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# The physiological roles of dietary fibre

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ABSTRACT

The term dietary fibre (indigestible carbohydrates of plant origin) encompasses a range of divergent compounds that differentially affect numerous important gastrointestinal and systemic bodily processes.

The main role of the gut is to absorb nutrients following digestion. Complex neurohumoral pathways control gut secretion and motility. Dietary fibres that inhibit intestinal digestive processes result in decreased upper GI transit times, which may affect satiety and satiation.

The large intestine houses a varied microflora. Dietary fibre is a major energy source for these bacteria, and therefore markedly affects microfloral diversity/toxicity. Dietary fibres can also affect innate immune responses of the gut mucosa both directly and indirectly.

Dietary fibre impacts all processes of the gut, which as a result may impact on cardiovascular/systemic health. As many commonly-used hydrocolloids are viscous, palatable dietary fibres, they have the potential to be used in acceptable foodstuffs that offer a wide range of added health benefits.



## 1. Introduction

During the 1950s–1970s, a series of independent observational studies suggested that dietary fibre intake was associated with a number of health benefits (Burkitt, 1952; Burkitt, Walker, & Painter, 1972; Cleave, 1956, 1968; Trowell, 1972, 1973, 1978). These observations highlighted previous historical observations on health benefits of consuming fibre-rich foods, such as those of Hippocrates and Dr. Allinson, and resulted in a rapid expansion of research into the epidemiology of dietary fibre intake and disease, and potential physiological mechanisms for reduced disease risk. This increased scientific interest quickly led to public and food industry acceptance of dietary fibre as being a beneficial nutrient to include within a healthy diet.

The term dietary fibre refers to a vast range of biophysically and biochemically divergent compounds, with varying effects on physiological parameters. It is difficult to attribute all health benefits of natural, fibre-rich foods noted in observational studies solely to their fibre content *per se*, for fibre supplementation of foods to be considered as a means to provide added health benefits. Therefore, it is necessary to consider how the physicochemical properties of different fibre types and sources impact on gastrointestinal and systemic physiology. This review will outline some of the key processes of the gut, and summarise previous evidence that different dietary fibres modulate these factors to varying degrees. In addition, this review will highlight the need for nutrition scientists to work more closely with the food industry to increase the potential for the development of safe, palatable and targeted fibre-enriched food products with proven health benefits that can be used to combat major health challenges such as obesity, cardiovascular disease and type II diabetes. Due to the association between dietary fibre consumption and human health, this review will focus on evidence in human studies, but will also discuss evidence from other appropriate models where relevant.

## 2. What is dietary fibre?

The term dietary fibre was first used in 1953 by Eben Hipsley in his observation publication noting populations with diets high in fibre-rich foods tended to also have lower rates of pregnancy toxaemia (Hipsley, 1953). Previously the analytical term "crude fibre" had been used to the portion of plant foods that escaped solvent, acid and alkali extractions (Trowell, 1976). "Dietary fibre" was first postulated to be "unavailable" plant material (i.e. that which escaped digestion and absorption in the human upper GI tract (Asp, 1987)). Conflict between what to define as dietary fibre, and how to analyse this, have made dietary fibre classification for public health and food industry purposes less defined (Buttriss & Stokes, 2008; McCleary et al., 2008). This has tended to lead the classification away from the original hypotheses and observations of dietary fibre intake within natural, unrefined foods resulting in health benefits. However, it must be noted that four key factors can be attributed to the range of physiological effects that dietary fibres bring about:

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1. The rheological/biophysical properties of dietary fibre within the gut/simulated gastrointestinal conditions, often referred to as "viscosity"(Dikeman & Fahey, 2006; Jenkins, Kendall, Axelsen, Augustin, & Vuksan, 2000).
2. The function of fibre within foods as a matrix (Englyst & Englyst, 2005; McDougall, Morrison, Stewart, & Hillman, 1996).
3. The biochemical characteristics of various dietary fibres, and their specific effects.
4. The effect of dietary fibres on the large bowel microfloral diversity and the associated by-products of fermentation.

As a result, this review will include dietary fibres or putative dietary fibres that have such functions, and will discuss the importance of each of these functions on the physiological impacts of dietary fibres.

## 3. Gastrointestinal absorptive and digestive processes

The main function of the gastrointestinal tract is to absorb nutrients from ingested foods. This function is preceded by a series of digestive processes that occur within different gut compartments. These enzymatic processes are governed by secretion of enzymes and associated co-factors, and through maintenance of the gut lumen at optimal pH conditions for digestion (Allen, Flemstrom, Garner, & Kivilaakso, 1993; Hersey & Sachs, 1995; Nauntofte, 1992). Gastrointestinal secretion of enzymes and other factors, alongside control of gut motility is governed by a series of complex neurohumoral pathways (summarised in Table 1) that tend to be driven by luminal content. Two main features of luminal content which appear to drive a major portion of gastrointestinal physiology are luminal chemical profile and luminal bulk. The nutrient/chemical profile of the gut lumen is sensed by specialised chemosensor enteroendocrine cells within the epithelium (Sternini, Anselmi, & Rozengurt, 2008), while mechanoreceptors (stretch activated neural cells) occurring within the myenteric and submucosal plexusues (Furness, 2000) are activated as a result of mechanical pressure from luminal contents.

The main absorptive area in the gut is the small intestine, which, from a dietary stand-point, is involved in the absorption of the subunits of digestible macronutrients (i.e. amino acids and some di/tripeptides from proteins, monosaccharides from carbohydrates and fatty acids/glycerol from di/triglycerides), as well vitamins, minerals and other micronutrients.

Ingested foods must be mechanically homogenised with digestive secretions in order to allow better hydrolysis of macronutrients, and, in some cases, to allow micronutrient release. Mastication in the buccal cavity begins this process, mixing food with salivary secretions. Within these secretions, α-amylase acts to commence digestion of starches (Pedersen, Bardow, Jensen, & Nauntofte, 2002; Schenkels, Veerman, & Amerongen, 1995). Salivary secretions also lubricate the passage of food along the oesophagus, due to the presence of fluid (Pedersen et al., 2002) and salivary mucins (Tabak, 1995).

Food boluses entering the stomach are maintained there for mixing with gastric secretions. A strongly acidic secretion allows denaturation of proteins and solubilisation of other factors. Gastric proteases (mainly pepsin) cleave amino bonds in proteins to form a range of shorter peptides and amino acids. Gastric lipase initiates digestion of dietary lipids, accounting for 10–40% of total triglyceride hydrolysis in healthy, adult individuals (Embleton & Pouton, 1997). The stomach also releases intrinsic factor from parietal cells, which must bind to vitamin B12 before this vitamin can be absorbed further down the gastrointestinal tract (Markle, 1996). By the time the majority of luminal contents leave the stomach, they have been processed into a creamy, homogenous slurry, known as chyme.

As luminal contents appear in the upper section of the small intestine (the duodenum), they are met with alkali (bicarbonate-rich) secretions from the liver, pancreas and intestinal crypts. Pancreatic exocrine secretions also contain a myriad of enzymes for digestion of all macronutrients (see Table 2), while hepatic secretions contain bile and other factors that act to emulsify lipids, greatly increasing their available surface area for pancreatic lipase action (Miled et al., 2000). Di- and trisaccharides are hydrolysed by α-glucosidase and other brush border-bound enzymes to convert them into monomers.

Monosaccharides, amino acids, minerals and water-soluble vitamins are transported across the luminal intestinal epithelium by specific carrier proteins. These uptake pathways are generally driven by a $Na^+$-gradient from lumen to serosa. Fat-soluble vitamins (Vitamins A, D, E and K), glycerol and fatty acids are all believed to diffuse through the hydrophobic barrier of the epithelium.

Small intestinal structure is ideally suited to a large absorptive capacity. The presence of the plicae circularis and villi increase the luminal absorptive area around 20–30 times of that of a smooth cylinder (Moffett, Moffett, & Schauf, 1993). A further increase in surface area is afforded to intestinal epithelia by the presence of a brush border (made up of around 2 billion microvilli/$cm^2$). This further increases the surface area of the small intestine by 20–40-fold (Moffett et al., 1993), providing an approximate surface area of 200 $m^2$ (Snyder et al., 1975), compared with ca. 0.05 $m^2$ in the stomach and ca. 6 $m^2$ in the large intestine (DeSesso & Jacobson, 2001; Snipes, 1997). Due to intestinal mixing, the epithelium has abundant contact with luminal contents to facilitate absorption.

Outside of the small intestine, some simple dietary factors, like glucose and alcohol may be absorbed more proximally (Manning & Evered, 1976; McIntyre & Thompson, 1992; Wilkinson, 1980). The large intestine is involved with salvage of low levels of other nutrients remaining luminally (Kunzelmann & Mall, 2002; Nordgaard & Mortensen, 1995). However, the main absorptive capacities of the large intestine are in the uptake of water (Ma & Verkman, 1999; Sandle, 1998) and microfloral metabolites, including short-chain fatty acids (SCFA), ammonia and urea. The action of dietary fibre on colonic SFCA absorption is dealt with later alongside its effects on the intestinal microflora (see Section 8).

## 4. Effects of dietary fibres on digestion and absorption

Classically, it has been postulated that the presence of any dietary fibre in the upper GI tract will result in a decreased rate of intestinal uptake of a range of nutrients. However, as the term covers a widely expanding range of moieties, it is necessary to consider what physicochemical factors of dietary fibres are important in these roles.

It appear that dietary fibre which are water-soluble and are either viscous or gel-forming under gastric/intestinal conditions reduce absorption rates more than low molecular weight, low viscosity fibres. Previous *in vitro* studies into the effect of dietary fibres on casein digestion by gastrointestinal proteolytic enzymes, showed that higher concentrations of dietary fibres in the casein solution resulted both in measured increased viscosity and reduced digestion rates over 6 h (El Kossori et al., 2000). In previous animal studies, Kimura et al., (1996) noted higher levels of cholesterol excretion in rats fed diets containing 1000 mg/kg of degraded alginates with molecular weights of 5 and 10 KDa compared to the effect of a diet with a lower molecular weight (1 kDa) alginate or a control (no fibre) diet (Kimura, Watanabe, & Okuda, 1996). While

**Table 1**
Overview of the major processes involved in digestion.

| Digestive process | Neurohumoral mediation of process | | | Description |
|---|---|---|---|---|
| | Major mediator(s) | Released from | Stimulus | |
| Cephalic phase | Acetylcholine | Vagal efferents | Thought, smell or sight of food | Drive salivary gland secretion and acid/pepsinogen secretion in stomach |
| Receptive relaxation | Acetylcholine | Vagal efferents | Food bolus entering stomach | Relaxation of stomach smooth muscle to increase luminal volume ready for meal |
| Gastric accommodation | Acetylcholine | Vagal efferents | Gastric distension | Relaxation of proximal stomach |
| Gastric mixing | Gastrin | Gastric G cells | High gastric pH, luminal $Ca^{++}$ and amino acids[a] | Potentiation of acid and pepsinogen release |
| | Acetylcholine | Vagal efferents | Gastric distension | Slow contractions in fundus drive grinding of stomach contents and mixing with digestive secretions |
| Gastrocolic reflex | Acetylcholine | Vagal efferents | Gastric distension | Increased motility of distal gut (ileum and colon) |
| Gastric emptying | Motilin | Intestinal enteroendocrine cells[b] | Possibly alkali in duodenum | Relaxes pyloric sphincter to increase gastro-duodenal flow rate |
| Gastro-ileal reflex | Unsure, possibly gastrin and motilin | Unsure | Unknown | Relaxation of ileocaecal sphincter |
| Duodenal digestion | Cholecystokinin | I cells in upper small intestine | Fats, amino acids[c], glucose or conjugated bile acids in intestinal lumen | Limits gastric emptying. Increases pancreatic enzyme secretion. Drives bile output from gall bladder. Increases intestinal motility |
| | Gastric Inhibitory peptide (GIP) | Duodenal and jejuna K cells | Fat, glucose, amino acids in intestinal lumen | Limits gastric emptying Increases gastric motility |
| | Secretin | Duodenal S cells | Fat and low pH in intestinal lumen | Limits gastric emptying. Increases bicarbonate secretion into intestine |
| Duodenocolic reflex | CCK | Duodenal I cells | Fat in intestinal lumen | Increased motility of distal gut (ileum and colon) |
| | GIP | Duodenal K cells | | |
| | Secretin | Duodenal S cells | | |
| Myenteric reflex | Acetylcholine and other neruotransmitters[d] | Enteric motor neurones | Luminal distension | Local stimulation drives intestinal peristalsis |
| Ileal brake | GLP-1 | Ileal L cells | Lipid and carbohydrate in ileal lumen | Reduction of small intestinal and gastric motility and digestive secretion |
| | PYY | Ileal L cells | | |
| Colonic brake | GLP-1 | Colonic L cells | Colonic luminal nutrients | |
| | PYY | Colonic L cells | | |

Table compiled with particular reference to (Buchan, 1999; Furness, 2000; Heijboer et al., 2006; Moffett et al., 1993; Wank, 1995).
[a] Tryptophan and phenylalanine have largest stimulatory effect.
[b] Occassionally referred to as M cells, but not to be confused with immunoregulatory M cells.
[c] Histidine and arginine have largest stimulatory effect.
[d] For further details, see review by Furness (2000).

such absorption-lowering effects can be beneficial in reducing energy uptake, it must also be noted that such factors are also likely to reduce the bioavailability of minerals, vitamins and phytochemicals. Kim et al., (1996) noted an increased Fe-uptake in rats fed a low molecular weight (89 kDa), highly esterified (75%) pectin compared with native, (higher molecular weight, less esterified) pectins (Kim, 1998; Kim, Atallah, Amarasiriwardena, & Barnes, 1996).

Few studies have assessed whether the presence or absence of a natural food matrix affects absorption rates of nutrients. While whole grain foods contain all the bran, wheat and germ components of their original grain source, it is normal that these components are separated and reconstituted following milling (Slavin, Jacobs, & Marquart, 2000). While this procedure tends to increase nutrient availability from these foods, as well as product safety, it also results in the vast majority of whole grain food products not having an integral food matrix. Previously published tables of the glycaemic indices of a range of foodstuffs show that there is very little difference between those cited for whole grain and refined grain sources (Atkinson, Foster-Powell, & Brand-Miller, 2008; Foster-Powell, Holt, & Brand-Miller, 2002). These data clearly suggest that the fibre content *per se* of foods does not directly equate to a food's glycaemic index. Evidence of the importance of a food matrix to the glycaemic index of grain foods comes from studies by Jenkins et al. (1988). Within these studies, the glycaemic response of adults with diabetes mellitus to consuming breads with varying levels of intact/cracked grains (i.e. unmilled) was assessed. Breads containing up to 75% intact/cracked grains were considered palatable, and the higher the level of these grains, the lower the mean glycaemic index. As an example, a wholewheat bread from this study had a glycaemic index of 92 ± SEM of 1, whereas a 75% intact/cracked grain (bulgur wheat) bread had a glycaemic index of only 69 ± 4 (Jenkins et al., 1988). While the removal of the fibre matrix of apples (as a result of pureeing or juicing) did not affect the postprandial appearance of glucose (or total sugars) in the plasma after a meal containing 60 g of available apple carbohydrate, a much larger insulinaemic response to the ingestion of juiced or pureed apples resulted in a subsequent fall in fasting glucose concentrations (Haber, Heaton, Murphy, & Burroughs, 1977). The loss of apple fibre matrix also resulted in decreased levels of satiety noted by participants within this study.

Hydrocolloid inclusion in foods has long been used to benefit product consistency or texture. Many food hydrocolloids are also dietary fibres by definition, and can be used to restructure foods (i.e. form a novel fibre-based food matrix), while at the same time offering highly acceptable food products to the consumer. Small human feeding studies have suggested that inclusion of food hydrocolloids like alginates (Torsdottir, Alpsten, Holm, Sandberg, & Tolli, 1991), guar gum (Fairchild, Ellis, Byrne, Luzio, & Mir, 1996; Leclèlre et al., 1994; Sierra et al., 2001; Torsdottir, Alpsten, Andersson, & Einarsson, 1989) and beta-glucan (Behall, Scholfield, & Hallfrisch, 2006; Jenkins, Jenkins, Zdravkovic, Wulrsch, & Vuksan, 2002; Nazare et al., 2009; Tosh, Brummer, Wolever, & Wood, 2008;

**Table 2**
Secreted factors that govern macronutrient digestion in each gastrointestinal segment.

| Segment and factor | Released from | Action | Approximate time for action |
|---|---|---|---|
| *Buccal cavity* | | | A few seconds |
| α-amylase | Salivary gland serous cells | Cleaves α-1,4 glycosidic linkages in starch | |
| *Stomach* | | | 1–4 h |
| Hydrochloric acid | Parietal cells | Denatures proteins, releasing bound nutrients | |
| Pepsin | Peptic (chief) cells | Cleaves peptide bonds in polypeptides | |
| Gastric lipase | | | |
| *Small intestine* | Pancreatic acinar cells | | 2–8 h |
| Trypsin | | Cleaves peptide bonds in polypeptides | |
| Chymotrypsin | | | |
| Carboxypetidases | | Cleaves peptide bonds at carboxyl end of polypeptide | |
| Aminopetidases | | Cleaves peptide bonds at carboxyl end of polypeptide | |
| Elastase | | Elastin digestion | |
| Collagenase | | Collagen digestion | |
| Pancreatic lipase | | Cleaves ester bonds in triaclyglycerol micelles at lipid:water interface | |
| Hydrolase | | Digests cholesterol esters | |
| Pancreatic amylase | | Cleaves α-1,4 glycosidic linkages in starch | |
| Bile acids | Hepatic acinar cells | Emulsification of lipids to increase lipid:water interface area | |
| Large intestine | Mixed colonic microflora | Ferment dietary fibre to absorbable short-chain fatty acids | 1–3 days |

Yokoyama et al., 1997) into test meals results in a blunting of postprandial glycaemic and insulinaemic responses.

## 5. Gastrointestinal motility and its control

The neural circuitry of the gut – the enteric nervous system – contains nearly as many neurones as the central nervous system (Otterson & Sarr, 1993), offering insight into the complexity of the control of gut function. Parasympathetic stimulation comes from vagal innervations along almost the entirety of the gut, except for the distal colon, which is innervated by sacral nerves (Camilleri & Ford, 1998; Edwards, 1997). This stimulation is predominantly excitatory to all areas of smooth muscle in the gut except for sphincters, while sympathetic innervations tends to inhibit everything except sphincter activity (Camilleri & Ford, 1998).

In between meals (the inter-digestive state), cyclic, phasic waves of peristalsis occur along the whole gut, known as the migrating motor complex, or MMC (Kellow et al., 2006; Kellow et al., 1999). During long fasting states, the MMC acts to purge the gut lumen of trace amounts of luminal contents of endogenous (e.g. mucus, sloughed cells, digestive secretions) and bacterial origin, and also clears any remaining dietary components (Otterson & Sarr, 1993). The MMC has three major phases, which only occur in the fasting state (except in ruminants); phase I – contractile quiescence, phase II – intermittent contractions, phase III – regular, high amplitude phasic contractions (Quigley, 2003). Upon consumption of a meal with adequate energy the MMC is interrupted by a fed pattern of contractility. This pattern of contractility is described below, and differs markedly between the stomach, small intestine and large intestine.

In very general terms, the presence of a bolus in a particular segment of the gut will result in an increase in muscular activity in distal organs of the gut in order to make room for the bolus, while at the same time decreasing motor activity in the proximal organs (or by reducing central drive for food ingestion), to allow full digestion of the current luminal contents. As with secretory activity, motility is driven by luminal content and bulk, assessed respectively by enteroendocrine and mechanoreceptor cells along the GI tract. Under normal conditions, this proximal negative feedback mechanism also acts to reduce the need for food intake via the gut–brain axis (Cummings & Overduin, 2007; Konturek, Konturek, Pawlik, & Brzozowki, 2004). The cephalic phase of digestion brings about changes in gut motility that allows the stomach to be empty and receptive to food boluses. Following entry of food into the stomach, luminal factors drive gut motility in a fashion that allows as full a digestion as possible of dietary components to occur. Gastrointestinal motility must either act to maintain luminal contents in one segment and allow them to better mix with digestive secretions or the absorptive epithelium (segmentation), or propel them aborally to the next section of the gut (peristalsis). These actions are based on two types of contraction within smooth muscle; those of short duration (phasic contraction) and those of a more sustained duration (which governs tone of the gastrointestinal segment) (Kellow et al., 2006, 1999). The main neurohumoral control mechanisms and reflex pathways are outlined in Table 1.

Within the stomach, motility is brought about by the co-ordinated movement of three muscular layers, an outer longitudinal layer that occurs over the distal two thirds of the stomach, a circular layer (present throughout the stomach), and an internal oblique layer that occurs only in the proximal half of the stomach (Shafik, El Sibai, Shafik, & Shafik, 2007).

As the stomach first receives food, isotonic relaxation of these muscle layers occurs, in a process known as receptive relaxation. Within this process, the stomach acts as a reservoir for meals of varying sizes. As food enters the stomach, it may further relax (gastric accommodation) in order to accept more food (Tack, Piessevaux, Coulie, Caenepeel, & Janssens, 1998). Subsequently, the gastric pacemaker (situated at the upper part of the greater curvature) initiates slow contractile waves that act to mix the food with gastric digestive secretions. To reduce flow into the duodenum, the pyloric region maintains a tonic contraction during this mixing phase. Tonic contraction in the antrum and body of the stomach results in movement of solid food particles to the distal section of the stomach, and release of liquid components into the duodenum through the pyloric sphincter (Hellstrom, Gryback, & Jacobsson, 2006).

During these ingestive phases, the main luminal drive to stop consuming food is believed to come from gastric distension (Geliebter, 1988; Jones et al., 1997; Kissileff, Carretta, Geliebter, & Pi-Sunyer, 2003; Phillips & Powley, 1996), which is sensed by stretch receptors (also known as mechanoreceptors) that occur both within the myenteric plexus that lies between the longitudinal and circular muscle layers, and the submucosal plexus (Furness, 2000).

Solid components are slowly broken down into smaller particles by mechanical action and gastric secretions until they become liquid enough to pass out of the stomach during gastric emptying. During gastric emptying, there is an increased frequency of

peristaltic contraction coupled with pyloric sphincter relaxation (Cuomo & Sarnelli, 2004). As well as neurohumoral factors secreted by the gut, control of gastric emptying is also fine tuned by systemically-produced hormones, such as leptin and factors such as plasma glucose concentrations. In general, these factors act to delay gastric emptying following ingestion of a meal (Hellstrom et al., 2006).

The rate of gastric emptying is governed by the luminal milieu of the duodenum, and possibly the stomach. While it is generally accepted that an increased energy content of a meal will reduce gastric emptying, some evidence exists to suggest that specific dietary components could act to affect gastric emptying rate. Marciani et al. (2001) demonstrated that gastric emptying was more affected by the nutrient density of a meal than meal viscosity in a crossover study in 12 healthy participants (Marciani et al., 2001). Intragastric infusion of a free fatty acid-based meal instead of a triglyceride-based meal significantly reduced gastric emptying rates and increased retention of contents in the proximal stomach in healthy human participants (Little et al., 2007). The amino acids L-tryptophan and phenylalanine are known to be strong stimulators of CCK release (one of the main drivers for delayed gastric emptying – see Table 1), but only tryptophan (>4 mM) was shown to reduce gastric emptying rates following instillation into the dog stomach (Stephens, Woolson, & Cooke, 1975).

The small intestine has integral layers of longitudinal and circular muscle running along its entire length. Slow wave MMC activity is believed to propagate in the proximal duodenum of the small intestine, which decrease in frequency from duodenum to ileum (Otterson & Sarr, 1993). During the intestinal phase of digestion, migrating clustered contractions will act to propel luminal contents approximately 10–30 cm distally along the small intestine each time (Otterson & Sarr, 1993). These waves of peristalsis occur alongside segmentation-based movements along the small intestine. Segmentation allows better mixing of intestinal contents, and is effected by short (2–4 cm) bands of circular muscle contraction and relaxation (Moffett et al., 1993). Movement within the villi, effected by vertical smooth muscle cells within each finger-like projection, ensures better contact of the absorptive surface with the luminal contents of the intestine (Hosoyamada & Sakai, 2005).

Duodenal luminal nutrient content not only acts as a drive for reduced gastric emptying (see above), but also as the luminal stimulus for a wide neurohumoral release from the duodenum that acts to govern much of the rest of the gut's motility. Secretin, motilin, GIP and CCK release as the chyme bolus enters the duodenum trigger reduced gastric activity, while also increasing intestinal motility distal to the duodenum, and increasing levels of hepatic and pancreatic secretions delivered into the duodenum (see Tables 1 and 2 for further detail).

In a human crossover study, Feinle et al. (1997) noted that isosmotic solutions of saline, glucose and maltodextrin (a mixture of short-chain, digestible saccharides) all reduced frequency of gastric phasic contraction equally (compared with an isotonic saline solution), while maltodextrin had a much greater effect on phasic amplitude and gastric tonic activity. Infusion of 10% and 20% lipid (fractionated soy bean oil) solutions also significantly lowered phasic and tonic activity in the stomach, with a marked effect on phasic frequency of contraction. Lipid concentration did not affect this action (Feinle, Grundy, & Read, 1997). When lipids were administered alongside lipase inhibitors, the action of lipids on gastric emptying was abolished, suggesting that the products of lipolysis governed gastric emptying (Feinle et al., 2003). Further evidence for this came from the studies of McLaughlin et al. (1999), who noted that only fatty acids of 12 carbons or above elicited CCK release in humans, suggesting that only specific sources and types of fats will inhibit gastric emptying rates (McLaughlin et al., 1999).

As digesta reaches the distal ileum, enteroendocrine cells drive the release of PYY (Adrian, Ferri, & Bacarese-Hamilton, 1985; Pironi et al., 1993) and GLP-1 (Holst, 2007; Layer & Holst, 1993; Schirra & Goke, 2005). These hormones are believed to drive reduced secretion and motility in the stomach and proximal small intestine. This so-called "ileal brake" mechanism (Pironi et al., 1993; Spiller et al., 1988; Spiller, Trotman, & Higgins, 1984) effectively acts as negative feedback control, so that the gut cannot damage itself as a result of over-secretion, and also effectively signals the end of the intestinal phase of digestion. Energy content of the luminal contents appears to drive the ileal brake mechanism, with lipid (Maljaars, Romeyn, Haddeman, Peters, & Masclee, 2009; Pironi et al., 1993; Spiller et al., 1988) and possibly carbohydrate (Layer, Peschel, Schlesinger, & Goebell, 1990; Tohno, Sarr, & DiMagno, 1995) presence in the ileal lumen affecting neuropeptide release and resulting in reduced gastric emptying/gut secretion levels. Fatty acids (oleic acid) appeared to be a more potent stimulus of the ileal brake mechanism than intact triglycerides (Spiller et al., 1988).

In the large bowel, longitudinal muscle instead occurs in three tonically contracted strips – the *taenia coli*. Areas of contraction along the *taenia coli* form a number of segmental pouches, known as haustra along the length of the bowel up to the sigmoid colon. Haustra may act as mixing segments to benefit absorption of water and trace nutrients, and possibly to aid fermentation. Haustra are also capable of driving mass propulsive movement of large volumes of luminal contents distally. Compared with the stomach and small intestine, there are two important differences to colonic/large intestinal motility. The first is that MMCs are not inhibited by food intake, and the second is that two lengths of phasic contraction occur; short duration and long duration (Sarna, 1993).

As previously stated, the distal colon is innervated separately from the rest of the gut. Motility also seems to be driven independently from the rest of the gut. In particular, defaecation reflexes are governed locally sigmoid colon/rectal sensation of luminal bulk, as well as extrinsically by sympathetic innervation (Bharucha, 2008; Rao, 2004; Shafik, El-Sibai, Mostafa, & Shafik, 2001).

Some evidence exists to suggest that luminal nutrients occurring in the colonic lumen result in further release of PYY and GLP-1, which results in a prolongation of reduced gastric emptying and gastrointestinal digestive secretion. This feedback inhibition mechanism has been dubbed the "colonic brake", and is suggested by both canine (Wen, Phillips, Sarr, Kost, & Holst, 1995) and human studies (Nightingale et al., 1996). In addition, SCFA levels in the lumen of the colon have been suggested to alter colonic motility in some species. Rodent experiments have shown that direct instillations of high concentrations (>100 mM) of butyrate and propionate, but not acetate, resulted in reduced peristaltic activity and increased tonic activity (Cherbut et al., 1998). These effects would be expected to benefit epithelial absorption, as they would increase the exposure time of luminal fluid alongside decreasing the radius of colonic lumen. Similar results did not occur in the human colon upon instillation of similar levels of SCFA (Cherbut, 2003).

## 6. Effects of dietary fibres on gut motility

Classically, dietary fibre is cited as reducing whole gut transit time, thereby increasing frequency of defaecation. While this model generally holds true for dietary fibres from natural food sources, which are often a mixture of fermentable and infermentable fibre types, fibre's laxative actions are entirely dependent on luminal bulk. Furthermore, it must also be noted that in many cases, the intake of dietary fibres may actually increase transit time within the upper GI tract (stomach and small intestine), due to the

previously discussed effects of reducing digestion rates. Furthermore, feed-forward and feedback from other portions of the gut as a result of fibre intake will also affect motility of the different organs of the gastrointestinal tract.

Prolongation of nutrient release into the intestinal lumen is likely to result in a lengthened phase of hormonal feedback from the duodenum, terminal ileum and colon, leading to a delay in gastric emptying. At the same time, prolonged transit within the stomach and small intestine are likely to drive increased motility (and therefore decreased transit time) distally.

The most researched area of the effects of dietary fibres on gastric motility is linked to gastric emptying. A range of studies have demonstrated that inclusion of viscous fibres in liquid test meals results in delayed gastric emptying, and are particularly consistent in the case of pectins in human studies (Holt, Heading, & Carter, 1979; Iftikhar, Washington, Wilson, MacDonald, & Homer-Ward, 1994; Lawaetz, Blackburn, & Bloom, 1983; Sandhu, El Samahi, & Mena, 1987; Schwartz, Levine, & Singh, 1982; Sanaka, Yamamoto, Anjiki, Nagasawa, & Kuyama, 2007; Schwartz et al., 1988). Previous human studies suggest that ispaghula husk does not have any affect on gastric emptying rates (Bianchi & Capurso, 2002; Frost, Brynes, Dhillo, Bloom, & McBurney, 2003; Jarjis, Blackburn, Redfern, & Read, 1984; Rigaud, Paycha, Meulemans, Merrouche, & Mignon, 1998). One previous study would also suggest that locust bean gum does delay gastric emptying (Darwiche, Bjoìrgell, & Almeìr, 2003). In the case of guar gums and alginates, some studies have shown them to have a significant prolongation of gastric emptying rate [guar – (French & Read, 1994; Holt et al., 1979; Russo et al., 2003) alginate – (Torsdottir et al., 1991)], while other studies showed no effect [guar – (Rydning, Berstad, Berstad, & Hertzenberg, 1985; Van Nieuwenhoven, Kovacs, Brummer, Westerterp-Plantenga, & Brouns, 2001), alginate – (Hoad et al., 2004)]. As a whole, this evidence suggests that the viscosity/gelation of dietary fibres within the gastric lumen is not an important factor in gastric emptying rates. A previous study by Marciani et al., (2001) suggested that the nutrient content of the luminal fluid was a more important predictor of gastric emptying rates than luminal viscosity (Marciani et al., 2001). In part, variance between studies could be as a result of differences in the methodologies used to assess gastric emptying, and procedural differences for the same methodology in different study centres (Szarka & Camilleri, 2009), as well as the use of different test meals (Tougas et al., 2000). However, it is unlikely that the physicochemical properties of single dietary fibres tested were comparable between studies. To date, no study has compared different analogues of the same dietary fibre type on gastric emptying. This type of experimentation would greatly benefit understanding of how the physicochemical characteristics of dietary fibres affect gastric emptying.

The effect of natural dietary fibre matrices on gastric emptying has not been investigated as intensively. In a study comparing the physiological effects of a mixed meal containing high levels of natural fibres (fruit, vegetables and whole grains) against one without these fibres (instead containing fruit and vegetable juice and refined grains), removal of natural fibre resulted in an mean decrease in gastric emptying rate of approximately 45 min (232 min with fibre compared with 186 without) in a crossover feeding trial in 8 healthy adult participants (Benini et al., 1995). A previous scintigraphic study has shown that consumption of coarsely-milled bran resulted in delayed gastric emptying of a rice test meal (compared to a bran-free control), whereas finely ground bran particles did not (Vincent et al., 1995).

Within the small intestine, the presence of intestinal bulk and nutrients would again be expected to result in a shift away from peristalsis towards intestinal mixing. This would be expected to result in slower small intestinal transit. However, few studies have directly assessed small intestinal transit in relation to dietary fibre intake in healthy humans. In a double-blind crossover trial where participants were fed crushed biscuits with or without 15 g of coarse wheat bran over four days. The bran significantly accelerated small intestinal transit time of a rice-based test meal (Hebden, Blackshaw, D'Amato, Perkins, & Spiller, 2002). In a similar study, addition of 15 g of coarse bran directly to a rice test meal also resulted in a significantly shortened small intestinal transit time (McIntyre, Vincent, Perkins, & Spiller, 1997).

A number of studies have assessed the impact of guar gum on small intestinal motility. The first incorporated 15 g of powdered guar into the normal meals of five non-symptomatic adults over two days. This study found no effect of guar inclusion on mouth to stroma transit rates (Higham & Read, 1992). A lack of effect of guar gum on small bowel motility was also noted in a study in non-ileostomised participants (Van Nieuwenhoven et al., 2001). Within small bowel manometry studies testing small bowel motor activity, inclusion of 5 g guar in either solid or liquid meals (glucose-based drink) resulted in a delayed reappearance of the MMC phase III compared to non-guar containing controls. This effect was not seen when guar was consumed with water alone (Schonfeld, Evans, & Wingate, 1997). These results would suggest the vehicle in which fibre is included is vital to its physiological effects.

A separate feeding study in ileostomy patients included either 10 g or 30 g of inulin a day dissolved in water (all taken at one time) alongside an analogous diet each day for three days (one without added inulin). Orocaecal transit rate was reduced by the higher inulin intervention compared with the lower amount and the inulin-free control (Bach Knudsen, & Hessov, 1995).

While "normal" gut transit within the stomach and small intestine is measured in hours, transit from the ileocaecal valve to anus is generally measured in days. Therefore, although the physiological boundaries of transit within each gut segment are not well characterised, the majority of time for whole gut transit occurs within the large intestine. Intake of dietary fibre-rich foods is generally associated with a reduced whole gut transit time. This is believed to be brought about by increasing luminal bulk, resulting in increased peristalsis. A more rapid large intestinal transit is believed to be beneficial in reducing the particularly high luminal toxicity that can occur within the large bowel (Blackwood, Salter, Dettmar, & Chaplin, 2000), which occurs as a result of a concentration of potentially damaging factors of endogenous, bacterial and dietary origin (Brownlee, Havler, Dettmar, Allen, & Pearson, 2003). The dietary fibres that will be best at bulking the luminal contents of the large bowel are those that are not well fermented by the colonic microflora, and those that have a high water-binding capacity (Chaplin, 2003). In particular, the bulking capacity of fibres in the lumen of the colon may reduce the damaging potential of luminal agents (e.g. secondary bile acids) that could cause damage to the underlying mucosa.

Initial observations noted that populations who consumed unrefined, fibre-rich foods had much more rapid gut transit than those that consumed refined grains (Burkitt et al., 1972). A large body of dietary studies supported this observation with strong evidence that intervention with high-fibre foods resulted in a large reduction in transit rate compared with mainly refined food consumption (Cummings et al., 1978; Cummings, Hill, & Jenkins, 1976; Lupton, Morin, & Robinson, 1993; Muller-Lissner, 1988; Stasse-Wolthuis, Hautvast, & Hermus, 1979; Tomlin & Read, 1988). A number of other studies have also noted increased faecal bulk with dietary inclusion of fibre-rich foods, but no impact on measured large intestinal/whole gut transit (Fleming, O'Donnell, & Perman, 1985; Jenkins et al., 1999; Nagengast et al., 1993; Stephen, Dahl, Sieber, Van Blaricom, & Morgan, 1995). Previous reports also

suggested that increased intake of fibre-rich foods acts to normalise both fast and slow colonic transit (Harvey, Pomare, & Heaton, 1973; Price, Davis, & Wilding, 1991), which may in part account for the discrepancies seen in some of these studies.

The impact of isolated dietary fibres on large intestinal/whole gut transit in humans is not well described in the literature. Supplementation of the diet with high levels of either pectin, alginate and β-glucan-rich oat hull fibre did not result in an impact on measured transit time in human studies, but did significantly increase faecal wet weight in each case (Anderson, Brydon, Eastwood, & Sedwick, 1991a; Cummings, Southgate, & Branch, 1979; Stephen, Dahl, Johns, & Englyst, 1997). In similar studies, inclusion on inulin, oligosaccharides and arabinogalactan had no effect on either faecal weight or transit time (Causey, Feirtag, Gallaher, Tungland, & Slavin, 2000; Robinson, Feirtag, & Slavin, 2001; Van Dokkum, Wezendonk, Srikumar, & Van Den Heuvel, 1999). Ispaghula husk is commonly used as the active ingredient in a range of bulk laxatives worldwide. While previous human studies agree that dietary supplementation with ispaghula increases total faecal output, stool consistency and faecal wet and dry weights (Ashraf, Park, Lof, & Quigley, 1995; Kumar, Kumar, & Vij, 1987; Marteau et al., 1994; McIntyre et al., 1997; Stevens, VanSoest, Robertson, & Levitsky, 1988). One study noted an improvement to colonic transit rate of ispaghula supplementation in constipated older persons (Cheskin, Kamal, Crowell, Schuster, & Whitehead, 1995), while another noted no benefit to the transit of chronic idiopathic constipation patients (Ashraf et al., 1995).

## 7. Gastrointestinal immune function

Along with, and as a result of its absorptive functions, the gastrointestinal tract is involved with a range of immune functions. The mucosa effectively partitions the rest of the body from digestive enzymes, large numbers of bacteria and assorted toxins that occur within the gut. The gut's mucosa has two main roles in immunity. Firstly, the mucosa samples luminal contents to assess the threat to the body. This is effected by the gut-associated lymphoid tissue or GALT (Nagler-Anderson, 2001). Like the mucosa lining the airways and distal genitor-urinary tract, the gut comes into contact with a wide range of external antigenic compounds. The epithelium of the small and large intestine contains specialised M cells (located within organised lymphoid follicles) that sample factors reaching the mucosal surface by reverse pinocytosis (Nicoletti, 2000). Sampled antigens are then transported to antigen-presenting cells in the underlying tissues, which link up with the systemic immune system to potentially elicit a response (Niedergang & Kraehenbuhl, 2000).

In general, the GALT must act to tolerate the wide range of damaging and benign agents it comes into contact with (Nagler-Anderson, 2001). It is believed a loss of tolerance to certain antigens, or hyper-responsiveness to non-damaging antigens may result in such conditions as inflammatory bowel disease (Chandran, Satthaporn, Robins, & Eremin, 2003a, 2003b) or food allergies (Kaminogawa, Hachimura, Nakajima-Adachi, & Totsuka, 1999; Vighi, Marcucci, Sensi, Di Cara, & Frati, 2008).

The gut epithelium must also protect itself from the luminal stress of damaging agents and shear forces (Allen & Flemstrom, 2005; Allen & Pearson, 1993). To do this, protective mucus is secreted along almost the entirety of the GI tract (excluding the oesophagus and possibly Peyer's patches). The factors driving intestinal mucus secretion are summarised in Fig. 1. Within the mouth, the active component of mucus, mucin, is secreted alongside other salivary secretions and acts as a lubricant. In the stomach and intestine, mucus is secreted as a protective barrier. This barrier is believed to be made up of two layers (Strugala, Allen, Dettmar, & Pearson, 2003; Taylor, Allen, Dettmar, & Pearson, 2004). The luminal (loosely adherent) layer acts as to lubricate the passage of contents aborally. Due to its constant removal by shear forces, the loosely adherent layer can act to entrap damaging agents and sequester them away from the underlying mucosa (Brownlee, Knight, Dettmar, & Pearson, 2007). The underlying (adherent) layer acts as a selective barrier to luminal factors reaching the mucosa, while still allowing the vital absorptive and secretive roles of the underlying tissues to occur (Pearson, Brownlee, & Taylor, 2004).

## 8. Effects of dietary fibre on gastrointestinal immunity

There is a distinct paucity of data regarding intake of dietary fibres in humans and immune function, associated with the gut or otherwise (Watzl, Girrbach, & Roller, 2005). Animal studies within this area are also sparse. Field et al., (1999) fed two groups of dogs analogous diets containing either "highly fermentable fibre" (a mixture of beet pulp, fructo-oligosaccharides and gum arabic) or an infermentable fibre source (wood cellulose) for two weeks. At the end of this time, jejuna GALT tissues were assessed histologically. The authors concluded that fermentable fibre intake affected the T-cell composition of GALT. In particular, the fermentable fibre diet resulted in increased T-cell mitogen response intra-epithelially and lower responses in underlying mucosal tissues (Field, McBurney, Massimino, Hayek, & Sunvold, 1999). Subsequent normal vs. germ-free murine studies suggested that dietary fibres effects on GALT are driven by the changes to the gut microflora they govern (Sharma & Schumacher, 2001).

No previous study has assessed the impact of dietary fibres on the human mucus barrier. Due to the invasiveness of procedures involved with measuring the mucus barrier directly, only indirect measures of changes, such as assessment of faecal mucin output would be appropriate in humans. The effects of different types of dietary fibres on the intestinal mucus barrier have previously been assessed in a range of animal models. Briefly, fibres and fibre sources such as alginates (Barcelo et al., 2000; Brownlee et al., 2005; Shimotoyodome, Meguro, Hase, Tokimitsu, & Sakata, 2001), ispaghula husk (Brownlee et al., 2003; Cabotaje, Shinnick, Lopez-Guisa, & Marlett, 1994; Strugala et al., 2003), wheat bran (Brownlee et al., 2003; Strugala et al., 2003), ulvan (Barcelo et al., 2000) and carrageenan (Shimotoyodome et al., 2001) all appear to benefit the protective potential of the colonic mucus barrier in comparison to the effect of no fibre (Barcelo et al., 2000; Brownlee et al., 2003; Cabotaje et al., 1994; Shimotoyodome et al., 2001), cellulose (Brownlee et al., 2003; Shimotoyodome et al., 2001), pectin (Barcelo et al., 2000; Brownlee et al., 2005, 2003) or gum arabic (Barcelo et al., 2000). These studies are reviewed in more detail elsewhere (Brownlee, Dettmar, Strugala, & Pearson, 2006).

## 9. The colonic microflora

The large intestine plays host to a large and diverse resident microflora. Over the last 10–15 years, 16 s ribosomal RNA analysis has allowed a more complete characterisation of the diverse bacterial species that make up this population (Eckburg et al., 2005). Around 95% of human colonic microflora (as estimated from faecal sampling) appear to be within *Bacteroides* and *Clostridium* phylogenic groups (Sghir et al., 2000; Suau et al., 1999), with less than 2% of the total microflora being made up of Lactobacilli and bifidobacteria (Sghir et al., 2000).

In general, the colonic microflora is partitioned from the rest of the body by the mucus layer and mucosa. Loss of this partitioning effect is associated with disease processes within the large intestine (Muller, Autenrieth, & Peschel, 2005; Nenci et al., 2007), but it is

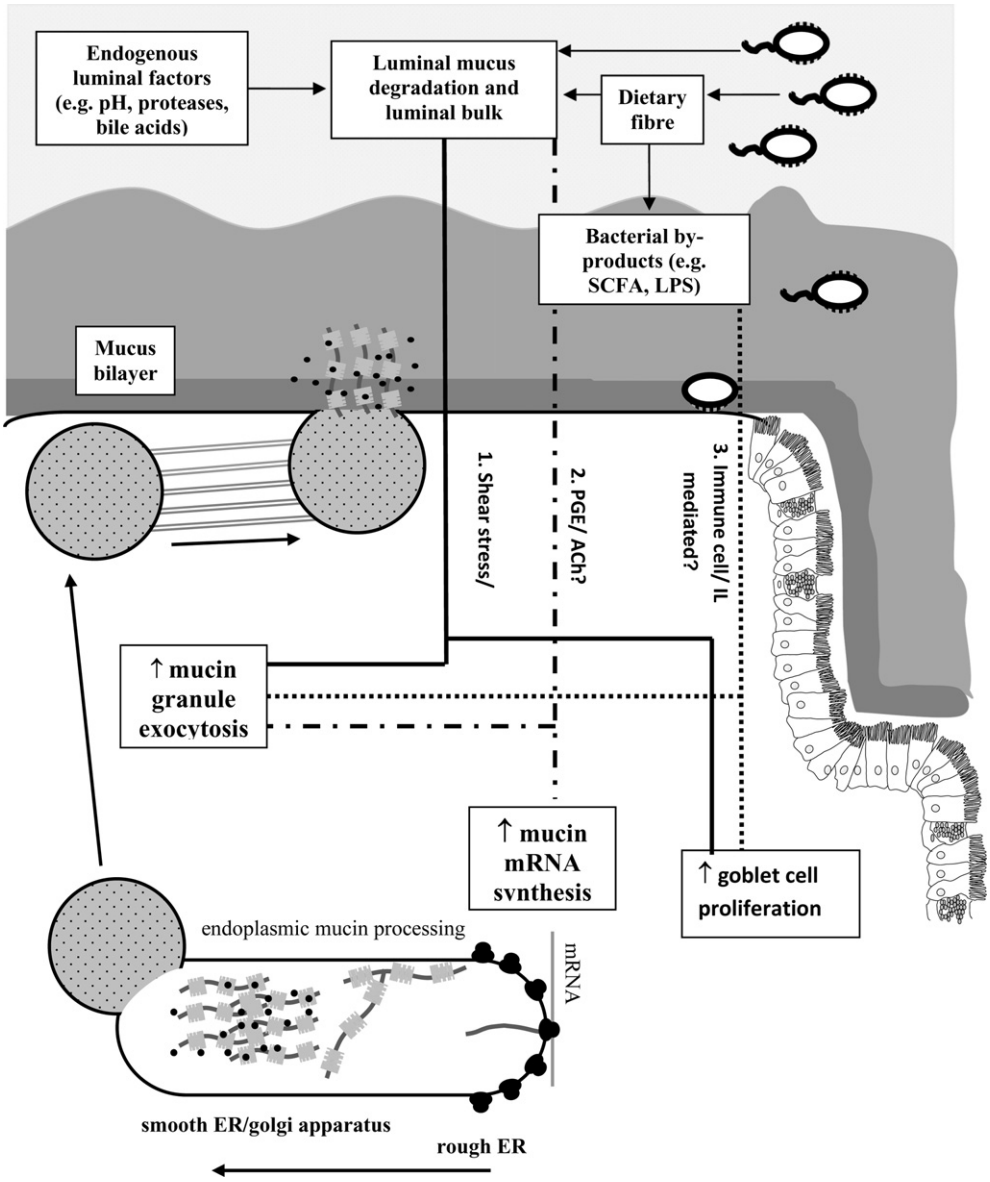


**Fig. 1.** Modulation of mucus barrier dynamics by luminal factors in the intestine. A complex interplay of luminal factors of bacterial, endogenous and dietary origin results in luminal bulk and mucus degradation. The epithelium may respond to increased decreased luminal stress by altering rates of mucin granule exocytosis, mucin mRNA synthesis or goblet cell proliferation. Luminal stress levels triggers three putative pathways that result in an epithelial response. 1. Shear stress or colonic distension results in triggering of the mucosal mechanoreceptors. 2. Either as a result of increased luminal bulk or as a result of enteroendocrine cell response to changes in the luminal milieu, prostaglandins/ acetylcholine are released. 3. Direct (e.g. bacterial adherence to epithelium) or indirect (e.g. chains to bacterial by-products) sensing of changes to the luminal bacterial populations by the gut-associated lymphoid tissues results in an immune-type response, driven by interleukins or other local mediators. These three pathways appear to drive different epithelial responses that drive mucin secretion. Within mucin production, proteins are synthesised with the rough endoplasmic reticulum, then glycosylated and polymerised. Prior to encapsulation in secretory vesicles, mucins are tightly packed into granules, in high calcium ion (black dots) concentration. It is believed that as the vesicle joins to the apical membrane, the mucins rapidly swell upon hydration and separate from $Ca^{++}$. SCFA = short-chain fatty acids, LPS = lipopolysaccharide, PGE = prostaglandins, ER = endoplasmic reticulum. Adapted from details given in (Allen & Flemstrom, 2005; Asker, Axelsson, Olofsson, & Hansson, 1998; Brownlee et al., 2006; Forstner, 1995).

unsure whether this is a cause or effect of the disease process. Within the healthy large intestine, the main way the colonic microflora interacts with the host is through its metabolites (Macfarlane & Macfarlane, 1997). Some of these metabolites are putatively damaging to the underlying mucosa, such as indoles, ammonia and amines while others are potentially beneficial to the host, including SCFA (Cook & Sellin, 1998; Mortensen & Clausen, 1996) and lignans that the mammalian gut can absorb (Mazur, Duke, Wahala, Rasku, & Adlercreutz, 1998; Rowland et al., 2003). SCFA are produced by bacterial fermentation of dietary carbohydrate sources, of which dietary fibre is the main type in the large intestinal lumen.

## 10. Effects of dietary fibre on the colonic microflora

Dietary fibre, as the major dry weight component of digesta reaching the caecum, plays a profound role on the number and diversity of bacteria that inhabit the large intestine. In the absence of dietary fibre or other luminal energy sources, resident bacteria in the colon will turn to large intestinal mucus as an energy source prior to attacking the underlying mucosa. As bacteria require the necessary enzymes to break down saccharide bonds of the diverse range of dietary fibres, fibre will clearly affect microfloral population dynamics. Increased levels of short-chain fatty acids (particularly butyrate) are believed to be of importance in the health

of the colonic epithelium, which is reviewed in detail elsewhere (Topping & Clifton, 2001). The presence of any fermentable dietary fibre is likely to lead to an increase in microfloral bifidobacterial and *Lactobacillus* levels, as these bacteria ferment carbohydrates.

Previous studies in humans have suggested dietary fibres like alginate (Terada, Hara, & Mitsuoka, 1995a), chitosan (Terada et al., 1995b), and inulin (Grasten et al., 2003) lead to a reduction in potentially harmful microfloral metabolites. A range of small human interventions with various fermentable dietary fibres have shown significant, but small, clinical benefit in a number of intestinal diseases and disorders either on their own or in combination with probiotics (Bittner, Croffut, Stranahan, & Yokelson, 2007; Fujimori et al., 2009; Molist et al., 2009).

## 11. Systemic effects of dietary fibre consumption

Of particular interest to the research community at the moment is the possibility of using dietary fibre-based interventions or therapies as a means of reducing risk of cardiovascular disease, type II diabetes and combating obesity. Strong observational evidence exists to suggest that populations who consume higher levels of dietary fibre have a significantly reduced risk of morbidity and mortality from the above conditions (Hu et al., 2001; Liu et al., 1999; Ludwig et al., 1999; Pietinen et al., 1996; Salmeron et al., 1997a, 1997b; Wolk et al., 1999). A range of approaches have been employed to test hypothetical ways in which dietary fibres could bring about the observed reduction of disease risks, including a number of well-designed studies that acute benefits of dietary fibre-based meal consumption, such as glycaemic index and effects on satiety. However, it is likely that acute responses to meals will not necessarily result in health benefits. The epithelium of the intestinal is believed to be completely replaced by new cells every several (2–3) days (Gordon, 1989). In particular, long-term animal feeding studies would suggest that higher dietary fibre intake results in increased crypt and villus length within the intestine (Hedemann et al., 2006; McCullough, Ratcliffe, Mandir, Carr, & Goodlad, 1998; Schedle, Pfaffl, Plitzner, Meyer, & Windisch, 2008) and increased digestive secretion output in pig and man (Dukehart, Dutta, & Vaeth, 1989; Jakob et al., 2000; Langlois, Corring, & Fevrier, 1987), which is suggestive of longer term adaptive responses of the body to dietary fibre intake that allow better absorption. Such adaptive mechanisms may offset any acute beneficial effects of dietary fibre intake.

In order to isolate "cause and effect" of dietary fibre intake and disease risk reduction over a longer period, it is necessary to carry out randomised, controlled trials, whereby a dietary intervention in human participants is made over a considerable length of time (a number of weeks or months), and robust markers of disease risk are assessed. To date, a number of studies have shown benefits of dietary intervention with fibre-rich foods on the metabolic profiles of participants (Esposito et al., 2004; Jang, Jong, Oh, Hyun, & Sang, 2001; Jenkins et al., 2008; King et al., 2007; McMillan-Price et al., 2006; Pittaway, Robertson, & Ball, 2008). While these studies all suggest dietary fibre to be a beneficial component within the dietary intervention, and would give reason to include fibre-rich foods within the diet, they still do not isolate the effects of dietary fibres from other compounds within these foods. A recent short-term (7-day), randomised crossover trial has demonstrated that consumption of a beverage containing 1.5 g of sodium alginate (in a formulation that gelled strongly in the stomach) resulted in a significantly reduced daily energy intake in 68 free-living adults (Paxman, Richardson, Dettmar, & Corfe, 2008). This study would suggest specific formulations of alginates may be a useful dietary fibre therapy to aid weight loss.

Fewer studies have managed to adequately model such effects, no doubt in part due to difficulties in controlling such studies and the production of appropriate test foods for frequent inclusion in the diet. To date, the US Food & Drug Administration has only granted specific health claims for (soluble) fibres with reducing cardiovascular disease risk for 3 g/day of β-glucan from whole oats or barley or 7 g/day of psyllium husk (2006 (referenced June 2009)), based on a number of supportive, randomised, controlled trials (Anderson et al., 1991b; Anderson, Riddell-Mason, Gustafson, Smith, & Mackey, 1992; Brown, Rosner, Willett, & Sacks, 1999; Davidson et al., 1991; Everson, Daggy, McKinley, & Story, 1992; Kestin, Moss, Clifton, & Nestel, 1990; Leadbetter, Ball, & Mann, 1991). Other sources of dietary fibre require similar randomised controlled trials to be carried out before health claims can be justified.

Animal studies would suggest that dietary fibres bind bile acids in the intestine (Sandberg et al., 1994; Seal & Mathers, 2001; Van Rosendaal, Shaffer, Edwards, & Brant, 2004). This effect results in increased faecal losses of bile acids, which are normally reabsorbed in the terminal ileum. As a result, the liver must produce new bile salts, for which cholesterol is used as a starting material. This interruption in the normal biliary circulation would also be expected to occur within the human gut, and offers insight into the potential of certain dietary fibres to reduce total body, and as a result plasma cholesterol concentrations.

## 12. Summary

Dietary fibres impact all aspects of gut physiology and are a vital part of a healthy diet. Initial observations linking dietary fibre to health extolled the benefits of consumption of fibre-rich foods rather than dietary supplementation with fibres. The beneficial effects of fibre come partly from its action as a food matrix in natural foods. Dietary fibre should not be considered as a singular entity, as it is a term that in fact encompasses a wide range of moieties with divergent physicochemical properties. Specific fibres may have specific effects on the gut, but their fermentability and viscosity also govern their physiological actions.

For the majority of dietary fibre types, there is a need for appropriately designed, randomised controlled trial data to demonstrate the long-term benefits to health that are suggested by acute physiological responses to fibre ingestion. There is also a need for development of novel fibre-rich foods that are both acceptable to the consumer and have proven health benefits. Development of such food products is only likely if academic and industry partners work closely together. From an academic's perspective, "palatability" of test formulations with dietary fibre is more a reflection of the edibility of the product, as opposed to the likeability. From an industry perspective, "palatability" revolves around a food being as tasty as possible. The most palatable fibre formulations are likely to be low molecular weight compounds that evidence would suggest will have limited health benefits outside of their fermentability. In terms of dietary fibre product acceptability, it is also necessary to consider gastrointestinal consequence of ingestion rather than just taste tests. Many fibres (particularly those that are rapidly fermented) could result in gas production, bloating and diarrhoea.

Many hydrocolloids used by the food industry are dietary fibres. These ranges of gums, from various sources, are used extensively as thickeners, stabilisers and emulsifiers due to the biophysical characteristics and their high palatability. This would suggest they may be ideal candidate dietary fibres to assess for health benefits and novel fibre-rich foods.

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