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The impact of dietary fibres on the physiological processes governing small intestinal digestive processes

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ABSTRACT

This review is the second in a series of three articles considering how different types of dietary fibre may affect gut function and health, focusing on the impact of dietary fibre intake on the small intestinal digestive processes. While the small intestinal structure supports the large proportion of gastrointestinal absorption that occurs there, the processes of digestion of macronutrients are largely dependent on the exocrine secretions of the pancreas and liver. The impact of dietary fibre, either as isolates or an integral part of foods such as fruits and vegetables, is therefore also considered on the exocrine functions of these accessory organs.

The physiological processes of these three interconnected organs of digestion are outlined and the evidence that dietary fibre impacts on these processes is considered. Evidence for the association of long-term dietary fibre intake on health outcomes related to these organs is also evaluated.

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1. Introduction

This review is a second of a series of three aimed at pooling together some of the recent evidence that dietary fibre impacts many major aspects of gastrointestinal physiology. The first of these reviews (Brownlee, 2014) focused on the effect of various types of dietary fibre on gastrointestinal processes from the mouth to the stomach. Here, the authors focus on the impact of dietary fibres on the physiology of the small intestine and its accessory organs (the liver and pancreas). Similar to the previous review, this article will consider the impact of a wide range of dietary fibres both as isolates and within fibre-rich foods on the physiological function of this section of the gastrointestinal tract. A subsequent review within this journal will focus on how dietary fibres impact on the physiological processes of the large intestine.

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The small intestine is the major site of absorption of the end products of the digestive process due to its enormous surface area (a result of three levels of anatomical folding (Helander & Fändriks, 2014)) and orchestrated motility (WorsØe et al., 2011). To allow macronutrient digestion, the small intestine must also act as a major site of digestion, although this process is almost entirely dependent on exocrine secretions from the pancreas and liver (Chandra & Liddle, 2013; Maldonado-Valderrama, Wilde, MacIerzanka, & MacKie, 2011; Nawrot-Porabka, Jaworek, Leja-Szpak, Kot, & Lange, 2015). These accessory organs are not only important in allowing appropriate intestinal digestion to occur but also in orchestrating the body's utilisation of the products of digestion and possibly linking dietary intake to many longer-term systemic disease trajectories (Strowig, Henao-Mejia, Elinav, & Flavell, 2012; Unger, Clark, Scherer, & Orci, 2010). Although the ideas that dietary fibres can affect the processes of digestion and absorption and can impact on systemic health are not new, the putative mechanisms through which such effects could be brought are often poorly characterised and do not always agree with longer-term studies on health outcomes.

This review therefore aims to explore the potential of dietary fibres to impact short-term changes in small intestinal, pancreatic and hepatic function and to evaluate longer-term impacts on health outcomes related to the correct functioning of these organs in relation to the processes of digestion.

2. Dietary fibres and the commencement of the intestinal phase of digestion

During the non-digestive state, small intestinal motility is limited to a series of migrating motor complexes which are believed to maintain aboral movement of luminal contents to reduce the build-up of secreted mucus and other secreted factors and to limit the potential of microbial infiltration into the underlying mucosa (Birchenough, Johansson, Gustafsson, Bergström, & Hansson, 2015; Pelaseyed et al., 2014). Motility patterns rapidly shift as a result of the pyloric sphincter allowing chyme to enter the duodenum. This action also heralds the commencement of the intestinal phase of digestion. The motility pattern within the intestinal phase of digestion consists of migrating, clustered contractions that tend to move contents along the small intestine up to 30 cm at a time at varying frequencies (Otterson, Leming, Fox, & Moulder, 2010; Otterson & Sarr, 1993). Segmentation along the small intestine ensures adequate mixing of luminal contents with digestive juices and improves the chances of contact with the villi (Gwynne, Thomas, Goh, Sjövall, & Bornstein, 2004; Huizinga & Lammers, 2009). The villi themselves pass through the luminal content in waving motions, as a result of the contraction of both the muscularis mucosae as well as the action of the surrounding smooth muscle layers below the mucosa (Schulze, 2015). This smooth muscle in the intestine features distinct, continuous bands of longitudinal and circular muscle along the entire length of the duodenum, jejunum and ileum (Huizinga & Lammers, 2009). Within the intestinal phase of digestion, a series of reflexes act to control the distal and proximal motility of the gut (Brownlee, 2011). The myenteric reflex, driven by local distension of the gut wall by a bolus of digesta, results in a local increase in the number and strength of contractions mediated by the myenteric plexus within the smooth muscle layers (Fujita et al., 2004). The myenteric reflex is initiated by the interstitial cells of Cajal and is driven by release of acetylcholine (Klein et al., 2013), leading to the orchestration of contraction and relaxation events in the immediate vicinity. Separate neurohumorally-driven feedback mechanisms also occur, as a result of digesta entering the duodenum. This impacts on gastric activity and also leads to a reflex increase in motility of the jejunum, ileum and large intestine (Furness, Rivera, Cho, Bravo, & Callaghan, 2013) to ensure that subsequent segments of the gut are prepared to receive the incoming luminal content.

As acidic digesta (chyme) enters the duodenum from the stomach, this triggers perhaps the key event in the commencement of the intestinal phase of digestion. Cholecystokinin (CCK) is released from the enteroendocrine I cells in the duodenum, resulting in an increase in pancreatic secretion (Wang, Prpic, Green, Reeve, & Liddle, 2002) and release of bile from the gall bladder (West & Mercer, 2004). Alongside secretin, motilin and gastric inhibitory peptide, CCK release appears to orchestrate the small intestinal digestive processes and may also have roles in the reduction of gastric secretion and increased intestinal motility (Ellis, Chambers, Gwynne, & Bornstein, 2013; West & Mercer, 2004). Previous studies note that the presence of luminal amino acids is key to CCK release (Daly et al., 2013). The presence of low concentrations of amino acids in chyme, as a result of protein digestion in the stomach, is possible but may also be dependent on the presence of basal protease activity in the intestine (Nishi, Hara, Hira, & Tomita, 2001).

Studies in animals and humans have noted that some types of dietary fibre appear to impact on circulating concentrations of CCK. Human studies have particularly focused on the acute, postprandial impact of fibre ingestion on plasma CCK levels. Purified preparations of cellulose and hydrolysed guar gum were shown to significantly increase the amplitude and time length of postprandial CCK peaks (Geleva, Thomas, Gannon, & Keenan, 2003; Heini et al., 1998), while flaxseed fibre and polydextrose appeared to have no effect (Kristensen et al., 2013; Olli et al., 2015). A similar increase in postprandial CCK response was noted upon inclusion of dried bean flakes (which increased dietary fibre content by almost 12 g/100 g) in a test meal compared to the bean-free control (Bourdon et al., 2001). A three-month supplementation of two different dosages of oat β-glucan within an energy restricted diet did not result in greater increases to fasting plasma CCK compared to energy restriction alone and did not benefit weight loss (Beck, Tapsell, Batterham, Tosh, & Huang, 2010).

A study in rats noted that the presence of guar gum and fructo-oligosaccharides within chow reduced *ad libitum* energy intake. This effect was removed when competitive ligands of CCK receptors were given to the animals (Rasoamanana et al., 2012), strongly suggesting that the impact of fibres was mediated by the action of CCK. However, it must be noted that postprandial plasma CCK concentration was not measured within this study. Consumption of a standard chow incorporating resistant starch did not impact the food intake of rats who were intraperitoneally dosed with a specific amount of CCK (Shen et al., 2009), suggesting that there was no indirect impact of dietary fibre inclusion on the sensitivity of the test animals to CCK responses.

Secretin is a hormone secreted from the enteroendocrine S cells within the duodenum. Its release causes increases in pancreatic exocrine secretion and is linked to reduction in feelings of hunger and reduced gastric emptying rates (Sekar & Chow, 2013). Secretin is believed to be the first hormone ever to be isolated (Dockray, 2014). Secretin therefore exerts a number of similar roles to those of CCK. Motilin is mainly released from duodenal enteroendocrine cells (Goswami, Tanaka, Jogahara, Sakai, & Sakata, 2015). While secretin tends to reduce gastric emptying, motilin tends to increase pyloric sphincter relaxation to increase flow into the duodenum, with peak motilin concentrations occurring at the same time the gastric phase of digestion is being completed (Ozaki et al., 2009). Classical studies have highlighted that both hormones are released over a relatively short time frame (less than 30 min) in response to intestinal acidification, with secretin reaching a peak faster than motilin and maintaining values above baseline levels of a longer period of time (Ozaki et al., 2009), suggesting that their