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Nutrition Discussion Forum

Approaches to health via lowering of postprandial glycaemia

A recent issue of the *British Journal of Nutrition* reflects a marked change in the importance we now attach to the role of dietary carbohydrates in both managing and retarding the development of non-communicable diseases, with four commentaries: *Choosing your carbohydrates to prevent diabetes* (Mathers, 2002); *Low carbohydrate does not mean low glycaemic index!* (Wolever, 2002); *Diet, satiety and obesity treatment* (Tremblay *et al.* 2002); *Handbook of dietary fibre* (Cummings, 2002). Among these there are important messages on public health and expressions of hope for the future. However, the commentaries on starchy staples and glycaemic index (GI) have implications beyond diabetes and may be more easily understood in a broader context that aims for low postprandial glycaemia, eventually by more than one approach based on diet together with weight restriction and/or reduction.

Certainly one can welcome a possible role for low-GI-starch foods in the prevention of type 2 diabetes mellitus (DM), as suggested by Mathers (2002). The weight of published evidence to date, nevertheless, is in diabetic subjects (not pre-diabetic or glucose-intolerant subjects), is based largely on intervention trials with diets characterised analytically as low-GI-carbohydrate (not necessarily lower-GI-starch as sometimes intended) and measurement of biochemical risk factors (not clinical outcomes, either microvascular or macrovascular). Advice to consume low-GI-starch foods can also elevate intake of sugars (G Livesey, unpublished results), many of which have GI values less than common starch-rich staple foods. Interestingly, there is anecdotal evidence in favour of using low-GI-starch foods in diabetic subjects in a non-western setting, where the likelihood of a spontaneous intake of more sugars would seem less: physicians of ancient India treated DM with barley and Bengal gram (Kapur & Kapur, 2001), which we now know to be amongst the lowest-GI-starch foods available. Efficacy of such treatment on clinical outcome was unreported and now possibly unknown without further study.

There is a scarcity of published dietary intervention studies with low-GI foods that examine prevention of clinical disease in subjects at risk, such as those who are glucose intolerant. However, there appears to be improved pancreatic β-cell function with low-GI-carbohydrate diets among some subjects who are glucose intolerant (Wolever & Mehling, 2002). Moreover, in the STOP-NIDDM randomised trial the glycaemic response to diet was reduced by slowing digestion with an α-glucosidase inhibitor (acarbose), and there were both significantly fewer conversions of glucose-intolerant to type 2 DM patients and more reversions to normal (Chiasson *et al.* 2002). Mathers' (2002) comments are presumably a call to further arm us with similar knowledge as may be got from more research on diets. Hence, however good the outlook, we do not know yet the level of disease reduction that will come from intervention with low-GI diets (let alone low-GI-starch foods).

Nevertheless, the future burden of disease from diabetes (obesity, stroke and heart disease too, which may be greater problems) is expected to overburden government (and private) health budgets. Waiting for conclusive proof on the magnitude of efficacy of low-GI-carbohydrate foods on clinical end points may therefore be unwise, given the suggested absence of risk from reduced postprandial glycaemia (American Diabetes Association, 2001), a positive outlook (c.f. Canadian Diabetes Association, 2000; Buykens *et al.* 2000, 2001) and prospective evidence from epidemiological studies that low-GI diets (again not uniquely low-GI-starch diets, though inclusive of whole-grain cereals) appear to lower the advent of type 2 DM, CHD and possibly stroke (all disease outcomes rather than biochemical markers of disease) (Salmerón *et al.* 1997*a*,*b*; Liu *et al.* 2000*a*,*b*, 2002). Further, we have to consider whether reduced glycaemia might benefit a majority of the population (Khaw *et al.* 2001) and not just those believed to be at greatest risk. The last paper (Khaw *et al.* 2001) indicates glycosylated haemoglobin (HbA1c) is an important prospective marker of macrovascular disease. This, in addition to microvascular diseases seen in diabetics, and the need to control glycaemia more broadly than for diabetes prevention, supports earlier epidemiological work on glycaemic index (or glycaemic load) and CHD.

Interpretation of the epidemiological evidence is difficult. It is unclear whether the advent of type 2 DM and CHD is linked to the GI of the carbohydrate ingested or to glycaemic load (i.e. GI × carbohydrate intake). The latter seems to hold the stronger association with both advent of diseases (Salmerón *et al.* 1997*a*,*b*; Liu *et al.* 2000*a*) and C-reactive protein, a marker for CHD risk (Liu *et al.* 2002). Yet, this might not have been expected: as with low-carbohydrate intake, low glycaemic load could imply higher fat intake, causing damage that obscures the true strength of benefit from low glycaemia. Such obscuring might explain why sometimes (e.g. Meyer *et al.* 2000) the association between disease and glycaemia does not always manifest itself strongly.

Wolever (2002) rightly points out that low GI (as applied to carbohydrate) does not mean the same as low carbohydrate. GI describes carbohydrate quality not quantity, and so low GI cannot imply low-fat intake. Indeed, low GI can imply any level of fat intake, which makes it important to specify that healthy diets are those that are of high intake of low-GI carbohydrates. However, over-focusing on GI limits our scope of vision. We may: (1)

fail to recognise the importance of low postprandial glycaemia as a real health benefit that can outweigh other health risks; (2) become overcautious in using all forms of carbohydrate (even damaging high-GI starch) to ensure fat as a health risk is displaced from the diet (here meaning total fat because displacement is on an energy basis without discrimination over the type of fat) (c.f. American Diabetes Association, 2002); (3) fail to consider or follow up other dietary approaches to lowering postprandial glycaemia. Thus, Tremblay *et al.* (2002) rightly point out that postprandial glycaemia can be reduced by methods other than replacing one carbohydrate source with another of lower GI value; their example study was exchange of protein and carbohydrate (Dumesnil *et al.* 2001).

Several possibilities for lowering postprandial glycaemia exist: should absence of benefit be encountered, it might be traced to co-modification of other obscuring risk factors, outlined as follows.

Some carbohydrate foods that elicit a high glycaemic response (high GI) in peripheral blood may be replaced by carbohydrate foods that elicit a lower response (low GI). Applied to starch-rich staple foods, this advice also increases the intake of unavailable carbohydrates (dietary fibre), which might also benefit health. Since the total benefit of low-GI carbohydrate is conditional on maintaining a presence of carbohydrate to displace fat from the diet, it makes no sense for low-GI-food recipes to be rich in fat. To make life more interesting and easier, advice to consume more low-GI-carbohydrate foods brings exposure to a wider variety of nutritious foods (Wolever, 1997) and can reduce conflict in families that include a diabetic patient (Gilbertson *et al.* 2001). For a useful trail of analytical reviews, as opposed to commentaries, see Miller (1994), Wolever (1997), Frost & Dornhorst (2000) and Jenkins *et al.* (2002). There seems to be little risk from such advice.

Some food-ingredient carbohydrates might be partly replaced by alternatives such as lower-energy, low-glycaemic sugars, polyols and non-digestible polysaccharides. These tend to be used as carbohydrate replacers when low-GI starches are not suitable; again, risk to health is unknown and gastrointestinal disturbances are minimal when consumed in realistic amounts. This particularly applies to sugar-free sweets when sweets are desired, and to snack foods and fruit preserves when lower energy, lower glycaemia and lower insulinaemia are desirable. It might be thought that the lower-energy-value foods, due to unavailability of the carbohydrate present, would be compensated by other energy sources at later meals. It is notable, therefore, that intervention studies in free-living subjects indicate unavailable carbohydrate is substantially more effective at reducing body weight, on a weight-for-weight basis, than replacing dietary fat with carbohydrate (G Livesey, unpublished results), such benefit being limited by the quantity that can be readily consumed. Both energy restriction and body weight reduction improve prospects of health (Christiansen *et al.* 2000).

Viscous polysaccharides might be used to slow absorption and reduce postprandial glycaemia; again, there is little health risk when foods are adequately formulated.

Some high-GI carbohydrate might be replaced by protein (e.g. Tremblay *et al.* 2002), particularly vegetables or fish, but not meat. In addition to improved atherogenic risk factors after 1 week of treatment, the replacement of starch in bread with protein for 3 months showed improved carbohydrate tolerance and decreased HbA1c in type 2 DM patients (Stilling *et al.* 1999). Improvement in insulin sensitivity has also been seen in diabetic patients with kidney disease (Gin *et al.* 2000), though not consistently so (Stefikova *et al.* 1997). Reduced food intake (energy, glycaemic load and saturated fat) (Dumesnil *et al.* 2001) may partly explain the improved biochemistry even prior to body-weight reduction (or *visa versa* or both with possible spiralling improvement). Certain comments may be warranted in regard to high-protein diets: (1) while higher protein intake would be contraindicated by nephropathy, on a population or other group basis, the potential for use in prevention of type 2 DM and prevention and intensive management of CHD and possibly obesity is substantial, and so the approach merits exploration to establish longer-term efficacy and limit the risks, both for high-risk groups and in population samples; (2) the results at present might seem to support the hypothesis of McCarthy (2000) that protein–carbohydrate interactions promote insulin secretion and obesity, although raising the concentration of one putative interactant while lowing the concentration of the other provides no information about the existence or possible importance of the interaction; (3) replacement of high-GI carbohydrate with protein does not automatically mean a McDougall type or similar ketotic diet where carbohydrate is highly restricted. An intake requirement for carbohydrate (which could be low-GI carbohydrate) for the purpose of optimal health nevertheless may be helpful, though setting the value is problematic; (4) knowledge about high-protein diets is limited, and so protein should not for the present replace low-GI carbohydrate as the preferred means to displace high-GI carbohydrates. Eventually, protein might be seen as an energy source to help replace some saturated fat; (5) risks of higher protein (20–40 % energy) possibly include adequacy of Ca retention, kidney stone formation due to lower citrate excretion from carbohydrate (Reddy *et al.* 2002) and possibly higher advent of type 1 DM.

Some high-GI carbohydrates could be replaced by monounsaturated fats (Garg, 1998; Luscombe *et al.* 1999; American Diabetes Association, 2002). However, this might be objectionable when low-GI carbohydrates can be used instead, because the higher energy density might elevate energy intake and body weight (for which there is little evidence for monounsaturated fats) and reduced pancreatic β-cell function among glucose-intolerant (and potentially type 2 diabetic) subjects (Wolever & Mehling, 2002). Monounsaturated fats may have a role to replace saturated fats with advantage as the latter may promote insensitivity to insulin, at high intakes at least (Vessby *et al.* 2001), and so reduce long-term glycaemic control. Combinations of high-monounsaturated fats (replacing particularly C_{12}, C_{14} and C_{16} fats) and low-GI carbohydrate diets (replacing high-GI carbohydrate) for optimal metabolic control and health deserves examination. In some food products, replacement of saturated fats with low-energy, low-fat alternatives has a place.

It is possible to replace high-GI carbohydrate with saturated fats (but do not do it!), and so reduce glycaemia acutely. This approach would certainly be objectionable for reasons related to body weight, CHD and diabetes control. More specifically, in respect of postprandial glycaemia it would lead to a loss of insulin sensitivity and impairment of β-cell function, so increasing long-term postprandial glycaemia.

Restriction of food intake to reduce body weight inevitably increases the chance of lowering carbohydrate intake and so postprandial glycaemia. The risks appear to be minimal other than for possible concern for adequate Ca, Fe, Mg and Zn intakes, and for growth in children.

Increased physical activity may also reduce postprandial glycaemia via demand for glucose and improved insulin sensitivity (Ratzmann *et al.* 1981; Yamanouchi *et al.* 1995). There is an acute risk of hypoglycaemia in unprepared diabetics.

A theoretical improvement in glycaemia might be expected from nibbling. In the free-living situation, this may be obscured by a sedentary social context such as watching television. However, redistribution of food intake throughout the day would neither affect glycaemic index nor glycaemic load from food. Little benefit is seen in type 2 DM on blood glucose concentrations (Arnold *et al.* 1997), although long-term studies, particularly on HbA1c, are absent. This should not overshadow improvement in the blood lipid profile (Jenkins *et al.* 1989) and possibly body weight (Kirk, 2000; Westerterp-Plantenga *et al*. 2002).

When it is critical to reduce postprandial glycaemia, it can also be achieved partly by inhibitors of carbohydrate digestion, the drug equivalent of simultaneously lowering both carbohydrate GI and availability (Brooks *et al.* 1998), and by appropriately designed insulin and drug therapy (Kapur & Kapur, 2001; Kelley, 2002). General risks from drug-based approaches (other than inhibitors of digestion) occur when doses exceed need, and include more episodes of hypoglycaemia, body-weight gain and greater cardiovascular risk dependent on the drugs used.

The list is not exhaustive and the examples have yet to be fully or adequately researched. Nevertheless, it is clear that reduced postprandial glycaemia can be attempted in a number of different ways, with overall benefit likely to be limited by the associated risks accompanying the method(s) chosen. It does seem that postprandial glycaemic reduction offers greater preventive and therapeutic potential than does reduction of glycaemic index of available carbohydrates alone, but also greater risks when achieved by some approaches. Given the severity of these non-communicable diseases, their prevalence, expected overall rising trend and limited health budgets, we may have to welcome several approaches to dietary therapy with the goal of reduced postprandial glycaemia, allowing each approach and combination of approaches to find appropriate effective niches, from acute treatment to near lifelong exposure.

Physical activity, reduced food intake, limitation of the fat content of the diet and raised dietary fibre intake are established preventative and therapeutic strategies in the public domain with broadly adequate (though too limited and inaccurate) food labelling. At present however, the public are not informed about how carbohydrate-containing foods affect postprandial glycaemia, or about ways to limit postprandial glycaemia by choice of foods and food preparation methods. With certain foods (e.g. potatoes, rice; Foster-Powell *et al.* 2002) this is not easy as there is great variety and preparation methods markedly affect the value. However, these need not be seen as obstacles, rather they are opportunities for improvement of foods and diets.

An increasing realisation of the relationship between HbA1c, dietary glycaemic load and non-communicable diseases is truly very remarkable given that just over a decade ago starch in particular (and perhaps available carbohydrate in general) seemed as though it was just a make-weight, health-bland source of energy: good, it seemed, only because it kept fat out of the mouth (Department of Health, 1990). Postprandial glycaemia is now emerging as one of the major risks that the public encounters; meanwhile, consumers are neither empowered via food labelling or local authoritative food tables nor advised to protect themselves. In addressing this problem, we ought not now to lose sight of other possible dietary approaches and we ought to consider a role for lower postprandial glycaemia as contributing to the benefits of both increased physical activity and energy restriction.

Geoffrey Livesey
Independent Nutrition Logic
Pealerswell House
Wymondham
Norfolk NR18 0QX
UK

References

Arnold L, Mann JI & Ball MJ (1997) Metabolic effects of alterations in meal frequency in type 2 diabetes. *Diabetes Care* **20**, 1651–1654.

American Diabetes Association (2001) Post-prandial blood glucose [a consensus statement]. *Diabetes Care* **24**, 775–778.

American Diabetes Association (2002) Evidence based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care* **25**, Suppl. 1, s149–s198.

Brooks B, Molyneaux Zilkens R, Ross G & Yue DK (1998) The use of acarbose in Type 2 diabetes patients in secondary failure: effect on glycaemic control and diet-induced thermogenesis. *Diabetes Research and Clinical Practice* **42**, 175–180.

Buyken AE, Toeller M, Heitkamp G, Irsiglert C, Hollert F, Santeusanio F, Stehle P & Fuller JHEURODIAB IDDM Complication Study Group (2000) Carbohydrate sources and glycaemic control in type 1 DM. *Diabetic Medicine* **17**, 351–359.

Buyken AE, Toeller M, Heitkamp G, Karamanos B, Rottiers R, Muggeo M & Fuller JH (2001) Glycaemic index of the diet of European outpatients with type-1 diabetes: relations to glycated hemoglobin and serum lipids. *American Journal of Clinical Nutrition* **73**, 574–581.

Canadian Diabetes Association (2000) Guidelines for the management of DM in the new millennium. *Canadian Journal of Diabetes Care* **23**, 56–69.

Chiasson JL, Josse RG, Gomis R, Hanefeld M, Karasik A & Laakso M (2002) Acarbose for prevention of type 2 DM: the STOP-NIDDM randomised trial. *Lancet* **359**, 2072–2077.

Christiansen MP, Linfoot PA, Neese RA & Hellerstein MK (2000) Effect of dietary energy restriction on glucose production and substrate utilization in type 2 diabetes. *Diabetes* **49**, 1691–1699.

Cummings (2002) Book Review: Handbook of dietary fibre. *British Journal of Nutrition* **88**, 216–217.

Department of Health (1990) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom. Report on Health and Social Subjects* no. 41. London: H. M. Stationery Office.

Dumesnil JG, Turgeon J, Trembley A, Poirier P, Gilbert M, Gagnon L, St-Pierre S, Garneau C, Lemieux I, Pascot A, Bergeron J & Després J-P (2001) Effect of a low-glycaemic index–low-fat–high-protein diet on the atherogenic metabolic risk profile of abdominally obese men. *British Journal of Nutrition* **86**, 557–568.

Foster-Powell K, Holt HA & Brand-Miller JC (2002) International tables of glycemic index and glycemic load values: 2002. *American Journal of Clinical Nutrition* **76**, 5–56.

Frost G & Dornhorst A (2000) The relevance of glycaemic index to our understanding of dietary carbohydrates. *Diabetic Medicine* **17**, 336–345.

Garg A (1998) High-monounsaturated-fat diets for patients with DM: a meta-analysis. *American Journal of Clinical Nutrition* **67**, 577S–582S.

Gilbertson HR, Brand-Miller JC, Thorburn AW, Evans S, Chondros P & Werther GA (2001) The effect of flexible low glycemic index dietary advice versus measured carbohydrate exchange diets on glycemic control in children with type 1 diabetes. *Diabetes Care* **24**, 1137–1143.

Gin H, Rigalleau V & Aparicio M (2000) Lipids, protein intake, and diabetic nephropathy. *Diabetes Metabolism* **26**, Suppl. 4, 45–53.

Jenkins DJA, Kendall CWC, Augustin LSA, Franceschi S, Hamidi M, Marchie A, Jenkins AL & Axelsen M (2002) Glycemic Index: overview of implications in health and disease. *American Journal of Clinical Nutrition* **76**, 2665–2735.

Jenkins DJ, Wolever TM, Vuksan V, Brighenti F, Cunnane SC, Rao AV, Jenkins AL, Buckley G, Patten R & Singer W, Corley P & Josse R (1989) Nibbling versus gorging: metabolic advantages of increased meal frequency. *New England Journal of Medicine* **321**, 929–934.

Kapur A & Kapur K (2001) Relevance of glycemic index in the management of post-prandial glycaemia. *Journal of the Association of the Physicians of India* **49**, 42–45.

Kelley DE (2002) Approaches to preventing mealtime hyperglycaemic excursions. *Diabetes, Obesity and Metabolism* **4**, 11–19.

Khaw K-T, Wareham N, Luben R, Bingham S, Oakes S, Welch A & Day N (2001) Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European Prospective Investigation of Cancer and Nutrition (EPIC-Norfolk). *British Medical Journal* **322**, 1–6.

Kirk TR (2000) Role of dietary carbohydrate and frequent eating in body-weight control. *Proceedings of the Nutriton Society* **59**, 349–358.

Liu S, Manson JE, Buring JE, Stampfer MJ, Willett WC & Ridker P (2002) Relation between diet with a high glycaemic load and plasma concentrations of high-sensitivity C-reactive protein in middle-aged women. *American Journal of Clinical Nutrition* **75**, 492–498.

Liu S, Manson JE, Stampfer MJ, Rexrode KM, Hu FB, Rimm EB & Willett WC (2000*a*) Whole grain consumption and risk of ischemic stroke in women: A prospective study. *Journal of the American Medical Association* **284**, 1534–1540.

Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH & Manson JE (2000*b*) A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *American Journal of Clinical Nutrition* **71**, 1455–1461.

Luscombe ND, Noakes M & Clifton PM (1999) Diets high and low in glycaemic index versus high monounsaturated fat diets: effects on glucose and lipid metabolism in NIDDM. *European Journal of Clinical Nutrition* **53**, 473–478.

McCarthy MF (2000) The origins of western obesity: a role for animal protein? *Medical Hypotheses* **54**, 488–494.

Mathers JC (2002) Choosing your carbohydrates to prevent diabetes. *British Journal of Nutrition* **88**, 107–108.

Meyer KA, Kushi LH, Jacobs DR, Slavin J, Sellers TA & Folsom AR (2000) Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. *American Journal of Clinical Nutrition* **71**, 921–930.

Miller B (1994) Glycaemic index in diabetes. *American Journal of Clinical Nutrition* **59**, 747s–752s.

Ratzmann KP, Zander E, Witt S & Schulz B (1981) Investigation of insulin sensitivity in early diabetes III. The effect of a combined physical training and diet programme on body weight, serum lipids and insulin sensitivity in obese asymptomatic diabetics. *Endokrinologie* **77**, 233–241.

Reddy ST, Wang C-Y, Sakhaee K, Brinkley L & Pak CYC (2002) Effect of low-carbohydrate, high-protein diets on acid–base balance, stone-forming propensity, and calcium metabolism. *American Journal of Kidney Diseases* **40**, 265–274.

Salmerón J, Ascherio A, Rimm EB, Coldiz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL & Willet WC (1997*a*) Dietary fibre, glycaemic load, and risk of NIDDM in men. *Diabetes Care* **20**, 545–550.

Salmerón J, Manson JE, Stampfer MJ, Coldiz GA, Wing AL & Willet WC (1997*b*) Dietary fibre, glycaemic load, and risk of non-insulin-dependent diabetes in women. *Journal of American Medical Association* **277**, 472–477.

Stefikova K, Spustova V, Gazdikova K, Krivosikova Z & Dzurik R (1997) Dietary protein restriction in combination with angiotensin converting enzyme inhibitor improves insulin resistance in patients with chronic renal disease. *International Journal of Urology and Nephrology* **29**, 497–507.

Stilling B, Mehlsen J, Hamberg O, Larsen JJ, Gram NC & Madsbad S (1999) Effect of a new starch-free bread on metabolic control in NIDDM patients. *Nutrition, Metabolism and Cardiovascular Disease* **9**, 98–101.

Tremblay A, Dumesnil JG & Després J-P (2002) Diet, satiety and obesity treatment. *British Journal of Nutrition* **88**, 213–214.

Vessby B, Unsitupa M, Riccardi G, Rivellese AA, Tapsell LC, Nalsen C, Berglund L, Louheranta A, Rasmussen BM, Calvert GD, Maffetone A, Pedersen E, Gustafsson IB & Storlien LH (2001) Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. *Diabetologia* **44**, 312–319.

Westerterp-Plantenga MS, Kovacs EM & Melanson KJ (2002) Habitual meal frequency and energy intake regulation in partially temporally isolated men. *International Journal of Obesity and Related Metabolic Disorders* **26**, 102–110.

Wolever TMS (1997) The glycaemic index: Flogging a dead Horse? *Diabetes Care* **20**, 452–456.

Wolever TMS (2002) Low carbohydrate does not mean low glycaemic index! *British Journal of Nutrition* **88**, 211–212.

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.

Yamanouchi K, Shinozaki T, Chikada K, Nishikawa T, Ito K, Shimizu S, Ozawa N, Suzuki Y, Maeno H, Kato K, Oshida Y & Sato Y (1995) Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to reduce body weight but also to improve insulin sensitivity. *Diabetes Care* **18**, 775–778.