

diabetes is a growing problem in the population: according to Diabetes UK there are 3% (1.8m) diagnosed cases (approximately 250,000 with type 1 and over 1.5m with type 2) and another estimated 0.75-1m undiagnosed cases of type 2 diabetes. No statistics are available for the athletic population

diabetes:

should athletes be worried? how to balance blood sugar levels

report: Kathryn Bistany

what is diabetes?

Diabetes is a syndrome or group of symptoms arising from failure to regulate the metabolism of glucose by means of the pancreatic hormone, insulin. This occurs due to a lack of insulin because the pancreas does not produce enough, fails to produce any or the body fails to make proper use of the insulin that is available. Diabetes is classified as insulin-dependent (type 1) and non-insulin-dependent (type 2). This paper will focus on the latter and will ignore any genetic predisposition to the disease.

the glycaemic index and diabetes

The Glycaemic Index (GI) can be considered as a measure of carbohydrate quality. It measures the postprandial (after a meal) glycaemia (plasma glucose) raising potential of a single food by expressing the rise in glycaemia in response to a 50g available carbohydrate portion of that food as a percentage of the rise in response to a 50g available carbohydrate portion of a reference food (white bread or glucose).

Foods high on the GI result in a sharp rise of plasma glucose, with a high demand for insulin, followed by a more or less rapid fall of glucose. Foods that are low to moderate on the GI produce a slower rise, with a lower demand for insulin, and a more gradual decline in plasma glucose.^{1,2}

Those in favour of carbohydrate quality, argue that GI is a robust measurement, predicts the relative glycaemic response to mixed meals and is easy to follow and implement. In contrast, opponents who favour giving priority to carbohydrate quantity argue that GI is highly variable, not physiological, cannot reliably predict mixed meal responses and is difficult to learn or follow.³

Despite some opposition to low-GI intervention in type 2 diabetes, the interventions are clinically efficacious in diabetes therapy over

the mid to long-term. The Canadian Diabetes Association, Diabetes Australia, Diabetes UK and the European Association for the Study of Diabetes all support the application of the GI concept in the management of diabetes.

insulin resistance

Insulin resistance, a component of the Insulin Resistance Syndrome, also known as Syndrome X and the Metabolic Syndrome, is associated with type 2 diabetes.^{4,5,6} No statistics for insulin resistance are available in the UK, although, according to Diabetes UK, a national register may be set up in the future.

Obesity is the most significant factor leading to insulin resistance with visceral obesity having a particularly strong negative correlation.⁵ It can be reversed with diet modification based on a low fat intake and limiting refined carbohydrates without the need of caloric restrictions. Physical activity is an important factor in reversing the problem.⁷

Mechanisms leading to insulin resistance are unclear, although the abnormal accumulation of certain fats in the liver (hepatic steatosis) is a contributing factor.⁸

In a study by Pan et al⁹ skeletal muscle triglyceride (mTG) appeared to be another important factor in predicting insulin resistance. Trained athletes and animals show the same or higher levels of muscle triglycerides as sedentary controls but have improved insulin action. The authors postulated that this could be due to the distribution of triglyceride. Endurance exercise increases both the mitochondrial volume and distribution in skeletal muscles. In trained dogs, mitochondria appear virtually in direct contact with triglyceride droplets whereas no such association with mitochondria was found in untrained animals. As a result, trained individuals may have an improved ability to mobilise fats.

Research into sucrose and fructose on animals has consistently shown that high sucrose and fructose diets decrease insulin sensitivity.⁵ Studies on humans have been inconsistent.

In a large cohort study by Janket et al¹⁰ 38,480 initially healthy postmenopausal women were followed for an average of six years. The researchers accrued 918 incident cases of type 2 diabetes but found no definitive influence of sugar intake on the risk of developing type 2 diabetes. It was noted, however, that the median follow-up time of six years might not have been long enough to detect a very subtle relationship between sugar intake and incidence of type 2 diabetes.

Assessment on humans is thought to be more complicated because of other factors affecting insulin sensitivity. Some studies found that those consuming a diet consisting of large amounts of sweets and desserts were at increased risk of developing diabetes. However, the diet also included high amounts of saturated fats (red meat, fries, dairy products) which is known to be associated with decreased insulin sensitivity.⁵

No studies have shown a negative effect of sucrose on insulin sensitivity. One explanation for this lack of correlation could be that recruitment of volunteers for nutrition studies is notoriously difficult and many studies have a young or a highly health-orientated population. Both groups are likely to be physically active. Given the strength of the positive influence of physical exertion on insulin sensitivity, such persons are inclined to be resistant to the negative effects of diet. However, this does suggest that the promotion of physical activity may have a greater influence on insulin sensitivity than diet.⁵

Another possible explanation is that the GI concerns only the first two hours of the postprandial period. It is postulated that a GI defined by a 4-6 hour postprandial period would alter the ranking of sucrose in a GI table to a higher level.^{5,11} Neither sucrose (a disaccharide: glucose bonded to fructose) nor fructose (a monosaccharide) are high on the GI.

Studies based on high fructose versus high glucose diets have shown that the high fructose diets produce an increase in plasma triacylglycerol, plasma cholesterol, VLDL and LDL cholesterol concentrations, all of which are a risk factor in cardiovascular disease.^{12,13,14} In addition, some of these effects were seen in men but not women.¹³ The reason for this difference is not clear. Although not all studies are consistent with these findings, the positive data cannot and should not be dismissed as it may be of considerable clinical importance. It is also important to note that some individuals are more sensitive to fructose than others.¹⁴

the risk for athletes

Are athletes at risk of developing type 2 diabetes as a result of their high intake of fructose, sucrose and high glycaemic foods? Although the scientific evidence to-date does not support this notion, athletes may be at risk of developing insulin resistance which is associated not only with diabetes but also with coronary heart disease, hypercholesterolaemia, hypertension, dysglycaemia, osteoarthritis and impaired glucose tolerance.^{2,4,5,6}

An over-consumption of refined carbohydrates, over-processed foods, saturated fats and processed vegetable fats are all associated with insulin resistance.^{6,7,11,15} The majority of adult athletes we have consulted to-date, over-consume the above with the possible exception of saturated fats. However all our adolescent athletes consumed large amounts of saturated fats.

Although some athletes are becoming more informed on the importance of nutrition for both their long-term health and their performance, there are still a large number who are uninformed

or misinformed on nutritional issues. Particularly distressing is the lack of knowledge amongst adolescent athletes, which needs to be urgently addressed, not only by nutritionists and dieticians, but also by coaches and parents.

One procedure that can be immediately implemented by everybody is that of chewing our food thoroughly and eating more slowly: it appears that prolonging absorption time by increasing the length of time to complete a meal, consuming smaller and more frequent meals or drinking a beverage over a prolonged period of time all improve glucose tolerance.¹⁵

In summary, to minimise the risk of insulin resistance, the following points should be adhered to:

- limit sugars and high GI carbohydrates to just before, during and just after exercise
 - at other times, consume a large variety of foods avoiding repeating the same food on any one day
 - try to include colourful foods at every meal
- eat fresh rather than ready-made as often as possible
- limit all saturated fats found in dairy products and fatty meats
 - avoid fried foods
 - avoid junk foods
 - dilute fruit juices. ^{fn}

references

- Sheard NF, Clark NG, Brand-Miller JC, Franz MJ, Pi-Sunyer FX, Mayer-Davis E, Kulkarni K, Geil P (2004) Dietary carbohydrate (amount and type) in the prevention and management of diabetes. *Diabetes Care* 27(9):2266-71.
- Salmerin J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC (1997) Dietary fiber, glycaemic load, and risk of NDDM in men. *Diabetes Care* 20(4): 545-550.
- Sievenpiper JL, Vuksan V (2004) Glycemic index in the treatment of diabetes: the debate continues. *Journal of the American College of Nutrition* 23(1): 1-4.
- Festa A, Hanley AJG, Tracy RP, D'Agostino R, Haffner SM (2003) Inflammation in the prediabetic state is related to increased insulin resistance rather than decreased insulin secretion. *Circulation* 108(15):1822-30.
- Daly M (2003) Sugars, insulin sensitivity, and the postprandial state. *The American Journal of Clinical Nutrition* 78(4): 865S-872S.
- Liu S, Manson JE, Buring JE, Stampfer MJ, Willett WC, Ridker PM (2002) Relation between a diet with a high glycemic load and plasma concentrations of high-sensitivity C-reactive protein in middle-aged women. *The American Journal of Clinical Nutrition* 75:492-498.
- Roth JL, Clohisy M (2002) The metabolic syndrome: where are we and where do we go? *Nutrition Reviews* 60(10; part 1): 335-7.
- Montminy M & Koo SH (2004) Outfoxing insulin resistance? *Nature* 432(7020):958-959.
- Pan DA, Lillioja S, Kriketos AD, Milner MR, Baur LA, Bogardus C, Jenkins AB, Storlien LH (1997) Skeletal muscle triglyceride levels are inversely related to insulin action. *Diabetes* (46): 983-988.
- Janket SJ, Manson JE, Sesso H, Buring JE, Liu S (2003) A prospective study of sugar intake and risk of type 2 diabetes in women. *Diabetes Care* 26(4):1008-15.
- Daly ME, Vale C, Walker M, Littlefield A, Alberti KGMM, Mathers JC (1998) Acute effects on insulin sensitivity and diurnal metabolic profiles of a high-sucrose compared with a high-starch diet. *American Journal of Clinical Nutrition* 67(6): 1186-96.
- Swanson JE, Laine DC, Thomas W, Bantle JP (1992) Metabolic effects of dietary fructose in healthy subjects. *The American Journal of Clinical Nutrition* 55(4):851-856.
- Bantle JP, Raatz SK, Thomas W, Georgopoulos A (2000) Effects of dietary fructose on plasma lipids in healthy subjects. *The American Journal of Clinical Nutrition* 72(5):1128-34.
- Hollenbeck CB (1993) Dietary fructose effects on lipoprotein metabolism and risk for coronary artery disease. *The American Journal of Clinical Nutrition* 58(5 Suppl):800S-809S.
- Hung T, Sievenpiper JL, Marchi A, Kendall CWC, Jenkins DJA (2003) Fat versus carbohydrate in insulin resistance, obesity, diabetes and cardiovascular disease. *Current Opinion in Clinical Nutrition and Metabolic Care* 6(2):165-176.