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Slowly Digestible Starch--A Review

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Abstract:

The link between carbohydrate intake and health is becoming increasingly important for consumers, particularly in the areas of glycemic index (GI) and extended energy-releasing starches. From a physiological point of view, slowly digestible starch (SDS) delivers a slow and sustained release of blood glucose along with the benefits resulting from low glycemic and insulinemic response. SDS has been implicated in several health problems, including diabetes, obesity, and cardiovascular diseases (metabolic syndromes). It may also have commercial potential as a novel functional ingredient in a variety of fields, such as nutrition, medicine, and agriculture. The present review assesses this form of digestion by analyzing methods to prepare and evaluate SDS, factors affecting its transformation, its health benefits, and its applications.

Keywords: slowly digestible starch, structure, digestibility, glycemic response, health claim

INTRODUCTION

In human nutrition, starch plays a major role in supplying metabolic energy, which enables the body to perform a multitude of functions. Based on the rate and extent of digestibility, starches have been classified into rapidly digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS) [1, 2]. Starch can be quantified into different fractions using the *in vitro* Englyst assay: the starch fraction digested within 20 min of incubation is classified as RDS, the starch fraction digested between 20 and 120 min corresponds to SDS, and the remaining fraction that is not further digested is RS (Fig.1 a). RDS induces a fast increase in blood glucose and insulin levels, which can induce a series of health complications, such as diabetes and cardiovascular diseases. SDS is slowly digested throughout the small intestine, resulting in a slow and prolonged release of glucose into the blood stream, coupled to a low glycemic response. This starch type may be helpful in controlling and preventing hyperglycemia-related diseases. Moreover, RS is the starch portion that cannot be digested in the small intestine, but instead is fermented in the colon as dietary fiber, which may prevent disease and lead to better colonic health (Fig.1 b) [1, 3-5].

Obesity and diabetes have become major public health concerns worldwide, with the number of cases increasing exponentially in recent years [6]. The multi-factorial etiology of this worldwide epidemic, and the idea that diet may contribute to it, is now well recognized. New developments in food and nutritional science have led to the conclusion that slowing down the rate of digestion of glucose from ingested carbohydrate sources helps to blunt glycemia, reduces insulin requirements,

and causes satiety [7]. The food industry has been developing a new, slowly digestible carbohydrate (SDC) [8-11]. Some examples of commercially available products include isomaltulose, trehalose, oligo-alternan, pullulans, sucromalt, as well as other slowly digestible syrups. All of these products claim to have a slow and extended postprandial level of glucose after intake, although they differ in molecular structure, functional properties, and potential application in conjunction with SDS [10-15]. The present review focuses on SDS as an SDC and analyzes its digestibility, preparation, physiological effects, and potential application.

DIGESTION OF SDS

Starch digestion and absorption consists of essentially three phases: the intraluminal phase, the brush border phase, and the glucose absorption phase. Starch is ingested, then enzymatically hydrolyzed, and finally absorbed as glucose for energy metabolism within the upper gastrointestinal tract.

Unlike most starch components, molecules of SDS are not disrupted in the oral cavity by salivary α-amylase, in the stomach lining by gastric acid, or through the vigorous grinding action resulting from gastric motility. The process of digestion is initiated after acidic chyme moves into the small intestine through gastric emptying, and most digestion of SDS occurs in the duodenum. SDS is hydrolyzed by enzymes secreted from the pancreas and is converted into small linear oligomers and α-limit dextrins. These amylolytic degradation products from SDS, made up predominantly of disaccharides (maltose), diffuse from the lumen into the brush border membrane, where the final

digestion to glucose occurs through the action of the mucosal enzyme complexes containing sucrase-isomaltase (SI) and maltase-glucoamylase (MGAM) [15, 16]. Quezada-Calvillo et al. reported that MGAM and SI account for more than 85% of starch α-glucogenesis, while α-amylase serves only as an amplifier of mucosal starch digestion. MGAM has a higher α-glucogenic activity than SI, but it is inhibited by mealtime concentrations of luminal maltodextrins[17, 18]. In this way, MGAM regulates the total rate of starch α-glucogenesis. Compared to the N-terminal subunit of MGAM, the C-terminal subunit of MGAM has greater catalytic efficiency due to its higher affinity for glucan substrates and larger number of binding configurations at the active site [19]. Ao et al. have also shown that hydrolysis by pancreatic α-amylase is not required in order for the N-terminal subunit of MGAM to degrade native starch granules in the human small intestine [20]. In summary, SDS is degraded primarily by recombinant human enzymes: α-glucosidases, including small intestinal MGAM; SI; and pancreatic α-amylase.

ANALYTICAL METHODS FOR SDS DIGESTION

In order to monitor the rate and extent of starch digestion or the intestinal absorption of starch-derived glucose, several available methods are available, and they vary in their degree of invasiveness and accuracy.

In Vitro Approaches

Measuring Nutritional Starch Fractions

The method most widely used to quantify nutritionally important starch fractions was developed by Englyst et al.[1], and it involves stimulation of the human digestion system. In this procedure, the various types of starch constituents are determined by controlled enzymatic hydrolysis with pancreatic amylase and amyloglucosidase at 37 °C and the released glucose is measured using glucose oxidase. SDS is calculated by subtracting the amount of glucose hydrolyzed after 120 min from the amount of glucose hydrolyzed after 20 min. This technique yields values for rapidly available glucose (RAG) and slowly available glucose (SAG), thereby providing a description of the rate of glucose release from the food being tested [21].

A novel method from The Netherlands Organization for Applied Science Research (TNO) made two major improvements to the Englyst test: a mixture of microbial enzymes and analysis of the amount of glucose released at specific time points. One study comparing various pure carbohydrates to different kinds of carbohydrate- containing food products showed that the GI values obtained with the TNO method correspond well with results obtained from human studies [22]. Guraya et al. reported that the digestibility of SDS can be determined by measuring the rate of starch hydrolysis due to porcine α-amylase [23, 24]. The hydrolysate (maltose) can be measured using the colorimetric dinitrosalicylic acid (DNS) method. In this approach, SDS is calculated as follows: SDS% = $(B\text{-}A)/C \times 100$, where A is the mg of maltose produced upon digestion of starch after 1 h has passed; B is the maximum mg of maltose produced after a certain time, with no further increase in maltose observed; and C is the total starch expressed in mg of maltose. Other

investigators have attempted to improve the Guraya procedure and confirmed the maximum mg of maltose produced after 10 h [25]. Few comparisons have been performed using the same samples. However, only the method of Englyst has been validated using *in vivo* data and there are some drawbacks to this authoritative method. These include its troublesome demand for a wide variety of substrates, its lengthy procedure and poor reproducibility when the technician is not extensively trained, its requirement for specific equipment, and the fact that some of the enzymes needed are not commercially available.

Measuring Viscosity

Various researchers have shown that changes in starch viscosity affect its digestibility. The controlled stress rheometers, a commercially available concentric cylinder viscometer, provides a sensitive indicator of the extent of dissolution of starch granules and of their subsequent enzymatic hydrolysis [26]. Gee and Johnson have shown that viscosity declines over the course of a simulated digestion and that relative viscosity values can predict to what extent intact foods raise the viscosity of partially digested gut contents[27]. The viscosity is inversely related to the digestibility of the partial α-amylase treated maize starch, which can function as SDS and RS consistent with its low GI [28]. According to the studies of Han and Hamaker as well as the work of Benmoussa et al., the extent of breakdown of swollen granules and the viscosity after gelatinized starch granule structure depends on how much amylopectin fine structure have disrupted[29, 30]. In those studies, breakdown viscosity was found to correlate negatively with SDS based on Rapid Visco Analyser

(RVA) profiles. Indeed, RVA method may serve as a tool for screening the slow digestion properties of different starches. Zhang et al. reported that a viscosity-based screening method based on RVA profiles can be used to assess the properties of SDS obtain from genetic mutants of maize [31].

Other Methods for Measuring Digestion Rate

Granfeldt et al. introduced a method for measuring the rate of *in vitro* starch digestion in products that is based on a peculiar 'as eaten' (chewing/dialysis test) structure, which shows promise for predicting the metabolic behavior of starchy foods[32]. In this method, the test starch substrate is chewed under standardized conditions, and then incubated with the proteolytic enzyme pepsin. Subsequently the mixture is transferred to dialysis tubing and incubated with pancreatic α-amylase for 3 h. Aliquots of the dialysate are collected at different times, and the degree of hydrolysis is calculated as the proportion of potentially available starch degraded to maltose. The hydrolysis index (HI) is calculated as the area under the hydrolysis curve (0-180 min) with the product as a percentage of the corresponding area with white wheat bread, chewed by the same person [32, 33]. Starch hydrolysis kinetic curves follow a first-order equation $C = C_{\infty}(1\text{-}e^{-kt})$, where C is the quantity of ingested starch digested at time t, C_{∞} is the potentially digestible starch fraction (less than 100), k is the fractional starch digestion rate, and t is the chosen time. Weurding et al. stated that the k of SDS among feedstuffs, during a study of starch digestive behavior in the small intestines of broiler chickens, was less than 1 h^{-1} in an *in vitro* test [34,35]. Wen et al. published a

method for measuring total carbohydrate digestion rate [36]. It involves homogenizing the starch samples with water, transferring them to a dialysis bag, and incubating with salivary α-amylase for various times. Maltose concentration is determined using a standard curve of maltose content versus absorbance (the Somogyi-Nelson method), and the carbohydrate digestion rate is determined as: rate ($mg \cdot g^{-1} \cdot h^{-1}$) = $x/[w \cdot (1-m) \cdot t]$, where x is the carbohydrate amount in the diluted dialysate by reference to the standard curve, w is the weight of sample, m is the moisture of the starch, and t is the reaction time. These methods have been used to provide the *in vitro* values for some starchy foods, but not for SDS, which may need to be confirmed in the future.

In Vivo Approaches

Glycemic Index (GI)

The concept of GI was introduced by Jenkins et al. in an attempt to classify carbohydrate-based foods according to the postprandial glucose responses that they elicit [37]. The GI is defined as the incremental area under the blood sugar response curve (the change in blood glucose level 2 h after a meal) of 50 g available carbohydrate portion of a test food expressed as percentage of the response to the same amount of carbohydrate from a standard food (either white bread or glucose) ingested by the same subject [7, 38]. The related term of glycemic load (GL) has been proposed to take into account differences in carbohydrate content among foods, meals, or diets [39, 40]. GL is calculated by multiplying the GI of a food by the amount of total dietary carbohydrate per serving and it therefore serves as an indicator of the ability of the carbohydrate to raise blood glucose

levels or global dietary insulin demand. A food-based 'GI', the relative glycemic potency (RGP), has recently been proposed to overcome the limitations of GI and is defined as the theoretical glycemic response to 50 g of a food, expressed as a percentage of the response to 50 g of glucose [41]. In addition, the glycemic glucose equivalent (GGE), defined as the weight of glucose with the same glycemic impact as a given weight of food, has been proposed as a practical measure of relative glycemic impact [41, 42].

Given the glycemic response to SDS, which shows a delayed appearance of the blood glucose peak and a prolonged, moderate elevation of glucose after the peak, the extended glycemic index (EGI) has been proposed to measure the slow digestion of starchy foods *in vivo* on an extended time scale [43]. In addition, the equation introduced by Granfeldt and Goñi et al. has been used to predict the GI from the HI: GI = 0.862 × (HI) + 8.198 and GI = 0.549 × (HI) + 39.71, respectively [44, 45]. HI and GI correlate significantly in cereal and legume products [32]. Data from the hydrolysis of novel starch products *in vitro* are useful in predicting glycemic responses *in vivo* [46]. Englyst et al. demonstrated that the relationship between RAG and glycemic response is significantly positive, and that RAG accounts for 70% of the remaining variance in glycemic response [47, 48]. However, the combination of SAG and fat accounts for 73.1% of the variance in GI, with SAG as the dominant variable. Using a plain sweet biscuit, Garsetti et al. observed a similar relationship between starch digestibility *in vitro* and responses *in vivo* [49].

Insulin Index (II)

Insulin secretion is largely assumed to be proportional to postprandial plasma glucose responses. Cumulative changes in insulin responses for food are quantified as the incremental area under the 120 min response curve; the incremental area is calculated using the trapezoidal rule, with fasting concentrations taken as the baseline and truncated at zero [50]. The insulin index (II) has been suggested as a concept for dietary management of people with diabetes. The calculated equation of insulin index is similar to the equation developed by Jenkins for calculating GI values. Using the *in vitro* method, Granfeldt et al. showed a significant correlation between HI and II [32]. In cereal products, the combination of RAG and protein contribute equally to account for 45.0% of the variance in II [48].

FACTORS INFLUENCING THE FORMATION OF SDS

Starch Structure

Starch is a semicrystalline material synthesized as roughly spherical granules in plant tissues. These granules consist of alternating concentric layers of ordered and dense crystalline and less ordered amorphous regions (lamellas) extending from the hilum to the surface of the granules. The crystalline regions are formed from the short branch chains of amylopectin molecules arranged in clusters; these crystalline regions are interspersed with amorphous regions that constitute branching points of amylopectin and amylose. The sub-chains of the amylopectin molecules can be classified into three types according to their length and branching points (Figure 2). The shortest A chains, for which the degree of polymerization (*DP*) is 6-15, have no branching points

as ‘outer’ chains. The B chains are branched by A chains or other B chains (e.g., B1, B2, and B3, depending on their respective length and the number of clusters they span). There is only one C chain per amylopectin molecule, which is identifiable because it contains a single reducing terminal. Within this structure, the branch-points are located in the region of low molecular order, and the linear chains lie within the region of high molecular order. These linear chains can then form double helices to make up the crystal structure. On the basis of wide-angle X-ray diffraction scattering studies, native starch is classified into A, B, C, and V types (Figure 3). The A type is characteristic of most starches of cereal origin, while the B type is typically found in potatoes, other root starches, amylomaize starches, and retrograded starch. The C type, which is a combination of the A and B types, is commonly found in smooth pea and various bean starches. The V type can be found only in amylose helical complex starches after starch gelatinization and the formation of complexes with lipids or related compounds.

Using the Englyst method, Zhang et al. showed that native cereal starch is an ideal SDS, since its structure causes them to be digested slowly [55, 56]. They found that the semicrystalline A-type structure of native cereal starches, including the distribution and perfect crystalline regions in both crystalline and amorphous lamellae explains this slow digestion. The high proportion of SDS in cereal starches was also found to correlate with a higher fraction of short A chains with *DP* 5-10. The mechanism of slow digestion of native cereal starch involves digestion from inside out and layer-by-layer: enzymatic digestion begins at the surface pores and interior channels, and then

side-by-side digestion gradually enlarges the channel by simultaneously digesting crystalline and amorphous regions. Native starch is digested more slowly than processed (gelatinized) starch; the latter has lost its crystalline structure, allowing greater accessibility to enzymes without the obstructions caused by α-glucan associations, such as double helices (especially in crystallites), or by amylose-lipid complexes in cereal starches [57]. Hamaker et al. and Zhang et al. reported that dispersed, amylopectin fine structures with high branching density, either long or short internal chains as well as shortened terminal non-reducing ends, lead to slow digestion, which is the chemical entity that is responsible for a slow digestion property because of the inherent molecular structure of amylopectin [58, 59]. In an *in vivo* study by Seal et al.[46], the plasma glucose response after consuming raw maize starch is slow and sustained, which is characteristic of a typical glycemic response curve of SDS. The structure of SDS may consist of imperfect crystallites and amylopectin with a high branching density and pattern, and this is most likely the cause of the slow digestion.

Heat and Moisture

Heat and water content are important factors in the formation of SDS. When native starches are heated in excess water, the granules undergo a characteristic structural reorganization (gelatinization). The extent of gelatinization depends on the water content, temperature, time, and degree of shear during the process. As previously described, native starch (especially A-type) is an ideal SDS, and the slow digestibility of starch changes in cooked or processed starchy food.

Incomplete gelatinization can be achieved by lowering the heating temperature, decreasing the water content, or shortening the processing time of the starch. In this way, some of the nutritional and low-GI benefits of SDS may be retained. Chung et al. reported that when partially gelatinized waxy rice starch pastes, containing 5% starch on a dry weight basis, are heated at different temperatures (60, 65, or 70 °C for 5 min), they show different digestion rates after retrogradation [60]. The amounts of SDS and RS positively correlate with the relative melting enthalpy of the partially gelatinized starch samples. In cereal products, such as barley porridges, parboiled rice, biscuits, and pasta, the degree of gelatinization or limited-swelling starch, which is determined mainly by the moisture level, cooking time, and temperature, influences the formation of SDS and moderates glycemic response [44, 49, 61-63].

Heat-moisture as one of the hydrothermal treatments usually refers to the incubation of starch granules at low moisture levels (<35% w/w) for a certain period of time at a temperature above the glass transition temperature, but below the gelatinization temperature. At the same time, annealing is performed in excess water or at an intermediate water content (≥40% w/w) [64, 65]. Heat-moisture treatment does not destroy granule structure, but it alters the crystalline packing of starch; for example, the B type of potato starch can be converted to the A or C type, while the annealing processes modify the binding forces between the crystallites and the amorphous matrix [66]. Therefore, hydrothermal treatment can be used as a method to form SDS. Anderson et al. adjusted non-waxy and waxy rice starches to 20% moisture (wet basis), heated them to their melting

temperature (T_m) in a differential scanning calorimeter, and held them there for 60 min [67]. They found that the starches were digested more slowly than unheated samples. Moreover, microwave heating produces only minimal changes in digestibility, even though it causes a re-association of amylopectin branch chains [68]. Severijnen et al. used these principles to study the production of a sterilized liquid product with a low GI [69]. When the modified high amylose starch is heated above 120 °C for 4-5 min, the SDS proportion increases and reaches a maximum, where it remains for at least several months when stored at 4 °C. According to a patent of Woortman and Steeneken [70], high SDS content can be achieved when producing a gellable starch product by heating starch with an amylose content below 50% to at least 170 °C under mildly acidic conditions, and then rapidly cooling it.

Interactions of Starch with Other Components

Interactions of starch with different components present in the food system are known to influence the formation of SDS or SDS-state food; the latter refers to the form of food in which starch is slowly digested. The two most important forms of starch interaction with other constituents involve formation of starch-protein interactions and starch-lipid complexes. Starch-protein interaction in the protein matrix is thought to reduce the rate of α-amylolysis in cereal and legume products [71-74]. According to Colonna et al. and Granfeldt and Björck [72, 75], a viscoelastic and dense gluten network surrounds the starch granules in pasta products and reduces the access of amylases to the starch, and it also restricts the swelling and leaching of starch during

boiling. In cooked sorghum porridge, encapsulation of starch in sheet-like and web-like protein structures reduces the access of degrading enzymes to the starch. This explains the lower starch digestibility and slower kinetics of digestion [76]. The interaction between starch and protein also limits the availability of the starch in white bread made from regular flour, while gluten-free bread elicits a higher glycemic and insulinemic response [71].

In a study by Holm et al. [77], amylose in starch molecules forms complexes with lysolecithin, and these complexes are degraded slowly and are completely absorbed in the gastrointestinal tract of rats within 120 min. As a result, they produce lower plasma glucose and liver glycogen than does free amylose. Murray et al. evaluated apparent digestibility in ileal-cannulated dogs that were fed enteral diets containing a debranched amylopectin-lipid complex (V-complex) or RS [78]. They found that the ileal and total tract digestibilities of carbohydrate for the control, V- complex, and RS diets were 89%, 76% and 43%, respectively, which indicates that consuming a diet containing V-complex diet lowers the carbohydrate digestibility and, subsequently, the serum glucose and insulin responses.

In addition to interactions with protein and lipid, starch may also interact with soluble fibers (guar gum, psyllium, β-glucans, or pectin), anti-nutrients (enzyme inhibitors, tannins, phytates, saponins, or lectins), sugars, and organic acids [3, 74, 79]. Brennan et al. reported that the rate of starch hydrolysis slows significantly when the starch granules and surrounding bread matrix are coated with a layer of galactomannan mucilage, which acts as a physical barrier to amylase-starch

interactions and subsequent release of hydrolyzed products [80]. In addition to its effect on digesta viscosity, guar gum may significantly reduce the rise in postprandial glycemic response that results from the reduction in the rate of gastric emptying. Starch blockers (α-amylase inhibitors) may inhibit *in vitro* α-amylase activity or may bind to starch substrate, indicating that these substances have the potential to interfere with starch digestion *in vivo* and thereby modulate the glycemic effect of SDS [81, 82].

Processing Conditions

Processing techniques may affect both post-processing/cooking processes and storage conditions (retrogradation), influencing SDS formation in food, i.e. the formation of SDS-state foods. This fact is of great importance for the food industry, since it offers the possibility of increasing the SDS content of processed food and foodstuffs, such as the use of whole grains in bread or paste, wherein the cellular layers surrounding the starch granules are intact and present an organized food form to physical hindrance of enzyme accessibility. Autoclaving, baking, pressure-cooking, flaking, and parboiling, among other methods, are known to influence starch digestibility and the yield of SDS [83-86]. Holm et al. demonstrated that starch in flaked whole grain wheat is less available than that in boiled, popped, and steam-cooked wheat in an *in vitro* assay using pepsin and pancreatic α-amylase [84]. This starch elicited lower plasma glucose and plasma insulin *in vivo*. In a study of Casiraghi et al. [85], both parboiled and quick-cooking parboiled rice are digested more slowly with a lower GI than polished rice, which has a starch availability for

α-amylase similar to that of white bread. Autoclaving of red kidney beans was shown to increase the metabolic response (blood glucose and insulin levels) compared to boiling at atmospheric pressure, and this result may be due in part to the thermal or mechanical alteration of the botanical structure of the seeds and also to the release of physically inaccessible starch as a result of the mechanical disruption of cell walls [87, 88]. Granfeldt et al. reported that thick rolled oats cause lower metabolic responses than reference bread or thin flakes [89]. Boiling and pressure- cooking significantly decrease the levels of SDS in three varieties of rice (Doongara, Inga and Japonica) and the amylose content affects starch digestibility, which has been attributed to the process of retrogradation [90].

According to Guraya et al. [23], when 10% non-waxy and waxy starch suspensions are debranched with pullulanase, followed by heating, cooling to allow crystallization or gelling, and then stirred, the digestibility decreases after cooling, because of the prevention or slowing of the formation of crystalline structures or double helices. Freezing of debranched, cooled waxy and non-waxy starch does not affect this decrease in digestibility. During pullulanase debranching and retrogradation treatment of the cooked waxy maize starch suspensions, short-term retrogradation occurs as a result of the gelation and crystallization of the amylose fraction, leading to maximum SDS formation; in contrast, long-term retrogradation due to the amylopectin fraction occurs during storage of starch gels [91]. Chung et al. showed that the retrogradation changes the enzymatic

digestion behavior of the waxy rice starch samples, leading to significant changes during the initial stages of digestion [60].

PREPARATION OF SDS

For starchy products with high SDS, structural modification of starch molecules (double helical structures and crystallites) can be achieved using physical, enzymatic, chemical, genetic, or multiple methods.

Physical Modification

Physical treatments for preparation of SDS include hydrothermal treatment, recrystallization, polymer-entrapment, and extrusion. Shin et al. reported that when granular sweet potato starch with 50% moisture content is heated to 55 °C, the amount of heat-stable SDS relative to that of raw starch increases by 200% [92]. Hydrothermal treatment of granular sweet potato starch alters its structure from C_b type to A-type as a result of the starch crystallites melting and subsequent recrystallization. This structure change reduces the relative crystallinity and converts a fraction of amorphous amylose into the crystalline form, thereby increasing enzyme susceptibility. According to Guraya et al.[24], the maximum SDS (44%) is produced using higher enzyme concentration, shorter debranching time, and rapid cooling and storage at 1 °C. This process favors the nucleation step of crystallization and the formation of SDS, while higher temperature favors the propagation and maturation of crystals, resulting in RS formation. Shin et al. observed a similar result in the storage of waxy sorghum starch that had been debranched with isoamylase [25]. The SDS contained

optimum product at a level of 27% after isoamylase treatment for 8 h and storage at 1 °C for three days. The resulting SDS fraction may consist primarily of amorphous regions and a small portion of imperfect crystallites. Miao et al. showed that controlled retrogradation with partially debranched waxy maize starch can be used to make SDS, which occurs upon the formation of imperfect, low-density B-type crystallites [91]. Debranching treatment of waxy starches forms a great number of short chains available for chain re-alignment and cross-linking, and it favors the formation of double helices that aggregate into ordered crystalline structures via hydrogen bonding and/or hydrophobic interactions during cooling, leading to the formation of more SDS. Similarly, SDS can be obtained through storage of cooked-debranched starch, and subjecting the starch samples to additional hydrothermal treatment increases the amount of boiling-stable SDS [93]. Recent studies have shown that retrogradation correlates with the SDS content of mutant maize; this maize has a higher proportion of long amylopectin chains and linear branched chains of amylopectin with *DP* 9-30. This type amylopectin molecule probably acts as an anchor point to slow the digestion of branched-chain fractions with *DP* > 30, which as physical entities are the primary constituent of the slowly digestible portion of the starch [59]. Abrahamse et al. developed a sterilized food product containing a high level of SDS by melting amylose and then rapidly cooling before storing or drying it [94].

Using entrapment or encapsulation of the starch in the structured protein network of noodles to reduce digestibility, Venkatachalam and Hamaker showed that biopolymer-entrapped starch

microspheres can be used as novel slowly digestible carbohydrate ingredients that lead to a moderate and extended glycemic response [95]. Starch encapsulated spheres with a maximum SDS concentration of 44% were prepared by dropping a homogeneous mixture of 1% (w/w) sodium alginate and 5 g of starch into a 2% (w/v) $CaCl_2$ solution. This technology was patented by Hamaker et al. [96], who used it to create starch entrapped in biopolymer matrix compositions, which provides boiling-stable, slowly digestible starch that can serve as a source of fermentable dietary fiber with health benefits. Factors such as biopolymer type and concentration, as well as microsphere size and starch type, have been manipulated to obtain defined amounts of SDS ranging from 23 to 50%. An SDS product can be generated by using partially gelatinized or partially plasticized materials to form a low-swelling network linking mixed crystallites consisting of short-chain amylose (DP < 300) and basic starch. This network can be formed through cooking or mixing processes, especially extrusion [97]. In addition, according to the patent of Winowiski et al. [98], SDS can be generated in feed by adding a reducing carbohydrate to comminuted cereal grain, heating the mixture, then drying. In other words, physical modifications of the starch structure that affect enzyme binding and the rate of digestion can be used to modulate starch digestibility and form SDS.

Enzymatic Modification

Controlled enzymatic treatment of starch with pullulanase, isoamylase, α-amylase, β-amylase, or transglucosidase is an alternative approach to changing the chain-length of starch supramolecular

structure in order to achieve appropriate digestibility and glycemic response. According to a disclosed patent of Shi et al. [99], a slowly digestible starch can be prepared by debranching amylose-containing starches using pullulanase or isoamylase. In the case of waxy starches, higher concentrations of debranching enzyme and shorter debranching time are more suitable for debranching starch to form SDS [24]. According to Han et al.[28], a low GI maize starch with some branched structure can be developed by partial α-amylase treatment and retrogradation, since slow digestibility was retained after cooking. Short chains of amylopectin and non-crystalline amylose are rapidly digested, while DP_n 121 chains show the greatest resistance to digestion, followed by DP_n 46 chains. A similar trend was reported in the formation of SDS from native and commercial starches by controlling the hydrolysis of gelatinized starch with α-amylase [100]. Recently, van der Maarel et al. filed a patent for "novel slowly digestible storage carbohydrate", which is produced by treating a native root or tuber starch, comprising more than 90% amylopectin, with a branching enzyme derived from a microorganism with a branching degree of at least 8.5-9% [101]. Daniel and Marie-Helene showed that soluble, highly branched glucose polymers containing a larger proportion of α-1,6 glucoside linkages, produced using a branching enzyme, are an SDS-state food that can regulate digestion [102]. Ao et al. reported that both the increase in branch density and the crystalline structure of starch enhance its slow digestibility through the partial shortening of amylopectin A and B1 chains (exterior chains), as well as linear chains of amylose, through the action of β-amylase and maltogenic α-amylase [103]. This correlates closely with an increase in the

number of α-1, 6 linkages and a decrease in the number of α-1, 4 linkages. The enzyme-treated starch contains B- and V-type crystalline structures, which increase the resistance of the starch to digestion. These studies suggest that enzymatic debranching of the exterior chain length of amylopectin in order to change its molecular structure can form starch with higher proportions of SDS.

Chemical Modification

In many processes, starch is modified by chemical methods to improve functionality and create commercially valuable, starch-based products. The most common chemical modification processes are acid treatment, cross-linking, oxidation, and substitution, including esterification and etherification. Some studies have focused on such treatments in SDS production. In the patent of Ian et al.[104], a chemically modified starch is achieved using propylene oxidation, acetylation, octenyl succinic anhydride (OSA) modification, phosphorylation, dextrinization, or combinations of such treatments to yield less than 25% blood glucose at 20 min and 30-70% at 120 min after ingestion, indicating controlled glucose release over an extended period of time and more constant glucose levels. Wolf et al. reported an effect of chemical modification on the rate and extent of digestion of common starch (27% amylose), waxy and dull waxy starch (0% amylose), and a high-amylose variety (50% amylose) [105]. The extent of starch digestion was significantly reduced using dextrinization, etherification, and oxidation, except for cross-linking, whereas the rate of starch digestion was not markedly affected by any chemical modifications. These chemical

modifications generated RS rather than SDS, and increasing the degree of modification decreased the extent of digestion. According to Shin et al. [106], the optimal conditions for using citric acid treatment to produce rice starch enriched in heat-stable SDS (54.1%) is to add 2.62 mmol of citric acid to 20 g of starch, which is then incubated at 128.4 °C for 13.8 h. Esterification with OSA has been shown to be the most potent method of modifying waxy starch to form SDS, followed by combined modifications (crosslinking-hydroxypropylation or acetylation) and crosslinking [107]. Based on the results of Wolf et al.[108], OSA-modified starch shows markedly low glycemic response during human trials, consistent with the extended glucose release profile of SDS. Dry heating (130 °C) of OSA-starch increases the SDS content and decreases the RS content, which consistent with the results of acid-treated rice starch cooking reported by Shin et al. [106]. He et al. also showed that a higher level of SDS (42.8%) was produced by subjecting OSA-starch to heat-moisture treatment (10% moisture, 120 °C for 4 h) than by treating OSA-starch (28.3%) with the Englyst method [43]. The modified starch products with attached OSA molecules may act as uncompetitive inhibitors to reduce the enzyme activity and thereby cause slow digestion. As these studies show, chemical modifications can be used to prepare SDS, but clinical and toxicological trials need to be performed in order to evaluate the safety and efficacy of SDS consumption.

Genetic Modification

Genetic modification of starch biosynthesis involves developing a strategy to generate new cultivars with desired functionality through extensive breeding and characterization of the

resulting varieties. Genetically controlled factors that affect the type of starch produced include starch structure, starch content, interacting cell components, and starch granule architecture. Waxy starches may be more suitable for making SDS, since their fine amylopectin structure--the distribution of branches and chain length--is more critical for SDS formation [24]. Moallic et al. developed a novel SDS (long-chain amylopectin starch) from maize by overexpressing a particular enzyme involved in starch biosynthesis [109]. This starch, with high granule crystallinity, has few short chains and more intermediate and long chains. Zhang et al. also showed that genetic mutants containing amylopectin starch molecules with either a higher proportion of short chains with $DP < 13$, particularly A chains with *DP* 5-9, or a higher proportion of long chains with $DP \geq 13$, particularly intermediate to long B chains with $DP > 30$, contain a greater proportion of SDS than wild type [31]. According to a study by Benmoussa et al. [30], formation of SDS positively correlates with the presence of both long and intermediate/short chains, respectively, while it negatively correlates with the lowest proportion of extremely short chains. They also found that starch granules channels can regulate starch digestibility, since starch granules with channels are digested from the interior outward, and more extensive channelization gives the enzymes more access to substrate [110]. Therefore, genetic engineering has the potential to produce ideal starch with a high SDS content.

BENEFICIAL PHYSIOLOGICAL EFFECTS OF SDS

Many studies have suggested that choosing carbohydrates with a low GI (GI ≤ 55) has beneficial effects on various aspects of physiology and metabolic disorders involved in chronic non-transmissible disease. As described above from Fig. 1, low-GI diets yield a more stable diurnal profile, reducing postprandial hyperglycemia and hyperinsulinemia, as well as attenuating late postprandial rebound in circulating, non-esterified fatty acids (NEFA), all of which are factors that exacerbate these metabolic syndromes (Table 1). Lower glucose and insulin levels are associated with improved risk profiles, including insulin sensitivity, β-cell function, high-density lipoprotein cholesterol, oxidative status, prothrombotic factors, and endothelial function [3, 5-7].

A moderate postprandial glycemic and insulinemic response due to SDS implies that SDS-rich foods may provide wide health benefits by reducing common chronic diseases related to diet, such as diabetes and pre-diabetes, cardiovascular diseases, and obesity ('metabolic syndromes'). SDS-rich foods may exert these effects by reducing the stress on regulatory systems related to glucose homeostasis [115]. According to Seal et al.[46], the plasma glucose concentration and serum insulin concentration in both healthy adults and diet-controlled type 2 diabetic subjects rose faster, and showed a maximum glucose change approximately 1.8 times greater, when rapidly hydrolyzed starch was digested than when slowly hydrolyzed starch was digested. As reported by Ells et al. [4], SDS consumption leads to a low and sustained glycemic and insulinemic response as well as low NEFA, which can decrease cholesterol. Such a response can contribute to the prevention and treatment of diabetes and the complications of this metabolic syndrome. There was

a reduction in plasma triacylglycerols, phospholipids levels, and epididymal adipocyte volume as well as a tendency towards lower plasma insulin levels after consumption of low-GI starchy food compared to an isoenergetic isoglucidic high-GI diet for five weeks [117]. High-GI starch is thought to increase fatty acid synthase activity and lipogenesis by reducing the amount of hepatic phosphoenolpyruvate carboxykinase mRNA, and this starch may have undesirable long-term metabolic effects [118]. Mixed meals containing SDC induce low glycemic and insulinemic responses, and they also reduce the postprandial accumulation of both hepatically and intestinally-derived, triacylglycerol-rich lipoproteins (apolipoprotein B-100 and B-48) in obese subjects with insulin resistance [119]. Rapidly and slowly digestible carbohydrates differ considerably in the stimulation of incretin hormonal secretion. SDS induces the late and prolonged glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) response from 3-5 hours after ingestion, which may indicate that SDS modulates glucose homeostasis and regulation of energy storage in the late postprandial phase [120].

SDS and Diabetes Mellitus (DM)

Postprandial hyperglycemia leads to insulin resistance and eventual pancreatic β-cell failure, which result in non-insulin-dependent diabetes mellitus (NIDDM), which comprises 90% of diabetes cases. Therefore, reducing meal-associated hyperglycemia is one goal in the prevention of DM. Pharmaceutical approaches have shown that reducing the rate of carbohydrate digestion attenuates the postprandial glucose response. A glucosidase inhibitor (Acarbose) and *Phaseolus*

vulgaris α- amylase inhibitor (Phase 2®) are effective at reducing postprandial hyperglycemia; they act by reducing starch bioavailability and colonic fermentation [82, 121, 122]. Because of the poor nutritional view of nutritionist, this method should be restricted, while the use of SDS is preferred [123]. Kaufman et al. reported that raw (uncooked) cornstarch as an SDS source as part of an evening snack may diminish the nighttime and morning hypoglycemia associated with insulin-dependent diabetes mellitus (IDDM), without causing hyperglycemia [124]. This method of ingesting a complex carbohydrate to regulate and stabilize the blood glucose level has been patented [125]. Two other patents have appeared that use native cornstarch by itself to prepare medical foods for improving impaired glucose tolerance and preventing hypoglycemia- induced diabetes [126, 127]. Clinical studies have shown that a low-GI diet results in a modest improvement in long-term glycemic control in normolipidemic, well-controlled individuals or overweight patients with NIDDM [128, 129]. In randomized controlled trials, a low-GI food proved more beneficial in the management of diabetes than a conventional or high-GI food, through reduction of HbA1c or fructosamine levels [130]. Consumption of low-GI, starchy foods decreases the glucose and insulin responses throughout the day and improves glucose utilization, lipid profile, and capacity for fibrinolysis, suggesting therapeutic potential for the treatment of diabetes [131, 132]. SDS can reduce meal-associated hyperglycemia, and hence it should be recommended for the prevention and management of DM.

SDS and Cardiovascular Disease (CVD)

High carbohydrate consumption is associated with increased serum triglycerides and low HDL-cholesterol levels, both of which are hallmarks of the metabolic syndromes and an increased risk for certain cardiovascular diseases [133]. High-carbohydrate and high-GI diets are linked to a risk of coronary heart disease in women during a large prospective study [134]. Cross-sectional studies showed that low-GI diets are associated with a high concentration of HDL-cholesterol, especially in women [135]. In addition, in a well-controlled study involving type 2 diabetic patients, several parameters were found to be significantly lower after a low-GI diet than after a high-GI diet: the fasting plasma total, the activity of serum cholesterol, LDL cholesterol, free fatty acids, apolipoprotein B, and plasminogen activator inhibitor 1 [129, 132]. Bouché et al. reported that five weeks of a low-GI diet improves some plasma lipid parameters, decreases total fat mass, and tends to increase lean body mass without altering body weight; these changes are accompanied by a decrease in the expression of some genes implicated in lipid metabolism [136]. In this way, such a diet may help healthy, slightly overweight subjects and it may play a role in the prevention of metabolic diseases and their cardiovascular complications. In conclusion, SDS can have an impact on postprandial blood glucose, insulin levels, and the resulting metabolic syndromes, such as CVD and coronary heart disease (CHD).

SDS and Glycogen Storage Disease (GSD)

GSD is any one of several hereditary metabolic disorders that result from enzyme defects. It affects glycogen synthesis or degradation in muscles, liver, and other cell types. Type I GSD is

associated with the absence or deficiency of glucose-6- phosphatase, which results in hypoglycemia during fasting. Uncooked native maize starch has been administered overnight to patients as a continuous dietary source of glucose, an approach that has proven effective as an oral therapy for preventing nighttime hypoglycemic episodes [137, 138]. Qi and Tester reported that a therapeutic food composition comprising a waxy and/or hydrothermally treated starch may be used to treat or prevent hypoglycemia in patients susceptible to hypoglycemic episodes, such as patients with GSD, type 1 diabetes, or liver disease [127]. Giving GSD patients cornstarch processed by controlled heat-moisture treatment induces euglycemia that lasts longer and leads to metabolic control that is better in the short term, compared to uncooked cornstarch [139]. In these cases, SDS can be a significant aid in the treatment of GSD.

SDS and Weight or Obesity Control

Obesity is becoming more prevalent and is associated with an increase in mortality and morbidity due to DM, hypertension, CVD, stroke, and cancer. In theory, low-GI foods may benefit body weight management by promoting satiety and fat oxidation at the expense of carbohydrate oxidation [112, 116, 140, 141], which would support the hypothesis of nutritional benefits from SDS consumption. Epidemiologic and clinical intervention studies show that a low-GI diet increases satiety to a greater extent and reduces plasma insulin responses more than a high-GI diet [112, 142, 143]. Consumption of overall high-GI starch favors adipogenesis more than does low-GI starch, leading to a greater risk of obesity, since digestion and absorption of low-GI starch may be slower than that

of high GI starch. Slower digestion leads to reduced lipogenesis in adipose and liver tissues, whereas the apparent digestibility of low-GI starch is similar to that of normal cornstarch [144]. A diet high in rapidly absorbed carbohydrates leads to an increase in the total body and hepatic fat deposition, hyperinsulinemia, and an elevation in the concentration of plasma triacylglycerol. In this way, consumption of a low-GI diet decreases the risk of non-alcoholic fatty liver disease (NAFLD) in humans [145]. The oxidation pattern of unavailable and slowly digestible carbohydrate helps to modulate feelings of hunger, and the high-unavailable carbohydrate diet suppresses feelings of hunger to a greater extent than does the low-unavailable carbohydrate diet during the postprandial periods [14]. Thus, low-GI meals appear to improve access to stored metabolic fuels and promote satiety [146-148]. *Ad libitum* studies in overweight or obese adults and children show that low-GL diets correlate with marked weight benefits, loss of adiposity, and reduced food intake [148]. Long-term randomized controlled trials showed that a low-GI diet can be used to prevent and treat obesity in children [149]. Therefore, these studies identify SDS as a major component of strategies to prevent obesity.

SDS and Physical Exercise

To avoid hypoglycemia, central fatigue, and exhaustion associated with endurance sports activities, such as marathons, oral glucose may need to be taken before prolonged vigorous exercise in order to increase endurance. Consumption of a low-GI and carbohydrate-rich food may attenuate the insulin-mediated metabolic disturbances associated with carbohydrate intake prior to

exercise, facilitating the maintenance of carbohydrate availability [150]. Therefore, SDS may be beneficial in products used by athletes to enhance performance by providing an increased, more consistent source of systemic energy via extended glucose release [99, 100].

SDS and Cognitive Performance

Brain processes that provide accurate perception and memory of environmental events enable organisms to function successfully depending on the enough glucose from food. A number of studies performed in adults have shown that missing breakfast may impair performance in tasks that test reaction time, spatial memory, and immediate word recall [151]. Lang et al. invented a breakfast cereal product (biscuit or cracker) containing more than 12% (w/w) SDS with the goal of improving cognitive performance in people, particularly in children and adolescents, specifically with regard to memory retention, attention, concentration, vigilance, and mental well-being [152]. Glucose regulation has been associated with cognitive performance in elderly subjects with normal glucose tolerance, and dietary carbohydrates result in enhanced cognition in subjects with poor memories or β-cell function independently of plasma glucose [153]. A recent study involving low-GI breakfasts containing SAG rather than RAG showed improvement in cognitive performance later in the morning [154]. A literature review that focused on physiological effects of starches concluded that administration of glucose may influence both memory and mood, particularly when intense metabolic demands are placed on the brain [155].

SDS and Other Health Implications

The digestion of alginate-entrapped starch microspheres as an SDS source in the alimentary canal generates short-chain fatty acids, such as acetic, propionic, and n-butyric acid, which help to prevent colon cancer and produce less energy [96, 156]. Low pH in human dental plaque caused by bacterial fermentation of sugars into acids induces dental cariogenesis. The pH has been shown to decrease the least after ingestion of whole-grain foods containing considerable amounts of SDS [9, 111]. Raw cornstarch can also be used to maintain normoglycemia in children with nesidioblastosis [157]. Intake of low-GI diets for 10 weeks reduces LDL cholesterol, which is beneficial for preventing ischemic heart disease [158]. Consumption of low-GI carbohydrates during pregnancy not only reduces the risk of gestational DM in healthy pregnant women, but improves the long-term outcomes of infants [159].

APPLICATIONS OF SDS

SDS, as a new functional component or ingredient in novel product development, can be widely used in edible solid or liquid processed food products, nutritional supplements, and drug preparations (tablet, emulsion, and suspension). The amount of SDS added is selected in order to achieve the desired functional properties, digestibility, and glucose release rate, or some desirable balance of these parameters. SDS can be used in the form of a powder as an ingredient in a variety of edible products to modulate the rapid glucose release that is typical of many processed starchy foods, such as cakes, bread, cookies, pastries, pasta, pizza, cereals, chips, fries, candy, muesli,

dressings, fillings, icing, sauces, syrups, soups, gravies, puddings, custards, cheese, yogurts, creams, beverages, dietary supplements, diabetic products, sports drinks, nutritional bars, energy bars, as well as food for children and babies [93, 97, 99-101]. A new slow-digesting rice starch (Ricemic) has been developed at the USDA ARS Southern Regional Research Center, and used to maintain a stable blood-sugar level in diabetics, to provide athletes with a steady energy supply to maintain endurance, and to replace fat in non-frozen dairy products [160, 161]. To date, starch-based cereal foods and whole-kernel foods have been developed with both a low GI and a high SDS load, for example the EDP® ("energy delivered progressively") range of plain biscuits developed by experts from Danone Vitapole. These products are currently marketed in several European countries (Belgium, The Netherlands, France, Spain, Italy, Czech Republic, and Slovakia), as well as in China, Malaysia, and Russia. Jolly-Zarrouk et al. reported an extended energy beverage containing much SDS (1.5-15 times) prepared by hydrothermal treatment (20-35% moisture, 100-110 °C for 20-60 min), such as Milo® beverage, Nesquick® beverage, Migros® drink, or orange juice from Nestlé [162]. Numerous reports describe how to produce SDS, while studies regarding the mechanism and molecular structural basis of slow digestion are fewer. In addition, most of the reported SDS materials show low thermal stability when used in food processing. The current challenge for the food industry is to develop new technologies to make tailor-made carbohydrate foods with low GI and appropriate amounts of heat-stable SDS.

Diabetic snack bars are available that are formulated with uncooked cornstarch either to prevent hypoglycemia or to reduce postprandial hyperglycemia: Extend Bar (Clinical Products, Ltd.), Nite Bite Timed-Release Glucose Bar (ICN Pharmaceuticals, Inc.), Gluc-O-Bar (APIC, USA, Inc.), Ensure Glucerna (Rose Products Division, Abbott Laboratories), and Choice DM (Mead Johnson Nutritionals) [163]. In addition, given the characteristics of enzymatic digestion in the upper gastrointestinal tract, SDS is used as a novel, starch-based biodegradable carrier that may prove useful in oral drug delivery systems specifically targeting the small intestine [93]. For example, SDS may be able to serve as a biomacromolecule film-former for pharmaceutical/nutraceutical coatings to allow complete release in the small intestine.

In addition to its applications in food and medicine, SDS can also be used as feedstuff material. Based on a patent of Winowiski et al. [98], feed for ruminants that is rich in SDS may reduce the rate of digestion by rumen microbes, thereby reducing the effect that rapid consumption of fermentable grains can have on rumen pH and fiber digestion. This may provide a more even flow of fermentable starch to support microbial metabolism, and it may increase the proportion of starch from cereal grain consumption that ultimately arrives in the small intestine. Compared to consumption of RDS, consumption of SDS results in improved protein and energy utilization in broiler chickens, such as superior feed conversion in amino acid level [164, 165]

CONCLUSIONS

Accumulating epidemiological data indicate that a diet characterized by low-GI foods has beneficial metabolic effects and the potential to reduce insulin resistance and improve certain metabolic conditions. Consuming a much wider range of low-GI foods is necessary to achieve a well-balanced, low-GI diet. However, there are few commercially available low-GI products on the market, which severely fails to meet the growing needs of people with diabetes, obesity, and related disorders. SDS as a novel functional component in products delivers a slow and prolonged release of glucose when digested, resulting in a lower GI. It not only helps to fill the existing scarcity of low-GI foods available, but it also maintains glucose homeostasis and prevents metabolic syndromes. Although SDS is potentially beneficial to health, the slow digestion and structural properties of starch need to be further elucidated in order to increase the SDS content in processed food.

ABBREVIATIONS

CHD, coronary heart disease; CVD, cardiovascular disease; DM, diabetes mellitus; DNS, dinitrosalicylic acid; DP, degree of polymerization; EGI, extended glycemic index; HI, hydrolysis index; GCE, glycemic glucose equivalent; GI, glycemic index; GIP, glucose-dependent insulinotropic polypeptide; GL, glycemic load; GLP-1, glucagon-like peptide-1; GSD, glycogen storage disease; IDDM, insulin-dependent diabetes mellitus (type 1 diabetes); II, insulin index; MGAM, maltase-glucoamylase; NEFA, non-esterified fatty acids; NIDDM, non-insulin-

dependent diabetes mellitus (type 2 diabetes); OSA, octenyl succinic anhydride; RAG, rapidly available glucose; RDS, rapidly digestible starch; RGP, relative glycemic potency; RS, resistant starch; RVA, Rapid Visco Analyser; SAG, slowly available glucose; SDC, slowly digestible carbohydrate; SDS, slowly digestible starch; SI, sucrase-isomaltase; TNO, The Netherlands Organization for Applied Science Research.

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List of Figure Legends

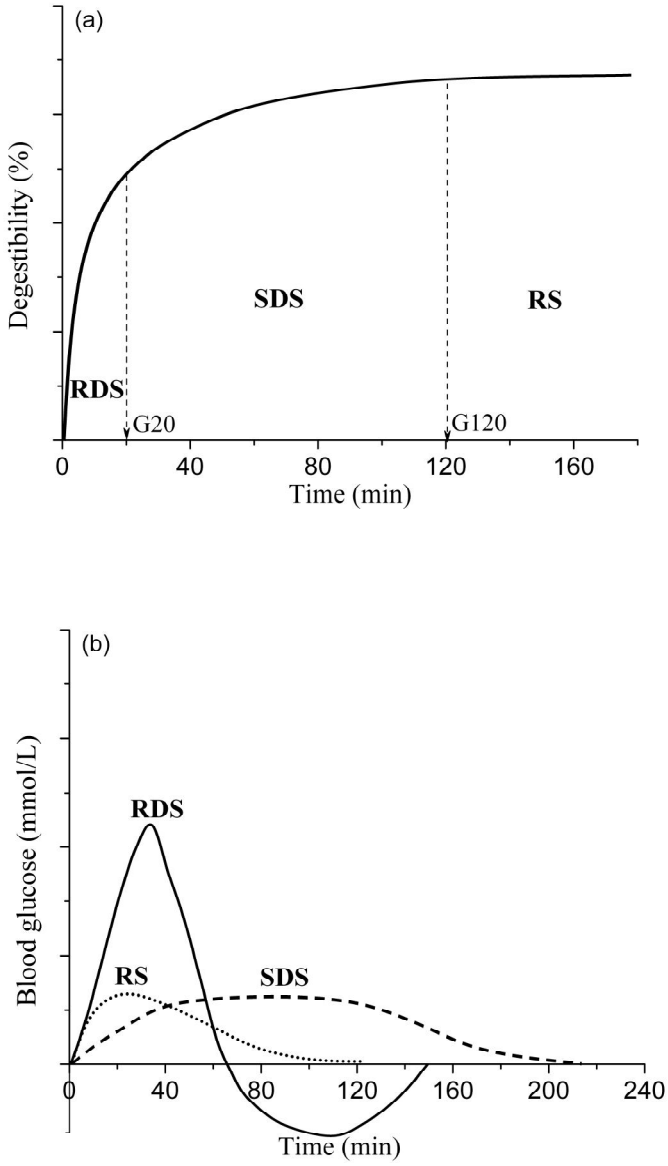

Fig.1 Classification of the bioavailability of nutritional starch fractions. (a) *In vitro* digestion using the Englyst assay, (b) *in vivo* glycemic response to RDS, SDS, and RS.

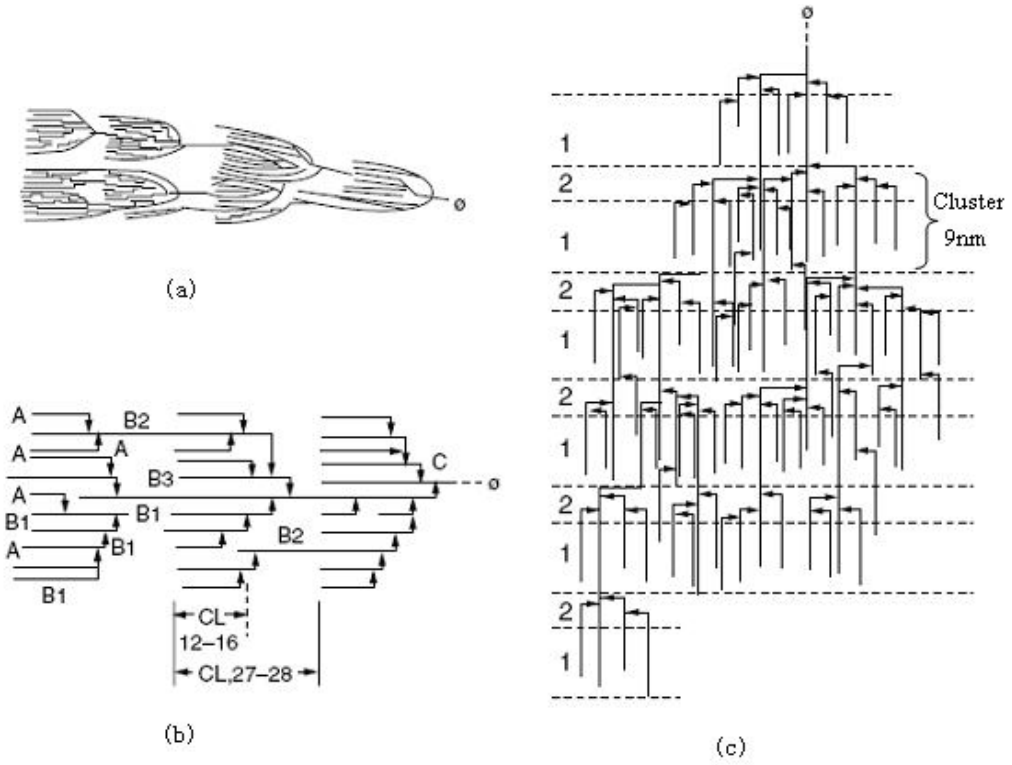

Fig.2 Schematic models of the sub-chains within an amylopectin molecule as proposed by (a) French [51], (b) Hizukuri [52], and (c) Robin et al. [53] (Reproduced with permission). Ø, reducing end; CL, chain length; A chain, outer chain; B1, B2, and B3 chains, inner chains; C chain, backbone chain; 1, crystalline region or high molecular order; 2, amorphous region or low molecular order.

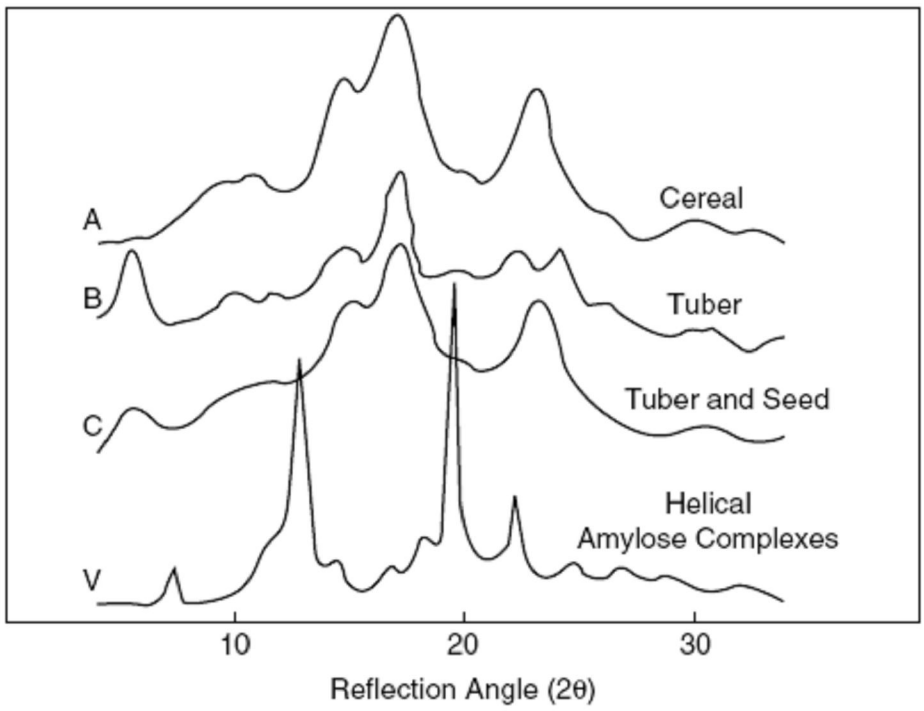

Fig.3 X-ray diffraction diagram of different starches [54] (Reproduced with permission).

List of Table Captions

Table 1 Physiological effects of low-GI foods.Table 1 Physiological effects of low-GI foods

Physiological benefits	**Implications for Health**
Improved metabolic modulation	Diabetes mellitus
Improved glucose tolerance	Cardiovascular disease
Prevention of hypoglycemia and hyperglycemia	Glycogen storage disease
Improved postprandial glycemic and insulinemic response	Dyslipidemia
Reduced blood lipid level	Cancer
Reduced cariogenic potential	Inflammation
Reduced glycosylation of body protein	Athletic performance
Prolonged satiety	Cognitive function
Prolonged physical performance during endurance exercise	Weight management
Delayed aging	Dental care

Source: Truswell et al. [111]; Björck et al. [3, 9]; Ludwig [112], Jenkins et al. [113]; Pi-Sunyer [79]; Augustin et al. [114]; Sievenpiper et al. [115], and Bell et al. [116].