Food microstructure and digestion

Elizabeth Troncoso and José Miguel Aguilera review the influence that food structure has on digestive behaviour

Introduction

The fundamental role of food structure in understanding the behaviour of foods during processing and eating is well established (1). New interest has arisen regarding the function that food structure may play once foods are inside the body and, consequently, in our nutrition, health and well-being (Fig. 1). Attention is further supported by the increased belief that foods and not nutrients are the fundamental unit of nutrition (2). Thus, the release of nutrients from the food matrix (the composite containing the nutrients in a food) as well as the interactions between food components and restructuring phenomena during transit in the digestive system becomes far more important than the original contents of nutrients. In this respect, Lundin *et al*. (3) have recently stated that there is an emerging interest in the impact of food structure on digestive behaviour and its relationship to human nutrition, because the interactions between individual macronutrients (protein, fat and carbohydrate) control in many cases the rate of digestive processes, such as proteolysis and lipolysis, influence satiety and condition the absorption of nutrients. This article summarises some of the major findings presently available on the effect of food microstructure on the digestive process.

Food structures

We ingest nutrients in the form of microstructures spanning in size from nanometres to a few millimetres (4). In plant tissue, major food components such as starch, storage proteins and most lipids are contained in discrete, homogeneous packets embedded in the cellular cytoplasm. Other food molecules are hierarchically assembled into functional structures as is the case of the cellulose and pectin in plant cell walls and of collagen and the myofibrillar proteins in muscle tissues. Some key food molecules are bound or enclosed in organelles, and thus are not readily available. Moreover, during processing or cooking new structures are formed and further complex interactions, yet to be resolved, develop from the molecular level to the micron scale (see Fig. 2). This is the chemical and structural panorama of the food we eat and digestion has to liberate and breakdown molecules to a state in which they can be absorbed, but in so doing restructuring and interactions take place complicating further our understanding of nutrient release and its bioaccessibility (5). For instance, the presence of an insoluble state (e.g., clotted milk proteins, precipitated polysaccharides or highly viscous fibre) is known to delay gastric emptying (6). In summary, when it comes to nutrition, health and well-being, the microstructure of foods within our bodies matters.

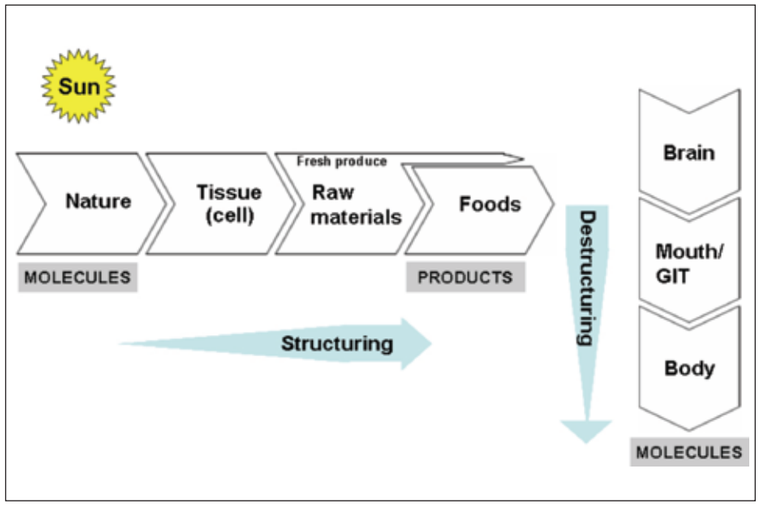

Figure 1. Foods we eat: from molecules to products and back to molecules. While food processing induces structure formation, digestion is basically a destructuring step.

The reactor inside our bodies

The gastrointestinal tract (GIT) is a versatile multi-compartment reactor that operates on a variable solid/liquid feed but delivers more or less standardised products. The main organs of the GIT include the mouth, the stomach and the small intestine. The GIT is connected to the vascular, lymphatic and nervous systems allowing the regulation of the digestive response, delivery of absorbed compounds to organs in the body and the regulation of food intake (7). In the GIT the structure of foods undergoes major size reduction facilitating the release of nutrients embedded in the food matrix so they may be subjected to enzymatic action and eventually absorbed. Mouth and stomach are the major compartments where foods are disintegrated into smaller

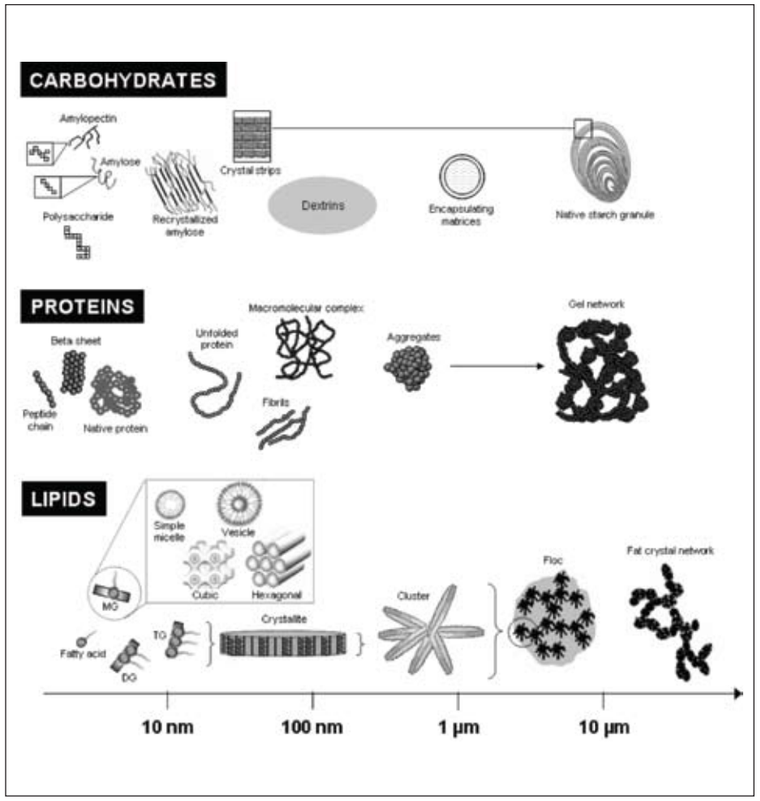

Figure 2. Some hierarchical structures of major food components as may be present in foods.

sizes, whereas the small intestine is the major site of macromolecular breakdown and nutrient absorption. In the GIT, both mechanical forces and chemical reactions breakdown ingested food into small molecules, and the kinetics of digestion depends on the particular physicochemical characteristics of the flowing contents as well as on physiological conditions. Disintegration and dissolution are affected by the formulation and processing conditions (e.g. shear, mixing, heat) used at the manufacturing/preparation stage (8). Using starch as an example, equal amounts consumed in foods prepared using different added ingredients (e.g. presence of sugar) and water contents, and subject to diverse forming and heating conditions will differ in their kinetics of digestion. Before absorption in the gut, any compound of interest has to be bioaccessible, meaning that it has to be in a molecular dispersed state, colloidal form, or within a micellar system in the case of hydrophobic materials.

Food structures in the mouth

The oral step is rapid but plays an important role in reducing particle size, mixing, hydrating and lubricating the contents with saliva so that a bolus is formed and swallowed. Mastication increases the surface area of food pieces but it may be quite inefficient in breaking open some plant tissues as demonstrated by the presence of intact cells of nuts in the faeces. While a small portion of starch is hydrolysed by the enzyme α-amylase due to the short retention time, almost no protein or fat digestion occurs in the mouth.

Food structures in the stomach

The motility of the stomach mixes the ingested bolus with gastric juice, which contains acid and digestive enzymes (e.g. gastric lipase, pepsin and mucin), inducing further reduction of particle size, remixing and phase separation. Dietary lipid is dispersed into a finely divided emulsion by the peristalsis of the stomach, creating a lipid-water interface where enzymatic hydrolysis by gastric lipases takes place. In healthy humans, gastric lipase leads to the hydrolysis of 10-30% of ingested triacylglycerols (TAGs), the main hydrolytic products being diacylglycerols (DAGs) and free fatty acids (FFAs). It has been shown *in vitro* that some components of dietary fibre interfere with the lipid emulsification process, leading to a smaller area of "clean" interface and consequently to lower TAG lipolysis (9). Likewise, the nature of a pre-existing adsorbed layer on fat droplets conditions hydrolysis. Proteins stabilising an emulsion may undergo conformational changes by the acidic environment of the stomach, especially if they are denatured and become aggregated, thus affecting lipolysis. The emulsion leaving the stomach contains emulsified lipids as fine droplets (i.e. $< 0.5\ \mu m$ in diameter) as well as a hydrolysed lipid fraction.

The action of pepsin on proteins results in a mixture of polypeptides, oligopeptides and some free amino acids (FAAs). In any case, the efficacy of proteolysis in the stomach depends on the structural changes of individual proteins or protein assemblies, as affected by the lower pH and ionic strength. For example, casein, unlike whey proteins, coagulates in the stomach and as a result the overall gastric emptying time for casein is longer. This reduced postprandial increase in plasma amino acids compared to that of non-coagulating whey proteins, has led to the concept of "fast" and "slow" proteins (10).

Food structures in the intestine

The peristaltic motor activity propels the chyme along the length of the intestine where it is exposed to pancreatic enzymes (such as pancreatic lipase, co-lipase, trypsin, chymotrypsin and carboxipeptidases) and bile salts, while mixed with phospholipids and sloughed intestinal cells. In the case

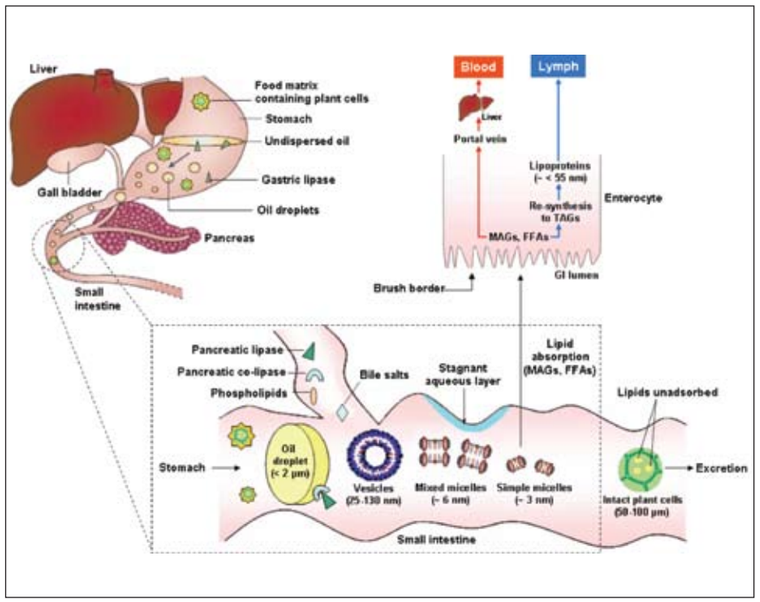

Figure 3. Schematic diagram of the digestive process of an oily plant food. The dynamics of digestion of fatty foods in the GIT leads to the breakdown of complex structures, which disassemble during transit or may remain intact. Adapted from (12).

of lipids, the combined action of bile and pancreatic juice brings about a marked change in the physicochemical form of the luminal lipid emulsion. Pancreatic lipase is secreted into the duodenum, and in the presence of co-lipase it hydrolyses the remaining TAGs to form monoacylglycerols (MAGs) and FFAs. Pancreatic lipase (and gastric lipase as well) acts on emulsified substrates, thus, the physicochemical properties of the emulsion-water interface largely determine the extent of enzyme binding and consequent lipolysis. Finally, lipid digestion continues in the small intestine with desorption and dispersion of insoluble lipid into an absorbable form. Digested lipids are solubilised in the lumen of the intestine into at least two types of nanostructures: bile salt micelles and unilamellar vesicles. These assemblies deliver digested lipids to an aqueous-enterocyte layer where they are subsequently absorbed into the enterocyte´s brush border membrane lining the surface of the small intestine (11). Figure 3 shows a scheme of the digestive process for lipid foods in the GIT.

The final steps in the digestion of proteins take place in the intestinal lumen and are associated with small intestinal mucosal cells, which contain a number of peptidases. The collective result of the action of proteases in the GIT is to reduce the majority of dietary protein to a mixture of FAAs, dipeptides and tripeptides, making them available to the various carrier-transporters of the brush border membrane. However, the physical and chemical state of proteins, affected by the environment of the GIT or previous proccesing (e.g. thermal treatment), might change the intestinal transport properties as has been shown for allergenic proteins. Harsh conditions in the GIT, interaction with lipids and the formation of larger aggregates contribute to the allergenicity of plant food proteins (13). Thermal denaturation not only affects the physical state of proteins but also their susceptibility to the pepsin and the trypsin/chymotrypsin mixture.

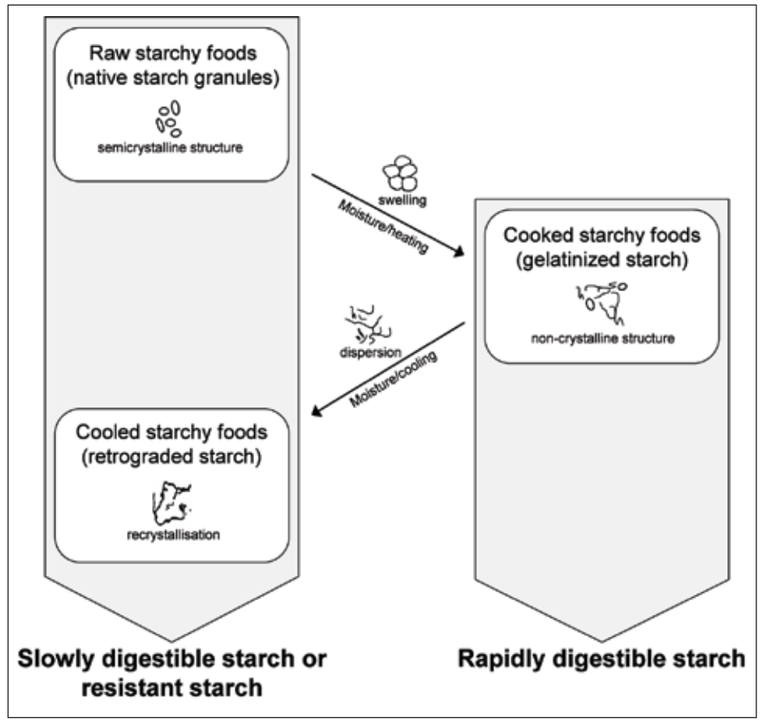

Figure 4. Digestibility of starch in the small intestine according to the structural changes of starch granule induced by processing.

The presence of plant hydrocolloids (e.g. pectins) and their nonspecific interactions with some proteins reduces the accessibility of proteases to cleavage sites.

Complex carbohydrates are digested to monosaccharides, mostly glucose, galactose and fructose, prior to absorption in the small intestine. Intestinal digestion occurs through reactions mediated by pancreatic α-amylases and by disaccharidases anchored to the brush border surface of the enterocytes. Enterocytes are then responsible for the complete absorption of the sugars into the body and once absorbed, galactose and fructose are mostly converted to glucose for metabolism or storage (14). The physical form of food is the major determinant of the rate of digestion of both starches and sugars. The extent of starch digestion within the small intestine is variable, depending on its physical form and a substantial amount of undigested starch enters the colon, where may be fermented by bacteria or simply appear in faeces. The degree of gelatinisation of starch granules and of recrystallisation of the released polymers is of vital importance in the breakdown to sugars because both phenomena influence the susceptibility to enzymatic degradation. In this context, a classification of starchy foods based on intrinsic factors (i.e. physical structure) has been proposed for starch digestibility in the small intestine as it may vary from a rapid digestion to indigestibility (i.e. resistant starch) (15) (Fig. 4). The incomplete digestion of starch is related to the matrix surrounding starch, the nature and physicochemical properties of the starch *per se* at the granule and molecular levels (e.g. granule size and amylose:amylopectin ratio), and the presence of other dietary components (e.g. sugar, dietary fibre and lipid). The "encapsulation" of starch granules by a protein network is an important factor in explaining the slow degradation of starch by α-amylase in pasta, which may limit its accessibility due to different structural factors, such as porosity, compactness and tortuosity of the protein matrix, and structure of starch.

Conclusions

As foods rather than nutrients become a primary consideration in the search for better health and well-being, the role of food structure becomes even more prominent. A better understanding of the structural changes in the gut and interactions taking place will allow the rational design of foods and bioactive supplements for specific conditions. This is not an easy task since we eat meals that vary in the quantity and quality of food components as well as in the type of matrices in which they are contained. Experimental apparatus that simulate the dynamics in the GIT on real foods are urgently needed to rapidly screen the effects of food microstructure and then proceed to validation by expensive *in vivo* human experiments.

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