childhood diabetes in association with first attendance at school. Our data (table I) suggest that the relative frequencies of HL-A 8 and W 15 vary according to age at onset. For example, the ratio between cases positive for HL-A 8 and those positive for W 15 in the 11-15 years age group was higher than with other age groups. This deserves more-detailed prospective investigation in combination with virological studies. If this variability is real a possible implication is that there are different alleles at the diabetogenic locus which interact with different viral agents. Thus one allele might be in linkage disequilibrium more with HL-A 8 and another with W 15.

If virus-mediated damage to pancreatic β-cells is important there are several possible ways in which genes in the HL-A chromosomal region might influence the outcome. Genes could exert their influence by affecting membrane virus receptor sites; by determining cross-antigenicity with viral antigens; or by determining the nature of the immune response to virus infection in a manner analogous to that shown for Ir genes in the H-2 chromosomal region of mice.[13] The mechanism of action of the proposed HL-A-linked diabetogenic gene, however, must remain speculative. The finding that a disproportionate number of sibships show the affected siblings to have both HL-A chromosomes identical implies that genes influencing susceptibility to diabetes are often inherited from both parents. If this is confirmed by a larger study it would follow that the gene action is such that the susceptibility is greater when two such genes are inherited than when only one is present.

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High Intestinal Lactase Concentrations in Adult Arabs in Saudi Arabia

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Summary

The maximum rise in blood glucose after 50 g lactose by mouth was determined in 40 adult Arabs. Out of 30 Bedouin, urban Saudi, and Yemeni and 9 of mixed ancestry (usually partly African), 25 (83%) and 2 (22%) respectively showed an increase of over 1·1 mmol/l (20 mg/100 ml). In common with most northern Europeans and Hamitic people of northern Africa, Arabs in Saudi Arabia usually have high intestinal lactase concentrations in adult life. This persistence of high levels probably originated in the Arabian peninsula. Its selective advantage may have been associated with the fluid and calorie content of camels' milk, which is important for survival in desert nomads.

Introduction

Most northern Europeans and their descendants and the Hamitic people of northern Africa have high intestinal lactase concentrations in adult life.[1 2] Other ethnic groups and all other mammalian species have low concentrations, this not being determined by environmental factors.[1] Most adult Arabs[3 4] and Sephardic, oriental, and Ashkenazi Jews[5] on the eastern Mediterranean littoral have low concentrations.

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Patients and Methods

The table summarizes the clinical details of 40 Arab inpatients at the King Abdul Aziz Hospital in Riyadh. They were admitted consecutively and agreed to be investigated after an explanation of the procedure. None had evidence of gastrointestinal disease, malnutrition, or diabetes mellitus. Designation of ethnic and national status was based on dialect, a detailed family history, and assessment of physical features including general appearance, stature, skin colour, hair distribution and appearance, and nasal contours. People with a mixed ethnic background are known locally as *Khadiry* (five of the nine in this study had distinct African characteristics). Fourteen patients had a 25-g D-xylose absorption test; in 13, the 90-minute blood xylose concentration was over 2·3 mmol/l (35 mg/100 ml) and the five-hour urinary excretion over 33·4 mmol (5 g).

After a 10-hour overnight fast, two capillary blood samples were taken in duplicate with a 10-minute interval. Lactose 50 g (Analar, B.D.H.; glucose content of a 100 g/l solution <20 gm/l) diluted in 500 ml water was then given by mouth. Further capillary samples were taken, again in duplicate, at 15, 30, 60, and 90 minutes. Blood glucose concentrations after immediate deproteinization were estimated in duplicate by a hexokinase method (Boehringer, Mannheim, 15931). Standard glucose curves were constructed for each series of determinations. Symptoms were sought during the following 24 hours.

Results

The maximum rise in blood glucose (table) usually occurred 30 or 60 minutes after lactose, but in nine and seven patients with a rise of more and less than 1·1 mmol/l (20 mg/100 ml) respectively, it occurred at 90 minutes. The rise of 9·7 mmol/l (176 mg/100 ml) in one of the Bedouin was associated with a fasting concentration of 4·8 mmol/l (87 mg/100 ml). The mean fasting concentration of 6·8 mmol/l (124 mg/100 ml) in one of the *Khadiry* was associated with previously undiagnosed diabetes mellitus. Four patients with a rise of less than 1·1 mmol/l responded normally to the xylose test; the one abnormal result was associated with a glucose rise of 5·1 mmol/l (91 mg/100 ml).

Details of 40 Arabs and Summary of Results

Group	No. Studied			Mean Age in Years (Range)	Mean Body Weight in kg (Range)	Estimated Daily Milk Intake (ml)				No. with Symptoms after Milk	Mean Fasting Blood Glucose in mmol/l (Range)	Mean Maximum Rise in Blood Glucose in mmol/l (Range)	No. (%) of Patients with Maximum Blood Glucose Rise >1·1 mmol/l
	M.	F.	Total			0	<250	250—500	>500				
Bedouin	9	5	14	34 (18-60)	54 (44-73)	3		10	1	3	4·5(3·7-5·6)	3·2(0·2-9·7)	12 (85·7)
Urban Saudi	5	3	8	40 (17-65)	51 (39-64)	2		6		3	4·2(3·4-4·7)	2·4(0·8-4·7)	7 (87·5)
Khadiry	4	5	9	33 (14-60)	51 (35-74)	1		8		2	4·6(3·6-6·8)	1·2(0·1-4·2)	2 (22·2)
Yemeni	8		8	31 (16-40)	51 (43-64)	1	1	6		4	4·3(3·9-5·0)	2·2(0·2-4·7)	6 (75·0)
Syrian	1		1	48	53			1		1	4·5	0·6	

Conversion: SI to Traditional Units—Glucose: 1 mmol/l ≈ 18 mg/100 ml.

Four Bedouin, two urban Saudis, seven *Khadiry*, and three Yemenis had symptoms (colic or diarrhoea or both) after lactose; 11 out of 13 with a rise in glucose of less than 1·1 mmol/l had symptoms.

Discussion

An increase in blood glucose after 50 g lactose by mouth gives a good indication of intestinal lactase activity.[1] Adult Arabs on the Arabian peninsula[6] constitute the third major group to be shown to have high lactase activity; they seem, therefore, to be ethnically distinct from eastern Mediterranean populations. It seems likely that high lactase in most northern Europeans and the Hamitic people of Africa originated there.

The selective advantage of lactase in adult life in evolution is unknown. Bedouin have for thousands of years lived as nomads in the deserts of Arabia, and for long periods calorie and fluid intake have been largely derived from camels' milk. Adults with a high lactase concentration are likely, therefore, to have had an increased survival rate. In severe gastrointestinal infections active absorption of monosaccharides is coupled with water absorption[7]; thus it seems possible that adults with high lactase concentrations who lived largely on milk could have survived epidemics such as cholera. That the selective advantage of lactase has been associated with increased calcium absorption in groups not exposed to strong sunlight[8] is untenable.

Childhood malnutrition, especially marasmus, is common in many areas where adult hypolactasia is prevalent and high lactase concentrations fall soon after birth.[1] Though marasmus is common in Arab children in eastern Mediterranean countries, where adult hypolactasia is prevalent, it seems to be uncommon in Riyadh.

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Osteomalacia Presenting as Chorea

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Summary

A 16-year-old epileptic developed chorea. He had osteomalacia, hypocalcaemia, and hyperphosphataemia, which were due not to hypoparathyroidism but to vitamin D deficiency—itself secondary to longstanding dietary deficiency and anticonvulsant drug administration.

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Introduction

Choreiform movements are a rare but well described feature of hypocalcaemia[1 2] though in all reported cases the hypocalcaemia was due to hypoparathyroidism. We report here a case of osteomalacia accompanied by hypocalcaemia and hyperphosphataemia which presented with severe generalized choreiform movements. Several factors, including anticonvulsant drugs, were probably implicated in the causation of the underlying osteomalacia and hypocalcaemia.

Case Report

EARLY HISTORY

The patient was born on 7 July 1957. Delivery was normal and he weighed 6 lbs (2·7 kg). Between the ages of 2 and 5 years he had several febrile convulsions. When he was 11 he was investigated for abdominal pain and constipation. Physical examination showed nothing abnormal, but an iron deficiency anaemia (9·6 g/dl) and a low serum albumin