Invited commentary

Choosing your carbohydrates to prevent diabetes

Type 2 diabetes is on the increase. By the year 2010 the number of new cases is expected to double to reach 270 million, of whom 4 million will be in the UK. Until relatively recently, type 2 diabetes was perceived as a disease of middle-aged and older people, but this form of diabetes has now been reported in children in several countries worldwide including the UK (Ehtisham et al. 2000; Fagot-Campagna et al. 2001). The first reported cases in the UK were among children of South Asian or Arabic origin (Ehtisham et al. 2000) but the recent report of type 2 diabetes in four white UK teenagers (Drake et al. 2002) set alarm bells ringing (Dyer, 2002). It is well recognised that type 2 diabetes has a very strong association with obesity (National Audit Office, 2001) and it is unlikely to be a coincidence that all the UK cases of type 2 diabetes were overweight or obese (Ehtisham et al. 2000; Drake et al. 2002). Economic growth is fuelling a rapid demographic and nutritional transition in many developing countries resulting in increased prevalence of obesity in children as well as adults (Uauy et al. 2001), so that the developing world is likely to have to bear the bigger part of the burden of type 2 diabetes. Prevention is usually better (and cheaper) than cure, so what can be done to stem the tide of type 2 diabetes?

Impaired glucose tolerance (IGT) is diagnosed following a 75 g oral glucose tolerance test in which the 2 h blood glucose value is intermediate between the cut-off for normality, i.e. 7.0 mmol/l, and the diabetic cut-off, i.e. 11.1 mmol/l (American Diabetes Association, 1997). It is believed that IGT is an intermediate stage in the natural history of type 2 diabetes (Saad et al. 1991) and it is a central element of the insulin resistance syndrome known to be associated with a high risk of macrovascular disease (World Health Organization, 1999). Individuals with IGT have a 3–9 % per year greater than normal risk of developing type 2 diabetes (Edelstein et al. 1997), but progression to diabetes is not inevitable. Major risk factors for progression from IGT to type 2 diabetes include obesity, abdominal fat distribution, family history of diabetes, physical inactivity, high-fat diet, insulin resistance and increasing age. Data from NHANES III in the USA suggest that almost 7 % of the adult population has IGT, with similar rates for men and women but higher rates among non-Hispanic blacks and Mexican-Americans than among non-Hispanic whites (Harris et al. 1998).

IGT is an attractive population group in which to test diabetes prevention strategies and recent studies provide good evidence that lifestyle modifications and/or drugs can delay or prevent the conversion of IGT to type 2 diabetes. For example, in the Da Qing study in China, a randomised controlled intervention with diet and/or exercise in men and women (mean age 60 years) who had IGT resulted in significantly reduced risk of diabetes after 6 years than in controls (Pan et al. 1997). Middle-aged Finnish IGT subjects who underwent a combined intervention with diet and exercise had a 58 % reduction in risk of type 2 diabetes after 4 years (Tuomilehto et al. 2001). Both intervention studies aimed to lower body weight in participants with BMI > 25 kg/m² in addition to reducing intakes of fat.

Among of the major aims in the management of IGT are prevention of worsening of insulin sensitivity and other cardiovascular risk factors with the longer-term hope of reducing the risk of progression to diabetes. It is widely accepted that a lower-fat diet may contribute to achieving these aims, but there is considerable controversy as to whether higher-carbohydrate diets could be contra-indicated because of their association with raised plasma triacylglycerol concentrations and exacerbation of the metabolic abnormalities of insulin resistance (Reaven, 1997). It is highly likely that not all carbohydrates behave similarly in this respect. For example, there is moderately consistent evidence that high intakes of sucrose or fructose may have adverse effects on insulin sensitivity particularly in those who are genetically susceptible (Daly et al. 1997). In the study reported in this issue of the British Journal of Nutrition, Wolever & Mehling (2002) hypothesised that the rate and extent of digestion of carbohydrates in the small bowel will determine the rate at which glucose appears in the blood stream and so the need for the pancreas to secrete insulin. They compared advice to select high-carbohydrate diets with a low or high glycaemic index (GI; surrogate for rate of glucose uptake from the gut) with advice to select a low-carbohydrate–high-monounsaturated fatty acid diet on aspects of insulin action over a 4-month period in men and women with IGT. After adjusting for baseline values, the improvement in the glucose disposition index was significantly greater for those on the high-carbohydrate–low-GI diet than for those on the low-carbohydrate–high-GI monounsaturated fatty acid diet and there was no adverse effect on plasma triacylglycerol concentrations. Wolever & Mehling’s (2002) study underlines the importance of considering not only the amount but also the nature of the carbohydrate in the diets of those with IGT. It appears likely that high-carbohydrate–low-fat diets will have no adverse effects on those with IGT if the sources of that
carbohydrate are relatively unprocessed starchy staples rich in slowly digested starch (Englyst et al. 2000). Indeed, such low-energy-dense diets may be helpful in reducing body fatness (Wolever & Mehling, 2002), which is likely to be a major determinant of reduced risk of progress to diabetes.

Young children who are overweight and obese have impaired insulin sensitivity and are at enhanced risk of developing type 2 diabetes (Young-Hyman et al. 2001). Public health measures to tackle this problem will need to be multi-factorial with increasing physical activity as a major target. However, it will also be important to encourage prudent eating habits, which should include ample intakes of low-fat, starchy staples with low GI. The search for genetic determinants of type 2 diabetes continues unabated (see e.g. Carlsson et al. 2001; Luo et al. 2001) and it is probable that there will be important diet: gene interactions in the aetiology of this disease, understanding of which may allow better targeting of dietary and other lifestyle advice in the longer term.

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References
Reaven GM (1997) Do high carbohydrate diets prevent the development or attenuate the manifestations (or both) of syndrome X? A viewpoint strongly against. Current Opinion in Lipidology 8, 23–27.