The application of resistant starch in food in dietary intervention for type 2 diabetes

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Abstract. Characterized by persistent hyperglycemia due to progressive insulin resistance, type 2 diabetes (T2D) remains a major metabolic disorder affecting over 500 million individuals worldwide and remaining a significant role in global morbidity and healthcare costs. As dietary interventions gain attention in T2D management, resistant starch (RS), which is fermentable dietary fiber, has emerged as a functional ingredient with glycemic regulatory potential. This review examines the classification, structural characteristics, and physiological functions of RS, highlighting the distinct metabolic benefits of RS2 and RS3. Mechanistically, RS improves glucose homeostasis by delaying gastric emptying, enhancing insulin sensitivity, shaping the structure of gut microbiota and stimulating generation of short chain fatty acids, thus collectively influence lipid metabolism, inflammatory pathways and appetite control. However, current studies show significant variability in intervention duration, RS dosage, and individual response, which limit translational consistency. Future research should prioritize standardized intervention designs, explore synergistic interactions with other dietary components, and investigate long-term outcomes mediated by the gut-brain axis. Overall, RS holds promise as a targeted dietary strategy in T2D management, with implications for functional food innovation and personalized nutrition.

Keywords: Resistant starch (RS), Type 2 diabetes (T2D), Insulin sensitivity, Gut microbiota, Inflammation.

1. Introduction

T2D has became a major global health challenge, especially leading to over 90% of diabetes cases worldwide [1]. With the character of insulin resistance, chronic hyperglycemia, and progressive β-cell dysfunction, T2D is significantly contributing to risks of cardiovascular disease, kidney damage, and other metabolic complications [2]. Though pharmacological treatments remain essential, more and more attention has been paid to dietary interventions that can modulate metabolism and offer preventive therapeutic benefits. In this context, resistant starch (RS) is a form of starch that resist small intestine enzymatic digestion, which has gained increasing scientific interest on T2D control, for its potential to influence glucose metabolism, gut microbiota composition, inflammation, and appetite regulation in individuals. Moreover, RS content in food is highly variable and influenced by elements including amylose content, botanical origin, processing method, and storage conditions, which are crucial in determining its functional efficacy in T2D intervention.

RS can be classified into five types (RS1 to RS5) based on their structural and physicochemical properties [3]: RS1 is Physically entrapped starch , RS2 is native granular starches, RS3 refers to retrograded starch, which are physically modified , RS4 is starch undergone chemical modifications while RS5 comprises complexes formed between amylose and lipids [3].Owing to their small intestine enzymatic digestion resistance, all RS types can reach the colon intact, where they are then fermented by the gut microbiota. This fermentation yields short-chain fatty acids (SCFAs), primarily acetate, butyrate, propionate, which are believed to mediate many of RS’s health effects. Their mechanisms of T2D actions will be elaborated in later sections [4]. These mechanisms are further discussed in later sections.

There have been stuties demonstrated that high-amylose maize RS2 can enhance sensitivity of insulin and control of glycemic in individuals with obesity or T2D [5]. These beneficial effects are believed to be mediated through delayed glucose absorption, enhanced insulin signaling, and RS

biotransformation via intestinal microbiota in the colon to produce SCFAs such as butyrate—metabolites increasingly shown to regulate glucose metabolism and improve gut barrier function in T2D [6].

Beyond glycemic benefits, it is also found that RS could act as a prebiotic and reshape the gut microbiotamodulate gut microbiota composition and diversity, which selectively promotes beneficial SCFA-producing bacteria to grow, such as Faecalibacterium prausnitzii, Roseburia, and Bifidobacterium spp [7]. then used as anti-inflammatory agent [8]. This microbial modulation is believed to improve gut barrier function, reduce intestinal permeability, and downregulate systemic inflammation—factors closely associated with T2D pathogenesis [8]. However, a research in 2017 suggested that the metabolic benefits of RS, notably improvements in insulin levels, may not always be entirely dependent on microbial fermentation. This indicate that additional host-mediated pathways such as bile acid signaling and adipose tissue remodeling may also be involved [9].

Chronic inflammation, a hallmark of T2D pathogenesis, may be alleviated by RS via its anti-inflammation and antioxidant effects. RS consumption has been shown to has been shown to lower circulating levels of CRP, TNF-α, and IL-6, as well as oxidative markers such as MDA, alongside improved antioxidant capacity [8]. Furthermore, RS consumption has been found to enhance satiety-related gut hormone responses. For example, the intake of RS2-enriched wheat is follwed by elevated PYY and reduced GIP levels . However, the evidence for actual improvements in self-reported satiety remains inconsistent and warrants further investigation [10].

In spite of these promising findings, significant gaps still exist in the understanding of RS’s role in T2D prevention and treatment. Variability in RS types, individual gut microbiota profiles, food processing methods, and study designs all contributed to inconsistent clinical outcomes. Thus, long-term and large-scale randomized clinical trials are needed for establishing optimal dosage, intervention duration and population-specific effects of RS. There’s also challenges in incorporating RS into functional foods while maintaining palatability and stability [11].

In light of these considerations, this review aims to offers an integrative overview of current research on RS, particularly focus on its classification, mechanisms of T2D managing action —including effects on insulin sensitivity, gut microbiota, inflammation, and appetite—and the opportunities and challenges involved in developing RS-based functional foods. By integrating molecular, clinical, and technological perspectives, this paper seeks to highlight the potential of RS as a novel and accessible dietary strategy for mitigating the global burden of T2D.

2. Structural Characteristics and Classification of RS

2.1. Classification of RS

RS is mainly classified into five types , each characterized by distinct physicochemical properties, digestive resistance mechanisms, and food sources. These classifications are essential for understanding their physiological behavior and guiding functional food applications.

2.1.1 RS1 – Physically Inaccessible Starch

RS1 is defined as starch physically encapsulated within unbroken plant cell structures. which prevents enzymatic hydrolysis by limiting α-amylase access [3,11]. This resistance is primarily due to structural barriers and can be diminished by processes such as milling, grinding, or cooking.

Metabolically, RS1 slows carbohydrate digestion in the small intestine, thereby attenuating postprandial glycemic spikes and potentially enhancing satiety [6]. Although its fermentability is lower than that of other RS types, it still contributes to SCFA production to a limited extent [4]. Preserving the cellular integrity of plant tissues is essential for maintaining RS1 functionality in food processing [11].

2.1.2 RS2 – Native Granular Starch

RS2 typically consists of ungelatinized starch granules characterized by B-type crystallinity and elevated amylose content, both of which contribute to its enzymatic resistance during small intestinal transit—a feature central to its metabolic functionality [3]. This type is abundant in raw starchy foods and high-amylose maize (HAM-RS2), a well-researched source for its metabolic benefits [3,6].
Studies have found that RS2 could improve the sensitivity of insulin and lowers HOMA-IR in overweight individuals [5]. Compared to RS1, it more effectively reduces postprandial glucose levels [11].

RS2 also promotes SCFA production—particularly butyrate—which enhances gut barrier integrity and insulin signaling [4,6]. Its relatively high fermentability and well-established clinical relevance make RS2 a strong candidate for functional food applications in type 2 diabetes (T2D) management.

2.1.3 RS3 – Retrograded Starch

RS3 forms when gelatinized starch is cooled, promoting retrogradation of amylose and amylopectin into semi-crystalline regions (mainly B- and V-type), which hinder α-amylase digestion [3,11]. This type naturally arises in cooked-then-cooled foods like rice, pasta, and potatoes [3].

RS3 is widely consumed due to its prevalence in common culinary practices. Clinical studies have associated RS3 intake with elevated butyrate levels and improved insulin sensitivity in overweight individuals [5,12]. It also enhances the growth of butyrate-producing microbes and supports gut barrier integrity [6].

Its metabolic effects are largely attributed to SCFA-mediated pathways, including GPR41/43 activation, GLP-1 release, and reduced pro-inflammatory signaling [6,8,13]. These mechanisms highlight RS3's role in glycemic regulation and T2D prevention [12].

2.1.4 RS4 – Chemically Modified Starch

RS4 encompasses starches chemically altered through esterification, cross-linking, or etherification, resulting in resistance to enzymatic digestion and stability under heat or mechanical processing [11]. Products such as NOVELOSE® and VERSAFIBE™ are commonly used in fiber-enriched processed foods.

Unlike natural RS types, RS4 can be custom-designed to deliver targeted metabolic effects, making it especially valuable in clinical nutrition. Randomized trials have demonstrated that RS4 consumption lowers post-ingestive glucose and insulin excursions in overweight populations [14].

In addition, RS4 intake has been linked to anti-inflammatory benefits, such as reduced CRP and TNF-α levels, and enhanced butyrate production [15]. These findings position RS4 as a functional ingredient for improving both glycemic regulation and systemic inflammation in T2D management.

2.1.5 RS5 – Amylose–Lipid Complexes

RS5 is composed of amylose–lipid complexes, typically involving monoacylglycerols, that form V-type crystalline structures with limited enzymatic accessibility [5,15]. These complexes are usually found in high-amylose starches processed with lipids, for example, in fried or emulsified starchy foods.

Recent research suggests RS5 contributes to metabolic regulation via gut microbiota modulation and increased SCFA synthesis—particularly butyrate [4]. Beyond glucose regulation, RS5 has shown potential to enhance satiety and attenuate inflammation [11].

Its dual benefits on insulin sensitivity and lipid metabolism—especially in the context of high-fat diets—make RS5 a promising component in T2D-focused dietary interventions [4,11].

Each RS type exhibits distinct structural and physiological features, contributing differently to glycemic regulation and gut health. While RS2 and RS3 are most commonly studied for their effects on insulin sensitivity and SCFA production, RS4 and RS5 offer standardized, functional applications through technological modification. In the following sections, we examine the mechanisms by which RS influences glucose metabolism, inflammation, and the gut microbiota in type 2 diabetes.

Table 1. RS types and their relevance to T2D

RS Type	Structural Characteristics	Typical Food Sources	Fermentability	Key Metabolic Functions	Relevance to T2D Management
RS1(Physically Inaccessible Starch)	Starch trapped in intact plant cell walls; physically blocks α-amylase access	Whole grains (brown rice, wheat berries), legumes (lentils, chickpeas), coarsely ground cereals	Low	Slows digestion, mild SCFA production, promotes sxatiety	Reduces postprandial glucose, supports whole food-based interventions
RS2(Native Granular Starch)	Raw starch granules with high amylose and crystalline B-type structure	Unripe bananas, raw potatoes, plantains, high-amylose maize (HAM-RS2)	High	Enhances insulin sensitivity, increases butyrate, modulates lipids	Most studied RS type; improves HOMA-IR and fasting glucose
RS3(Retrograded Starch)	Gelatinized and cooled starch forms resistant B-/V-type double helices	Cooled cooked starches: refrigerated rice, pasta, potatoes, bread crust	High	Boosts butyrate production, enhances gut barrier, lowers HbA1c	Demonstrated increase in insulin sensitivity (~15%) and butyrate
RS4(Chemically Modified Starch)	Esterified or cross-linked starch resists enzymatic hydrolysis even after processing	Functional foods: fiber-enriched bread, bars, meal replacements (e.g., NOVELOSE®, VERSAFIBE™)	Medium–High	Reduces postprandial glucose, lowers CRP & TNF-α, engineered effects	Clinically effective for glycemic control and inflammation reduction
RS5(Amylose–Lipid Complexes)	V-type crystals formed by amylose-lipid helices; steric hindrance blocks enzymes	Fried or lipid-rich starchy foods, amylose-stearic acid complexes	Moderate	Enhances satiety, modulates gut microbiota, increases SCFAs	Potential synergy in glucose/lipid metabolism, esp. with high-fat meals

2.2. Factors Affecting RS Content in Food

The RS content in food is not fixed—it is significantly influenced by both intrinsic and extrinsic factors. Key intrinsic parameters include the amylose-to-amylopectin ratio, botanical origin, and granule morphology, which determine the inherent digestibility of the starch (Table 1). For example, high-amylose maize tends to generate more RS2 and RS3 under specific processing conditions due to its dense crystalline structure and retrogradation capacity [3,5].

Extrinsic factors such as thermal processing, storage duration, pH, and moisture availability also play crucial roles. Boiling and pressure cooking can substantially alter RS levels by modifying starch

crystallinity; interestingly, a 2024 study found that pressure cooking enhanced RS formation more effectively than boiling in legumes and tubers, likely due to accelerated retrogradation post-cooling [16]. Food matrix restructuring techniques, including dual heat–shear treatment and extrusion followed by cooling, have also proven effective in significantly increasing RS3 content. These methods provide practical strategies for developing functional foods enriched with resistant starch [17].

Additionally, post-cooking storage at low temperatures (e.g., refrigeration for 24–48 h) promotes starch retrogradation, increasing RS3 content in cooked staples like rice and pasta [11]. pH adjustment during food preparation may also alter RS stability, as acidic environments tend to disrupt crystal formation, while neutral or slightly alkaline conditions favor RS retention [3].

Optimizing these variables allows for tailored manipulation of RS content in everyday foods, thereby enhancing their physiological functions—particularly glycemic modulation and SCFA-mediated benefits relevant to T2D management. Understanding these factors thus informs both clinical nutrition strategies and industrial product development in the context of resistant starch application.

3. The Nutritional Function of RS in Type 2 DiabetesaA

Resistant starch (RS) is a fermentable carbohydrate that exerts multi-faceted benefits on glucose metabolism, gut microbiota, inflammation, and weight control—all critical in managing type 2 diabetes (T2D). This section examines its effects on insulin sensitivity and glycemic control, gut microbiome modulation, inflammation and oxidative stress reduction, and appetite regulation and body weight management.

3.1. Factors Affecting RS Content in Food

a. Delayed Glucose Absorption

RS resists digestion in the small intestine, thus reducing the glycemic load of meals. Analyses have shown that RS1 and RS2 intake significantly lower postprandial glucose area-under-the-curve (AUC) by approximately 0.65 standard mean difference (SMD) in acute settings, and 0.31 SMD chronically; fasting blood glucose also decreases by ~0.31 SMD with ⩾6 g/day RS consumption [8,11]. Mechanistically, dense and semi-crystalline structure of RS limits α-amylase accessibility and slows starch hydrolysis, delaying glucose release into circulationz [11].

b. Enhanced Insulin Sensitivity

Chronic RS2 intake (20–40 g/day) has been associated with significant reductions in fasting insulin and HOMA-IR, with fasting insulin SMD reaching −0.40 [5]. RS2 has been shown to upregulate insulin-signaling molecules (IRS-1, IRS-2, and AKT) in the liver and skeletal muscles, thereby facilitating glucose utilization. In addition, RS has also been shown to influences insulin sensitivity via gut microbial modulation and bile acid–FXR signaling [6].

c. Incretin Hormone Modulation

RS-derived SCFAs, notably butyrate and propionate, have been implicated in the stimulation of incretin hormones such as PYY and GLP-1, thereby modulating insulin dynamics and appetite regulation, contributing to improved glycemic control [10,13].

3.2. Regulation of Intestinal Flora

a. Prebiotic Fermentation and SCFA Production

Once in the colon, RS serves as a selective substrate for beneficial saccharolytic bacteria, notably members of Bifidobacterium, Roseburia, and Faecalibacterium, leading to an elevated SCFA yield that underpins its metabolic advantages [4,11]. These SCFAs maintain colonic epithelial integrity, fuel colonocytes, reinforce tight junctions, and attenuate systemic metabolic endotoxemia [13,18].

Butyrate, in particular, has been shown to activate antioxidant pathways via Nrf2 signaling, contributing to improved host metabolic resilience [18].

b. Microbial Diversity Restoration

An 8-week RS2 intervention (40 g/day) in overweight adults led to a mean weight reduction of about 2.8 kg and enhanced insulin sensitivity, associated by increased abundance of Ruminococcus bromii and Bifidobacterium adolescentis—microbial taxa associated with gut barrier restoration and secondary bile acid metabolism [6].

c. Barrier Integrity Enhancement

RS fermentation enhances intestinal barrier function through upregulation of mucin (MUC2) and tight junction proteins (claudins and ZO-1), thereby reducing intestinal permeability and systemic inflammation [6]. Preclinical studies using pulse-derived RS also reported elevated CLDN1 and ZO-1 expression, with concomitant reductions in serum endotoxins and oxidative stress markers, potentially mediated by butyrate-induced Nrf2 activation [18].

d. Microbiota-independent Effects

The metabolic benefits of RS are not solely dependent on microbial fermentation. In germ-free or antibiotic-treated animal models, RS supplementation still significantly enhanced insulin sensitivity, suggesting the involvement of microbiota-independent mechanisms. These effects have been linked to modulation of host bile acid signaling pathways and immune responses in adipose tissue, highlighting RS’s potential to directly influence host metabolism beyond gut microbial mediation (Figure 1) [10].

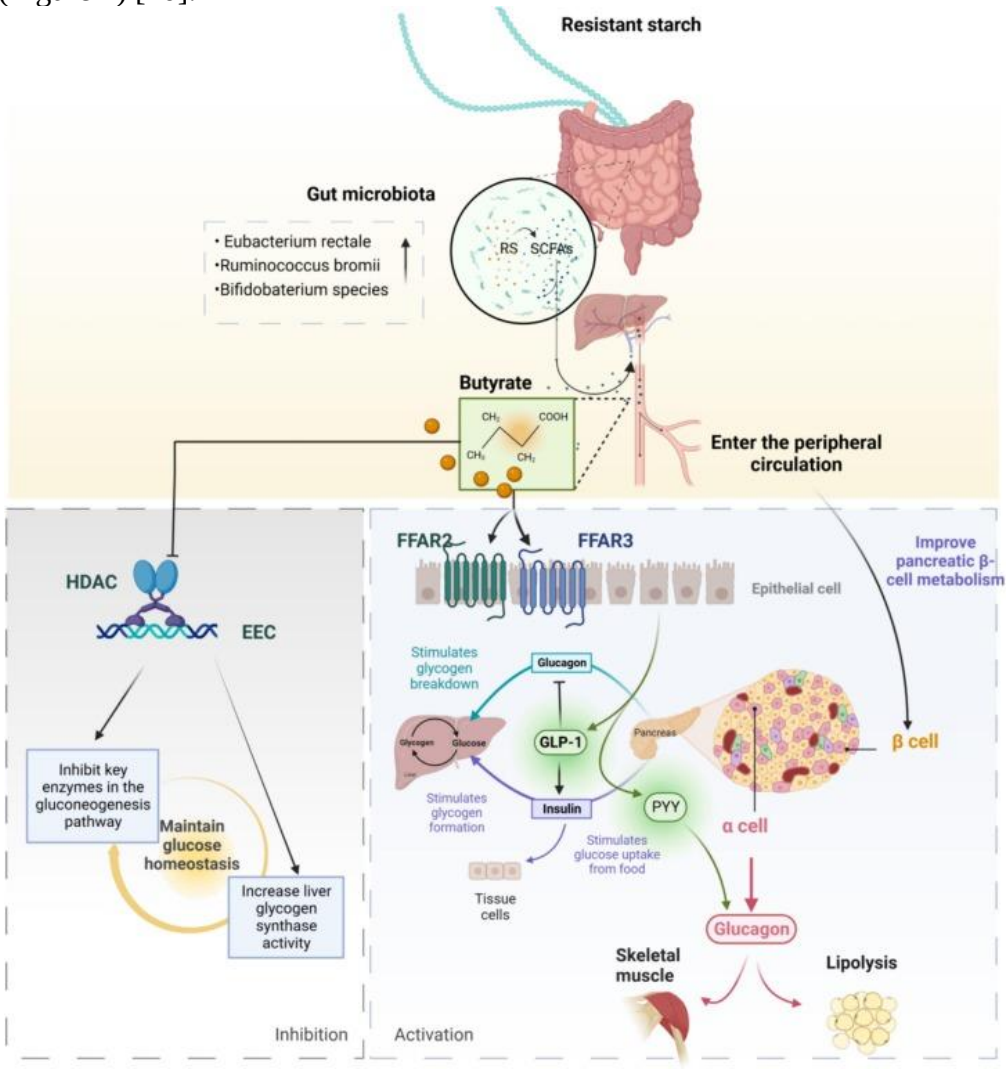


Fig. 1 Anti-diabetic mechanism of resistant starch via gut microbiota modulation.

3.3. Anti-inflammatory Properties & Reduced Oxidative Stress

a. Suppression of Systemic Inflammation

Chronic low-grade inflammation marked by elevated TNF‑α and IL‑6 impairs insulin signaling in T2D. RS supplementation consistently reduces these cytokines in both animal and human studies. For example, a pig feeding study reported significant decreases in TNF‑α and IL‑6 following RS diets . Chemically modified RS4 also lowered hs‑CRP and improved lipid profiles in metabolic syndrome patients [15]. A recent meta-analysis confirmed that RS intake significantly lowered TNF‑α (SMD ≈ −0.71) and IL‑6 (SMD ≈ −0.61) in individuals with T2D and metabolic syndrome [19].

b. Enhanced Antioxidant Capacity

Oxidative stress is another hallmark of T2D that contributes to β-cell dysfunction and insulin resistance. Evidence indicates that RS intake enhances host antioxidant capacity by upregulating enzymatic defenses—including SOD, catalase, and GPx—potentially via SCFA-induced AMPK–Nrf2 signaling activation, while lowering oxidative markers like malondialdehyde (MDA). These effects are mediated in part by butyrate-induced activation of the AMPK–Nrf2 pathway, which promotes antioxidant gene expression and suppresses reactive oxygen species (ROS) generation [9,12].

c. Immune–Metabolic Modulation

Beyond local gut effects, fermentation-derived SCFAs, especially butyrate, exert systemic immune-modulatory functions through multiple mechanisms. Butyrate inhibits histone deacetylases (HDACs), thereby enhancing acetylation of histones and promoting the transcription of anti-inflammatory genes [13]. Moreover, SCFAs facilitate the expansion of regulatory T (Treg) cells and suppress the pathway of NF-κB, consequently suppresses the production of pro-inflammatory cytokines.. Butyrate also modulates the NLRP3 inflammasome and promotes systemic immune tolerance through the Nrf2–Keap1 pathway [20].

3.4. Appetite Suppression and Obesity Alleviation

A modest yet accumulating body of evidence suggests RS may aid weight control—an important factor in T2D—by altering satiety signals and digestive kinetics. Hughes et al. showed RS2 supplementation (~30–40 g/day) increased PYY and reduced GIP, although self-reported satiety remained unchanged [10]. Clinical trials in overweight individuals reported modest weight reduction (~1.5–3 kg over 6–8 weeks) with RS-enriched diets [5,12]. These effects are believed to result primarily from slower gastric emptying and increased secretion of satiety hormones via SCFA-GPCR signaling. Given the heterogeneity in individual responses and study designs, this area warrants longer-term, well-controlled trials with behavioral and metabolic endpoints.

4. Discussion

Although resistant starch (RS) shows promise in modulating glycemia and improving insulin sensitivity [5,9], its clinical utility remains limited by several key challenges.

One major issue is the high inter-individual variability in metabolic response, likely driven by differences in gut microbiota composition and RS fermentability. For instance, study have reported that RS supplementation altered the gut microbial structure and facilitated weight loss in some individuals, but not in all subjects, suggesting that baseline microbial signatures influence efficacy [21].

Moreover, food processing and cooking techniques significantly reduce RS content, which undermines its functional effects in habitual diets. For example, studies have shown that boiling or high-temperature processing can reduce RS levels by over 50% [17]. These losses complicate dose standardization in clinical trials and consumer products.

Another limitation is the short duration and limited scale of most existing trials. While small-scale studies indicate improved postprandial glucose and insulin sensitivity after RS intake [12,22], few have assessed long-term outcomes such as HbA1c, lipid metabolism, or T2D incidence. Behavioral endpoints — such as changes in satiety perception or dietary adherence — are also underexplored [10].

To fill these gaps, future studies should incorporate long-term randomized controlled designs with multi-omics profiling to identify responders and clarify underlying mechanisms [6,13]. Additionally, co-formulation strategies combining RS with probiotics or polyphenols may enhance efficacy by leveraging complementary gut-mediated pathways [4,7,20]. Finally, technological advances, such as enzyme-resistant modifications or microencapsulation, could improve RS stability and bioavailability in processed foods [17].

5. Conclusion

RS demonstrates multifaceted potential in the dietary management of T2D, primarily through its roles in improving glycemic control, enhancing insulin sensitivity, modulating gut microbiota, and mitigating chronic inflammation. Among the RS types, RS2 and RS3 have shown the most consistent metabolic benefits, though The metabolic effectiveness of RS is modulated by multiple variables, including plant origin, technological treatments, and interactions within the food matrix.

Despite promising outcomes in controlled trials, significant challenges hinder the translational application of RS. These include individual variability in glycemic response, loss of RS content during thermal processing, and limited data from long-term, large-scale human studies. Moreover, the inconsistency in dosage, intervention duration, and RS type across current studies complicates the interpretation and generalization of findings.

Future efforts should focus on identifying optimal RS formulations that retain functional properties post-processing, establishing dose-response relationships, and exploring synergistic effects with other dietary bioactives such as polyphenols and probiotics. Personalized nutrition strategies leveraging microbiome profiling may further improve intervention precision. Addressing these aspects through interdisciplinary, longitudinal research will be pivotal to unlocking RS's full therapeutic potential in both the prevention and management of T2D.

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