RESISTANT STARCH - IMPLICATIONS FOR HEALTH

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Summary

Dietary fibre is not the only dietary constituent to resist digestion in the small intestine of humans. Most starch-containing foods contain a fraction of starch which also escapes digestion and arrives in the colon undigested. This undigested starch i.e. resistant starch (RS) may have important implications along the entire length of the gastrointestinal tract.

I. INTRODUCTION

The pioneering work of Burkitt and Trowell (Trowell 1990) emphasised the important role 'dietary roughage' has in protecting against a number of chronic degenerative diseases including cardiovascular diseases, non-insulin dependent diabetes mellitus (NIDDM) and bowel diseases such as bowel cancer, constipation, diverticulosis and haemorrhoids. Indeed the role of dietary fibre in the maintenance of health is now well recognised. However another component of the diet, starch, has received little research attention in relation to these diseases.

Until quite recently it was believed that all starch was digested and absorbed in the small intestine. We now know that this is not the case and, depending on the type of diet consumed, significant amounts of starch reaches the large bowel undigested (Englyst and Cummings 1985; 1986; 1987). This 'resistant starch' (RS) has been defined as 'the sum of starch and products of starch degradation not absorbed in the small intestine of healthy individuals' (European Concerted Action of Resistant Starch [EURESTA], June 1990). In reaching the bowel undigested RS may have similar physiological effects as 'dietary fibre'. This paper will discuss the potential implications to our health of starch reaching the large bowel undigested.

II. IDENTIFICATION OF RESISTANT STARCH

Resistant starch was first identified by Englyst and colleagues while developing a method for measuring dietary fibre as non-starch polysaccharides (NSP) (Englyst et al. 1982). During this procedure Englyst discovered that certain foods which had been processed (i.e. white bread and boiled potatoes) gave higher values for NSP than their uncooked counterparts. This apparent increase in NSP was shown to be due to starch. This 'resistant' starch fraction was defined as starch 'resistant to dispersion in boiling water and hydrolysis with pancreatic amylase and pullulanase'. Resistant starch was only available for enzymic hydrolysis after previous solublisation with DMSO or alkali (Englyst et al. 1982).

This early work by Englyst also recognised that the current AOAC method for measuring 'dietary fibre' also includes some forms of RS in its estimation (see Englyst and

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Cummings 1987). It follows that the health benefits which have been attributed to 'dietary fibre' not be due solely to NSP and may, in part at least, be due to starch. This highlights the need to chemically define 'dietary fibre' as NSP. The NSP definition of 'dietary fibre' is gaining wider acceptance in this field of research, which is essential if the physiological effects attributable to starch and those of NSP are to be clearly identified.

III. CATEGORIES OF RESISTANT STARCH

Four categories of resistant starch have now been identified.

(a) RS_1 - physically trapped starch

This category refers to starch granules that are physically inaccessible to digestion, examples include whole or partly ground grains, seeds, cereals and legumes. This form of RS is released when food is ground, chewed or milled (see Englyst and Kingman 1990). Indeed, one of the consequences of the improved milling techniques developed during this century (aimed at producing a more palatable and refined product), has been to greatly reduce this form of RS in our diet.

(b) RS_2 - resistant starch granules

RS_2 includes ungelatinised starch granules (see Englyst and Kingman 1990). Gelatinisation of starch granules occurs during cooking and food processing in the presence of excess water and heat, where starch granules swell and eventually rupture releasing the starch polymers (amylose and amylopectin) (Holm et al. 1987). Fully gelatinised granules are readily digested. The susceptibility of a starch granule to gelatinisation and digestion depends upon the crystalline nature of the starch granule. The crystalline characteristics of starch granules are in turn determined by X-ray diffraction. Common starches are grouped into three categories; A-type (eg. cereal starch), B-type (eg. raw potato, green banana starch and high amylose maize starch) and C-type (eg. legumes starch) which is intermediate between A- and B-types. The A-type starch granule is digested without cooking while B-type is poorly digested until firstly gelatinised by cooking. Starch granules present in some varieties of high amylose maize require extremely harsh conditions (up to 154-171°C) for complete disruption of the granule. These are temperatures not achieved during conventional cooking practices and, as a result, some starch granules in high amylose maize starches will reach the colon intact. High amylose maize is commonly used in research into the physiological effects of RS, and some ungelatinised starch granules will be measured as 'dietary fibre' using the present AOAC method for 'dietary fibre' determination.

(c) RS_3 - retrograded amylose and amylopectin

This involves the process of re-crystallisation (or retrogradation) of the starch polymers (amylose and amylopectin) which occurs with cooling after gelatinisation. Retrograded amylose is highly resistant to digestion and exhibits an X-ray diffraction which is characteristic of B-type crystalline forms of starch (Miles et al. 1985a; Miles et al. 1985b). RS_3 is solubilised by treatment with DMSO or KOH (Englyst et al. 1982). This form of RS is generated by food processing and examples can be found in bread and cooled boiled potato (Englyst et al. 1982), and also cycles of autoclaving and cooling both high amylose (Sievert and Pomeranz 1989) and wheat (Berry 1986) starches. The current AOAC method for measuring dietary fibre will include retrograded amylose in its estimation of dietary fibre.

(d) RS_4 - chemically modified starch.

This type of starch has recently been included in the definition of RS. While the first three types occur naturally in the human diet. RS_4 is frequently used by the food industry to improve various characteristics of food products including stability on re-heating and changes in pH. These modifications can include cross-linking and substitution (see Bjorck et al. 1988). Chemically modified starches (CMS) are commonly used in the baby food industry (Filer 1988). The various forms of CMS can vary greatly in their degree of digestibility (see Bjorck et al. 1988).

IV. MEASUREMENT OF RESISTANT STARCH

Before any investigations into studying the physiological effects of resistant starch could be conducted, it was clearly important to have a reliable technique for measuring its levels in foods. A number of assays have now been developed (Johansson et al. 1984; Siljestrom and Asp 1985; Berry 1986; Bjorck et al. 1986; Tovar et al. 1990; Englyst et al. 1992; Muir and O'Dea 1993) and some of these have recently been reviewed (see Champ, 1992). Generally all of these approaches involve degradation of starch with alpha-amylase followed by conversion of the resulting products, i.e. dextrins and maltose, to glucose by incubation with amyloglucosidase, and finally glucose determination.

The assay developed in this laboratory (Muir and O'Dea 1993) mimics crudely the conditions of starch digestion. Food is prepared normally and chewed, before incubation with pepsin for 30 minutes, followed by a 15 hour incubation with alpha-amylase and amyloglucosidase at 37°C. Any starch that remains after this process represents RS. This assay has been validated in individuals with an ileostomy (an in vivo model for small intestinal digestion in humans; Muir and O'Dea 1993). A comparison of the in vitro assay and the in vivo ileostomy model is shown in Figure 1. The range of foods tested to date contain a number of different types of RS (RS_1, RS_2, RS_3), and includes a number of individual foods (i.e. rice, pearl barley, Cornflakes, baked beans) and mixed meals (low and high RS meals, see Fig. 1). The high RS meal contained uncooked green banana flour, unprocessed/coarsely milled wheat and high amylose maize kernels (i.e. 'Himaize'). The low RS meal included gelatinised green banana flour, processed/finely milled wheat and low amylose maize kernels (see Fig. 1).

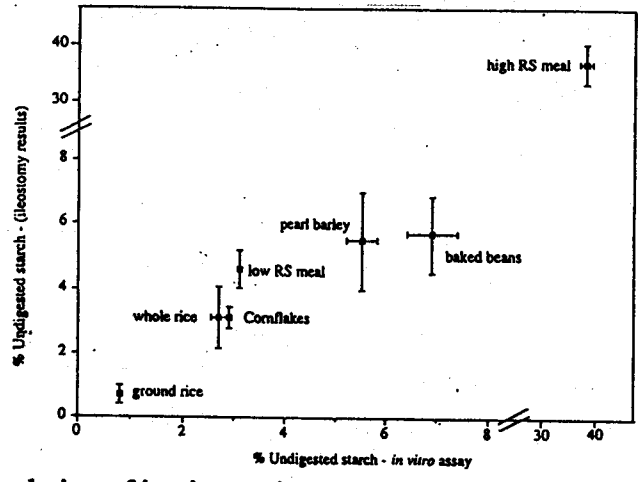

Fig. 1. Correlation of in vitro resistant starch assay with the human ileostomy results for starch escaping digestion (mean ± SE).
([1] Adapted from Muir et al, 1993).

(a) Manipulation of RS in food

Levels of RS present in food can be manipulated by the choice of food processing and varying the plant variety. Food processing techniques which can affect levels of RS include; milling, cooking and cooling cycles, pH changes and autoclaving. Because there is a direct relationship between the amylose content of a cereal and the levels of RS (Sievert and Pomeranz 1989; Berry 1986), it follows that plant breeding programs aimed at increasing the amylose content of grains and cereals will correspondingly also increase their RS content. This has been achieved quite successfully with varieties of maize. These principles of food processing and choice of plant variety were used to greatly affect the levels of RS in the range of food shown in Figure 2.

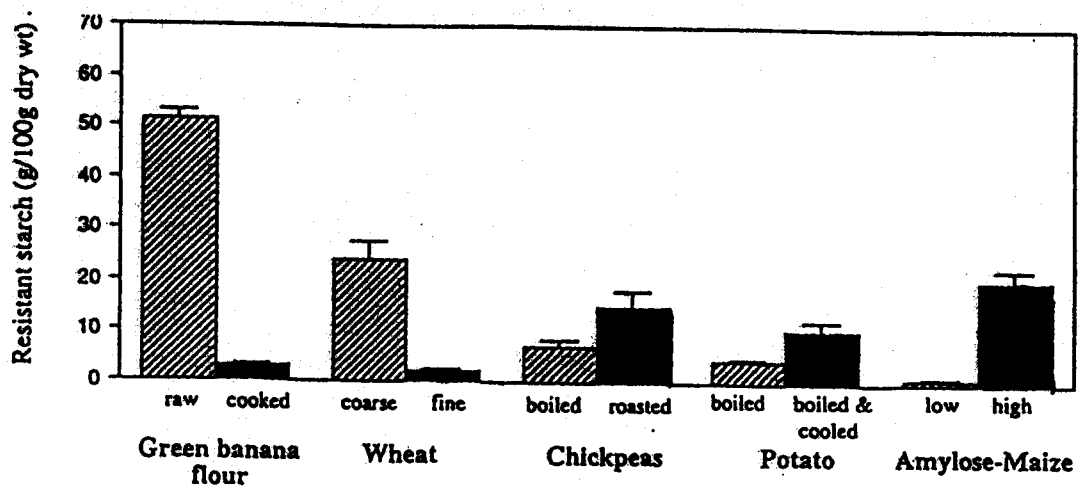

Figure 2. Manipulation of resistant starch levels in a range of starch-containing foods[1] ([1] Adapted from Muir et al, 1993).

There is little data on the levels of RS in a typical Western diet, although estimates have been made that approximately 5% of the starch in the U.K diet is resistant to digestion. In Australia the consumption of starch is low, around 110g per day (CSIRO Division Of Human Nutrition 1993). Because the major dietary sources of starch in the Australian diet are cereal products, potatoes, rice and bread, all of which have a level of RS of approximately 2-3% (see Englyst and Cummings 1985; 1986; 1987; Muir and O'Dea 1993), it is expected that roughly 3% of the starch in a typical Australian diet is RS. This level of RS could be raised through increasing the quantity of starch consumed, modifying the way in which the food has been processed and choice of plant variety. Figure 3 compares the levels of RS and NSP in a range of

commonly used foods. It can be seen from this figure that, depending on the foods consumed, RS can make a significant contribution to the human diet.

However, before recommending the need to increase the RS content of the diet it is clearly important to established what (if any) physiological effects the consumption of RS does have, and whether or not RS shares any health benefits with NSP.

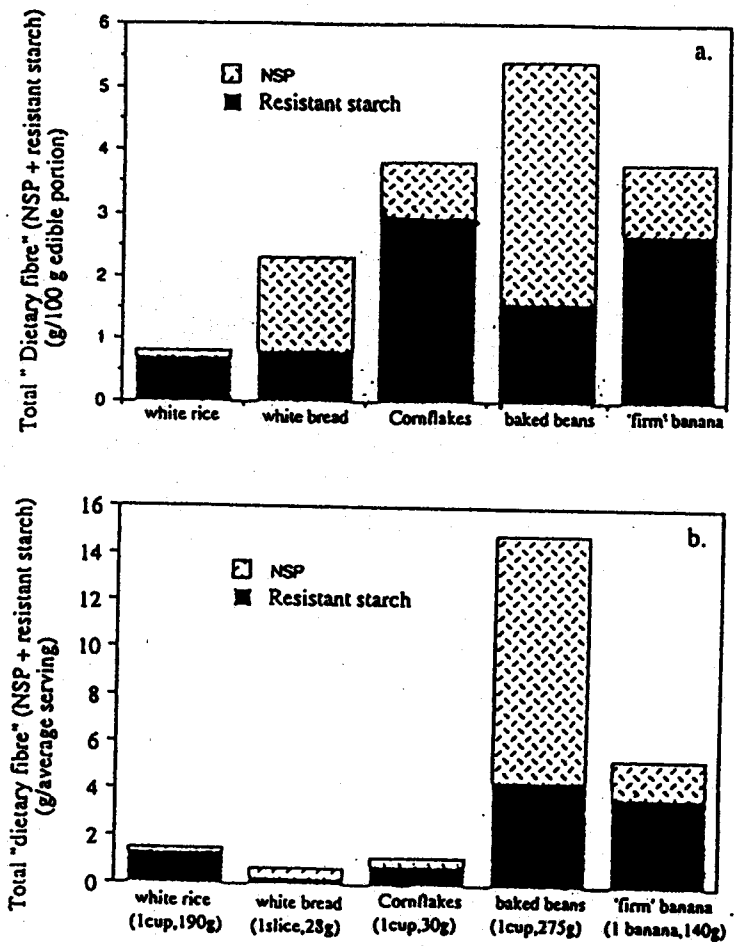

Figure 3. Levels of resistant starch and NSP in a range of foods. ([1] Adapted from Muir et al. 1993).

IV. SMALL INTESTINAL EFFECTS OF RESISTANT STARCH

Carbohydrate-rich foods which are slowly digested and absorbed are also associated with an increased starch malabsorption (Jenkins et al. 1987). Indeed, up to 20% of ingested carbohydrate has been shown to be mal-absorbed in individuals with an ileostomy who consume slowly digested starch-rich foods (Jenkins et al. 1987).

Starch-containing foods that are slowly digested and absorbed along the length of the small intestine result in reduced post-prandial glucose and insulin responses (Jenkins et al. 1987). The therapeutic potential of slowed rates of carbohydrate digestion for patients with NIDDM has been investigated by selecting starch-containing foods which are slowly absorbed

or by inhibiting alpha-glucosidase activity with Acarbose (Jenney et al. 1993; Hanefield et al. 1991; Brand et al. 1991; Wolever et al. 1992). Both of these approaches have been demonstrated to result in significant improvements in glycaemic control and lipid levels in subjects with NIDDM (Jenney et al. 1993; Hanefield et al. 1991; Brand et al. 1991; Wolever et al. 1992). Because these slowly digested and absorbed carbohydrates also produce improvements on lipid profiles (i.e. lowering cholesterol and triglyceride), suggests that foods that lower the glycaemic impact of a meal may also have long-term benefits in the treatment of hyperlipidaemia.

Starch-containing foods produce lower glycaemic responses for a range of reasons including: the physical form or the food (e.g. pureeing, grinding, chewing); the starch structure (e.g. amylose/amylopectin ratio, degree of gelatinisation); food processing (thermal extrusion, milling, canning, cooking/cooling cycles); composition of the meal or food (e.g presence of fat, soluble NSP, protein,); antinutrients (e.g. phytic acid) and the presence of amylase inhibitors (see O'Dea 1990). A number of these factors will also have a significant impact on the levels of RS. Indeed, one possible explanation for the lower glycaemic and insulinemic responses after meals containing foods high in amylose starch (Goddard et al. 1984; Behall et al. 1988; Brand Miller et al. 1992) is the formation of RS produced after the heating and subsequent cooling of the high-amylose product to form RS_3.

The presence of significant amounts of RS in food will produce lower glycaemic and insulinemic responses merely by lowering the level of the starch available for digestion. It follows that there is the potential for foods high in RS to provide another dietary means by which the glucose and insulin responses can be blunted.

(a) Colonic fermentation

There may be certain benefits to be derived from starch reaching the large bowel. In the colon undigested starch (and NSP) undergo fermentation by the resident luminal anaerobic bacteria (MacFarlane and Cummings 1991). The products of fermentation include; gases (CO_2, CH_4, H_2), the short chain fatty acids (SCFA, acetate, propionate and butyrate), lactate and branched chain fatty acids (isobutyrate, isovalerate) (MacFarlane and Cummings 1991). SCFA are the principal end-products of colonic fermentation, with acetate the major SCFA found in the human colon. The SCFA are produced in the approximate molar ratio of 60:25:15 (for acetate, propionate and butyrate respectively), although this can vary depending on the nature of the polysaccharide being fermented (see below). SCFA are rapidly absorbed from the colonic lumen, where most of the butyrate is utilised as an important energy source by the colonocyte- the main cell type in the colon (Roediger 1982). Acetate and propionate enter the portal blood stream and are transported to the liver.

Some have suggested that the metabolism of SCFA may contribute to improvements in the glycaemic and lipid metabolism in diabetic subjects fed diets high in starch and dietary fibre (Venter and Vorster 1989; Anderson and Bridges 1984; Chen et al. 1984). For example, it has been shown in animal models that intragastric infusions of acetate reduce plasma glucose levels (Asplund et al. 1985) and intra-portal infusions of propionate stimulate insulin secretion (Brockman 1982). In contrast, rectal infusions of SCFA have not had this effect (Wolever et al. 1991, Wolever et al. 1989), possibly suggesting that site of absorption is an important factor in these systemic effects. There is also some evidence that propionate may inhibit lactate gluconeogenesis (Anderson and Bridges 1984), resulting in reduced glucose output and inhibition of hepatic cholesterol synthesis directly (Anderson and Bridges 1984; Wright et al. 1990: Wolever et al. 1991). Acetate, the principal SCFA in the peripheral circulation, has been shown to stimulate the rate of gluconeogenesis in rat hepatocytes (Anderson and Bridges 1984). Carbohydrate fermentation has also been associated with enhanced suppression of hepatic glucose production and free fatty acid levels in healthy subjects (Thorburn et al. 1993).

V. LARGE INTESTINAL EFFECTS OF RESISTANT STARCH

Because RS reaches the colon one of the most immediate questions is whether or not RS behaves like other 'dietary fibres' in influencing bowel function. We have recently completed a study in which healthy individuals were fed three high RS meals over a period of 24 hours. There was a significant increase in breath hydrogen and the serum SCFA-acetate consistent with increased bacterial activity in the colon (submitted).

(a) Products of colonic fermentation and bowel health

SCFA have several possible actions relevant to the health of the large bowel. They are rapidly absorbed by the colonic mucosa promoting water and sodium absorption (Ruppin et al. 1980), thereby preventing osmotic diarrhoea. The production of SCFA and lactate leads to the acidification of the lumen (see MacFarlane and Cummings, 1991). The pH can reach as low as 4.8-5.0. This in turn can affect other processes including (Gustafsson 1982; Schulz et al. 1993): absorption of magnesium and calcium, the ionisation of SCFA, epithelial proliferation, the balance of bacterial species, the bacterial metabolism of bile salts and ingested carcinogens and the activity of bacterial enzymes, eg. β-glucosidase, β-glucuronidase, (responsible for de-conjugation and re-activation of potential mutagens). It is believed that some of these actions provide a degree of protection against bowel cancer (Bingham 1990). Instillation of SCFA have also been used successfully in the treatment of post operative complications of colonic surgery such as ileal pouchitis and diversion colitis and may also be beneficial in the treatment of ulcerative colitis (Rabassa et al. 1992). In addition, there is some evidence that SCFA may also have effects on the colonic motility. Both propionic and butyric acids have been shown to induce contraction in isolated rat muscle strips (Yajima 1985).

Butyrate may be important for the metabolic welfare of the epithelium of the large bowel. Butyrate is absorbed by a specific transport system on the colonocyte (Mascolo et al. 1991), this process is enhanced at lower pH (Reynolds et al. 1993). The absorption of butyrate also stimulates sodium absorption and is associated with bicarbonate excretion (Reynolds et al. 1993). Butyrate also has a range of effects which may be particularly relevant to bowel cancer. Butyrate 'stabilises' DNA via effects on histone deacetylase and methylation of DNA (Candido et al. 1978), it induces differentiation in a range of mammalian cells including colorectal cancer cell lines (Whitehead et al. 1987) and reduces their growth rate. Butyrate has also been shown in vitro to down-regulate oncogenes c-ras and c-mvc (see Young 1991).

Both in vivo (Englyst et al. 1987) and in vitro (Scheppach et al. 1988) studies have shown starch to be a richer source of butyrate via fermentation than the highly fermentable soluble-NSP. For example, in batches of human faecal slurries, 29% of the total SCFA production is butyrate when starch is the substrate, compared with 8%, 3% and 2% from arabinogalactan, xylan and pectin respectively (Englyst et al. 1987) . We have similar evidence in rats fed four diets; (i) low RS, (ii) high RS (RS_2 - high amylose maize ['Hi-maize']), (iii) guar gum and (iv) cellulose. The results clearly demonstrated that guar and the high RS diets were highly fermentable and produced the highest levels of SCFA in both the caecal and faecal samples. The high RS produced, preferentially, an increase in the total pool of butyrate as well as a higher molar ratio of butyrate (63:14:23 for acetate:propionate:butyrate) in the faeces when compared to the control (75:15:10), while guar preferentially increased propionate (57:30:13) when compared to the control (low RS) diet (manuscript in preparation).

This high production of butyrate when starch is fermented in the colon has also been demonstrated in humans. Individuals fed acarbose, a glucosidase inhibitor which causes starch malabsorption, produced a 50% increase in the amount of butyrate recovered in the faeces (Scheppach et al. 1988). Moreover, one recent report (Munster 1992) found that the addition of a high amylose maize starch drink (ie. high in RS) to a diet reduced cell proliferation, raised SCFA (including butyrate) and lowered bile acid excretion in the faeces of human subject.

(b) Effect of resistant starch on bowel function

Undigested starch has been reported to have a range of effects in the large bowel; it increases faecal bulking, lowers colonic pH, and increases colonic and faecal SCFA and lactate concentrations (see Muir et al. 1993).

We are currently completing a study into the effect RS has on bowel function in humans. In this study two diets which differed greatly in resistant starch concentration were fed to 11 human volunteers with no history of bowel disorders or carbohydrate intolerances. The high RS diet included cornbread made from high amylose maize kernels ('Hi-maize'), coarsely ground unprocessed wheat seed and uncooked green banana flour. The low RS diet contained low amylose maize kernels, processed wheat and cooked green banana flour (see Fig 2). The 'dose' of RS was assigned on the basis of usual energy intake (2 g RS/420 kJ of energy). An average of six and 41 grams of RS was given for the low RS and high RS dietary period respectively. The two diets were fed to the volunteers for three weeks each, in a randomised crossover design. Dietary food diaries were kept throughout the study to monitor compliance to the protocol. Levels of macronutrients (ie. carbohydrate, fat, protein and both soluble and insoluble-non starch polysaccharides) were held constant and only RS levels changed for both dietary periods. Three day faecal collections were carried out during the third week of each study period.

Greater levels of starch were detected in the faeces of subjects following the high RS diet, confirming that the diet was successful in achieving significant levels of starch reaching the colon. The results collected to date show that RS; had a mildly laxative effect, produced a change in the consistency of the stools (ie. softer) and had a marked impact on lowering faecal pH (6.9 ± 0.1 reduced to 6.3 ± 0.1, [mean $\pm$ SE, n=11] for the low RS and high RS diets respectively). Total faecal SCFA's were also increased during the high RS diet. This increase was mainly due to changes in acetate and butyrate, while levels of propionate, iso-butyrate, iso-valerate and valerate were not significantly affected (manuscript in preparation). There was also a good correlation between levels of starch in the faeces and butyrate excreted, consistent with the suggestion that starch is a good substrate for the production of butyrate, via fermentation. There was however, no significant change between the two diets in the levels of lactate (both D- and L- forms) in the faeces (manuscript in preparation). Participants also recorded a greater incidence of flatulence during the high RS diet. Indeed the levels of starch present in the faeces (5%) indicated that the majority (approximately 95%) of the RS fed to the subjects was fermented.

These results suggest that RS is having effects on the gastro-intestinal tract in humans which are comparable to some fermentable dietary fibres.

VI. THE FUTURE

Future work in this area must concentrate on the different types of RS which may have different physiological effects. Already come differences are becoming apparent between raw potato starch and raw high amylose maize starch in effects on bowel physiology in the rat. Regional sites of fermentation may have important implications, particularly in relation to colon cancer. Consequently it is important to study events occurring in both the proximal colon and distal colon. Also the interactions between RS and other components of the diet (i.e. protein and soluble-NSP and insoluble-NSP) must be considered.

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