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The Glycemic Index in the Management of Obesity

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This issue of *Endocrinology Rounds* discusses the role of dietary carbohydrates (CHOs) and the glycemic index in the management of body weight. It is commonly thought that diets eliciting low blood-glucose responses enhance weight loss by increasing satiety and fat oxidation. These effects have also been ascribed to foods with a low glycemic index (GI). Evidence is accumulating that low-GI foods may be more useful for weight loss than moderate reductions in CHO intake, but the mechanisms for this are unclear. Low-GI foods have inconsistent effects on short-term satiety and food intake, and studies are often confounded by other factors; as a result, it is impossible to ascribe the observed differences in satiety/food intake to the differences in blood glucose *per se*. Foods with a low GI usually result in low insulin responses, and low insulin may promote fat oxidation and reduce appetite through increased leptin sensitivity. However, counteracting this is the evidence that adults with high blood-insulin levels have lower food intake and gain less weight than those with normal levels. Low-GI starchy foods are digested slowly, leading to reduced energy absorption and increased colonic fermentation. The latter may have direct and indirect effects on the gut, pancreatic and adipose hormones that regulate energy intake and energy expenditure. This issue of *Endocrinology Rounds* on dietary CHOs and the glycemic index shows how low-GI foods may be helpful in the management of obesity; furthermore, this is consistent with current advice for weight management, which is to avoid energy-dense foods and to choose starchy, whole grain, high-fibre foods more often.

It is commonly believed that postprandial blood glucose and insulin concentrations influence the regulation of appetite, energy balance, and fat metabolism. A number of diet books¹ and scientific papers^{2,3} have been based on the premise that diets, which elicit low-blood glucose and insulin responses, will promote weight loss and assist with weight management. However, there are several different possibilities by which the glucose and insulin impact of a diet can be reduced, not all of which have the desired effect. One way to reduce the response to blood glucose and insulin is to consume low-GI foods.

Definition of GI and GL

GI is defined as $100 \times F/G$, where F is the incremental area under the curve (AUC) elicited by a 50-g available-CHO portion of a test food and G is the AUC elicited by 50 g of glucose taken by the same subject.^{4,5} The term "low-glycemic index" has sometimes been used incorrectly to describe low-glucose responses⁶ or low-CHO diets;⁷ this leads to confusion and misunderstanding about whether GI, as correctly defined, has any role to play in body-weight reduction. There are 2 implications from the correct definition of GI:

- GI is a *qualitative* indicator of the blood-glucose-raising ability of the available CHO in a food, independent of the amount of food consumed
- GI only applies to high-CHO foods.



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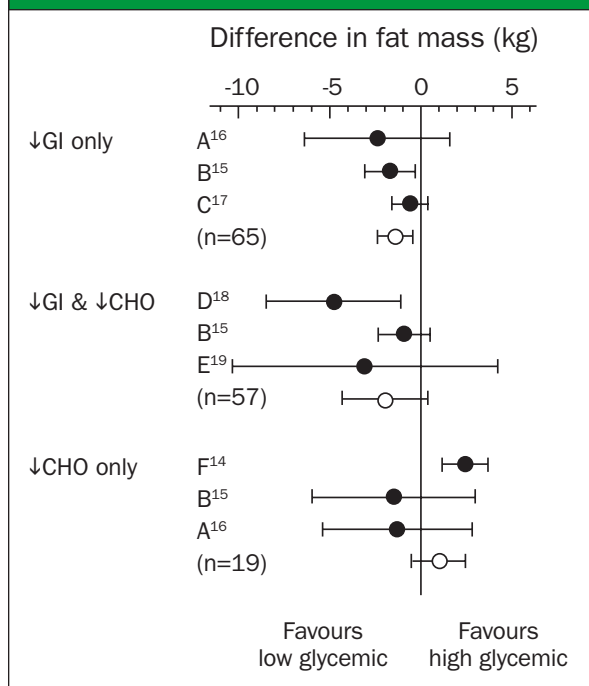
To obtain a *quantitative* index of how much food will raise blood glucose, both the amount of food consumed and its GI must be known; this is popularly assessed as glycemic load (GL) and $GL = g \times GI / 100$ where g is the grams of available CHO consumed and GI is the GI of the food. Because GL is quantitative, some find GL to be more useful than GI,⁸ however, the view proposed here is that GI is more useful than GL, precisely because it indicates the quality of dietary CHO.⁹

One problem with giving dietary advice based on GL is that it can be altered by changing either the amount of CHO or its GI (or both). We have shown that reducing CHO intake does not have the same effect as reducing GI on either second-meal glycemic responses in normal subjects,^{10,11} beta-cell function in subjects with impaired glucose tolerance,¹² or long-term blood glucose, lipids, and C-reactive protein in subjects with diabetes.¹³ This begs the question as to whether reducing GL by different methods has the same effect on body-weight regulation.

Low-GL diets in the management of body weight

Six recent studies lasting ≥ 10 weeks have examined the effects of low-GL and low-calorie diets on body fat in overweight and obese subjects.¹⁴⁻¹⁹ These studies used different methods of reducing GL, including: lowering GI with no reduction in CHO intake; decreasing both GI and CHO intake; and a modest reduction in CHO (10%-20% of energy) with no change in GI. The diets incorporating low-GI foods tended to reduce body fat compared with the controls, but the most consistent effects were seen with low-GI as opposed to a combination of low-GI and low-CHO (Figure 1). There was no consistent effect of a modest reduction in CHO intake. Most of these studies were underpowered, with < 25 subjects on each diet. The largest study was the Carbohydrate Ratio Management in European National diets (CARMEN) study¹⁴ with about 80 subjects on each diet; this trial indicated that a low-CHO diet (42% energy) significantly increased body weight and body fat compared with a higher CHO (49%), low-fat (25%) diet. Another notable study, with > 30 subjects on each diet, compared the effects of 3 different methods of reducing GL in the management of high body weight.¹⁵ Reducing diet GL by reducing GI, without changing the amount of dietary CHO, had, if anything, a greater effect in reducing body weight and body fat in overweight subjects than a comparable reduction in GL achieved by reducing CHO intake, or a larger reduction in GL achieved by reducing both GI and CHO intake. In addition, reducing GI alone had a more favourable effect on the blood-lipid profile.¹⁵

Figure 1: Change in body fat mass (means and 95% confidence intervals) in overweight or obese subjects given low-energy, low-glycemic load (GL) diets.



Reduced GL was achieved either by reducing glycemic index (GI) with no change in % energy from carbohydrate (CHO), or by reducing both GI and CHO, or by reducing CHO with no change in GI. Filled circles represent individual studies. Open circles represent weighted means for the 3 studies in each category.

In this discussion, no reference has been made to low-CHO diets in which CHO intake is reduced to $< 40\%$ of energy because, although such diets have a low GL, the focus is more on the amount of CHO rather than its nature. In addition, such diets are often considered not in line with general nutritional recommendations and, as such, probably do not belong in a discussion about low-GI diets. Nevertheless, there is evidence that such low-CHO diets result in greater weight loss over 6 months than conventional low-fat diets,^{20,21} but afterwards, weight tends to be regained and the results at 1 year are similar.^{21,22} Two recent studies compared 4 different, popular, weight-loss diets; a very low- (Atkins), very high- (Ornish), or intermediate- (ZoneTM) CHO diet, or a healthy diet approach (Weight Watchers[®] or United States Department of Agriculture [USDA] Pyramid). The first study revealed that the amount of weight lost over 1 year was unaffected by the amount of CHO, but was related to the degree of compliance, which was greater on the more moderate diets.²³ However, the second study demonstrated that, after 1 year, significantly more weight was lost on the Atkins diet than with the other 3 diets.²⁴

Do all low-GI foods affect body weight equally?

Clinical trials suggest that low-GI diets may enhance weight loss. While many factors may affect the GI of different foods, not all of these factors have the same potential effects on body-weight management. The 2 major mechanisms leading to low GI are the nature of the monosaccharide absorbed and the rate of absorption.²⁵ Fructose has a GI of approximately 20; it elicits glucose and insulin responses equivalent to about 20% of those with glucose.^{6,26} Consequently, the GI of sucrose (composed of glucose and fructose in equal proportions) has a GI of ~60, which is the mean of the GI values for glucose and fructose. However, preloads of fructose do not elicit feelings of satiety, nor do they reduce short-term food intake as much as an equivalent amount of glucose.²⁷ As a result, although incorporating fructose into a diet is an easy and very successful means of reducing dietary GI, it may not be effective in eliciting weight reduction. However, no studies have examined the effects on body weight by reducing GI with high-fructose foods compared with slowly digested starchy foods.

The GI values of starchy foods are directly related to their rate of digestion *in vitro*.²⁸ However, there are many factors in foods that affect the rate of starch digestion and absorption, such as starch structure (amylose vs amylopectin), the degree of gelatinization, chemical or enzymatic modifications, particle size, and the presence of dietary fibre or other components.^{29,30} There is little information about the effect of most of these factors on appetite and body-weight control, and those data that do exist are inconsistent. Dietary fibre is a good example of this problem.

In general, the effect of purified fibres on glucose and insulin responses is related to the ability of the fibre to increase the viscosity of the test meal.³¹ Thus, adding viscous fibres to foods (eg, psyllium³² or guar³³) reduces glucose responses, whereas nonviscous cereal fibre,³⁴ resistant starch,^{35,36} or fructo-oligosaccharides³⁷ have little or no effect. Therefore, if low glucose and insulin responses lead to increased satiety and reduced food intake, then one would expect viscous fibres to have a greater ability than nonviscous fibres to increase satiety and reduce food intake, but this is not the case. When the effects of a soluble fibre (psyllium) on short-term appetite were compared head-to-head with those of insoluble fibre (wheat bran), the soluble fibre elicited a significantly smaller suppression of food intake than the insoluble fibre.³⁸ Viscous soluble fibres have been demonstrated to increase satiety in some studies,³⁹ to have little or no effect in others,⁴⁰ and to have the opposite effect of increasing food intake in others.⁴¹ Similarly, nonviscous cereal fibre has been shown to reduce food intake in some studies,⁴² but to

Table 1: Some examples of low-GI food effects on satiety and/or food intake, illustrating inconsistent effects and confounding factors

Intervention	Effect on glucose	Effect on appetite*	Confounding factors
Bean vs potato ⁴⁵	–	Hunger –	Protein
Wheat particle size ⁴⁶	–	Satiety ±	None
Quick-cook rice ⁴⁷	+	Satiety ±	None
High-amylose rice ⁴⁷	–	Satiety ±	None
7 foods ⁴⁸	Low GI α increased satiety		Fat, protein, fibre
37 foods ⁴⁹	GI and satiety not related high insulin α low food intake		Fat, protein, fibre
High amylose ⁵⁰	–	Satiety +	None
Glucose vs starch ⁵¹		Satiety +	None
Low- vs high-GI breakfast cereal ⁵²	–†	Food intake –	Fibre
French fries vs mashed potato plus oil ⁵³	–	Satiety ±	Energy density

* + = significant increase; – = significant decrease; ± = no significant difference; α = significant correlation.

† Corn flakes (GI=85) vs All Bran® (GI=53), glucose response not measured in the study.

have no effect in others.⁴³ Interestingly, oligofructose, which has no effect on blood glucose, increased satiety in a pilot study.⁴⁴ The apparent inconsistencies in the results may be caused by widely different methods used in the various studies, but there is certainly no clear relationship between the effects of fibre on glucose and its effect on short-term appetite.

What is the mechanism by which low-GI diets reduce body weight?

Low-GI diets may influence body weight via short-term (ie, meal-to-meal) or long-term (ie, adaptation over weeks or months) mechanisms. The most common mechanism ascribed to low-GI foods is that they increase short-term satiety and reduce short-term food intake because of the reduced blood glucose and/or insulin responses they elicit. However, the results are not consistent and there are confounding factors present in many studies (Table 1).⁴⁵⁻⁵³ Ludwig⁵⁴ recently cited 16 studies as evidence that low-GI foods reduce appetite, but it is impossible to attribute this to reduced glucose or insulin *per se* because of confounding factors, such as differences in fibre, volume, protein, or the amount of chewing required. In addition, there are several different hypotheses relating to glucose and insulin responses that are not consistently supported by the evidence. Sometimes, the reduction in peak glucose and insulin is considered important, sometimes the lack of undershoot (related to dynamic falls in glucose), and sometimes the prolonged absorp-

Table 2: Classification of glycemic index for some foods

	High GI	Intermediate GI	Low GI
Breads	White wheat (71)	Stoneground (62)	Pumpernickel (50) (whole rye kernels)
	Whole wheat (70)	Rye crispbread (65)	
	Rice cakes (88)	Pita bread (57)	
	Corn flakes (84)	Shredded wheat (69)	Oat bran (55)
Breakfast cereals	Puffed rice (86)	Oatmeal (61)	Red River® (49)
	Puffed wheat (74)		All Bran Buds® (psyllium) (47)
Potatoes	Instant mashed (88)	French fries* (64)	Yam/sweet potato (51)
	Baked/boiled (73)	Potato salad (cold) (56)	
Grains	Cornmeal (70)	Couscous (65)	Parboiled rice (50)
		Polished rice (69)	Bulgur† (48)
		Macaroni (64)	Spaghetti (41)
			Pearled barley (25)
Legumes			Legumes (25-52)

* Frozen French fries cooked and consumed hot.

† Cracked wheat.

tion. The timing of the effects on satiety (early, late, continuous) does not correspond consistently with the timing of glucose/insulin effects. In addition, the relationship between glucose/insulin responses and satiety varies for different types of foods,⁴⁹ strongly suggesting that factors other than postprandial glucose and insulin are important in determining short-term satiety responses.

The factors regulating food intake are complex and include numerous hormones secreted by the gut (eg, glucagon-like peptide-1 [GLP-1], peptide YY [PYY], cholecystokinin [CCK]), the pancreas (eg, insulin), and adipose tissue (eg, leptin). It has long been debated as to whether hyperinsulinemia is a cause⁵⁵ or an effect⁵⁶ of obesity; this debate has not been resolved. Evidence that hyperinsulinemia is a physiological adaptation to obesity that helps to limit further weight gain is found in our recent study suggesting that hyperinsulinemic subjects have less food intake in response to a preload of glucose than those with low insulin.⁵⁷ Further, insulin resistant, hyperinsulinemic adults gain less weight than insulin sensitive adults.⁵⁸ In contrast, insulin as a cause of obesity is supported by evidence that insulin-resistant children gain more weight than insulin-sensitive children.⁵⁹ In addition, suppressing insulin responses with octreotide elicits weight loss in obese subjects, an effect associated with reduced energy intake,⁶⁰ which was suggested to be due to increased leptin sensitivity. Therefore, the role of insulin in the long-term control of appetite is unclear.

Other potential long-term mechanisms for an effect of low-GI foods on body-weight regulation are related to increased colonic fermenta-

tion, including a reduced efficiency of energy absorption⁶¹ or, potentially, the effect of colonic fermentation on hormones that regulate energy balance.⁶²

Practical application of the GI

GI is not the only attribute of foods that should be considered when planning a healthy diet; in fact, it should only be used to compare foods with similar nutritional profiles. GI is most appropriately used when comparing starchy carbohydrate foods, such as breads, grains, legumes, and tubers. The difficulty in using GI is in knowing the GI value of a specific food; GI cannot be predicted reliably from the composition of a food because it may be affected by differences in processing, cooking, and other factors. However, enough is known to enable practical use of GI and applying GI is simple. It is based on the concept of food exchanges, selecting foods with a lower GI, or having at least one serving of a low-GI food with every meal. This is entirely consistent with current dietary advice for weight management that is focused on controlling portion size, using energy-dense foods less often, and choosing bulky, whole-grain, starchy, high-fibre foods more frequently. High-GI foods do not have to be excluded because mixing high- and low-GI foods will produce a meal with an intermediate response. Examples of some high-, intermediate-, and low-GI foods are shown in Table 2. Information, practical tips and recipes based on GI can be obtained from the Canadian Diabetes Association website,⁶³ and from popular books on GI written by Dr. Jennie Brand-Miller.⁶⁴

Conclusions

It is apparent that low-GI foods may assist in weight management; however, the mechanisms for this effect are not completely understood. The effects of low-GI foods on short-term satiety and food intake are not likely to be due to reduced glucose or insulin responses *per se*, but rather to other factors; nevertheless, these factors may contribute to long-term reductions in food intake. A reduced rate of starch digestion leads to small reductions in energy absorption and may have direct and indirect effects on gut, pancreatic and adipose hormones that regulate energy intake and energy expenditure.

References:

1. Gallop R. *The G.I. Diet*. Mississauga, ON: Random House; 2004.
2. Dumesnil JG, Turgeon J, Tremblay A, et al. Effect of a low-glycaemic index–low-fat–high protein diet on the atherogenic metabolic risk profile of abdominally obese men. *Br J Nutr*. 2001;86(5):557-568.
3. Bell SJ, Sears B. Low-glycemic-load diets: impact on obesity and chronic diseases. *Crit Rev Food Sci Nutr*. 2003;43(4):357-377.
4. Jenkins DJ, Wolever TM, Taylor RH, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr*. 1981;34(3):362-366.
5. Brouns F, Björck I, Frayn KN, et al. Glycaemic index methodology. *Nutr Res Rev*. 2005;18(1):145-171.
6. Wolever TMS. Glycemic index vs glycemic response: non-synonymous terms. *Diabetes Care*. 1992;15(10):1436-1437.
7. Wolever TM. Low carbohydrate does not mean low-glycaemic index! *Br J Nutr*. 2002;88(2):211-212.
8. Monro J. Redefining the glycemic index for dietary management of postprandial glycaemia. *J Nutr*. 2003;133(12):4256-4258.
9. Wolever TMS. *The Glycaemic Index: A Physiological Classification of Dietary Carbohydrate*. Wallingford, UK: CABI Publishing; 2006.
10. Jenkins DJ, Wolever TM, Taylor RH, et al. Slow release carbohydrate improves second meal tolerance. *Am J Clin Nutr*. 1982;35(6):1339-1346.
11. Wolever TM, Bentum-Williams A, Jenkins DJ. Physiological modulation of plasma free fatty acid concentrations by diet: Metabolic implications in non-diabetic subjects. *Diabetes Care*. 1995;18(7):962-970.
12. Wolever TMS, Mehling C. High-carbohydrate/low-glycaemic index dietary advice improves glucose disposition index in subjects with impaired glucose tolerance. *Br J Nutr*. 2002;87:477-487.
13. Wolever TMS, Gibbs AL, Mehling C, et al. The Canadian trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. *Am J Clin Nutr*. 2008;87(1):114-125.
14. Saris WH, Astrup A, Prentice AM, et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study. The Carbohydrate Ratio Management in European National diets. *Int J Obes Relat Metab Disord*. 2000;24(10):1310-1318.
15. McMillan-Price J, Petocz P, Atkinson F, et al. Comparison of 4 diets of varying glycemic load on weight loss and cardiovascular risk reduction in overweight and obese young adults: a randomized controlled trial. *Arch Intern Med*. 2006;166(14):1466-1475.
16. Raatz SK, Torkelson CJ, Redmond JB, et al. Reduced glycemic index and glycemic load diets do not increase the effects of energy restriction on weight loss and insulin sensitivity in obese men and women. *J Nutr*. 2005;135(10):2387-2391.
17. Sloth B, Krog-Mikkelsen I, Flint A, et al. No difference in body weight decrease between a low-glycemic-index and a high-glycemic-index diet but reduced LDL cholesterol after 10-wk ad libitum intake of the low-glycemic-index diet. *Am J Clin Nutr*. 2004;80(2):337-347.
18. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med*. 2003;157(8):773-779.
19. Das SK, Gilhooly CH, Golden JK, et al. Long-term effects of 2 energy-restricted diets differing in glycemic load on dietary adherence, body composition, and metabolism in CALERIE: a 1-y randomized controlled trial. *Am J Clin Nutr*. 2007;85(4):1023-1030.
20. Samaha FF, Iqbal N, Seshadri P, et al. A low-carbohydrate as compared with a low-fat diet in severe obesity. *N Engl J Med*. 2003;348(21):2074-2081.
21. Foster GD, Wyatt HR, Hill JO, et al. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med*. 2003;348(21):2082-2090.
22. Stern L, Iqbal N, Seshardi P, et al. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med*. 2004;140(10):778-785.
23. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA*. 2005;293(1):43-53.
24. Gardiner CD, Kiazand A, Alhassan S, et al. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women. The A-Z Weight Loss Study: a randomized trial. *JAMA*. 2007;297(9):969-977.
25. Wolever TMS. Carbohydrate and the regulation of blood glucose and metabolism. *Nutr Rev*. 2003;61(5 Pt 2):S40-S48.
26. Lee BM, Wolever TMS. Effect of glucose, sucrose and fructose on plasma glucose and insulin responses in normal humans: comparison with white bread. *Eur J Clin Nutr*. 1998;52(12):924-928.
27. Akhavan T, Anderson GH. Effects of glucose-to-fructose ratios in solutions on subjective satiety, food intake, and satiety hormones in young men. *Am J Clin Nutr*. 2007;86(5):1354-1363.
28. Jenkins DJ, Ghafari H, Wolever TM, et al. Relationship between rate of digestion of foods and postprandial glycaemia. *Diabetologia*. 1982;22(6):450-455.
29. Würsch P. Starch in human nutrition. *World Rev Nutr Diet*. 1989;60:199-256.
30. Björck I, Elmståhl HL. The glycaemic index: importance of dietary fibre and other food properties. *Proc Nutr Soc*. 2003;62(1):201-206.
31. Jenkins DJ, Wolever TM, Leeds AR, et al. Dietary fibres, fibre analogues and glucose tolerance: importance of viscosity. *Br Med J*. 1978;1(6124):1392-1394.
32. Wolever TM, Vuksan V, Eshuis H, et al. Effect of method of administration of psyllium on the glycemic response and carbohydrate digestibility. *J Am Coll Nutr*. 1991;10(4):364-371.
33. Wolever TM, Jenkins DJ, Nineham R, Alberti KG. Guar gum and reduction of post-prandial glycaemia: effect of incorporation into solid food, liquid food, and both. *Br J Nutr*. 1979;41(3):505-510.
34. Jenkins DJ, Wolever TM, Taylor RH, Barker HM, Fielden H, Gassull MA. Lack of effect of refining on the glycemic response to cereals. *Diabetes Care*. 1981;4(5):509-513.
35. Ranganathan S, Champ M, Pechard C, et al. Comparative study of the acute effects of resistant starch and dietary fibers on metabolic indexes in men. *Am J Clin Nutr*. 1994;59(4):879-883.
36. Jenkins DJ, Vuksan V, Kendall CW, et al. Physiological effects of resistant starches on fecal bulk, short chain fatty acids, blood lipids and glycemic index. *J Am Coll Nutr*. 1998;17(6):609-616.
37. Brighenti F, Casiraghi MC, Canzi E, Ferrari A. Effect of consumption of a ready-to-eat breakfast cereal containing inulin on the intestinal milieu and blood lipids in healthy male volunteers. *Eur J Clin Nutr*. 1999;53(9):726-733.
38. Delargy HJ, O'Sullivan KR, Fletcher RJ, Blundell JE. Effects of amount and type of dietary fiber (soluble and insoluble) on short-term control of appetite. *Int J Food Sci Nutr*. 1997;48(1):67-77.
39. Hoad C, Rayment P, Spiller RC, et al. In vivo imaging of intragastric gelation and its effect on satiety in humans. *J Nutr*. 2004;134(9):2293-2300.
40. Kovacs EM, Westterterp-Plantenga MW, Saris WH, Goossens I, Geurten P, Brouns F. The effect of addition of modified guar gum to a low-energy semisolid meal on appetite and body weight loss. *Int J Obes Relat Metab Disord*. 2001;25(3):307-315.
41. Keogh JB, Lau CW, Noakes M, Bowen J, Clifton PM. Effects of meals with high soluble fibre, high amylose barley variant on glucose, insulin, satiety and thermic effect of food in healthy lean women. *Eur J Clin Nutr*. 2007;61(5):597-604.

42. Samra RA, Anderson GH. Insoluble cereal fiber reduces appetite and short-term food intake and glycemic response to food consumed 75 min later by healthy men. *Am J Clin Nutr*. 2007;86(4):972-979.
43. Silberbauer C, Frey-Rindova P, Langhans W. Breakfasts with different fiber and macronutrient contents do not differentially affect timing, size or microstructure of the subsequent lunch. *Z Ernährungswiss*. 1996;35(4):356-368.
44. Cani PD, Joly E, Horsmans Y, Delzenne NM. Oligofructose promotes satiety in healthy human: a pilot study. *Eur J Clin Nutr*. 2006;60(5):567-572.
45. Leathwood P, Pollet P. Effects of slow releases of slow release carbohydrates in the form of bean flakes on the evolution of hunger and satiety in man. *Appetite*. 1988;10(1):1-11.
46. Holt SH, Miller JB. Particle size, satiety and the glycaemic response. *Eur J Clin Nutr*. 1994;48(7):496-502.
47. Holt SH, Miller JB. Increased insulin responses to ingested foods are associated with lessened satiety. *Appetite*. 1995;24(1):43-54.
48. Holt S, Brand J, Soveny C, Hansky J. Relationship of satiety to postprandial glycaemic, insulin and cholecystokinin responses. *Appetite*. 1992;18(2):129-141.
49. Holt SH, Brand Miller JC, Petocz P. Interrelationships among postprandial satiety, glucose and insulin responses and changes in subsequent food intake. *Eur J Clin Nutr*. 1996;50(12):788-797.
50. van Amelsvoort JMM, Weststrate JA. Amylose-amylopectin ratio in a meal affects postprandial variables in male volunteers. *Am J Clin Nutr*. 1992;55(3):712-718.
51. Anderson GH, Catherine NLA, Woodend DM, Wolever TM. Inverse association between the effect of carbohydrates on blood glucose and subsequent short-term food intake in young men. *Am J Clin Nutr*. 2002;76(5):1023-1030.
52. Warren JM, Henry CJK, Simonite V. Low glycemic index breakfasts and reduced food intake in preadolescent children. *Pediatrics*. 2003;112(5):e414-419.
53. Leeman M, Östman E, Björck I. Glycaemic and satiating properties of potato products. *Eur J Clin Nutr*. 2008;62(1):87-95.
54. Ludwig DS. Dietary glycemic index and obesity. *J Nutr*. 2000;130(2S Suppl):280S-283S.
55. Sims EAH. Insulin resistance is a result, not a cause of obesity: Socratic debate: the con side. In: Angel A, Anderson H, Bouchard C, Lau D, Lieter L, Mendelson R, eds. *Progress in Obesity Research: 7. 7th International Congress on Obesity*. London, UK: John Libbey and Co.; 1996:587-592.
56. Ravussin E, Swinburn BA. Insulin resistance is a result, not a cause of obesity: Socratic debate: the pro side. In: Angel A, Anderson H, Bouchard C, Lau D, Lieter L, Mendelson R, eds. *Progress in Obesity Research: 7. 7th International Congress on Obesity*. London, UK: John Libbey and Co; 1996:173-178.
57. Samra RA, Wolever TM, Anderson GH. Enhanced food intake regulatory responses after a glucose drink in hyperinsulinemic men. *Int J Obes (Lond)*. 2007;31(8):1222-1231.
58. Swinburn BA, Nyomba BL, Saad MF, et al. Insulin resistance associated with lower rates of weight gain in Pima Indians. *J Clin Invest*. 1991;88(1):168-173.
59. Odeleye OE, de Courten M, Pettitt DJ, Ravussin E. Fasting hyperinsulinemia is a predictor of increased body weight gain and obesity in Pima Indian Children. *Diabetes*. 1997;46(8):1341-1345.
60. Velasquez-Miery PA, Cowan PA, Arheart KL, et al. Suppression of insulin secretion is associated with weight loss and altered macronutrient intake and preference in a subset of obese adults. *Int J Obes Relat Metab Disord*. 2003;27(2):219-226.
61. Jenkins DJ, Cuff D, Wolever TM, et al. Digestibility of carbohydrate foods in an ileostomate: relationship to dietary fiber, in vitro digestibility, and glycemic response. *Am J Gastroenterol*. 1987;82(8):709-717.
62. Reimer RA, McBurney MI. Dietary fiber modulates intestinal proglucagon messenger ribonucleic acid and postprandial secretion of glucagon-like peptide-1 and insulin in rats. *Endocrinology*. 1996;137(9):3948-3956.
63. Canadian Diabetes Association. Glycemic index – A new way of looking at carbs. http://www.diabetes.ca/Section_About/glycemic.asp. Accessed April 21, 2008.
64. Brand-Miller J, Wolever TMS, Foster-Powell K, Colagiuri S. *The New Glucose Revolution: The Authoritative Guide to the Glycemic Index – the Dietary Solution for Lifelong Health*. New York, NY: Marlowe & Company; 2003.

Upcoming Scientific Meetings

6 – 10 June 2008

68th Scientific Sessions

American Diabetes Association

San Francisco, California

CONTACT: http://professional.diabetes.org/Congress_Display.aspx?TYP=9&SID=39&CID=58000

15 – 18 June 2008

ENDO 2008

The Endocrine Society

San Francisco, California

CONTACT: <http://www.endo-society.org/endo/>

6 – 9 July 2008

European Society of Human Reproduction and Embryology

24th Annual Meeting

Barcelona, Spain

CONTACT: info@eshre.com

15 – 18 October 2008

Canadian Diabetes Association (CDA)/Canadian Society of Endocrinology and Metabolism (CSEM) Professional Conference and Annual Meetings

Montreal, Quebec

CONTACT: http://www.diabetes.ca/Section_Professionals/ConfIndex.asp

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