Simple and Complex Carbohydrates

David J.A. Jenkins, MD, Alexandra L. Jenkins, Thomas M.S. Wolever, MD,
Lilian H. Thompson, PhD, and A. Venkat Rao, PhD

Traditionally, the factors surrounding the assimilation of carbohydrate foods were considered to be relatively uncomplicated. Such foods were considered to be either simple (sugars) or complex (starches) carbohydrates. The former are absorbed rapidly and cause large rises in blood glucose after ingestion. The latter are absorbed more slowly and produce flat blood glucose responses (Figures 1 and 2). But thinking in this area has changed. Considerable differences are now recognized among sugars and starches, with substantial overlap between these two classes of carbohydrates.[1-6]

Much of the impetus for this change came from the studies of Helmut Otto's group[5,6] and from Phyllis Crapo, Gerald Reaven, and their associates.[2,3] By the mid-1970s even the importance of chain length of glucose polymers was called into question. Walhqvist demonstrated that feeding three carbohydrate forms —glucose, a five-unit glucose polymer, or liquid starch—all resulted in comparable glycemic and insulinemic responses in healthy volunteers.[7] The expected differences associated with increasing complexity were therefore not observed. This finding focused attention on the factors that might alter starch digestibility in the context of food systems where large differences in glycemic response are observed after feeding equicarbohydrate portions.[1-6] It has also stimulated interest in the possible physiologic and metabolic consequences of feeding more slowly absorbed foods with lesser glycemic impact.[8] From this interest, the concept of lente carbohydrate starchy foods has evolved.[8]

Presented by Dr D.J.A. Jenkins, Professor, Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, Canada.

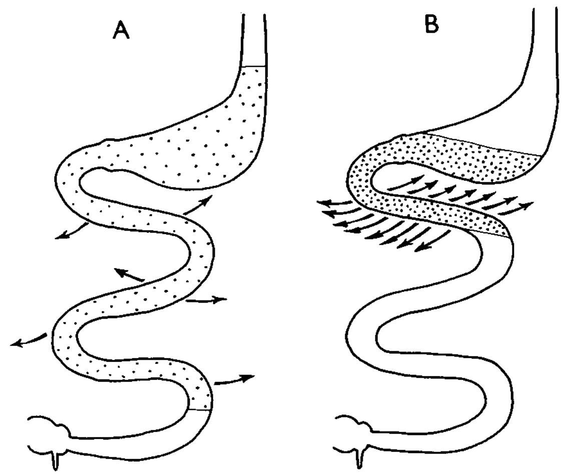

Figure 1. Stomach and small intestine showing a) slow absorption of energy-dilute nutrient in a fiber-rich "primitive" diet, and b) rapid absorption of energy-dense nutrient from low-fiber modern Western foods.

Differences in Rates of Digestion and Glycemic Response

Over the past five years, it has become increasingly clear that different starchy foods are digested at different rates (Figure 3).[9] The rates of in vitro digestion using pooled human digestive juices also appear to relate well to the in vivo glycemic responses observed after feeding equicarbohydrate portions of these foods to normal[9] and diabetic[10] volunteers.

Certain starchy foods, especially legumes, were digested slowly in vitro and also produced flat glycemic responses in vivo. Up to threefold differences among starchy foods were observed in rates of digestion and degree of blood glucose increase.[9] What was the relationship between rate of digestion and the glycemic response? Assuming that similar amounts of starch were absorbed, the same amount of glucose should eventually appear in the circulation. Thus, with slowly absorbed carbohydrate, the blood glucose response would be prolonged and flattened, but the area would not be altered. But this does not appear to be the case, probably because tissues clear glucose more efficiently during the latter stages of glucose absorption.

Lente Carbohydrate: Physiological Effects of Slowly Absorbed Carbohydrate

Isotopic studies undertaken in human beings to assess postprandial hepatic glucose uptake shed light on the disparity in the rate of absorption of glucose and peripheral blood glucose levels.[11] In addition to demonstrating a very limited role for the liver in taking up glucose from the gut, these studies also demonstrated that high glucose levels were being absorbed when peripheral blood glucose levels had returned to basal.[11] The implication was that, with time, glucose assimilation rates by peripheral tissues more closely resembled rates of glucose absorption from the gut. Thus if the absorption time could be prolonged by slowing the rate of absorption, then a lower blood glucose response would be seen in peripheral blood. Barium, given with a glucose load, was found to be still emptying from the stomach long after the fasting blood glucose level had returned to baseline.[12]

Other evidence supports this concept.[13] A noninsulin-dependent diabetic patient was given 240 g glucose over 12 hours either as three 80 g boluses 4 hours apart or by sipping 5 g glucose every 15 minutes. After the boluses there were higher peak rises in blood glucose, and a greater insulin response area and urinary glucose loss, compared with the more continuous mode of ingestion.[13] In addition, the RQ showed a progressive rise rather than the rises and falls corresponding with the bolus administration (Figure 4).[13] After a small rise, blood glucose response on the "continuous feeding" showed a progressive fall; by 12 hours the level was considerably below the elevated fasting value despite the continued ingestion of glucose over the entire period.[13]

Slow release of carbohydrate in the first meal results in a reduction in glycemic response area to a standard carbohydrate challenge given to normal volunteers 4 hours later.[14,15] This improvement in carbohydrate tolerance can also be ascribed to the continued slow absorption of carbohydrate from the first meal resulting in reduced FFA and ketone bodies prior to the standard carbohydrate challenge.[14] The effect may relate to the Staub[16]-Traugott[17] phenomenon, in which one meal is recognized as facilitating the uptake of the next one shortly after it.

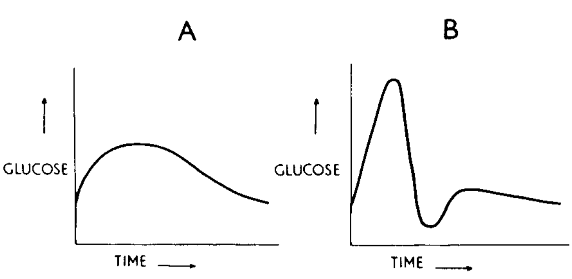

Figure 2. Postprandial glycemia following a) slow absorption from starchy, fiber-rich meals, and b) rapid absorption with undershoot due to excessive insulin release following refined fiber-depleted carbohydrate foods.

Factors Affecting Starch Absorption and Glycemic Response

Gastric emptying and small intestinal absorption. Initially, studies focused on gastric emptying as the mechanism by which absorption could be delayed.[18,19] Studies with viscous fiber emphasized the importance of gastric emptying. Food studies, such as a comparison between whole and ground rice, demonstrated a good correlation between gastric emptying rate and postprandial glycemia.[20] In diabetics with gastroparesis, fiber had little further effect in reducing postprandial glycemia.[21] This was probably the result of reduced frequency of

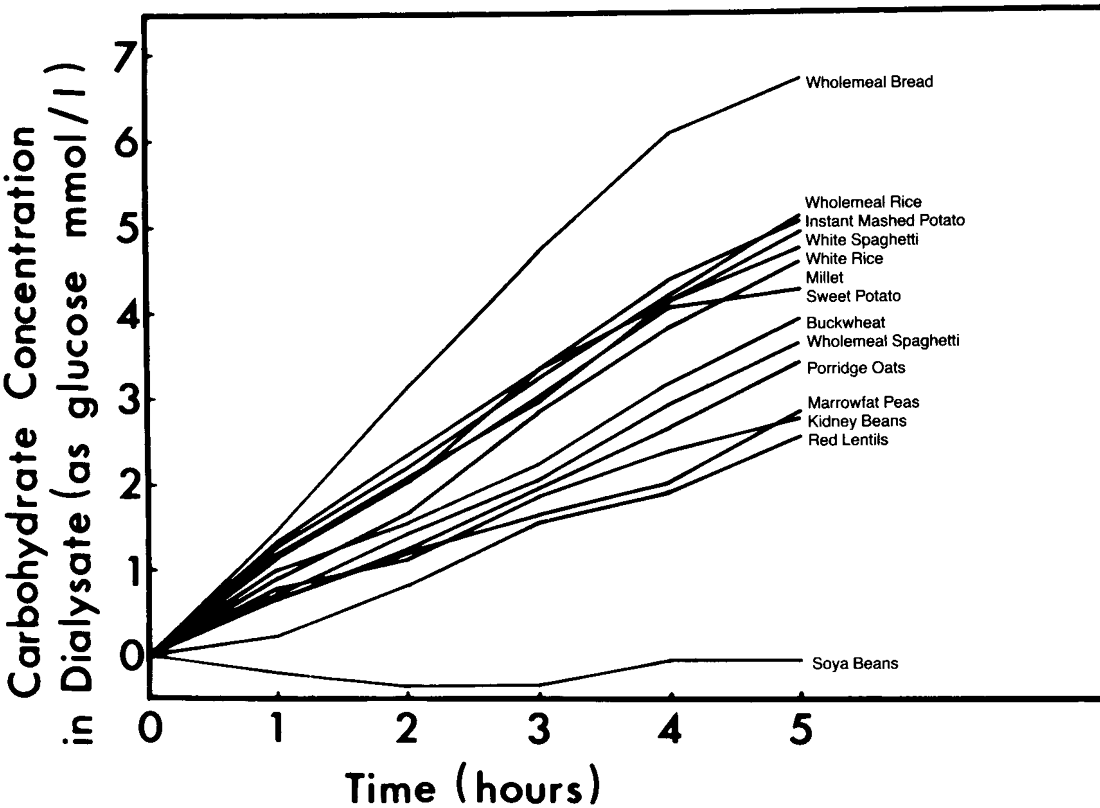

Figure 3. The increase in concentration of 5 hours of the products of starch digestion, measured as glucose after acid hydrolysis, subsequent to incubation of 2 g available carbohydrate portions of foods with pooled human saliva and pancreatic juice.

migrating motor complexes associated with both reduced gastric emptying and small intestinal motility.[22] Certainly the diabetic patients with gastroparesis had flatter postprandial responses in the control situation than did their peers, who had no evidence of autonomic neuropathy of the gut.[21] However, subsequent detailed studies with viscous fiber have failed to confirm the correlation between delayed gastric emptying and flattened glycemic response;[23] they emphasized the importance of small intestinal events explored in jejunal perfusion studies.[23,24] In these, an increase in the thickness of the unstirred water layer was seen with pectin[24] while with guar an overall impedence of luminal diffusion was demonstrated.[23] These latter studies also demonstrated impeded uptake of Na^+ and Cl^- in the presence of viscous fiber.[23] In food studies, it is also likely that small intestinal effects are of great importance. With legumes, delayed gastric emptying does not relate to their marked effect in flattening the glycemic response.[20] However, it is difficult to separate the effects of motor activity in the stomach and small intestine, since the migrating motor complex activity triggered in the stomach will have an effect on intestinal absorption.[22] Both gastric and small intestinal events are probably important, the balance of one vs the other depending on the food.

The effects on gastric emptying are not only seen in the acute situation. Administration of 20 g pectin daily for 4 weeks significantly reduced gastric emptying even in the absence of fiber.[25]

Food factors affecting digestion. The rate of digestion of foods may be altered by many factors (Table I). These include mechanisms of action that are still being elucidated. It is likely that the length of the list will increase as additional food constituents are found to have physiological effects. Current knowledge of these food factors is of limited value, because

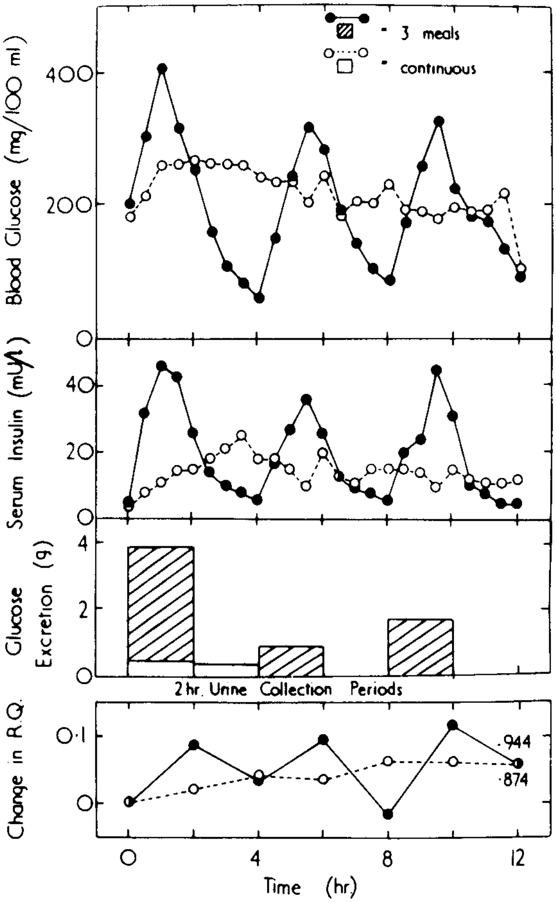

Figure 4. Blood glucose, insulin, 2-hour urinary glucose losses and respiratory quotient (RQ) measured over 12 hours are shown in a diabetic controlled by diet. On one occasion, 80 g glucose drinks were taken at 0, 4, and 8 hours. On another, 5 g glucose in solution was taken every 15 minutes by continuous sipping.

no conclusive food tables exist to document their concentration in foods. Also different types and mixtures of fibers, lectins, tannins, saponins, etc, with unique physiological effects, exist in differing proportions in different foods.

Thus, white spaghetti is digested more slowly and gives half the glycemic response of white bread.[26,27] Although the use of durum wheat in spaghetti may be important, food form in allowing penetration of enzymes is likely to be a determining factor. Particle size also plays a major part. Whole rice is more slowly digested and raises the blood glucose less than ground rice.[28,29] Pumpernickel bread made with whole rye kernels results in a flatter blood glucose response than wholemeal rye bread made from ground flour.*

The higher the amylose content of a starch, the less rapidly it is digested, and the less it raises the blood glucose. Thus, legumes are digested less rapidly than bread and release more maltose and glucose and less maltotriose than bread as markers of their higher amylose content.[30] Rice with a high amylose content raises the blood glucose less than rice with a higher amylopectin content.[31]

Processing, such as cooking, or modification of the starch will alter digestibility[32] as will the natural starch nutrient interactions present in the food. Although blending cooked lentils or cooking them for 60 minutes (three times as long as necessary) had little effect on digestibility or glycemic response, heating and drying them for 12 hours enhanced both the rate of digestion and the postprandial blood glucose rise.[30]

Early on it was recognized that the non-nutrient components in foods might be important determinants of absorption and subsequent metabolic events. Much interest has been stimulated in this regard by studies of dietary fiber. Fiber, especially viscous fiber, alters the activity of enzymes involved in luminal digestion[33] and reduces postprandial glycemia and insulin responses of healthy volunteers[34] and diabetics.[35] In the long term, fiber was demonstrated to improve many aspects of diabetic control[36-38] in addition to reducing serum lipids.[36-40]

Less well-recognized are the effects of the

TABLE I
Factors Affecting Starch Digestibility

Food Form	**Fiber (type)**
Particle size	Antinutrients:
Nature of the starch:	phytate
amylose content	lectins
amylopectin content	tannins
degress of gelatinization	saponins
processing	enzyme inhibitors
Starch-nutrient interactions (protein, fat)	

* *DJA Jenkins et al, unpublished data.*

so-called antinutrients often associated with dietary fiber. In their own right they may exert a powerful influence on digestion absorption and subsequent postprandial nutrient and endocrine fluxes. Thus lectins, although heat labile, are found in appreciable concentrations in many starchy foods[41] and relate well to glycemic responses to foods tested in both normal and diabetic volunteers.* When added to bread, kidney lectin (but not concanavalin A) reduced the rate of in vitro digestion.* Similarly, phytic acid added to bread in the quantities found in beans reduced the rate of digestion in vitro and the glycemic response in vivo. The effect could be minimized by adding Ca^{2+}.[42]

Effects of slow-release (lente) carbohydrate foods. No studies exist in which a single lente carbohydrate food has been selected as such for inclusion in the diet to study its effects on carbohydrate metabolism. However, legumes as a class are more slowly digested in vitro than are other starchy foods[9,10] such as cereals and potatoes. They also result in flatter glycemic responses when fed to normal[9] or diabetic volunteers.[10] Their long-term use has been associated with reduced C-peptide excretion,[43] improved diabetic control,[44] and lower blood lipid profiles.[45] Legumes are rich sources of both dietary fiber and the antinutrients that modify the rate of digestion. Although it may not be possible to ascribe their effects to specific nutrients, legumes are, in general, good sources of lente carbohydrate.

The Glycemic Index

Because of the difficulty in predicting the physiological effects of starchy foods from our present knowledge of their components it was considered useful to classify foods in terms of glycemic response. The relative Δ blood glucose could reflect differences in their rates of digestion and other metabolic characteristics. For this reason the glycemic index (GI) concept was introduced.[46]

$$GI = \frac{\Delta \text{ blood glucose from food}}{\Delta \text{ blood glucose from standard carbohydrate}} \times 100$$

The reference carbohydrate was glucose[4] but bread baked from a flour of known composition has proved more acceptable.[46] A relatively small number of foods has been classifed by this means.[46] More are required for such data to be useful in dietary manipulations. However, in initial studies where low-glycemic-index starchy foods have been exchanged in the diets of patients with hyperlipidemia for those with a higher value, significant reductions were seen in blood lipids (both cholesterol and triglyceride).[47] This approach may prove useful in guiding the choice of starchy foods when these are to be increased in the diets of patients with hyperlipidemia, including diabetics.

Summary

The rates of digestion of starchy foods differ, and relate to the glycemic response they produce. Many factors—such as the nature of the starch, the food form, processing, and fiber and antinutrient content—determine these effects through alterations in gastic emptying and small intestinal absorption. These differences suggest that, through selection of specific starchy foods, the diet can be manipulated to achieve certain metabolic effects. To aid this approach, foods are now being classified in terms of their glycemic index. Initial studies using low-glycemic-index foods to treat diabetes and hyperlipidemia are promising.

However, before any general application of this approach can be made, many more foods must be assessed; a better understanding of the mechanism of action of the factors which control the rate of absorption must be obtained; and, the scope of usefulness of controlling the rate of nutrient release in longer term clinical studies must be determined. □

1. PA Crapo and JM Olefsky, *N Engl J Med* 309: 44-45, 1983
2. PA Crapo, G Reaven, and J Olefsky, *Diabetes* 26: 1178-1183, 1977
3. PA Crapo, OG Kolterman, N Waldeck, GM Reaven, and JM Olefsky, *Am J Clin Nutr* 33: 1723-1728, 1980
4. DJA Jenkins, TMS Wolever, RH Taylor, HM Barker, H Fielden, JM Baldwin, AC Bowling, HC Newman, AL Jenkins, and DV Goff, *Am J Clin*

Nutr 34: 362-366, 1981
5. H Otto, G Bleyer, M Pennartz, G Sabin, G Schauberger, and R Spaethe in *Diabetik Bei Diabetus Mellitus*. H Otto and R Spaethe, Editors. Verlag Hans Huber, Bern, 1973
6. G Shauberger, UC Brinck, G Suldner, R Spaethe, L Niklas, and H Otto, *Diabetes* 26: 415, 1978
7. ML Wahlqvist, EG Wilmshurst, CR Murton, and EN Richardson, *Am J Clin Nutr* 31: 1998-2001, 1978
8. DJA Jenkins, *Diabetes Care* 5: 634-641, 1982
9. DJA Jenkins, H Ghafari, TMS Wolever, RH Taylor, HM Barker, H Fielden, AL Jenkins, and AC Bowling, *Diabetologia* 22: 450-455, 1982
10. DJA Jenkins, TMS Wolever, MJ Thorne, AL Jenkins, GS Wong, RG Josse, and A Csima, *Am J Clin Nutr* 40: 1175-1191, 1984
11. J Radzuik, TJ McDonald, D Rubenstein, and J Dupre, *Metabolism* 27: 657-669, 1978
12. G Cahill, *Nutrition Reviews* 44: 40-43, 1986
13. DJA Jenkins, TMS Wolever, RH Taylor, W Kannan, D Sarson, and SR Bloom, *Am J Clin Nutr* 37: 153-154, 1983
14. DJA Jenkins, TMS Wolever, R Nineham, DL Sarson, SR Bloom, J Ahern, KGMM Alberti, and TDR Hockaday, *Diabetologia* 19: 21-24, 1980
15. DJA Jenkins, TMS Wolever, RH Taylor, C Griffiths, K Krzeminska, JA Lawrie, CM Bennett, DV Goff, DL Sarson, and SR Bloom, *Am J Clin Nutr* 35: 1339-1346, 1982
16. A Staub, *Z Klin Med* 91: 44, 1921
17. K Traugott, *Bedentung fur die Lieberfunction Klin Worhenschr* 1: 892, 1922
18. S Holt, RC Heading, DC Carter, LF Prescott, and P Tothill, *Lancet* i: 636-639, 1979
19. AR Leeds, DN Ralphs, P Boulos, F Ebied, GL Metz, J Dilawari, A Elliott, and DJA Jenkins, *Proc Nutr Soc* 37: 23A, 1978
20. I Torsdottir, M Alpsten, D Andersson, RJM Brummer, and H Andersson, *Hum Nutr: Clin Nutr* 38C: 329-338, 1984
21. NS Levitt, AI Vinik, AA Sive, PT Child, and WPU Jackson, *Diabetes Care* 3: 515-519, 1980
22. DG Thompson, DL Wingate, M Thomas, and D Harrison, *Gastroenterology* 82: 51-55, 1982
23. NA Blackburn, JS Redfern, H Jarjis, AM Holgate, I Hanning, JHB Scarpello, IT Johnson, and NW Read, *Clin Sci* 66: 329-336, 1984
24. B Flourie, N Vidon, CH Florent, and JJ Berrier, *Gut* 25: 936-941, 1984
25. SE Schwartz, RA Levine, A Singh, JR Scheidecker, and NS Track, *Gastroenterology* 83: 12-17, 1982
26. DJA Jenkins, TMS Wolever, RH Taylor, HM Barker, H Fielden, and MA Gassull, *Diabetes Care* 4: 509-513, 1981
27. DJA Jenkins, TMS Wolever, AL Jenkins, R Lee, GS Wong, and R Josse, *Diabetes Care* 6: 155-159, 1983
28. K O'Dea, P Snow, and P Nestel, *Am J Clin Nutr* 34: 1991-1993, 1981
29. K O'Dea, PJ Nestel, and L Antonoff, *Am J Clin Nutr* 33: 760-765, 1980
30. DJA Jenkins, MJ Thorne, K Camelon, AL Jenkins, AV Rao, RH Taylor, LU Thompson, J Kalmusky, R Reichert, and T Francis, *Am J Clin Nutr* 36: 1093-1101, 1982
31. MS Goddard, G Young, and R Marcus, *Am J Clin Nutr* 39: 388-392, 1984
32. O Wurzburg, *Nutrition Reviews* 44: 74-79, 1986
33. G Isaksson, I Lundquist, and I Ihse, *Gastroenterology* 82: 918-924, 1982
34. DJA Jenkins, AR Leeds, MA Gassull, B Cochet, and KGMM Alberti, *Ann Int Med* 86: 20-23, 1977
35. DJA Jenkins, AR Leeds, MA Gassull, TMS Wolever, DV Goff, KGMM Alberti, and TDR Hockaday, *Lancet* ii: 172-174, 1976
36. DJA Jenkins, TMS Wolever, R Taylor, D Reynolds, R Nineham, and TDR Hockaday, *Brit Med J* 1: 1353-1354, 1980
37. A Aro, M Uusitupa, E Vontilainen, K Hersio, T Korhonen, and O Siitonen, *Diabetologia* 21: 29-33, 1981
38. U Smith and G Holm, *Atherosclerosis* 45: 1-10, 1982
39. TA Miettinen and S Tarpila, *Clin Chim Acta* 79: 471, 1977
40. DJA Jenkins, D Reynolds, B Slavin, AR Leeds, AL Jenkins, and EM Jepson, *Am J Clin Nutr* 33: 575-581, 1980
41. MS Nachbar and JD Oppenheim, *Am J Clin Nutr* 33: 2338-2345, 1980
42. JH Yoon, LU Thompson, and DJA Jenkins, *Am J Clin Nutr* 38: 835-842, 1983
43. BJ Burke, M Hartog, KW Heaton, and S Hooper, *Hum Nutr: Clin Nutr* 36C: 373-380, 1982
44. HCR Simpson, RW Simpson, S Lousley, RD Carter, M Geekie, TDR Hockaday, and JI Mann, *Lancet* i: 1-5, 1981
45. DJA Jenkins, GS Wong, R Patten, J Bird, M Hall, GC Buckley, V McGuire, R Reichert, and JA Little, *Am J Clin Nutr* 38: 567-573, 1983
46. DJA Jenkins, TMS Wolever, AL Jenkins, RG Josse, and GS Wong, *Lancet* ii: 388-391, 1984
47. DJA Jenkins, TMS Wolever, J Kalumsky, S Giudici, C Giordano, GS Wong, JN Bird, R Patten, M Hall, GC Buckley, and JA Little, *Am J Clin Nutr* 42: 604-617, 1985