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Abstract

Metabolic syndrome is a term linking the clinical profiles of some of the world's major health problems today: obesity, heart disease and diabetes. It is predicated on dietary patterns, and particularly on the delivery of fuel. The effects may be seen first in the development of abdominal obesity and insulin resistance leading to type 2 diabetes mellitus and coronary heart disease. This review examines the role resistant starch might play in the prevention and management of these conditions. Beginning with a definition of resistant starch, a critical review of the scientific literature is presented. Current knowledge suggests that resistant starch in the diet may assist in the prevention and management of conditions associated with the Metabolic Syndrome via its potential effects on delaying the delivery of glucose as fuel with subsequent fat utilisation and appetite control benefits. There is still a great deal of research to be undertaken in this area, but it is clearly warranted given the position of starches in the global food supply and the potential impact on population health.

Keywords

metabolic syndrome, resistant starch, diet

Disciplines

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Diet and Metabolic Syndrome: Where Does Resistant Starch Fit In?

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Metabolic syndrome is a term linking the clinical profiles of some of the world's major health problems today: obesity, heart disease, and diabetes. It is predicated on dietary patterns, and particularly on the delivery of fuel. The effects may be seen first in the development of abdominal obesity and insulin resistance leading to Type 2 diabetes mellitus and coronary heart disease. This review examines the role resistant starch might play in the prevention and management of these conditions. Beginning with a definition of resistant starch, a critical review of the scientific literature is presented. Current knowledge suggests that resistant starch in the diet may assist in the prevention and management of conditions associated with the metabolic syndrome via its potential effects on delaying the delivery of glucose as fuel with subsequent fat utilization and appetite control benefits. There is still a great deal of research to be undertaken in this area, but it is clearly warranted, given the position of starches in the global food supply and the potential impact on population health.

Metabolic syndrome is the name given to a cluster of conditions that are associated with Type 2 diabetes mellitus and coronary heart disease. A strong link has been observed between the development of abdominal obesity, abnormal glucose tolerance (high blood sugar levels despite high levels of circulating insulin, or insulin resistance), high blood pressure, and abnormal blood lipid levels (1). The concept of metabolic syndrome has also been demonstrated on a population level using factor analysis with the related conditions (2). It has been estimated from U.S. census data in the year 2000 that 47 million Americans suffered from metabolic syndrome, with greater proportions in older adults and in Mexican-Americans (3). Prevention of obesity is a major step in addressing the problem. Obesity itself is now a major public health concern throughout the world (4). It is listed in the top 10 global risks to health, with over a billion adults worldwide overweight, and 500 000 people in North America and Western Europe dying from obesity-related deaths each year (5). Current reference standards may be underestimating the problem in Asia (6), where there are concerns about increasing prevalence.

International health organizations and governments are taking the problem seriously, setting up monitoring systems and strategic initiatives (7, 8). There is recognition that environmental triggers have played a large part in the epidemic, where obesity rates have come to reflect normal physiological variability in a pathoenvironment (9). Although the etiology of obesity is complex, the metabolic core of the problem is an imbalance between food consumption and physical activity. This has very significant implications for the development of foods and food products. An expansion of the product range and modernization of production often reflects success; however, where food is concerned, these factors are attributed causes of obesity, particularly with the proliferation of convenience foods (10). This situation is not uncommon in the history of nutrition, where the relationship between food, dietary intake, and social conditions has changed. Earlier developments such as the modernization of agriculture and centralization of the food supply system have been linked to population nutrition health issues (famine in Ireland, pellagra in Italy, heart disease in Finland; 11). Resolution has been possible through thorough analysis of the problem, including its pathophysiological base, and responsive shifts to address the imbalance. The development of functional foods and functional ingredients, such as resistant starch, are pertinent elements in the contemporary analysis. Why people overeat is a vexing problem. The major influences on hyperphagia are seen as palatability (where fat plays a major role), amount of chewing (fiber requires more effort), volume, and satiating properties of the food (12).

Dietary bulk and taste influence intake (13). People tend to eat in standard portion sizes, not standard energy values (14), leading to the belief that energy density is really the key determinant of energy intake (14–16). In fact, cognitive, behavioral and sensory cues related to volume of food can interact with or override physiological cues for energy intake (15). Although there may be a difference between liking and wanting a food, it appears that increased variety combined with increased sensory characteristics has affected satiation (due to reduced habituation), supporting hyperphagia (17). Increased variety and more flavorsome snacks have been linked with increased obesity in the United States, but it is unknown if palatability, energy density, and variety influence energy intake and regulation, and the effect may be food group-specific (17).

The water and fat contents of food have the greatest impact on energy density (and in opposite directions), but in studies of dietary fat, variety, palatability, and fiber may confound results, and cognitive factors (form of food) may also play a part (16). Although a low-fat dietary strategy appears the favored approach to preventing overweight, ongoing research in a variety of experimental domains suggests that low fat plus high fiber may be more effective than either alone (16). This may be due to the metabolic consequences of fiber consumption, where the potential lies in the cascade of events resulting from a slower rate of delivery of glucose. As a component of fiber, if not a related element, resistant starch (RS) may play a role in this scenario. This review looks at why resistant starch is likely to be a positive inclusion in the dietary management of overweight related to metabolic syndrome. Extensive searches were conducted using currently available databases to identify and critique experimental studies involving RS in both short- and long-term interventions.

Definitions of Resistant Starch

RS is the sum of starch and products of starch degradation not absorbed in the small intestine (18). Starch granules provide the primary glucose storage mechanism for most plants. The starch granule is principally composed of 2 glucose polymers, amylose and amylopectin. Starches with a B- or C- type X-ray diffraction pattern, of which high amylose starch is one type, typically have an increased resistance to amylolysis. It is not the structural components of plants that cannot be digested, but rather the starch that slips through and interacts with the colonic microflora to result in the production of short-chain fatty acids (SCFA) in the large bowel. RS1 is that starch physically inaccessible to amylases (partly milled grains, seeds), RS2 is naturally resistant granular starch (raw potato, green banana, high amylose maize), RS3 is retrograded starch (cooked and cooled starch normally associated with cooked foods), and RS4 is chemically modified starches (used in processed foods; 19). In RS4 (20), the chemical modification interferes with the action of digestive amylases. High amylose starch is intrinsically more resistant than a high amylopectin starch (19). The concentration of SCFA (acetate, propionate, butyrate) formed from bacterial action on RS provides some of the indication of levels of RS, and assay procedures have attracted interest (19).

In the past, the quantification of RS has been seen as problematic when it was not considered a distinct chemical entity but rather a set of physical states that alter the rate of digestion of conventional starch (21). It could typically be qualified as part of total dietary fiber using conservative methods (21). Conservative methods approved by the AOAC INTERNATIONAL and similar organizations around the world serve as the basis for food labeling and regulation. To determine RS, AOAC recently approved an in vitro method that quantifies the amount of RS in food at the time of consumption (22). The American Association of Cereal Chemists has now also approved this method (AACC Method 2002.02).

Mechanisms of Action of Resistant Starch

With our current body of knowledge, the primary metabolic effect of RS is likely to be the slow release of glucose into the system, although the consequences of increased SCFAs in the gut from RS consumption have yet to be fully appreciated here. Through its presence in the gut, unabsorbed carbohydrates (CHO) may influence the absorption of other nutrients, which may affect the delivery of fuel, particularly glucose, from a meal. Insulin is secreted in response to rising glucose levels and acts to deliver glucose to cells, lowering blood glucose levels.

Supporting the use of glucose as fuel, insulin also inhibits the use of stored fat. Further, the associated products in circulation act on physiological signaling centers for hunger and satiety. On a metabolic level, a slower delivery of glucose would result in a lowered insulin response which, in turn, would improve access to stored fat, reduce (physiological) hunger, and promote weight management (23, 24). Improved use of fat stores can be observed through a lower respiratory quotient (RQ), a relative measure of oxygen uptake indicative of fat/carbohydrate fuel use. At the mechanistic level, a high RQ (a high reliance on CHO oxidation) after overfeeding may be associated with susceptibility to obesity (25–27), and the slower delivery of glucose at meals may influence consequent consumption and fuel utilization. A number of studies have explored this proposal, but rather than working with the composition of foods themselves, a great deal of this research has used the concept of glycemic index (GI), a ratio of the glycemic response produced by a defined amount of CHO from a food compared to a standard.

Human studies involving single foods, meal tests, and periods on whole diets have shown that relative hyperglycemia and impaired glucose tolerance have been observed after consumption of high GI diets compared with low GI diets (28, 29). However, studies with diets differing in GI have yielded inconsistent effects on whole body insulin resistance (29). Metabolic studies suggest that excess CHO after glycogen storage capacity is reached leads to *de novo* lipogenesis (new fat from CHO; 30), but it does not lead to reduced insulin sensitivity (31). This suggests that glycemic response alone may have less impact on the underlying disorder in the long term.

In the short term, high insulin responses from high GI foods suppress fat mobilization and reduce levels of free fatty acids, which may then promote hunger (32). The amount of dietary fiber may be linked to GI, as the increased satiety found after high fiber meals is also associated with a decreased insulin response, but there may also be other effects such as gastric distension and slow intestinal transit time (33). The possible effects of RS may also be seen here.

The effect of RS on decreasing the absorption rate of glucose has been demonstrated *in vitro* with a glucose control (34), and in test meals in humans ($n = 25$), with a white bread control, where the amylose content of starch (from cornstarch) in test bread was >50% (35). In another test meal study ($n = 5$ males) of raw potato starch (54.1% RS) and pregelatinized potato starch (100% digestible), significant reductions in postprandial glycemia and insulinemia, and short-term subjective sensations of satiety were observed with the RS meal (36). The satiating qualities of the foods would need further work on aspects previously discussed (volume, taste, energy density).

In a similar study of these same 2 foods ($n = 15$ males), RS intake was associated with lower diet-induced thermogenesis (energy costs associated with the metabolism of food) and glucose oxidation, and greater fat oxidation after 5 h ingestion, but adjusting for available CHO left no thermogenic effect (37). The higher fat oxidation may be indicative of the low energy content, but the results suggest a benign effect of RS on diet-induced thermogenesis. A further test meal study comparing RS (Lintner, 70% amylose) with the fibers cellulose and pectin on 6 males, found that RS acted similarly to cellulose in terms of metabolic responses (38), except for the production of SCFA, indicating an added benefit. These studies demonstrate the acute effects of RS on the delivery of glucose, but they do not give an indication of the effect of normal foods, and of mixed meals. The effects on second meals and of habituation would also be of interest.

Bioavailability and test meal studies have shown that it is possible to produce breads enriched with RS which show metabolic benefits (35, 39). It is important to bear in mind, however, that because RS depends on a physical state (21), recipes, portion size, cooking, processes, and cooling conditions still need attention in study design (39). Further consideration also needs to be given with mixed meal and whole diet studies. There are conceptual overlaps provided by GI and dietary fiber effects. For example, a study examining the second meal (lunch) effects of 7 breakfasts defined by GI, dietary fiber (β -glucan), and RS (high amylose barley, raw potato starch) compared to white bread in 10 healthy adults produced varied responses (40). Two of the 4 low GI breakfasts produced improved glucose tolerance in the second meal, and the highest

satiety score was achieved for the low GI + RS + fiber combination. This displays the difficulty in untangling categories and the individual effects of the CHO fraction, but does suggest expected synergies might eventuate. In fact, another more defined test meal study with 0–10% carbohydrate replacement by RS found a substantially greater total 6 h postprandial fat oxidation with 5% replacement, compared to no RS in a controlled 55% CHO meal (41). Both increased fat oxidation, and its reflection of a slower or lesser delivery of glucose would seem desirable in the management of overweight.

A 3-week crossover study of 23 overweight hypertriglyceridemic adults comparing high RS (high amylose) and low RS diets with a high fiber diet found no real effects on fasting clinical indices. A test meal at the end with high amylose starch comprising 33% of the CHO fraction produced significant but biologically small reductions in total postprandial plasma insulin concentrations (42). This study was not able to confirm the earlier results from 24 matched hyperinsulinemic and control subjects in a 14-week crossover trial containing high amylose (70% amylose) or control (30% amylose) core foods and ending with a test meal where 55% of the CHO was the refined starches. Here, chronic consumption of high amylose foods reduced insulin and triglyceride responses (43). The former study did, however, confirm the effect of commercially delivered RS in producing SCFAs, and this under free living conditions.

The form of RS may account for some of the differences seen in results. Where results of a supplemental study saw no difference in GI between RS2 and RS3 supplemented meals and controls, the authors concluded that these ingredients do not deliver “Lente” carbohydrate, and their benefits lie rather with the SCFA-producing properties (44). However, GI is a relative measure of glycemic response based on a 50 g available CHO reference standard, which does not necessarily reflect actual consumption. GI reflects the presence of dietary fiber, as determined by the appropriate *in vitro* method, but does not account for any physiological RS that is not quantified using this method. The glycemic response, which takes into account portion size, may produce different results.

Although all of these pieces of information are helpful, there is still a lagging doubt that not all the potential has been exposed. The principle of slow glucose delivery is desirable, and RS and GI appear to be related (45), but studies in both domains provide equivocal results. Part of the problem is that GI is a concept, not an ingredient, and it is limited by experimental definitions which are not set in free-living dietary consumption terms. Likewise, there may be challenges in measuring total dietary RS, but there is an opportunity to deliver RS into foods that supply substantial amounts of CHO to the population.

Population Nutrition

At the population level, although obesity is moderately inheritable, the rapidly increasing prevalence in the last 20 years could not be the result of genetic mutation in the population, but rather the result of an obesogenic environment (46). Because of its high energy density, dietary fat has been implicated, but shifts in patterns of fat consumption makes the picture difficult to capture (47). A U.S. Department of Agriculture survey of changes in food choice patterns in 1994–1996 ($n = 5649$) found that 45% of consumers ate less fat on meat and skin on chicken and chose chips less frequently, compared to 15% consuming baked/boiled potatoes without added fat, avoiding fat spreads on bread, using low fat rather than regular cheese, and choosing fruit for dessert (48). This pattern is apparent in other analyses showing that dietary sources of fat have moved from meat/dairy foods to fast/fried foods and grain-based mixed dishes (47). By the same token, a Canadian study of dietary patterns and obesity in 301 subjects 18–65 years old, and using principle component analysis, found food patterns leading to increased fat consumption were more likely to include French fries, hamburgers, and hot dogs rather than fatty meat and skin on chicken (49). This suggests that a global view of the whole diet will always be necessary because the effect of individual food choices will only have significance relative to other choices. Although much of the focus in obesity management is on reducing dietary fat, dietary factors other than fat may play an important role in weight regulation (24). Two major cohort studies of men ($n = 42,759$; 50) and women ($n = 65,173$; 51) found that increased glycemic load (indicating

amount and type of CHO) and low cereal fiber were associated with increased risk of Type 2 diabetes mellitus, a condition linked to overweight through insulin resistance (2). Although overweight people appear to consume a higher proportion of energy from fat than CHO, intakes of energy-dense high CHO foods may also lead to obesity (52).

The type of carbohydrate may also be an important consideration. The functions and consequent health benefits of CHO intake depend on the site, rate, and extent of digestion and/or fermentation in the gut (53). Categorizing different forms of CHO is a start to identifying differences. In the first instance, CHO can be described as absorbed or unabsorbed, where the total (at 16 kJ/g) or only part of the energy (at about 8.4 kJ/g) is available. In Western diets, about 89% of CHO is digested, (possibly because it is provided from starchy foods such as wheat and rice), and it provides by far the greatest source of energy (54). However, the type of starch preferred for use in food and industrial applications changes from country to country, depending on the prevailing economics of supply and availability. There are differences in the base starches used for food preparation in various parts of the world. Maize is used in all regions of the world, and almost exclusively in North America and Africa. Significant quantities of tapioca starch are used in Asia and South America. Wheat and potato starches are favored in Europe but are also used in smaller amounts in most other regions. There is now increasing potential to increase the amount of enhanced nutritional starches, such as those from high amylose maize, in foods consumed in all regions. The vast majority of maize starch (>99%) comes from regular or waxy maize. These totals do not reflect what is used in food as opposed to industrial purposes, but it does indicate potential regional differences in starches consumed, particularly between North America, Europe, and Asia. Another look at the dietary patterns of populations with low risk of “diseases of affluence” has shown higher starch, rather than fiber, intakes, indicating that RS may be more important than previously thought (55).

Conclusions

Metabolic syndrome is a term linking the clinical profiles of some of the world’s major health problems today. It is predicated on dietary patterns, and particularly on the delivery of fuel. The effects may be seen first in the development of abdominal obesity and insulin resistance leading to Type 2 diabetes mellitus and coronary heart disease. RS may have an important role to play in the prevention and management of these conditions on a number of levels. Current knowledge suggests this may result from its impact on the slower delivery of glucose, although the impact on metabolism of SCFAs in the gut is an exciting new area under exploration. At the metabolic level, the effect of RS on glucose delivery is likely to have a positive impact on insulin responses and thereby, the utilization of stored fat which, in turn, may influence appetite signals. These effects may influence overall food consumption, in support of energy balance. There is still a great deal of research to be undertaken in this area, but it is clearly warranted, given the position of starches in the global food supply and the potential impact on population health.

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References

- (1) Roth, J.L., Mobarhan, S., & Clohisy, M. (2002) *Nutr. Rev.* 60, 335–337
- (2) Meigs, J.B. (2000) *Am. J. Epidemiol.* 152, 908–911
- (3) Ford, E.S., Giles, W.H., & Dietz, W.H. (2002) *J. Am. Med. Assoc.* 287, 356–359
- (4) Seidell, J. (2000) in *Physical Activity and Obesity*, C. Bouchard (Ed.), Human Kinetics, Champaign, IL, pp 21–29

- (5) WHO (2002) *The World Health Report 2002: Reducing Risks, Promoting Healthy Life*, WHO, Geneva, Switzerland
- (6) Deurenberg-Yap, M. (2002) *Nutr. Diet.* **59**, 231
- (7) NHMRC (2002) *Draft Clinical Guidelines for Weight Control and Obesity Management in Adults*, Australian Government Publishing Service, Canberra, Australia
- (8) Cummings, S., Parham, E.S., & Strain, G.W. (2002) *J. Am. Diet. Assoc.* **102**, 1145–1155
- (9) Salbe, A. (2000) in *Physical Activity and Obesity*, C. Bouchard (Ed.) Human Kinetics, Champaign, IL, pp 69–102
- (10) NHMRC (1996) *Acting on Australia's Weight: A Strategic Plan for the Prevention of Overweight and Obesity*, Australian Government Publishing Service, Canberra, Australia
- (11) Crotty, P. (2002) in *Food and Nutrition*, M.L. Wahlqvist (Ed.), Allen and Unwin, Sydney, Australia, pp 20–33
- (12) Golay, A., & Bobbioni, E. (1997) *Int. J. Obes. Relat. Metab. Disord.* **21**, S2–11
- (13) Seidell, J. (1999) *Eur. Heart J. Suppl.* **1**, S118–S122
- (14) Zock, P.L., & Katan, M.B. (1999) *Br. J. Nutr.* **81**, 421–423
- (15) Rolls, B.J., & Bell, E.A. (1999) *Eur. J. Clin. Nutr.* **53**, S166–173
- (16) Yao, M., & Roberts, S.B. (2001) *Nutr. Rev.* **59**, 247–258
- (17) Raynor, H.A., & Epstein, L.H. (2001) *Psychol. Bull.* **127**, 25–41
- (18) Wisker, E., Daniel, M., Rave, G., & Feldheim, M. (2000) *Br. J. Nutr.* **84**, 31–37
- (19) Topping, D.L., & Clifton P.M. (2001) *Physiol. Rev.* **81**, 1031–1064
- (20) Brown, I.L., McKnight, K., & Moloney, E. (1995) *Food Aust.* **47**, 272–275
- (21) Haralampu, S. (2000) *Carbohydr. Polym.* **41**, 285–292
- (22) McCleary, B.V., & Monaghan, D.A. (2002) *J. AOAC Int.* **85**, 1103–1111
- (23) Brand-Miller, J.C., Holt, S.H., Pawlack, D.B., & McMillan, J. (2002) *Am. J. Clin. Nutr.* **76**, 281S–285S
- (24) Ludwig, D.S. (2000) *J. Nutr.* **130**, 280S–283S
- (25) Horton, T.J., Drougas, H., Brachey, A., Reed, G.W., Paters, J.C., & Hill, J.O. (1995) *Am. J. Clin. Nutr.* **62**, 19–29
- (26) Thomas, C.D., Peters, J.C., Reed, G.W., Abumrad, N.N., Sun, M., & Hill, J.O. (1992) *Am. J. Clin. Nutr.* **55**, 934–942
- (27) Westerterp, K.R. (1993) *Am. J. Clin. Nutr.* **57**, 759S–764S
- (28) Gilbertson, H.R., Brand-Miller, J.C., Thorburn, A.W., Evans, S., Chronodros, P., & Wether, G.A. (2001) *Diabetes Care* **24**, 1137–1143
- (29) Ludwig, D.S. (2002) *J. Am. Med. Assoc.* **287**, 2414–2423
- (30) Lammert, O., Grunnet, N., Fabre, P., Bjornsbo, K.S., Dich, J., Larson, L.O., Neese, R.A., Hellerstein, M.K., & Quistorff, B. (2000) *Br. J. Nutr.* **84**, 233–245
- (31) Macdonald, I.A. (1999) *Eur. J. Clin. Nutr.* **53**, S101–S106
- (32) Roberts, S.B. (2000) *Nutr. Rev.* **58**, 163–169
- (33) Sparti, A., Milon, H., Di Vetta, V., Schneiter, P., Tappy, L., Jecquier, E., & Schutz, Y. (2000) *Am. J. Clin. Nutr.* **72**, 1461–1468
- (34) Ou, S., Kwok, K., Li, Y., & Fu, L. (2001) *J. Agric. Food Chem.* **49**, 1026–1029
- (35) Behall, K.M., & Hallfrisch, J. (2002) *Eur. J. Clin. Nutr.* **56**, 913–920
- (36) Raben, A., Tagliabue, A., Christensen, N.J., Madsen, J., Holst, J.J., & Astrup, A. (1994) *Am. J. Clin. Nutr.* **60**, 544–551
- (37) Tagliabue, A., Raben, A., Heijnen, M.L., Deurenberg, P., Pasquali, E., & Astrup, A. (1995) *Am. J. Clin. Nutr.* **61**, 1070–1075
- (38) Ranganathan, S., Champ, M., Pechard, C., Blanchard, P., Nguyen, M., Colonna, P., & Krempf, M. (1994) *Am. J. Clin. Nutr.* **59**, 879–883
- (39) Hoebler, C., Karinth, C., Chiron, H., Champ, M., & Barry, J.L. (1999) *Eur. J. Clin. Nutr.* **53**, 360–366
- (40) Liljeberg, H.G., Akerberg, A.K., & Bjorck, I.M. (1999) *Am. J. Clin. Nutr.* **69**, 647–655 (
- (41) Higgins, J. (2002) in *Resistant Starch: Recent Research, New Possibilities*, Penford Australia Ltd., Sydney, Australia, pp
- (42) Noakes, M., Clifton, P., Nestel, P.J., Le Leu, R., & McIntosh, G. (1996) *Am. J. Clin. Nutr.* **64**, 944–951
- (43) Behall, K.M., & Howe, J.C. (1995) *Am. J. Clin. Nutr.* **61**, 334–340
- (44) Jenkins, D.J., Vuksan, V., Kendel, K.W., Wursch, P., Jeffcoat, R., Waring, S., & Mehling, C.C. (1998) *J. Am. Coll. Nutr.* **17**, 609–616
- (45) Bjorck, I., Granfeld, Y., Liljeberg, H., Tovar, J., & Asp, N.G. (1994) *Am. J. Clin. Nutr.* **59**, 699S–705S
- (46) Bouchard, C. (2000) in *Physical Activity and Obesity*, C. Bouchard (Ed.), Human Kinetics, Champaign, IL, pp 3–19
- (47) Popkin, B.M., Siega-Riz, A.M., Haines, P.S., Jahns, L., Galuska, D.A., Mendlain, J.M., & Heath, G.W. (2001) *Prev. Med.* **32**, 245–254
- (48) Serdula, M.K., Mokdad, A.H., & Williamson, D.F. (1999) *J. Am. Med. Assoc.* **282**, 1353–1358

- (49) Nolan, C.C., Gray-Donald, K., Shatenstein, B., & O'Loughlin, J. (1995) *Can. J. Public Health* **86**, 389–391
- (50) Salmeron, J., Ascherio, A., Rimm, E.B., Colditz, G.A., Spiegelman, D., Jenkins, D.J., Stampfer, M.J., Wing, A.L., & Willett, W.C. (1997) *Diabetes Care* **20**, 545–550
- (51) Salmeron, J., Manson, J.E., Stampfer, M. J., Colditz, G.A., Wing, A.L., & Willett, W.C. (1997) *J. Am. Med. Assoc.* **277**, 472–477
- (52) Blundell, J.E. & Stubbs, R.J. (1999) *Eur. J. Clin. Nutr.* **53**, S148–S165
- (53) Tharanathan, R.N. (2002) *Crit. Rev. Biotechnol.* **22**, 65–84
- (54) Jones, G.P. (2002) in *Food and Nutrition*, M.L. Wahlqvist (Ed.), Allen and Unwin, Sydney, Australia, pp 183–198
- (55) Topping, D.L. (2002) in *Resistant Starch: Recent Research, New Possibilities*, Penford Australia Ltd., Sydney, Australia, pp .VP, Author's Galley