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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 mM, and the diabetic cut-off, i.e. 11·1 mM (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 $> 25\,kg/m^2$ 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-monosaturated 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

American Diabetes Association (1997) Report of the Expert Committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* **20**, 1183–1197.

Carlsson M, Orho-Melander M, Hedenbro J & Groop LC (2001) Common variants in the beta2-(Gln27Glu) and beta3-(Trp64Arg)-adrenoreceptor genes are associated with elevated serum NEFA concentrations and type II diabetes. *Diabetologia* **44**, 629–636.

Daly ME, Vale C, Walker M, Alberti KGMM & Mathers JC (1997) Dietary carbohydrates and insulin sensitivity: a review of the evidence and clinical implications. *American Journal of Clinical Nutrition* **66**, 1072–1085.

Drake AJ, Smith A, Betts PR, Crowne EC & Shield JPH (2002) Type 2 diabetes in obese white children. *Archives of Disease in Childhood* **86**, 207–208.

Dyer O (2002) First cases of type 2 diabetes found in white UK teenagers. *British Medical Journal* **324**, 506.

Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, Haffner SM, Pettitt DJ, Sorkin JD, Muller DC, Collins VR & Hamman RF (1997) Predictors of progression from impaired glucose tolerance to NIDDM. *Diabetes* **46**, 701–710.

Ehtisham S, Barrett TG & Shaw NJ (2000) Type 2 diabetes mellitus in UK children – an emerging problem. *Diabetic Medicine* **17**, 867–871.

Englyst KN, Hudson GJ & Englyst HN (2000) Starch analysis in food. In *Encyclopedia of Analytical Chemistry*, pp. 4246–4262 [RA Meyers, editor]. Chichester: John Wiley & Sons Ltd.

Fagot-Campagna A, Narayan KMV & Imperatore G (2001) Type 2 diabetes in children exemplifies the growing problem of chronic diseases. *British Medical Journal* **322**, 377–378.

Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer HM & Byrd-Holt DD (1998) Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. *Diabetes Care* **21**, 475–476.

Luo TH, Zhao Y, Li G, Yuan WT, Zhao JJ, Chen JL, Huang W & Luo M (2001) A genome-wide search for type II diabetes susceptibility genes in Chinese Hans. *Diabetologia* **44**, 501–506.

National Audit Office (2001) *Tackling Obesity in England. Report by the Comptroller and Auditor General HC220*. London: The Stationery Office.

Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH & Howard BV (1997) Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* **20**, 537–544.

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.

Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Charles MA & Bennett PH (1991) A two-step model for the development of non-insulin-dependent diabetes. *American Journal of Medicine* **90**, 229–235.

Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M, Anuloa S, Capaitis Z, Moltchanov V, Hakumaki M, Mannelin M, Martikkala V & Sundvall J (2001) Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine* **344**, 1343–1350.

Uauy R, Albala C & Kain J (2001) Obesity trends in Latin America: transiting from under- to overweight. *Journal of Nutrition* **131**, 893S–899S.

Wolever TMS & Mehling C (2002) High-carbohydrate/low–glycaemic-index dietary advice improves glucose disposition index in subjects with impaired glucose tolerance. *British Journal of Nutrition* **87**, 477–487.

World Health Organization (1999) *Definition, Diagnosis and Classification of Diabetes mellitus and its complications. Report of a WHO Consultation. Part 1. Diagnosis and Classification of Diabetes Mellitus*. Geneva: WHO.

Young-Hyman D, Schlundt DG, Herman L, De Luca F & Counts D (2001) Evaluation of the insulin resistance syndrome in 5- to 10-year-old overweight/obese African-American children. *Diabetes Care* **24**, 1359–1364.