

With so much recent interest on dietary fibre, fibre-depleted starch foods have been said to be conducive to the development of diabetes in susceptible people with particular genotypes.<sup>22</sup> Certainly dietary fibre modifies carbohydrate absorption, and its favourable effect on diabetic control may be due to an effect on the incretin mechanism.

- <sup>1</sup> Bloom, S R, and Polak, J M, in *Gut Hormones*, ed S R Bloom, p 3. Edinburgh, Churchill Livingstone, 1978.
- <sup>2</sup> Creutzfeldt, S, *Diabetologia*, 1979, **16**, 75.
- <sup>3</sup> Brown, M, and Vale, W, *Endocrinology*, 1976, **98**, 819.
- <sup>4</sup> Stewart, I M, et al, *Thorax*, 1976, **31**, 278.
- <sup>5</sup> Kristensson, K, et al, *Acta Pathologica et Microbiologica Scandinavica*, 1971, **79**, sect A, 684.
- <sup>6</sup> Campbell, I W, et al, *Gut*, 1977, **18**, 462.
- <sup>7</sup> Kassander, P A, *Annals of Internal Medicine*, 1958, **48**, 797.
- <sup>8</sup> Wooten, R L, and Meriwether, T W, *Journal of the American Medical Association*, 1961, **176**, 1082.
- <sup>9</sup> Brownlee, M, and Kroopf, S S, *New England Journal of Medicine*, 1974, **291**, 1257.
- <sup>10</sup> Moore, J M, and Neilson, J, *Lancet*, 1963, **2**, 645.
- <sup>11</sup> Arapakis, G, et al, *British Medical Journal*, 1963, **1**, 159.
- <sup>12</sup> Malins, J M, and French, J M, *Quarterly Journal of Medicine*, 1957, **26**, 467.
- <sup>13</sup> Malins, J M, and Mayne, N, *Diabetes*, 1969, **18**, 858.
- <sup>14</sup> Berge, K G, Sprague, R G, and Bennett, W A, *Diabetes*, 1956, **5**, 289.
- <sup>15</sup> Scarpello, J H H, et al, *British Medical Journal*, 1976, **2**, 673.
- <sup>16</sup> Low-Beer, T S, *British Medical Journal*, 1973, **4**, 741.
- <sup>17</sup> Scarpello, J H B, Greaves, M, and Sladen, G E, *British Medical Journal*, 1976, **2**, 1225.
- <sup>18</sup> Green, P A, Berge, K G, and Sprague, R G, *Diabetes*, 1968, **17**, 385.
- <sup>19</sup> Condon, J R, et al, *British Medical Journal*, 1973, **4**, 423.
- <sup>20</sup> Tomkin, G H, *British Medical Journal*, 1973, **3**, 673.
- <sup>21</sup> Arvanitakis, C, Lorenzsonn, V, and Olsen, W A, *Journal of Laboratory and Clinical Medicine*, 1973, **82**, 195.
- <sup>22</sup> Trowell, H C, *Diabetes*, 1975, **24**, 762.

## Rickets in Asian immigrants

Rickets virtually disappeared from Britain in 1945 after milk was fortified with vitamin D. Nevertheless, it then reappeared in the early 1960s after fortification was stopped because of deaths from hypercalcaemia. Rickets and osteomalacia are now found mostly in Asian immigrants to Britain,<sup>1</sup> though their extent and severity are not known accurately. Florid rickets is seen most in Asian toddlers and adolescents, but congenital rickets also occurs, and osteomalacia is found in Asian adults of any age, but particularly pregnant women. Some studies have yielded prevalences of biochemical rickets of up to 50% in Asians<sup>2</sup> and a prevalence of clinical rickets of 5%.<sup>3</sup>

The Department of Health and Social Security does not agree that the problem is as serious as these figures suggest. Dr J G Ablett told the symposium on rickets held at the King's Fund Centre last week that he believes rickets is no more common among Asian immigrants to Britain than those remaining in the Indian subcontinent and that it should slowly disappear here when immigrants adapt to British conditions. Data from the Hospital Inpatient Enquiry and the Hospital Activity Analysis had shown, he said, that the crude admission rate to hospital for rickets has declined in the same time that the Asian population has more than doubled. Moreover, in his survey of general practitioners in areas with a high Asian population, most of them had thought that the incidence of rickets was decreasing.

The conviction that rickets in Asians is not as large a problem as had been suggested and that its frequency is decreasing rather than increasing seems to have been responsible for the recommendation by the working party on fortification of foods with vitamin D against fortification of chapaty flour. The

working party has also been worried that some sensitive adults would be in danger of developing vitamin D intoxication since young men eat much more chapaty flour than children. The conference heard that many researchers and public health officials were unhappy that the policy of fortification has been discarded: the responsibility for dealing with the problem in areas with a high Asian population is now on the local health authorities.

Clearly, at least in the short term, the diet of Asians must be supplemented with vitamin D. This can be achieved by encouraging Asians to eat more foods rich in vitamin D, by supplementing their diet with vitamin D preparations, or by a combination of the two methods. In areas of India where rickets is prevalent WHO policy is to supplement the diet, but this is seen as only a short-term policy; long term the policy is to encourage dietary change. In India most people eat traditional foods and avoid rickets; surely the lack of sunlight in Britain is not so severe as to make it impossible for a well-balanced traditional diet to be sufficient for Asian immigrants. For immigrants to change to a traditional British diet, which some claim may play a part in our high incidence of coronary artery disease, as well as certain gut diseases, might be to escape from the frying pan into the fire.

The Glasgow public health authorities are unimpressed with attempts to encourage Asians to change their diet and instead have started persuading them to take free vitamin D supplements. At the beginning of January they launched an energetic programme which combines training health professionals and reaching the Asian population through community groups, schools, newspapers, leaflets, films, and other methods. Other areas, including Leicestershire and Lancashire, have placed more emphasis on dietary change. The problem of a low intake of vitamin D is only one part of the nutritional problem among Asian immigrants: their intake of other fat-soluble vitamins and iron and protein may also be deficient. Thus all these various schemes for combating rickets and osteomalacia among Asian immigrants need careful evaluation if we are not to waste time and money.

<sup>1</sup> *British Medical Journal*, 1976, **3**, 444.

<sup>2</sup> Ford, J A, et al, *British Medical Journal*, 1972, **2**, 677.

<sup>3</sup> Goel, K M, et al, *Lancet*, 1976, **1**, 1141.

## Sir Ludwig Guttman at 80

The abilities of Sir Ludwig Guttman are diverse, which is why he has played the major part in dramatically improving (in only 30 years) the prognosis of paraplegia. Firstly, he has the gift of vision. In 1944, as the second world war produced a crop of young paraplegics, he was appointed director of the first spinal unit at Stoke Mandeville Hospital, and was able to see beyond the conventional view of the hopelessness of paraplegia.

Secondly, he is a gifted and scrupulous clinician and scientist. In a classic paper of 1945, reprinted in a special issue of *Paraplegia* honouring his 80th birthday,<sup>1</sup> he laid down the principles of managing paraplegia that prevail today and, doubtless, will continue to do so. He emphasised the importance of early active treatment that could best be undertaken in a special unit. Recognising that most paraplegics died from the results of pressure sores and urinary infections, he pioneered frequent turning and scrupulous skin cleaning. He also advocated intermittent urethral catheterisation and bladder