

³ *New Encyclopaedia Britannica*, vol 30, p 896. Chicago, Helen Hemingway Benton Publishers, 1973.

⁴ *Lancet*, 1975, **1**, 79.

⁵ Flatz, G, Rothauwe, H W, *Lancet*, 1973, **2**, 76.

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Ethnic background, milk consumption, and result of lactose tolerance test among 55 healthy adult Pakistanis

Groups	No studied	Estimated daily milk intake (ml)				Mean fasting blood glucose (mmol/l)	Mean maximum rise in blood glucose (mmol/l)	Maximum blood glucose rise of 1.1 mmol/l		No with gastro-intestinal upset
		0	250	250-500	500			No	%	
Punjabis	9	—	2	6	1	4.3	2.4	9	100	—
Sindhis	12	1	5	6	—	4.2	2.2	12	100	—
Baloochis	4	2	2	—	—	4.2	2.5	4	100	—
Pathans	15	3	10	2	—	4.5	1.8	15	100	1
Mohajirs	15	12	3	—	—	4.5	1.8	12	86	4

Conversion: SI to traditional units—glucose: 1 mmol/l $\approx$ 18 mg/100 ml.