Open Access

Recent Progress in Nutrition

LIDSEN Publishing Inc.

Review

The Case for Slow Energy Release Foods: Weight Management

Xin Qi *, Richard Tester

Glycologic Limited, Lambs Farm Business Park, Swallowfield, Reading, RG7 1UG, Berkshire, UK; E-Mails: e.qi@glycologic.co.uk; r.f.tester@glycologic.co.uk

* **Correspondence:** Xin Qi; E-Mail: e.qi@glycologic.co.uk

Academic Editor: Cristiano Capurso

Special Issue: Nutrition, Carbohydrate Intake and Health

Recent Progress in Nutrition
2026, volume 6, issue 3
doi:10.21926/rpn.2603016

Received: March 23, 2026
Accepted: July 23, 2026
Published: July 26, 2026

Abstract

Excess dietary calories relative to energy expenditure lead to increased body weight, a problem now widely described as an epidemic because of its association with numerous disease states. Although free digestible sugars are to the most extent absorbed relatively easily from foods in healthy individuals, this is not the case for starches; where the physico-chemical properties control the rate and extent of enzymatic hydrolysis in the small intestine. Thus, even when the quantity of starch in a food product is known this may not reflect its calorie provision due to its digestibility. This short review explores this 'glucose supply' issue and its implication for dietary calorie management. In particular, how controlling the rate and extent to which glucose is absorbed from the diet impacts upon body weight and as a consequence health; with a particular emphasis on slowly digestible starch (SDS) as a preferred dietary glucose source. Many Western dietary patterns favour starchy foods that are digested rapidly, producing pronounced postprandial peaks and troughs in blood glucose concentrations. The focus towards slowly digestible starch within the diet offers a more favourable pathway for glucose entry into the circulation, supporting more stable metabolic utilisation. The evidence suggests that this control of glucose absorption from the gut into the blood stream can influence positively body weight, body mass index (BMI) and associated

health conditions linked to body mass. These physical impacts associated with potential benefits on cognitive function.

Keywords
Slowly digestible starch; glycaemic response; starch digestibility; energy intake regulation; weight management

1. Obesity and Its Associated Disease Burden

Obesity leads to a shortened life span and is strongly associated with type-2 diabetes, cardiovascular disease, metabolic syndrome, certain cancers, kidney disease, obstructive sleep apnoea, gout, osteoarthritis and hepatobiliary disease [1, 2]. Importantly, reductions in disease risk occur proportionally with the degree of weight loss [1].

Data from NHS England [3] indicate that among adults aged 16 years and over, sixty-eight percent of men and sixty percent of women were overweight or obese in 2019. Among children, eighteen percent of boys and thirteen percent of girls were obese. Globally, the picture is similar. According to the World Health Organization [4], thirty-nine percent of adults aged 18 years were overweight in 2016, with thirteen percent classified as obese.

Excess body weight also impacts negatively on the development of diabetes. While type-1 diabetes is largely autoimmune in origin, body mass influences disease trajectory; type-2 diabetes is more directly associated with excess weight and adiposity [5].

Together, these findings emphasise the urgent need to develop sustainable strategies for weight management.

Although tackling obesity is a multi-stranded issue including dietary habits and exercise provision, from the food sector it includes reducing the calorie density of foods, portion sizes and frequency of eating. However, until now, there has been little focus on controlling the rate at which carbohydrate calories in the food may be digested with the associated physical and cognitive impacts. Sugar reduction in foods with associated sugar taxes in drinks (such as in the UK) do have an impact on calorie provision. Less work is undertaken in terms of product development on reducing the digestibility profile of starch in foods to make it in effect more like unprocessed (native) starch. Starch that is slowly digested (SDS) has a great potential to support calorie reduction strategies as although it provides calories in the diet it does so relatively slowly with associated physiological benefits. Including obesity and associated health issues.

Foods relatively rich in SDS are those in which there is much of the native structure in place and/or where processing induced modification is limited. These include legumes (lentils, chickpeas, beans), whole grains (oats, barley), some pasta, some nuts and seeds and green banana. Note that there is a blur between when starch is described as SDS or resistant starch (RS). Any SDS can be digested in the human small intestine but slowly over many hours whereas RS is transported to the colon and fermented.

2. Energy Balance and Body Weight Regulation

The concept of weight loss has been described succinctly by Higgins [6] and Aaseth et al. [2] where they report that for weight loss to be successful it is necessary to adopt a diet that creates a permanently negative and yet acceptable energy balance; where prolonged dietary adherence is crucial. Others have described how reduced energy input needs to be associated with increased energy output to be most effective [1]. Weight loss improves blood glucose profile abnormalities in obese patients with type-2 diabetes [7].

Although some evidence suggests that a low glycaemic load but relatively high-fat diet may facilitate weight-loss maintenance [8], intervention ultimately centres on calorie control, as all macronutrients (and ethanol) provide energy that must be regulated [9].

In adults, reduced intake of dietary sugars is associated with decreased body weight and *vice versa* [10]. However, where sugars are replaced isoenergetically with other carbohydrates, no comparable change in body weight is observed. This supports the view that excess calories - including those from dietary sugars - promote obesity, rather than any specific molecular effect of sugars themselves [11, 12]. Country-specific weight-control registries similarly recognise the need to reduce calories from digestible carbohydrates to manage body weight [13].

Thus, while calorie excess is central to obesity, the metabolic handling of those calories warrants closer examination.

3. Digestible Carbohydrates and Metabolic Health

Although energy balance reflects calories from all dietary sources - digestible carbohydrates 3.75 kcal/g, dietary fibre (classified often now in the literature as providing the equivalent of 2.00 kcal/g through colonic, fermentation), protein 4.00 kcal/g, alcohol 7.00 kcal/g and fat 9.00 kcal/g - many diets focus on the carbohydrate portion (restriction) as the major approach to reduce calorie intake and ultimately body weight.

There is increasing emphasis on plant-based eating patterns while limiting calories from digestible carbohydrates - including sugars, amorphous starch and starch derivatives. The evolution of man has led to several factors which increase the availability of digestible carbohydrates in the diet. This, often at the detriment of carbohydrates which function as dietary fibre (with the associated gut health and systemic impact). Contributing factors include food availability and affordability, pre-prepared foods, fast food consumption, sugary drinks, snacking between meals, excess calorie intake and insufficient physical activity.

Blüher [14] has captured the issues associated with obesity very succinctly in the recent review with discussion around the clear doubly impacting imbalance (albeit with regional variation) between (i) excess food supply and (ii) increased sedentary behaviour. Comparable recent obesity/diabetes related reviews identify similar cause and effect on body weight [15-17].

Digestible sugars and amorphous starch derivatives provide approximately 3.75 kcal/g energy for the body. In the UK, added and free sugars contribute between seven and thirteen percent of total energy intake [18]. The World Health Organization recommends limiting free sugars to less than ten percent of total energy intake, ideally below five percent [19]. The UK Scientific Advisory Committee on Nutrition similarly recommends no more than five percent of total energy intake should come from free sugars [20].

Definitions of free and added sugar [20] being:

- Free sugars '… all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and unsweetened fruit juices. Under this definition lactose when naturally present in milk and milk products is excluded.'
- Added sugars (USA definition): '… sugars and syrups that are added to foods during processing and preparation. Added sugars do not include naturally occurring sugars such as lactose in milk or fructose in fruits.'

The role of dietary sugars - including their relative glycaemic index and origin (such as glucose derived from starch digestion) - in health and disease has been reviewed in several recent publications [21-23]. The links between digestible carbohydrates and health are largely defined by their effects on blood glucose concentrations, obesity, and related metabolic disorders.

Sugars are rapidly absorbed from the intestine whereas less rapidly digested carbohydrates provide slow and sustained release of blood glucose along with the overall health benefits resulting from low glycaemic and insulinemic response [24]. A dysregulation in energy provision (includes amount and timing)/metabolism is intricately associated with the pathogenesis of a range of disorders including neurological, cardiovascular, metabolic syndromes, autoimmune disorders and cancer [25]. At the cellular level this (disfunction) reflects [25]:

- Autoimmune diseases: Increased energy consumption, glycolipid dysfunction, inflammation;
- Cancer: Increased glycolysis, glucose deprivation; glutamine deprivation;
- Cardiovascular disease: Mitochondrial dysfunction, increased glycolysis, myocardial cell injury;
- Diabetes: Insulin resistance, tissue damage; proliferation and metastasis;
- Neurodegenerative disorders: Mitochondrial dysfunction, decreased adenosine triphosphate (ATP) synthesis, increased cell death;
- Obesity: Increased lipogenesis; adipocyte proliferation, glycolipid disorder, inflammation.

Substantial evidence indicates that dietary fibre supports a favourable gut microbiota composition, conferring benefits to the host through a bidirectional symbiotic relationship that underpins both physical and psychological health. Conversely, diets high in sugars and refined carbohydrates have been linked to the development and progression of metabolic syndrome and related chronic diseases, as well as detrimental effects on mood and mental well-being [26, 27]. Furthermore, indigestible carbohydrates are of considerable physiological importance, enhancing gastrointestinal transit and acting as prebiotic substrates for beneficial microbial populations. Their fermentation by the gut microbiota generates short-chain fatty acids (SCFAs) and other bioactive metabolites that play key roles in maintaining intestinal and systemic health [28].

The regulation of body weight and the inter-individual variability in weight-related outcomes are determined by a complex interplay between genetic polymorphisms, epigenetic modifications, and alterations in hormonal signalling pathways and metabolic processes. These factors are further modulated by lifestyle behaviours, including nutritional intake, physical activity, sleep quality, and stress exposure, which collectively influence energy homeostasis and long-term body weight regulation [29].

Blood glucose responses following oral glucose tolerance tests differ markedly between metabolically healthy individuals and those with insulin dysregulation [30, 31]. Several nutrition-related disease states have been associated with excessive consumption of digestible starch, particularly in the context of obesity and its contribution to the development of type 2 diabetes and cardiovascular disease [32]. As starch constitutes a major source of dietary energy and generally

exerts limited effects on satiety, attention to the quantity consumed is important in preventing excessive energy intake and subsequent weight gain [32].

However, the relationship between dietary carbohydrates and metabolic disease is complex. Veit et al. [33] reported that current evidence does not support a direct association between dietary sugar intake and the incidence of type 2 diabetes. Notably, certain long-chain α-glucans may elicit a greater glycaemic response than sucrose. The authors concluded that excess energy intake and the consequent accumulation of body fat are more important determinants of diabetes risk than the consumption of specific carbohydrate types alone [33].

Importantly, digestible carbohydrates do not exert uniform metabolic effects. Their physiological impact is influenced by structural characteristics that determine the rate and extent of digestion, absorption, and subsequent glycaemic responses. Consequently, carbohydrate quality, in addition to quantity, is an important consideration in the prevention and management of metabolic disorders.

4. Starch Digestibility, Classification and Metabolic Implications

Starch digestibility is largely determined by its amorphous–crystalline ratio and its accessibility to digestive enzymes. Amorphous starch is digested rapidly and almost completely and can generate a high glycaemic index [34, 35]. Any SDS also contributes to the overall glycaemic response; however, by convention, the GI is determined from blood glucose measurements collected over a two-hour period following ingestion. As a result, a proportion of the glycaemic response attributable to more slowly digested starch fractions may not be fully captured within the standard assessment period. Consequently, GI values may not completely reflect the total glycaemic potential of SDS. Although SDS may ultimately produce a similar cumulative glycaemic exposure, expressed as the area under the blood glucose response curve (AUC), to an equivalent anhydrous glucose load (adjusted for starch moisture content and water of hydration), this response is typically distributed over a longer time frame than the conventional two-hour measurement period. In contrast, non-amorphous starch exhibits varying capacities to resist digestion and is defined collectively as ‘resistant starch’, of which five forms have been described [35-38] (Table 1).

Table 1 Resistant starch.

Type	Description
I	Native starch that is inaccessible to digestive enzymes because it is embedded in plant tissue.
II	Native starch granules where some starches/granules are more resistant to digestion than others due to their physico-chemical properties.
III	Retrograded (re-crystallised, staled) starch where amylose retrogrades much faster than amylopectin.
IV	Chemically modified.
V	Amylose-lipid inclusion complexes (which occur in native starch granules and during food processing).

Three principal fractions of starch were defined by Englyst et al. [39] (Table 2):

- Rapidly digestible starch (RDS)

- Slowly digestible starch (SDS)
- Resistant starch (RS)

Table 2 Rapidly digestible starch (RDS), slowly digestible starch (SDS) and resistant starch (RS).

Form	Duration of digestibility *in vitro* (min) according to Englyst et al. [39]
Rapidly Digestible Starch (RDS)	20
Slowly Digestible Starch (SDS)	20-120
Resistant Starch (RS)	>120

See Zhang and Hamaker [40, 41] and Miao et al. [24] for more details.

The *in vitro* classification system developed by Englyst and colleagues over many years has, for the most part, correlated successfully with *in vivo* trials [42]. Within this framework: RDS corresponds to digestion within twenty minutes (reflecting residence time in the mouth and small intestine residence); SDS is digested between twenty to one hundred and twenty minutes (reflecting small intestinal digestion); and RS resists digestion for more than one hundred and twenty minutes and passes into the colon.

Thorburn et al. [43] reported that there was a good correlation between starch digestibility and blood glucose concentration, linking starch structure directly to metabolic response. The Body Mass index (BMI) is often considered an indicator of adiposity [44]. Several studies have identified a positive correlation between fasting blood glucose levels and BMI [45-47], including in elderly populations [48]. Consistent with the metabolic relevance of glycaemic response, BMI has also been reported to correlate positively with dietary glycaemic index [49]. Furthermore, reducing dietary energy derived from carbohydrates, or lowering the glycaemic index of the diet, has been shown to promote fat loss and improve cardiovascular risk factors [50].

However, some findings appear contradictory. In cases of iatrogenic (investigation- or procedure-induced) hypoglycaemia among individuals with type 2 diabetes, patients with lower BMIs exhibited lower plasma glucose concentrations [51]. Similarly, Plečko et al. [52] reported that a BMI above normal was an independent predictive factor for reduced risk of hypoglycaemia in both diabetic and non-diabetic patients, with the converse also observed. These observations highlight the complexity of interactions between starch digestibility, glycaemic response, BMI, and metabolic regulation.

The balance between starch fractions that are readily digested and those that resist digestion is illustrated in Figure 1. Dextrins represent hydrolysed starches that are predominantly amorphous and therefore readily digested, although some may be modified to restrict digestion.

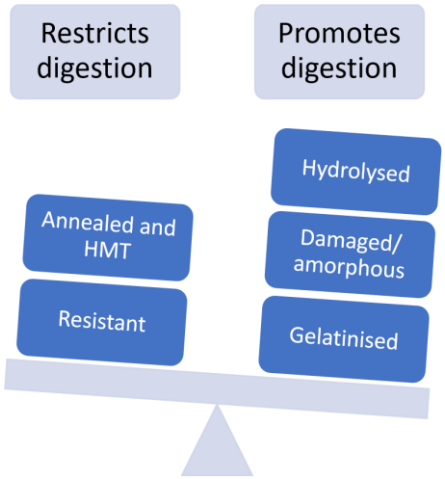

Figure 1 Starch digestive balance where some forms of processing make the starch less others more digestible (HMT = Heat-moisture treatment).

Importantly, some dietary intervention trials comparing starch and sugars do not clearly distinguish between starch forms or account for differences in digestibility, which complicates interpretation of body weight and metabolic outcomes [10]. These features are highlighted in Table 3.

Table 3 Forms of starch consumed in the diet of humans.

Form of Starch	Relative Digestibility
Uncooked in whole plant tissues	Resists digestion
Uncooked in processed plant tissues – especially flours	Resists digestion
Uncooked flours damaged starch fraction (caused by milling)	Relatively easily digested
Uncooked pre-extracted starch	Resists digestion
Acid or enzymatically hydrolysed	Increased digestibility
Chemically modified	Tends to be modifications that resist digestion
Gelatinised (disorganised/gelled progressively by heating in water)	Readily digested
Amorphous (lacking order such as present in native starch granules)	Readily digested
Retrograded (restructured after gelatinisation)	Resists digestion

See references [36-38] for more detail.

5. Indigestible Carbohydrates and Dietary Fibre

Indigestible carbohydrate refers to carbohydrate that resists digestion in the human small intestine. However, some forms, such as non-starch polysaccharides and RS, are fermented in the large intestine to produce short-chain fatty acids, which provide approximately 2 kcal/g of energy to the body. Common types of indigestible carbohydrates are presented in Table 4. These carbohydrates are more commonly referred to as dietary fibre.

Table 4 Sources and properties of common dietary fibre molecular species.

Name	Common sources	Physiochemical characteristics		
		Solubility	Viscosity	Fermentability
Cellulose	Plant cell walls	Insoluble	Non-viscous	Low
Arabinoxylan	Cereals, psyllium	Low-medium	Medium	High
β-glucan	Cereals, terrestrial fungi, seaweed	Low-medium	Medium-high	High
Pectin	Fruit and vegetables	High	Medium-high	High
Inulin	Cereals, fruits and vegetables	Medium-high	Low-high	High
Galacto-oligo saccharide	Dairy products and vegetables	High	Low	High
Resistant starch type-III	Retrograded starch in cooked foods	Low	Non-viscous to low	High

Adapted from Gill et al. [53].

Although the molecules classified as dietary fibre differ chemically in their sugar residue structures, residue modifications, bonding patterns, and molecular size, they are traditionally sub-classified nutritionally according to their solubility. More recently, however, colonic fermentability has been proposed as a more meaningful primary classification criterion than solubility [54, 55].

Both dietary fibre and RS play important roles in body weight regulation and weight loss, as well as in the prevention and management of type 2 diabetes [56, 57].

Chew and Brownlee [58] reviewed studies on the impact of dietary fibre - and fibre supplementation especially - on body weight/weight loss. Overall, they found that higher dietary fibre intake was associated with improved long term weight management, although the effectiveness of supplementation varied according to fibre type. Evidence of benefit was reported for glucomannan, chitosan, fibre complexes derived from *Opuntia ficus-indica* (prickly pear cactus; often enriched with acacia fibre), and soluble fibre blends containing konjac, sodium alginate, and xanthan gum. However, the authors noted that methodological differences among studies may have influenced the reported outcomes.

Several mechanisms have been proposed to explain the effects of dietary fibre on weight management [58]. These include: (i) increased satiety and satiation through bulking and intragastric gelation with potential regulation of cephalic and peripheral satiation-signalling hormones; (ii) decreased calorie uptake resulting from the indigestibility of fibre and its potential interference with macronutrient digestion and absorption; (iii) stimulation of the excretion of bile acids driving metabolic flux; (iv) increased energy expenditure during digestion itself and; (v) providing a substrate that alters the gut microbiota or the profile of fermentation by-products.

6. Slowly Digestible Starch and Body Weight Control

As an energy source, starch - particularly its slowly digestible and resistant forms - is considered significantly healthier than sugar when comparing their respective effects on physiological parameters, especially body weight [6, 40, 41, 59-64].

Consumption of SDS has been associated with improved calorie control through attenuation of the postprandial glycaemic response and prolongation of satiety [65]. SDS-rich foods are digested more slowly than conventional starches, resulting in more stable blood glucose concentrations and a sustained feeling of fullness. By extending satiety and reducing subsequent food intake, SDS may contribute to lower overall energy consumption and reduced weight gain [65].

The literature, including the detailed review by Raghunathan et al. [66], suggests that the effects of SDS on weight management are mediated through three interrelated physiological mechanisms:

i. The gradual release of glucose into the bloodstream, promoting a more stable insulin response and reducing the postprandial glycaemic excursions associated with fat accumulation;
ii. Stimulation of the secretion of satiety-related hormones, including glucagon-like peptide-1 (GLP-1) and oxyntomodulin (OXM); and
iii. Activation of gut-brain signalling pathways that enhance feelings of fullness and support appetite regulation.

Thus, overall, in theory, low glycaemic index (GI) foods, such as those containing SDS, may support body weight management by promoting satiety and enhancing fat oxidation [24]. SDS has also been suggested to exert a protective effect against diabetes [43].

When considering the relationship between SDS and the overall GI, it is important to recognise that the two are not synonymous. The SDS does not necessarily result in a low GI, as GI is influenced not only by the rate of starch digestion but also by the rate of blood glucose clearance [67].

The influence of RS, a dietary fibre fraction, on body weight management has been discussed above (Section 5). Furthermore, RS supplementation has been associated with weight loss and improved insulin sensitivity in overweight individuals, effects that appear to be mediated, in part, through beneficial modulation of the gut microbiota [68].

Both SDS and RS have been described by some authors as nutraceuticals because of their associated health benefits [69]. However, the utilisation of these starch fractions - particularly SDS - must take into account the impact of processing on their functionality and, consequently, their digestibility.

7. Mechanisms Underpinning Slow Energy Release

The SDS form of starch may blunt the postprandial rise and subsequent decline in plasma glucose and insulin concentrations, leading to prolonged energy availability and enhanced satiety [70]. As discussed in some detail in Section 6. This is therefore a mechanism that works after digestion, rather than through the control of digestive enzymes or subsequent retardation of glucose transfer through the gut into the blood stream. Similar conclusions have been drawn by Goux et al. [71] in terms of the two factors which they consider the digestive impact of SDS on blood glucose concentrations:

i. A diet with a high SDS content decreases the rate of glucose entering the blood stream which reduces the glycaemic and insulinemic responses in health;
ii. A relative reduction in blood glucose concentration reflects a reduction in the glycaemic and insulinemic response.

The relative contributions of starch fractions to glycaemic responses have been investigated by determining the glycaemic index (GI) of a range of cereal-based food products. Meynier et al. [72] demonstrated that glucose availability was strongly correlated with both GI and glycaemic response. Approximately 53% of the variability in GI could be explained by the digestibility profiles of RDS and SDS, together with dietary fat and fibre content. Among these factors, SDS was identified as the principal determinant of glycaemic response, accounting for 17% of the observed variance, compared with only 6% for RDS.

Human intervention studies have further demonstrated the physiological effects of SDS-rich foods. Compared with extruded cereal products, SDS-rich biscuits slowed glucose release and its subsequent appearance in the peripheral circulation, attenuated postprandial glucose excursions, and promoted a more sustained distribution of glucose absorption following breakfast [73]. These effects were accompanied by lower postprandial concentrations of glucose-dependent insulinotropic peptide (GIP) and insulin [73].

The influence of SDS on appetite regulation has also been examined. Vinoy et al. [74] reported that cereal products with a high SDS content reduced postprandial glucose and insulin responses while enhancing subjective feelings of satiety following breakfast.

The SDS form of starch has been shown to activate the gut - brain axis, thereby contributing to the regulation of energy intake [75]. In rodent models, SDS consumption alters feeding behaviour, characterised by reductions in both meal size and meal frequency. These behavioural changes are

accompanied by decreased hypothalamic gene expression of orexigenic neuropeptides and a concomitant increase in the expression of anorexigenic neuropeptides, suggesting a central mechanism through which SDS modulates appetite and satiety.

8. Glycaemic Stability: Hypoglycaemia and Hyperglycaemia

Hypoglycaemia, or low blood glucose, poses a significant risk to the body, which relies on glucose as its primary energy source. It is most commonly associated with insulin therapy [76] although inborn errors of metabolism, such as certain forms of glycogen storage disease (GSD), can also induce hypoglycaemia [35, 77]. Other conditions that can cause hypoglycaemia include hormone deficiencies and kidney failure [78]. In addition, prolonged or intense exercise may also induce hypoglycaemia [77]. Slowly digestible starch (SDS) can help restrict hypoglycaemia over extended periods [35]. In addition to its physiological effects, consumption of SDS has also been associated with improvements in cognitive performance ([79, 80], see below).

Hyperglycaemia can arise from several factors, including peripheral and hepatic insulin resistance, increased hepatic and renal glucose production, and high glucose loads from enteral feeds or intravenous infusions [81]. It is an independent predictor of mortality where the condition may induce decreased cerebral blood flow, intracellular acidosis and reduced adenosine tri-phosphate concentrations which may be evident in diabetes [81]. High intakes of digestible carbohydrate may accentuate hyperglycaemia in type-2 diabetics [82].

Evidence from animal studies supports a potential role for SDS in moderating these effects. Using streptozocin induced diabetic mice, Chen et al. [83] demonstrated that SDS feeding can improve the status with respect to hyperglycaemia and hyperlipidaemia. Similarly, human studies have shown that a diet containing a high proportion of SDS can improve glycaemic variability and reduce postprandial glycaemic excursions in patients with type-2 diabetes [84].

9. Cognitive Considerations

Unlike physiological health outcomes, where the relationship between blood glucose concentration and health parameters is increasingly well understood, the impact of glucose on cognition remains less clearly defined, although understanding is developing [85]. Both hypoglycaemia and hyperglycaemia impair cognitive performance, whereas maintaining a balanced and regulated blood glucose concentration, such as that provided by SDS, optimises cognition function [79, 80, 86-91]. The mechanisms linking blood glucose concentration to cognition and subsequent calorie consumption are not fully understood. However, it is reasonable to hypothesise that sustaining moderate blood glucose concentrations, as achieved with SDS, supports both brain and body functionality, including body weight regulation. The concept of the brain acting as a ‘glucostat’ has been the subject of debate for a very long time [85, 92, 93].

Studies in obese rats have indicated that SDS consumption can reduce daily food intake and in parallel suppress the expression of appetite-stimulating neuropeptide genes associated with the gut - brain axis [75]. These findings suggest that SDS-containing foods could be developed strategically to support obesity management, representing an intriguing opportunity for food producers, nutritionists, and consumers.

In their recent review of the role of carbohydrates in cognitive function, Arshad et al. [94] discussed the potential cognitive benefits of slowly digestible carbohydrate sources, although

without explicitly referring to slowly digestible starch (SDS). The authors noted that 'Whole grains and legumes are complex carbs [sic] that gradually release glucose, supporting long- term cognitive function and reducing fatigue when performing cognitively demanding tasks.' This perspective is consistent with the broader literature, which suggests that carbohydrate sources providing a sustained release of glucose may support cognitive performance by promoting greater glycaemic stability. However, while the proposed relationship between SDS-rich foods and cognitive function is biologically plausible, further well-controlled studies are required to better define the magnitude, mechanisms, and scope of these effects.

10. Future Outlook

The obesity epidemic is driving the food and drink sector to develop products with reduced caloric content through modifications to ingredients and portion sizes, alongside efforts to lower sugar content and alter carbohydrate digestion profiles more generally, including increasing dietary fibre content. By incorporating greater amounts of SDS into food products at the expense of sugars and RDS, energy remains available as glucose in the diet but is delivered in a more measured fashion, with the associated health benefits of reducing sugars and RDS.

The challenge is to ensure that pre-prepared foods remain sensorially desirable while making carbohydrate less readily and rapidly available during digestion. Interest in this area is growing within the food sector, with baked products, particularly biscuits, increasingly being developed with a high SDS content in mind. At present, however, it is not possible in most jurisdictions to make health claims relating to the rate of carbohydrate digestion. This reflects a number of factors, including inter-individual variation, differences in the amount and timing of consumption, interactions with other dietary components, and consumer-induced changes resulting from further processing or preparation.

Nevertheless, there is little doubt that the food industry is having to move beyond the post-war mindset of simply providing calories and protein towards a greater emphasis on controlling nutrient delivery and, in effect, helping to manage weight-related issues. This is occurring in parallel with the global uptake of medications designed to manage body weight by promoting rapid feelings of satiety and thereby reducing calorie intake. These include agonists of the incretin gut-derived hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), such as tirzepatide, which are already having a significant impact on diabetes management and the demand for bariatric surgery.

11. Conclusions

Controlling the rate at which glucose is released from the digestive tract into the bloodstream promotes a more stable state of glucose homeostasis. When glucose enters the circulation at a moderated pace, the body can balance more effectively its utilisation for immediate energy with its storage as glycogen. This improved metabolic regulation can positively influence body weight, BMI, and other physiological markers linked to conditions such as diabetes and cardiovascular disease. In addition, more stable glucose availability may also confer benefits for cognitive function.

From a food production perspective, enhancing the SDS content of food requires careful control of water to starch ratios and cooking times to restrict starch gelatinisation. Although native starches are not usually desirable to eat, they are relatively abundant in certain food categories, such as

some breakfast cereals and biscuits. Expanding the range of such products could support improvements in health, particularly in relation to weight management. Taxation has already impacted on the sugar content of soft drinks. There may also be an argument to tax foods (including ultra-processed) which are high in glycaemic response. While this is not correlated directly or inversely with SDS content, it reflects the broader need to control calorie intake (amount, frequency etc.) *and* the form in which carbohydrate derived calories are delivered within the diet.

Author Contributions

XQ: Writing – review & editing. RT: Writing – original draft. All authors have read and approved the published version of the manuscript.

Competing Interests

The authors have declared that no competing interests exist.

AI-Assisted Technologies Statement

Artificial intelligence (AI) tools were used solely for basic grammar correction and language refinement in the preparation of this manuscript. Specifically, OpenAI's ChatGPT was employed to improve the readability and linguistic clarity of the English text. All scientific content, data interpretation, and conclusions were developed independently by the author. The authors have thoroughly reviewed and edited the AI-assisted text to ensure its accuracy and accept full responsibility for the content of the manuscript.

References

1. Bray GA, Heisel WE, Afshin A, Jensen MD, Dietz WH, Long M, et al. The science of obesity management: An endocrine society scientific statement. Endocr Rev. 2018; 39: 79-132.
2. Aaseth J, Ellefsen S, Alehagen U, Sundfør TM, Alexander J. Diets and drugs for weight loss and health in obesity–An update. Biomed Pharmacother. 2021; 140: 111789.
3. NHS Digital. Health Survey for England 2019 [NS] [Internet]. London, UK: NHS England; 2020. Available from: https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2019.
4. World Health Organization. Obesity and overweight [Internet]. Geneva, Switzerland: WHO; 2025. Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
5. Al-Goblan AS, Al-Alfi MA, Khan MZ. Mechanism linking diabetes mellitus and obesity. Diabetes Metab Syndr Obes. 2014; 7: 587-591.
6. Higgins JA. Resistant starch and energy balance: Impact on weight loss and maintenance. Crit Rev Food Sci Nutr. 2014; 54: 1158-1166.
7. Kong DX, Xiao YX, Zhang ZX, Liu YB. Study on the correlation between metabolism, insulin sensitivity and progressive weight loss change in type-2 diabetes. Pak J Med Sci. 2020; 36: 1523-1528.

8. Ebbeling CB, Feldman HA, Klein GL, Wong JM, Bielak L, Steltz SK, et al. Effects of a low carbohydrate diet on energy expenditure during weight loss maintenance: Randomized trial. BMJ. 2018; 363: k4583.
9. Kirkpatrick CF, Bolick JP, Kris-Etherton PM, Sikand G, Aspry KE, Soffer DE, et al. Review of current evidence and clinical recommendations on the effects of low-carbohydrate and very-low-carbohydrate (including ketogenic) diets for the management of body weight and other cardiometabolic risk factors: A scientific statement from the national lipid association nutrition and lifestyle task force. J Clin Lipidol. 2019; 13: 689-711.e1.
10. Morenga LT, Mallard S, Mann J. Dietary sugars and body weight: Systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2013; 346: e7492.
11. Stanhope KL. Sugar consumption, metabolic disease and obesity: The state of the controversy. Crit Rev Clin Lab Sci. 2016; 53: 52-67.
12. Prinz P. The role of dietary sugars in health: Molecular composition or just calories? Eur J Clin Nutr. 2019; 73: 1216-1223.
13. Paixão C, Dias CM, Jorge R, Carraça EV, Yannakoulia M, De Zwaan M, et al. Successful weight loss maintenance: A systematic review of weight control registries. Obes Rev. 2020; 21: e13003.
14. Blüher M. Obesity: Global epidemiology and pathogenesis. Nat Rev Endocrinol. 2019; 15: 288-298.
15. Leitner DR, Frühbeck G, Yumuk V, Schindler K, Micic D, Woodward E, et al. Obesity and type 2 diabetes: Two diseases with a need for combined treatment strategies-EASO can lead the way. Obes Facts. 2017; 10: 483-492.
16. Bradley P. Refined carbohydrates, phenotypic plasticity and the obesity epidemic. Med Hypotheses. 2019; 131: 109317.
17. Safaei M, Sundararajan EA, Driss M, Boulila W, Shapi'i A. A systematic literature review on obesity: Understanding the causes & consequences of obesity and reviewing various machine learning approaches used to predict obesity. Comput Biol Med. 2021; 136: 104754.
18. Amoutzopoulos B, Steer T, Roberts C, Collins D, Page P. Free and added sugar consumption and adherence to guidelines: The UK National Diet and Nutrition Survey (2014/15-2015/16). Nutrients. 2020; 12: 393.
19. World Health Organization. Guideline: Sugars intake for adults and children [Internet]. Geneva, Switzerland: WHO; 2015. Available from: https://www.who.int/publications/i/item/9789241549028/.
20. Scientific Advisory Committee on Nutrition. SACN Carbohydrates and Health Report [Internet]. London, UK: GOV.UK; 2015. Available from: https://www.gov.uk/government/publications/sacn-carbohydrates-and-health-report.
21. Qi X, Tester RF. Is starch or maltodextrin "glucose?". Starch Stärke. 2018; 70: 1700304.
22. Qi X, Tester RF. Fructose, galactose and glucose-In health and disease. Clin Nutr ESPEN. 2019; 33: 18-28.
23. Qi X, Tester RF. Lactose, maltose, and sucrose in health and disease. Mol Nutr Food Res. 2020; 64: 1901082.
24. Miao M, Jiang B, Cui SW, Zhang T, Jin Z. Slowly digestible starch-A review. Crit Rev Food Sci Nutr. 2015; 55: 1642-1657.
25. Liu H, Wang S, Wang J, Guo X, Song Y, Fu K, et al. Energy metabolism in health and diseases. Signal Transduct Target Ther. 2025; 10: 69.

26. Giacco R, Costabile G, Riccardi G. Metabolic effects of dietary carbohydrates: The importance of food digestion. Food Res Int. 2016; 88: 336-341.
27. Clemente-Suárez VJ, Mielgo-Ayuso J, Martín-Rodríguez A, Ramos-Campo DJ, Redondo-Flórez L, Tornero-Aguilera JF. The burden of carbohydrates in health and disease. Nutrients. 2022; 14: 3809.
28. Rastall RA, Moreno FJ, Hernandez-Hernandez O. Dietary carbohydrate digestibility and metabolic effects in human health. Front Nutr. 2019; 6: 164.
29. Theodorakis N, Kreouzi M, Pappas A, Nikolaou M. Beyond calories: Individual metabolic and hormonal adaptations driving variability in weight management-A state-of-the-art narrative review. Int J Mol Sci. 2024; 25: 13438.
30. Heine RJ, Hanning I, Morgan L, Alberti KG. The oral glucose tolerance test (OGTT): Effect of rate of ingestion of carbohydrate and different carbohydrate preparations. Diabetes Care. 1983; 6: 441-445.
31. Kumar V, Gill KD. To perform glucose tolerance test. In: Basic concepts in clinical biochemistry: A practical guide. Singapore: Springer; 2018. pp. 63-66.
32. Svihus B, Hervik AK. Digestion and metabolic fates of starch, and its relation to major nutrition-related health problems: A review. Starch Stärke. 2016; 68: 302-313.
33. Veit M, van Asten R, Olie A, Prinz P. The role of dietary sugars, overweight, and obesity in type 2 diabetes mellitus: A narrative review. Eur J Clin Nutr. 2022; 76: 1497-1501.
34. Tester RF, Qi X. β-limit dextrin-Properties and applications. Food Hydrocoll. 2011; 25: 1899-1903.
35. Qi X, Ta MN, Tester RF. Savoury cracker development for blood glucose control and management. Bioact Carbohydr Diet Fibre. 2020; 24: 100249.
36. Qi X, Tester RF. Effect of native starch granule size on susceptibility to amylase hydrolysis. Starch Stärke. 2016; 68: 807-810.
37. Qi X, Tester RF. Effect of composition and structure of native starch granules on their susceptibility to hydrolysis by amylase enzymes. Starch Stärke. 2016; 68: 811-815.
38. Qi X, Tester RF. Heat and moisture modification of native starch granules on susceptibility to amylase hydrolysis. Starch Stärke. 2016; 68: 816-820.
39. Englyst KN, Englyst HN, Hudson GJ, Cole TJ, Cummings JH. Rapidly available glucose in foods: An in vitro measurement that reflects the glycemic response. Am J Clin Nutr. 1999; 69: 448-454.
40. Zhang G, Hamaker BR. Slowly digestible starch: Concept, mechanism, and proposed extended glycemic index. Crit Rev Food Sci Nutr. 2009; 49: 852-867.
41. Zhang G, Hamaker BR. Slowly digestible starch and health benefits. In: Resistant Starch: Sources, Applications and Health Benefits. Hoboken, NJ: John Wiley & Sons, Inc.; 2013. pp. 111-130.
42. Lehmann U, Robin F. Slowly digestible starch-its structure and health implications: A review. Trends Food Sci Technol. 2007; 18: 346-355.
43. Thorburn AW, Brand JC, Truswell AS. Slowly digested and absorbed carbohydrate in traditional bushfoods: A protective factor against diabetes? Am J Clin Nutr. 1987; 45: 98-106.
44. Nuttall FQ. Body mass index: Obesity, BMI, and health: A critical review. Nutr Today. 2015; 50: 117-128.
45. Agrawal N, Agrawal MK, Kumari T, Kumar S. Correlation between body mass index and blood glucose levels in Jharkhand population. Int J Contemp Med Res. 2017; 4: 1633-1636.

46. Sabet NS, Doustjalali SR, Khalaf AT, Zin KT, Hlaing TD, Chong MS, et al. Correlation between body mass index (BMI) and fasting blood glucose (FBG) level among undergraduate students. Res J Pharm Biol Chem Sci. 2018; 9: 609-617. Available from: https://www.rjpbcs.com/pdf/2018_9(3)/[76].pdf.
47. Doustjalali SR, Sabet NS, Amm AA, Sreemathi K, Linn N, Zin TK, et al. Correlation between body mass index (BMI) and fasting blood glucose (FBG) level among Malaysian adults age 40-60. Pak J Med Health Sci. 2020; 14: 1371-1375.
48. Sepp E, Kolk H, Loivukene K, Mikelsaar M. Higher blood glucose level associated with body mass index and gut microbiota in elderly people. Microb Ecol Health Dis. 2014; 25: 22857.
49. Ma Y, Olendzki B, Chiriboga D, Hebert JR, Li Y, Li W, et al. Association between dietary carbohydrates and body weight. Am J Epidemiol. 2005; 161: 359-367.
50. McMillan-Price J, Brand-Miller J. Low-glycaemic index diets and body weight regulation. Int J Obes. 2006; 30: S40-S46.
51. Cheng PC, Hsu SR, Tu ST, Cheng YC, Liu YH. Body mass index influences the plasma glucose concentration during iatrogenic hypoglycemia in people with type 2 diabetes mellitus: A cross-sectional study. PeerJ. 2018; 6: e4348.
52. Plečko D, Bennett N, Mårtensson J, Bellomo R. The obesity paradox and hypoglycemia in critically ill patients. Crit Care. 2021; 25: 378.
53. Gill SK, Rossi M, Bajka B, Whelan K. Dietary fibre in gastrointestinal health and disease. Nat Rev Gastroenterol Hepatol. 2021; 18: 101-116.
54. Dhingra D, Michael M, Rajput H, Patil RT. Dietary fibre in foods: A review. J Food Sci Technol. 2012; 49: 255-266.
55. Williams BA, Mikkelsen D, Flanagan BM, Gidley MJ. "Dietary fibre": Moving beyond the "soluble/insoluble" classification for monogastric nutrition, with an emphasis on humans and pigs. J Anim Sci Biotechnol. 2019; 10: 45.
56. Aziz AA, Kenney LS, Goulet B, Abdel-Aal ES. Dietary starch type affects body weight and glycemic control in freely fed but not energy-restricted obese rats. J Nutr. 2009; 139: 1881-1889.
57. Chambers E, Guess N, Viardot A, Frost G. Dietary starch and fiber: Potential benefits to body weight and glucose metabolism. Diabetes Manage. 2011; 1: 521-528.
58. Chew KY, Brownlee IA. The impact of supplementation with dietary fibers on weight loss: A systematic review of randomised controlled trials. Bioact Carbohydr Diet Fibre. 2018; 14: 9-19.
59. Aller EE, Abete I, Astrup A, Martinez JA, van Baak MA. Starches, sugars and obesity. Nutrients. 2011; 3: 341-369.
60. Gourineni V, Stewart ML, Skorge R, Sekula BC. Slowly digestible carbohydrate for balanced energy: *In vitro* and *in vivo* evidence. Nutrients. 2017; 9: 1230.
61. Gourineni V, Stewart ML, Skorge R, Wolever T. Glycemic index of slowly digestible carbohydrate alone and in powdered drink-mix. Nutrients. 2019; 11: 1228.
62. Cai M, Dou B, Pugh JE, Lett AM, Frost GS. The impact of starchy food structure on postprandial glycemic response and appetite: A systematic review with meta-analysis of randomized crossover trials. Am J Clin Nutr. 2021; 114: 472-487.
63. Guo J, Tan L, Kong L. Impact of dietary intake of resistant starch on obesity and associated metabolic profiles in human: A systematic review of the literature. Crit Rev Food Sci Nutr. 2021; 61: 889-905.

64. Bojarczuk A, Skąpska S, Khaneghah AM, Marszałek K. Health benefits of resistant starch: A review of the literature. J Funct Foods. 2022; 93: 105094.
65. Doan HT, Kim T, Cha M, Kim SJ. Synthesis and potential application of slowly digestible starch. J Funct Foods. 2025; 131: 106955.
66. Raghunathan R, Farahnaky A, Majzoobi M, Chandrapala J, Brennan C, Eri R, et al. Slowly digestible starch: A biochemical engineering perspective on functional food development and metabolic health. J Food Meas Charact. 2025; 19: 5197-5221.
67. Eelderink C, Schepers M, Preston T, Vonk RJ, Oudhuis L, Priebe MG. Slowly and rapidly digestible starchy foods can elicit a similar glycemic response because of differential tissue glucose uptake in healthy men. Am J Clin Nutr. 2012; 96: 1017-1024.
68. Li H, Zhang L, Li J, Wu Q, Qian L, He J, et al. Resistant starch intake facilitates weight loss in humans by reshaping the gut microbiota. Nat Metab. 2024; 6: 578-597.
69. Magallanes-Cruz PA, Flores-Silva PC, Bello-Perez LA. Starch structure influences its digestibility: A review. J Food Sci. 2017; 82: 2016-2023.
70. Sands AL, Leidy HJ, Hamaker BR, Maguire P, Campbell WW. Consumption of the slow-digesting waxy maize starch leads to blunted plasma glucose and insulin response but does not influence energy expenditure or appetite in humans. Nutr Res. 2009; 29: 383-390.
71. Goux A, Breyton AE, Meynier A, Lambert-Porcheron S, Sothier M, Van Den Berghe L, et al. Design and validation of a diet rich in slowly digestible starch for type 2 diabetic patients for significant improvement in glycemic profile. Nutrients. 2020; 12: 2404.
72. Meynier A, Goux A, Atkinson F, Brack O, Vinoy S. Postprandial glycaemic response: How is it influenced by characteristics of cereal products? Br J Nutr. 2015; 113: 1931-1939.
73. Péronnet F, Meynier A, Sauvinet V, Normand S, Bourdon E, Mignault D, et al. Plasma glucose kinetics and response of insulin and GIP following a cereal breakfast in female subjects: Effect of starch digestibility. Eur J Clin Nutr. 2015; 69: 740-745.
74. Vinoy S, Aubert R, Chapelot D. A cereal product high in slowly digestible starch increases subsequent feelings of satiety and decreases glucose and insulin responses. J Hum Nutr Food Sci. 2020; 8: 1132.
75. Hasek LY, Phillips RJ, Zhang G, Kinzig KP, Kim CY, Powley TL, et al. Dietary slowly digestible starch triggers the gut-brain axis in obese rats with accompanied reduced food intake. Mol Nutr Food Res. 2018; 62: 1700117.
76. Amiel SA. The consequences of hypoglycaemia. Diabetologia. 2021; 64: 963-970.
77. Douillard C, Mention K, Dobbelaere D, Wemeau JL, Saudubray JM, Vantyghem MC. Hypoglycaemia related to inherited metabolic diseases in adults. Orphanet J Rare Dis. 2012; 7: 26.
78. Kittah NE, Vella A. Management of endocrine disease: Pathogenesis and management of hypoglycemia. Eur J Endocrinol. 2017; 177: R37-R47.
79. Benton D, Ruffin MP, Lassel T, Nabb S, Messaoudi M, Vinoy S, et al. The delivery rate of dietary carbohydrates affects cognitive performance in both rats and humans. Psychopharmacology. 2003; 166: 86-90.
80. Benton D, Nabb S. Carbohydrate, memory, and mood. Nutr Rev. 2003; 61: S61-S67.
81. Brealey D, Singer M. Hyperglycemia in critical illness: A review. J Diabetes Sci Technol. 2009; 3: 1250-1260.

82. Garg A, Grundy SM, Koffler M. Effect of high carbohydrate intake on hyperglycemia, islet function, and plasma lipoproteins in NIDDM. Diabetes Care. 1992; 15: 1572-1580.
83. Chen YZ, Gu J, Chuang WT, Du YF, Zhang L, Lu ML, et al. Slowly digestible carbohydrate diet ameliorates hyperglycemia and hyperlipidemia in high-fat diet/streptozocin-induced diabetic mice. Front Nutr. 2022; 9: 854725.
84. Breyton AE, Goux A, Lambert-Porcheron S, Meynier A, Sothier M, VanDenBerghe L, et al. Starch digestibility modulation significantly improves glycemic variability in type 2 diabetic subjects: A pilot study. Nutr Metab Cardiovasc Dis. 2021; 31: 237-246.
85. Qi X, Tester RF. The impact of carbohydrate derived energy sources and metabolism on cognition. IJCMCR. 2025; 53; 005.
86. Cox D, Gonder-Frederick L, McCall A, Kovatchev B, Clarke W. The effects of glucose fluctuation on cognitive function and QOL: The functional costs of hypoglycaemia and hyperglycaemia among adults with type 1 or type 2 diabetes. Int J Clin Pract Suppl. 2002; 20-26. Available from: https://pubmed.ncbi.nlm.nih.gov/12166601/.
87. Warren RE, Frier BM. Hypoglycaemia and cognitive function. Diabetes Obes Metab. 2005; 7: 493-503.
88. Mortby ME, Janke AL, Anstey KJ, Sachdev PS, Cherbuin N. High "normal" blood glucose is associated with decreased brain volume and cognitive performance in the 60s: The PATH through life study. PLoS One. 2013; 8: e73697.
89. Weinstein G, Maillard P, Himali JJ, Beiser AS, Au R, Wolf PA, et al. Glucose indices are associated with cognitive and structural brain measures in young adults. Neurology. 2015; 84: 2329-2337.
90. Sünram-Lea SI, Owen L. The impact of diet-based glycaemic response and glucose regulation on cognition: Evidence across the lifespan. Proc Nutr Soc. 2017; 76: 466-477.
91. Nevo-Shenker M, Shalitin S. The impact of hypo-and hyperglycemia on cognition and brain development in young children with type 1 diabetes. Horm Res Paediatr. 2021; 94: 115-123.
92. Devarakonda K, Mobbs CV. Mechanisms and significance of brain glucose signaling in energy balance, glucose homeostasis, and food-induced reward. Mol Cell Endocrinol. 2016; 438: 61-69.
93. Yoon NA, Diano S. Hypothalamic glucose-sensing mechanisms. Diabetologia. 2021; 64: 985-993.
94. Arshad MT, Maqsood S, Altalhi R, Shamlan G, Mohamed Ahmed IA, Ikram A, et al. Role of dietary carbohydrates in cognitive function: A review. Food Sci Nutr. 2025; 13: e70516.