Resistant starch as a dietary strategy for metabolic and gut health: Implications for Asia-Pacific populations

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ABSTRACT

Background and Objectives: Metabolic diseases are rising rapidly across the Asia–Pacific region as traditional diets are displaced by refined, low-fibre foods. Resistant starch (RS) is a fermentable dietary component that modulates gut microbial activity and short-chain fatty acid (SCFA) production, representing a promising strategy for metabolic and gut health. However, responses to RS vary according to dose, RS type, and individual context. **Methods and Study Design:** This narrative review synthesised evidence from animal and human studies on RS classification, food sources, microbial interactions, and metabolic regulation. We evaluated how responses differ by dose, RS type, baseline microbiome composition, metabolic phenotype, and habitual diet, with particular attention to traditional Asia–Pacific staple foods and ongoing dietary transitions. **Results:** Across studies, RS supplementation at moderate-to-high doses improves insulin sensitivity, hepatic lipid accumulation, and markers of gut barrier function, primarily through SCFA-mediated and gut hormone pathways. However, responses are highly context-dependent, varying with baseline metabolic status, microbiome composition, habitual fibre intake and RS type. Individuals with metabolic disturbances or low fibre intake tend to show greater metabolic and microbial shifts. **Conclusions:** RS is a promising strategy for supporting metabolic and gut health in Asia–Pacific populations undergoing rapid nutritional transition. Culturally familiar RS-rich foods offer practical, regionally tailored intervention opportunities. Standardised characterisation, microbiome-stratified analyses and long-term human studies are needed to clarify who benefits most, under which conditions and through which mechanisms.

Key Words: resistant starch, gut health, gut microbiome, metabolic health, Asia–Pacific diets

INTRODUCTION

The global rise in chronic metabolic diseases represents a major public-health concern,[1, 2] with particularly rapid rises across the Asia–Pacific region.[3-5] Populations spanning South, East, and Southeast Asia, the Western Pacific and Oceania[6, 7] are experiencing rising rates of diabetes and related metabolic disorders, driven by rapid urbanisation, lifestyle change and dietary transitions.[8, 9] These trends highlight the need for accessible, evidence-based dietary strategies that can be widely adopted to support metabolic health in this region.

Emerging evidence implicates gut health as a key mechanistic link between diet and metabolic dysfunction.[10-18] The gut microbiota, epithelial barrier, and host metabolic

pathways operate as an integrated system regulating immune homeostasis, nutrient processing, and inflammation. Disruptions to this system, through gut dysbiosis and impaired barrier integrity, are associated with insulin resistance, dyslipidaemia, and chronic low-grade inflammation.[19-23]

Resistant starch (RS) has gained increasing attention as a microbiota-directed dietary component targeting the gut-metabolic axis. Unlike digestible starches, RS resists enzymatic breakdown in the small intestine and instead undergoes fermentation in the colon,[24] generating short-chain fatty acids (SCFAs) that support epithelial integrity, modulate immune responses, and influence glucose and lipid metabolism.[25, 26] These properties position RS as a promising nutritional strategy for reducing cardiometabolic risk.

The relevance of RS is particularly strong in Asia-Pacific populations, where traditional diets historically provided substantial RS through cooked-and-cooled rice, starchy root vegetables, legumes, plantains, and minimally processed grains. Dietary westernisation has reduced these sources in favour of refined and ultra-processed alternatives,[8, 9] coinciding with rising metabolic disease. Culturally familiar RS-rich foods, therefore, provide an attractive opportunity for regionally tailored interventions.

This review synthesises experimental and human evidence on RS and metabolic health, with focus on: (i) RS structural types and food sources; (ii) effects on gut microbiota, SCFA production, and gut barrier function; (iii) clinical evidence across health and disease; and (iv) the potential for culturally tailored RS interventions in Asia-Pacific populations.

TYPES AND SOURCES OF RESISTANT STARCH

Definition and classification of resistant starch

Resistant starch (RS) is a fraction of dietary starch that escapes digestion in the small intestine[27, 28] and undergoes fermentation by the colonic microbiota.[24] RS is classified into five structural types, based on the mechanism by which it resists small-intestinal digestion:[29]

-RS1: physically inaccessible starch trapped within intact plant cell walls.

-RS2: native granular starch with a crystalline structure that resists digestion until cooking gelatinises it.

-RS3: retrograded starch formed when cooked starches cool and recrystallise.

-RS4: chemically–modified starches engineered to resist digestion.

-RS5: amylose–lipid complexes produced during processing, including frying and extrusion.

These structural differences influence fermentation kinetics, SCFA production, and downstream physiological effects. Figure 1 summarises RS types, mechanisms of resistance, and key food sources, particularly examples within Asia Pacific diets.

Dietary sources and processing effects

The RS content varies widely and is strongly influenced by botanical source and processing, particularly cooking, cooling, and ripeness. For example, unripe bananas (e.g., green bananas) contain substantially more RS than ripe bananas.[30] Repeated cooking/cooling cycles markedly increase RS3 formation in starchy foods such as rice and potatoes,[31-33] while legumes provide consistent RS1 through intact cellular structures, with additional RS3 formed during cooling.[34, 35]

Across Asia Pacific populations, traditional dietary patterns rich in rice, legumes, and starchy tubers likely provided higher RS intakes than Western diets dominated by refined grains. Contemporary estimates suggest that Indian and Chinese diets yield 2–4 times higher (10–18 g/day) RS intake[36, 37] than in the United States (4.9 g/day),[38] Europe (3–6 g/day),[39, 40] and Australia (3–9 g/day).[41] As dietary westernisation progresses and reliance on refined grains increases, habitual RS exposure in the region has likely declined.

RESISTANT STARCH AND GUT MICROBIOTA

Diet is a major determinant of gut microbiota composition.[42] RS escapes enzymatic digestion in the small intestine and is fermented by colonic microbes, producing SCFAs and reshaping microbial ecology[43, 44] in a dose- and substrate-specific manner.[45]

Higher RS intake alters gut microbial diversity[46, 47] and has been associated with favourable metabolic outcomes, including slower weight gain in humans.[48] Although distinct RS types enrich different microbial groups, a consistent feature across studies is the enrichment of *Ruminococcus bromii*, a key "gateway degrader" that initiates RS hydrolysis[45, 47] and facilitates cross-feeding to butyrate-producing taxa such as Eubacterium rectale.[49, 50] These microbial shifts are accompanied by increased SCFA production, though the magnitude and profile of SCFAs vary according to RS type, dose and individual fermentative capacity.

Collectively, RS intake reshapes the gut microbiota, promotes SCFA production, and supports metabolic improvements related to obesity and weight regulation. However, substantial inter-individual variability – driven by baseline microbiota, habitual diet, RS type, and fermentative capacity – remains a defining feature of RS responsiveness. This variability

underscores the need for microbiome-informed interpretation and supports the rationale for personalised approaches to RS-based dietary interventions.

SHORT-CHAIN FATTY ACID PRODUCTION AND METABOLIC EFFECTS

The metabolic effects of RS are largely mediated through fermentation-derived SCFAs produced by the colonic microbiota.[51] The principal SCFAs – acetate, propionate, and butyrate – exert diverse effects on host metabolism, gut barrier integrity and endocrine signalling, positioning SCFA production as a key mechanistic link between RS intake and metabolic health.

Influence on glucose homeostasis and insulin sensitivity

SCFAs influence glucose homeostasis through both direct metabolic actions and gut hormone–mediated pathways.[52-54] Butyrate improves insulin sensitivity by enhancing mitochondrial function and increasing energy expenditure,[55] while acetate and propionate stimulate enteroendocrine secretion of GLP-1 and PYY via FFAR2 signalling.[52, 53]

In humans, RS supplementation (15–40 g/day) improves insulin sensitivity, with beneficial effects reported in populations with overweight, obesity, type 2 diabetes, and Metabolic Associated Steatotic Liver Disease (MASLD). Reported benefits include improved insulin sensitivity,[48, 56, 57] reduced intrahepatic triglyceride content,[58] and modest effects on body weight in some intervention contexts.[48, 56] These improvements coincide with shifts toward saccharolytic and butyrogenic gut microbiota[48] and bile acid remodelling.[58] supporting an integrated SCFA-driven and gut hormone–mediated mechanism that varies with dose, duration, and baseline metabolic status.

Beyond glycaemic control, RS fermentation also influences lipid metabolism and adiposity. In MASLD, RS supplementation reduces intrahepatic lipid accumulation and alters bile-acid profiles.[58] Animal studies further demonstrate enhanced hepatic cholesterol clearance, increased fat oxidation[59] and reduced adiposity alongside elevated SCFA production and circulating levels of GLP-1 and PYY.[60] Collectively, these findings support a plausible pathway linking RS fermentation to improved hepatic lipid handling, reduced ectopic fat, and increased energy expenditure.

Appetite regulation and satiety signalling

Appetite regulation provides an additional, though secondary, pathway linking RS fermentation to metabolic outcomes. In a key human trial, targeted colonic delivery of propionate increased postprandial GLP-1 and PYY secretion, reduced energy intake and

attenuated weight gain over 24 weeks.[54] Notably, acute gut hormone responses diminished over time, suggesting that longer-term benefits extend beyond satiety signalling alone. Consistent with this, animal studies demonstrate that acetate and propionate stimulate GLP-1 release primarily via FFAR2-dependent pathways, with RS supplementation reducing energy intake and adiposity alongside increased SCFA production.[52, 59, 60]

Variability in SCFA responses

A defining feature of RS interventions is substantial inter-individual variability in SCFA responses. While many trials report increased SCFA production following RS supplementation,[61-74] others show no change,[75-79] or decreases,[80] reflecting differences in RS type, dose, duration, and baseline microbiome composition. Interpretation is further complicated by weak correlations between faecal and circulating SCFA concentrations[81, 82-84] and methodological heterogeneity in SCFA measurement.[85] Together, these factors limit cross-study comparability and contribute to heterogeneity in reported metabolic outcomes.

Overall, RS influences metabolic regulation primarily through SCFA-mediated effects on glucose homeostasis, lipid metabolism, adiposity and appetite signalling. However, marked inter-individual variability – driven by microbiome composition, RS type and dose, and measurement limitations – remains a key challenge. These considerations underscore the importance of linking SCFA production to downstream physiological targets, particularly gut barrier function, which is addressed in the following section.

RESISTANT STARCH AND GUT PHYSIOLOGY

SCFAs generated through RS fermentation play a central role in maintaining intestinal barrier integrity and mucosal homeostasis. Among these, butyrate is a primary energy source for colonocytes and is consistently associated with enhanced epithelial integrity and reduced intestinal permeability across experimental models.86-88 SCFAs also modulate immune signalling by promoting anti-inflammatory pathways, supporting mucus-layer function and suppressing cytokine-driven inflammation, thereby reinforcing barrier resilience.[89, 90]

Although butyrate has received the greatest attention, acetate and propionate contribute to mucosal protection through complementary mechanisms,[89] highlighting that the physiological effects of RS fermentation reflect the combined action of SCFA mixtures rather than isolated metabolites. Consequently, RS-induced changes in SCFA profiles may differentially influence gut physiology depending on microbial composition and mucosal health.

Overall, RS supports gut physiological function through SCFA-mediated reinforcement of epithelial and immune processes; however, these effects are dose- and context-dependent,[91, 92] varying with RS type, fermentative capacity, microbiome composition and host factors. This underscores the importance of defining effective and safe RS dosing strategies and linking gut physiological responses to clinical outcomes, as examined in the following section on human intervention studies.

CLINICAL EVIDENCE AND HUMAN TRIALS

RS supplementation in healthy populations

In healthy populations, the effects of RS vary across the dose spectrum. Moderate-to-high doses (e.g., 22–40 g/day over 1.5–12 weeks) consistently improve peripheral insulin sensitivity,93 fasting glucose, and gut microbiota composition (e.g., enriching beneficial butyrate-producing taxa such as *Roseburia*, *Ruminococcus bromii*, and *Faecalibacterium prausnitzii*)[94] – without corresponding weight loss.[93, 94] These findings indicate metabolic benefits independent of body weight change. In contrast, lower doses (e.g., 3.5–7 g/day over 4–6 weeks) primarily confer gastrointestinal benefits, including enrichment of beneficial taxa such as *Bifidobacterium* and *Akkermansia* and improvements in bowel regularity, with less consistent evidence for increased SCFA production.[95-97] This pattern suggests that microbiota modulation might precede measurable fermentation outputs and systemic metabolic effects. Evidence also suggests an upper threshold for RS efficacy. In a dose-escalation trial, SCFA responses to RS4 increased up to ~35 g/day but plateaued at higher intakes, with no further gains observed at 50 g/day, potentially reflecting saturation of colonic fermentative capacity.[70]

RS supplementation in metabolic disorder populations

In metabolically compromised populations, higher RS doses are generally required to elicit clinically meaningful effects, with outcomes varying by metabolic phenotype. In adults with type 2 diabetes, supplementation with 40 g/day high-amylose maize RS2 for 12 weeks reduced postprandial glucose excursions and showed trends toward improved skeletal-muscle glucose uptake, despite no significant weight loss.[57] In prediabetes, similar-duration supplementation reduced inflammatory markers but produced limited improvements in insulin sensitivity or ectopic fat,[98] suggesting modest glycaemic and anti-inflammatory benefits in early dysglycaemia.

Stronger effects are observed in obesity and metabolic syndrome, particularly at higher doses and under energy-restricted interventions. Randomised trials using 30–50 g/day RS report reductions in visceral adiposity, improved insulin sensitivity and favourable lipid profiles, especially when RS replaces digestible starch.46, 48 These improvements are accompanied by enrichment of saccharolytic microbiota, increased faecal SCFAs and alterations in bile-acid metabolism that track closely with metabolic improvements.

Beyond glycaemic control, RS has demonstrated organ-specific metabolic benefits in conditions characterised by ectopic fat accumulation and metabolic toxicity. In MASLD, prolonged RS supplementation (20 g twice daily for 4 months) reduced intrahepatic triglyceride content and improved liver-related metabolic markers.[58] Lower RS doses have also reduced inflammation, oxidative stress and uraemic toxins, indicating benefits in metabolically vulnerable populations with altered gut–kidney axis.[99] Together, these findings indicate that RS acts primarily as a metabolic modulator rather than a weight-loss agent.

Meta-analyses support these trial-level observations, showing consistent improvements in fasting glucose, insulin resistance indices, inflammatory markers and selected lipid parameters across overweight, obese and diabetic populations, while pooled effects on body weight and energy intake remain small or non-significant.[100-105] The major beneficial effects of RS supplementation in humans are summarised in Table 1.

Dose–response, tolerability, and sources of variability

Across studies, RS supplementation is generally well tolerated. Mild gastrointestinal symptoms (e.g., bloating or flatulence) are the most commonly reported adverse effects and typically resolve with microbial adaptation.[95, 106, 107] Gradual dose escalation – beginning at ~10–15 g/day and increasing in 5–10 g increments – improves tolerability, even at higher target doses.[95, 106]

A consistent feature across trials is substantial inter-individual variability in metabolic responsiveness. Baseline microbiome composition strongly predicts response, with individuals lacking key RS degraders, such as *Ruminococcus bromii*, often showing minimal SCFA, GLP-1 or insulin responses, while those with higher baseline abundances demonstrate more robust metabolic benefits.[47, 48, 108] Habitual fibre intake and baseline microbial signatures further modify responsiveness, highlighting the importance of dietary context and microbial capacity.[109]

Heterogeneity is also influenced by RS type. RS2 has the strongest clinical evidence base, whereas RS3 from cooked–cooled foods is more food-based but difficult to achieve at

therapeutic doses, and RS4 varies widely in structure and physiological effects. Despite these differences, most trials treat RS as a uniform entity, limiting the interpretation of type-specific efficacy.[43, 110]

Evidence from Asia-Pacific populations

Evidence from Asia–Pacific populations highlight how habitual diet modifies RS effects. In Korean adults, higher dietary RS intake is associated with lower prevalence of metabolic syndrome independent of total carbohydrate intake. In Chinese intervention studies, supplemental RS2 improved insulin sensitivity, reduced visceral adiposity, and lowered intrahepatic triglyceride content despite moderate baseline RS intake from traditional diets.[48, 58] These findings indicate that RS supplementation remains effective even in populations with relatively high background intake, though response magnitude may differ from Western cohorts with lower habitual RS exposure.[58, 109] This context supports culturally tailored strategies that prioritise preservation and optimisation of traditional RS-rich foods alongside, or instead of, purified supplements.

REGIONAL AND CULTURAL PERSPECTIVES

Resistant starch in Asia-Pacific diets

Traditional dietary patterns across many Asia–Pacific populations have historically provided substantial RS, largely through staple starchy foods prepared with minimal processing. Indian and Chinese diets are estimated to supply approximately 10–15 g RS/day – around two-fold higher than intakes reported in Western settings – reflecting greater consumption of rice, legumes and tubers.[36, 38] Similar profiling in Korea confirms that traditional starchy foods remain major contributors to RS intake.[111, 112] These baseline intakes are relevant when considering supplementation strategies, as habitual diets in the region may already approach biologically meaningful RS exposure.

Key traditional staples such as rice, legumes, taro and green bananas can provide substantial RS when consumed in minimally processed forms or prepared in ways that promote starch retrogradation.[33, 113] Cooking-cooling cycles increase RS content in rice and reduce postprandial glycaemia,[33] while cold storage of parboiled germinated brown rice markedly increases RS formation through retrogradation.[114] Legumes such as lentils, chickpeas, black beans, and pinto beans retain meaningful RS after cooking and cooling.[35, 115] For root crops, taro corms contain 70–80% starch on a dry basis, and technological processing (heating, autoclaving, enzymatic debranching, retrogradation) can increase RS content up to

~35% with associated reductions in predicted glycaemic index.[116] Green banana flour contains particularly high RS (40–60 g/100 g dry basis), while taro can reach ~35% RS with appropriate processing.[116-119] Collectively, these data indicate that traditional Asia–Pacific foods can deliver substantial RS when appropriate varieties and preparation/storage methods are used, reducing reliance on purified supplements.

However, rapid nutritional transition is shifting carbohydrate quality across the region. Increased consumption of refined grains and ultra-processed foods, driven by urbanisation and globalised supply chains, is associated with greater metabolic syndrome risk.[120-122] These shifts suggest that habitual RS exposure has declined in many settings, potentially contributing to rising rates of obesity, type 2 diabetes and MASLD.

Potential for culturally tailored RS interventions

Embedding RS-focused strategies within existing food cultures offers a practical and potentially more sustainable alternative than reliance on supplementation alone. Across South and Southeast Asia, this may involve greater use of legumes and millets, alongside preparation methods such as cooling or reheating rice to increase RS content.[33, 35, 37, 115] In East Asia, traditional consumption of sweet potatoes, taro and mixed-grain porridges provides structurally diverse RS fractions,[112, 116] while Pacific Island staples such as taro, yam, cassava and green banana dishes offer high RS when minimally processed.[117-119] These culturally familiar approaches – steaming and cooling, overnight storage, mixed-grain cooking, selective use of less-polished cereals – can meaningfully increase RS intake without imposing unfamiliar eating patterns.

The rationale for regionally tailored interventions is further supported by evidence that gut microbiota composition in Asia–Pacific populations is strongly shaped by diet and urbanisation. Studies in Thailand and China demonstrate that traditional, fibre-rich diets are associated with a greater abundance of SCFA-producing bacteria, whereas Westernised dietary patterns and urbanisation are linked to loss of microbial diversity and fermentative capacity.[123-125] These findings underscore that diet–microbiota relationships are context-specific, and that RS-based interventions must account for local dietary and microbial backgrounds.

Baseline microbiome composition also appears to influence RS responsiveness.[123] Higher habitual fibre intake and specific microbial signatures predict stronger SCFA responses to fermentable substrates, whereas individuals adapted to low-fibre, refined-carbohydrate diets may require longer or more gradual RS exposure to achieve comparable benefits.[109, 126]

Persistent microbiota and host-tissue "memory" effects associated with longstanding obesity may further modulate responsiveness, supporting the positioning of RS as a cumulative, long-term dietary strategy rather than a short-term intervention.[127-130]

Taken together, RS interventions embedded within regional food cultures represent a promising approach to mitigating metabolic disease risk in Asia–Pacific populations undergoing rapid nutritional transition. Key knowledge gaps remain regarding optimal RS dosing across metabolic phenotypes, differential responsiveness across diverse dietary and microbiome backgrounds, and interactions between RS and region-specific microbial ecologies. Addressing these gaps will be essential for translating RS-based strategies into effective, culturally relevant dietary interventions.

CLINICAL IMPLICATIONS, FUTURE DIRECTIONS, AND RESEARCH GAPS

Standardisation and translation challenges

A major barrier to translating RS research into clinical and public health practice is ongoing heterogeneity in RS measurement, classification, and reporting. Although validated in vitro methods now exist to quantify starch escaping small-intestinal digestion,[131, 132] RS is still inconsistently assessed across studies and often measured in raw ingredients rather than foods as consumed. Given the strong influence of cooking, cooling and food structure on RS content, this limits comparability and clinical relevance.[133] In addition, incomplete reporting of RS type, dose and food matrix further complicates synthesis, while emerging evidence suggests that structure-based RS classifications alone may not adequately capture fermentation and microbiome responses.[134, 135] Future research should prioritise harmonised, food-relevant RS measurement, transparent reporting of RS characteristics, and frameworks that better link RS structure to biological function.

Collectively, these sources of heterogeneity—spanning RS type, dose, food matrix, population characteristics, and outcome measurement—limit cross-study comparability and limit the interpretability of pooled effect estimates, particularly in the context of substantial inter-individual variability.

Personalised nutrition and inter-individual variability

Substantial inter-individual variability is a consistent feature of RS intervention studies, with baseline gut microbiota composition, habitual fibre intake and metabolic status strongly influencing responsiveness. These findings challenge uniform intake recommendations and support a shift toward microbiota-informed, personalised approaches. Evidence that baseline

microbial signatures and dietary patterns predict SCFA and metabolic responses highlights opportunities for stratified trial designs and targeted interventions. Key priorities include identifying pragmatic predictors of responsiveness and testing strategies to enhance RS fermentation capacity in low responders, positioning RS as a flexible component of personalised nutrition rather than a fixed-dose intervention.

Toward clinical implementation

Beyond its role as a dietary fibre, RS may also complement emerging microbiome-directed therapies by supporting microbial stability and metabolic function in perturbed gut ecosystems. Early evidence suggests potential synergy with probiotics or other microbiome-modulating approaches,[95, 136, 137] although direct clinical validation remains limited. Future trials will need to define optimal RS type, dose and timing when used alone or in combination with microbiome-targeted interventions, and to establish whether RS can enhance the durability of metabolic benefits across disease contexts.

CONCLUSION

RS is a promising dietary component with beneficial effects on both metabolic and gut health, with outcomes dependent on RS type, dose, food matrix, and individual metabolic and microbiome context. Evidence from human and experimental studies indicates that RS is associated with improvements in metabolic health primarily through fermentation-derived pathways, including SCFA production, gut barrier support, and endocrine signalling, while exhibiting substantial inter-individual variability. These features are particularly relevant in populations undergoing dietary transition, including those across the Asia-Pacific region, where traditional RS-rich foods are increasingly displaced by refined carbohydrates. Progress in this field will depend on standardised RS characterisation, microbiome-stratified trial designs, and longer-term human studies to define when, for whom, and in what form RS can be most effectively deployed.

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REFERENCES

1. Chew NWS,Ng CH,Tan DJH, et al. The global burden of metabolic disease: Data from 2000 to 2019. Cell Metab. 2023: 35; 414-428.e413.
2. Zhang H,Zhou X-D,Shapiro MD, et al. Global burden of metabolic diseases, 1990–2021. Metabolism. 2024: 160; 155999.
3. Kerr JA,Patton GC,Cini KI, et al. Global, regional, and national prevalence of child and adolescent overweight and obesity, 1990–2021, with forecasts to 2050: a forecasting study for the Global Burden of Disease Study 2021. The Lancet. 2025: 405; 785-812.
4. Ranasinghe P,Mathangasinghe Y,Jayawardena R,Hills AP,Misra A Prevalence and trends of metabolic syndrome among adults in the asia-pacific region: a systematic review. BMC Public Health. 2017: 17; 101.
5. Mina T,Xie W,Low DY, et al. Adiposity and metabolic health in Asian populations: an epidemiological study using dual-energy x-ray absorptiometry in Singapore. The Lancet Diabetes & Endocrinology. 2024: 12; 704-715.
6. D'arcy P,Mayo L Fluid Frontiers: Oceania and Asia in Historical Perspective. The Journal of Pacific History. 2021: 56; 217-235.
7. Gaupholm J,Papadopoulos A,Asif A,Dodd W,Little M The influence of food environments on dietary behaviour and nutrition in Southeast Asia: A systematic scoping review. Nutr Health. 2023: 29; 231-253.
8. Harriden B,D'Cunha NM,Kellett J, et al. Are dietary patterns becoming more processed? The effects of different dietary patterns on cognition: A review. Nutrition and Health. 2022: 28; 341-356.
9. Popkin BM,Adair LS,Ng SW Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev. 2012: 70; 3-21.
10. Wu J,Wang K,Wang X,Pang Y,Jiang C The role of the gut microbiome and its metabolites in metabolic diseases. Protein & cell. 2021: 12; 360-373.
11. Han P,Gu J-Q,Li L-S, et al. The association between intestinal bacteria and allergic diseases—cause or consequence? Frontiers in Cellular and Infection Microbiology. 2021: 11; 650893.
12. Thingholm LB,Rühlemann MC,Koch M, et al. Obese individuals with and without type 2 diabetes show different gut microbial functional capacity and composition. Cell host & microbe. 2019: 26; 252-264. e210.
13. Da Silva HE,Teterina A,Comelli EM, et al. Nonalcoholic fatty liver disease is associated with dysbiosis independent of body mass index and insulin resistance. Scientific reports. 2018: 8; 1466.
14. Wang B,Jiang X,Cao M, et al. Altered fecal microbiota correlates with liver biochemistry in nonobese patients with non-alcoholic fatty liver disease. Scientific reports. 2016: 6; 32002.
15. Turnbaugh PJ,Bäckhed F,Fulton L,Gordon JI Diet-induced obesity is linked to marked but reversible alterations in the mouse distal gut microbiome. Cell host & microbe. 2008: 3; 213-223.

16. Kelley ST,Skarra DV,Rivera AJ,Thackray VG The gut microbiome is altered in a letrozole-induced mouse model of polycystic ovary syndrome. PloS one. 2016: 11; e0146509.
17. Guo Y,Qi Y,Yang X, et al. Association between polycystic ovary syndrome and gut microbiota. PloS one. 2016: 11; e0153196.
18. Schwimmer JB,Johnson JS,Angeles JE, et al. Microbiome signatures associated with steatohepatitis and moderate to severe fibrosis in children with nonalcoholic fatty liver disease. Gastroenterology. 2019: 157; 1109-1122.
19. Bauer TM,Schwacha H,Steinbrückner B, et al. Small intestinal bacterial overgrowth in human cirrhosis is associated with systemic endotoxemia. The American journal of gastroenterology. 2002: 97; 2364-2370.
20. Qin J,Li Y,Cai Z, et al. A metagenome-wide association study of gut microbiota in type 2 diabetes. nature. 2012: 490; 55-60.
21. Ruuskanen MO,Erawijantari PP,Havulinna AS, et al. Gut microbiome composition is predictive of incident type 2 diabetes in a population cohort of 5,572 Finnish adults. Diabetes Care. 2022: 45; 811-818.
22. Jiang W,Wu N,Wang X, et al. Dysbiosis gut microbiota associated with inflammation and impaired mucosal immune function in intestine of humans with non-alcoholic fatty liver disease. Scientific reports. 2015: 5; 8096.
23. Ansari I,Raddatz G,Gutekunst J, et al. The microbiota programs DNA methylation to control intestinal homeostasis and inflammation. Nature microbiology. 2020: 5; 610-619.
24. Van Hung P,Chau HT,Phi NTL In vitro digestibility and in vivo glucose response of native and physically modified rice starches varying amylose contents. Food Chemistry. 2016: 191; 74-80.
25. Abou-Samra M,Venema K,Ayoub Moubareck C,Karavetian M Gut microbiota composition of lean and obese Lebanese individuals. Benef Microbes. 2025: 16; 395-411.
26. Chen S,Cai S,Xu J, et al. Lack of Faecalibacterium prausnitzii and Bifidobacterium is associated with a higher risk of metabolic associated fatty liver disease in young-onset type 2 diabetes. International Journal of Diabetes in Developing Countries. 2024: 44; 167-174.
27. Englyst HN,Kingman S,Cummings J Classification and measurement of nutritionally important starch fractions. European journal of clinical nutrition. 1992: 46; S33-50.
28. Aston LM Glycaemic index and metabolic disease risk. Proceedings of the Nutrition Society. 2006: 65; 125-134.
29. Englyst HN,Hudson GJ The classification and measurement of dietary carbohydrates. Food Chemistry. 1996: 57; 15-21.
30. Gao H,Huang S,Dong T,Yang Q,Yi G Analysis of resistant starch degradation in postharvest ripening of two banana cultivars: Focus on starch structure and amylases. Postharvest Biology and Technology. 2016: 119; 1-8.

31. Moongngarm A,Tiboonbun W,Sanpong M, et al. RESISTANT STARCH AND BIOACTIVE CONTENTS OF UNRIPE BANANA FLOUR AS INFLUENCED BY HARVESTING PERIODS AND ITS APPLICATION. American Journal of Agricultural and Biological Sciences. 2014: 9.
32. Chen Z,Liang N,Zhang H, et al. Resistant starch and the gut microbiome: Exploring beneficial interactions and dietary impacts. Food Chem X. 2024: 21; 101118.
33. Sonia S,Witjaksono F,Ridwan R Effect of cooling of cooked white rice on resistant starch content and glycemic response. Asia Pac J Clin Nutr. 2015: 24; 620-625.
34. Tovar J,Björck IM,Asp N-G Incomplete Digestion of Legume Starches in Rats: A Study of Precooked Flours Containing Retrograded and Physically Inaccessible Starch Fractions12. The Journal of Nutrition. 1992: 122; 1500-1507.
35. García-Alonso A,Goñi I,Saura-Calixto F Resistant starch and potential glycaemic index of raw and cooked legumes (lentils, chickpeas and beans). Zeitschrift für Lebensmitteluntersuchung und - Forschung A. 1998: 206; 284-287.
36. Chen L,Liu R,Qin C, et al. Sources and Intake of Resistant Starch in the Chinese Diet. Asia Pacific Journal of Clinical Nutrition. 2010: 19; 274-282.
37. Platel K,Shurpalekar KS Resistant starch content of Indian foods. Plant Foods Hum Nutr. 1994: 45; 91-95.
38. Murphy MM,Douglass JS,Birkett A Resistant starch intakes in the United States. J Am Diet Assoc. 2008: 108; 67-78.
39. Brighenti F,Casiraghi MC,Baggio C Resistant starch in the Italian diet. Br J Nutr. 1998: 80; 333-341.
40. Tomlin J,Read NW The effect of resistant starch on colon function in humans. British Journal of Nutrition. 1990: 64; 589-595.
41. Roberts J,Jones GP,Rutishauser IHE,Birkett A,Gibbons C Resistant starch in the Australian diet. Nutrition & Dietetics: The Journal of the Dietitians Association of Australia. 2004: 61; 98+.
42. David LA,Maurice CF,Carmody RN, et al. Diet rapidly and reproducibly alters the human gut microbiome. Nature. 2014: 505; 559-563.
43. Bindels LB,Segura Munoz RR,Gomes-Neto JC, et al. Resistant starch can improve insulin sensitivity independently of the gut microbiota. Microbiome. 2017: 5; 1-16.
44. Qu W,Yuan X,Zhao J, et al. Dietary advanced glycation end products modify gut microbial composition and partially increase colon permeability in rats. Mol Nutr Food Res. 2017: 61.
45. Kieffer DA,Piccolo BD,Vaziri ND, et al. Resistant starch alters gut microbiome and metabolomic profiles concurrent with amelioration of chronic kidney disease in rats. American Journal of Physiology-Renal Physiology. 2016: 310; F857-F871.
46. Upadhyaya B,McCormack L,Fardin-Kia AR, et al. Impact of dietary resistant starch type 4 on human gut microbiota and immunometabolic functions. Scientific Reports. 2016: 6; 28797.
47. Martínez I,Kim J,Duffy PR,Schlegel VL,Walter J Resistant Starches Types 2 and 4 Have Differential Effects on the Composition of the Fecal Microbiota in Human Subjects. PLOS ONE. 2010: 5; e15046.

48. Li H,Zhang L,Li J, et al. Resistant starch intake facilitates weight loss in humans by reshaping the gut microbiota. Nature Metabolism. 2024: 6; 578-597.
49. Ze X,Duncan SH,Louis P,Flint HJ Ruminococcus bromii is a keystone species for the degradation of resistant starch in the human colon. The ISME journal. 2012: 6; 1535-1543.
50. Baxter NT,Schmidt AW,Venkataraman A, et al. Dynamics of Human Gut Microbiota and Short-Chain Fatty Acids in Response to Dietary Interventions with Three Fermentable Fibers. mBio. 2019: 10.
51. Guo J,Tan L,Kong L Multiple levels of health benefits from resistant starch. Journal of Agriculture and Food Research. 2022: 10; 100380.
52. Tolhurst G,Heffron H,Lam YS, et al. Short-chain fatty acids stimulate glucagon-like peptide-1 secretion via the G-protein-coupled receptor FFAR2. Diabetes. 2012: 61; 364-371.
53. Psichas A,Sleeth ML,Murphy KG, et al. The short chain fatty acid propionate stimulates GLP-1 and PYY secretion via free fatty acid receptor 2 in rodents. Int J Obes (Lond). 2015: 39; 424-429.
54. Chambers ES,Viardot A,Psichas A, et al. Effects of targeted delivery of propionate to the human colon on appetite regulation, body weight maintenance and adiposity in overweight adults. Gut. 2015: 64; 1744.
55. Gao Z,Yin J,Zhang J, et al. Butyrate improves insulin sensitivity and increases energy expenditure in mice. Diabetes. 2009: 58; 1509-1517.
56. Maki KC,Pelkman CL,Finocchiaro ET, et al. Resistant starch from high-amylose maize increases insulin sensitivity in overweight and obese men. J Nutr. 2012: 142; 717-723.
57. Bodinham CL,Smith L,Thomas EL, et al. Efficacy of increased resistant starch consumption in human type 2 diabetes. Endocr Connect. 2014: 3; 75-84.
58. Ni Y,Qian L,Siliceo SL, et al. Resistant starch decreases intrahepatic triglycerides in patients with NAFLD via gut microbiome alterations. Cell Metabolism. 2023: 35; 1530-1547.e1538.
59. So P-W,Yu W-S,Kuo Y-T, et al. Impact of Resistant Starch on Body Fat Patterning and Central Appetite Regulation. PLOS ONE. 2007: 2; e1309.
60. Belobrajdic DP,King RA,Christophersen CT,Bird AR Dietary resistant starch dose-dependently reduces adiposity in obesity-prone and obesity-resistant male rats. Nutrition & Metabolism. 2012: 9; 93.
61. Malcomson FC,Willis ND,McCallum I, et al. Resistant starch supplementation increases crypt cell proliferative state in the rectal mucosa of older healthy participants. British Journal of Nutrition. 2020: 124; 374-385.
62. Alfa MJ,Strang D,Tappia PS, et al. A randomized trial to determine the impact of a digestion resistant starch composition on the gut microbiome in older and mid-age adults. Clinical Nutrition. 2018: 37; 797-807.
63. Le Leu RK,Winter JM,Christophersen CT, et al. Butyrylated starch intake can prevent red meat-induced O6-methyl-2-deoxyguanosine adducts in human rectal tissue: a randomised clinical trial. British Journal of Nutrition. 2015: 114; 220-230.

64. Birkett A,Muir J,Phillips J,Jones G,O'Dea K Resistant starch lowers fecal concentrations of ammonia and phenols in humans. The American Journal of Clinical Nutrition. 1996: 63; 766-772.
65. Muir JG,Yeow EG,Keogh J, et al. Combining wheat bran with resistant starch has more beneficial effects on fecal indexes than does wheat bran alone. The American journal of clinical nutrition. 2004: 79; 1020-1028.
66. Bird AR,Vuaran MS,King RA, et al. Wholegrain foods made from a novel high-amylose barley variety (Himalaya 292) improve indices of bowel health in human subjects. British Journal of Nutrition. 2008: 99; 1032-1040.
67. Humphreys KJ,Conlon MA,Young GP, et al. Dietary manipulation of oncogenic microRNA expression in human rectal mucosa: a randomized trial. Cancer prevention research. 2014: 7; 786-795.
68. Clarke JM,Topping DL,Christophersen CT, et al. Butyrate esterified to starch is released in the human gastrointestinal tract. The American journal of clinical nutrition. 2011: 94; 1276-1283.
69. McOrist AL,Miller RB,Bird AR, et al. Fecal butyrate levels vary widely among individuals but are usually increased by a diet high in resistant starch. J Nutr. 2011: 141; 883-889.
70. Deehan EC,Yang C,Perez-Muñoz ME, et al. Precision Microbiome Modulation with Discrete Dietary Fiber Structures Directs Short-Chain Fatty Acid Production. Cell Host Microbe. 2020: 27; 389-404.e386.
71. Zhang L,Ouyang Y,Li H, et al. Metabolic phenotypes and the gut microbiota in response to dietary resistant starch type 2 in normal-weight subjects: a randomized crossover trial. Scientific reports. 2019: 9; 4736.
72. Hald S,Schioldan AG,Moore ME, et al. Effects of arabinoxylan and resistant starch on intestinal microbiota and short-chain fatty acids in subjects with metabolic syndrome: a randomised crossover study. PLoS One. 2016: 11; e0159223.
73. Abell GC,Cooke CM,Bennett CN,Conlon MA,McOrist AL Phylotypes related to Ruminococcus bromii are abundant in the large bowel of humans and increase in response to a diet high in resistant starch. FEMS microbiology ecology. 2008: 66; 505-515.
74. Jenkins DJ,Vuksan V,Kendall CW, et al. Physiological effects of resistant starches on fecal bulk, short chain fatty acids, blood lipids and glycemic index. Journal of the American College of Nutrition. 1998: 17; 609-616.
75. Sasidharan BK,Ramadass B,Viswanathan P, et al. A phase 2 randomized controlled trial of oral resistant starch supplements in the prevention of acute radiation proctitis in patients treated for cervical cancer. Journal of Cancer Research and Therapeutics. 2019: 15; 1383-1391.
76. Johnstone AM,Kelly J,Ryan S, et al. Nondigestible carbohydrates affect metabolic health and gut microbiota in overweight adults after weight loss. The Journal of nutrition. 2020: 150; 1859-1870.
77. Worthley DL,Le Leu RK,Whitehall VL, et al. A human, double-blind, placebo-controlled, crossover trial of prebiotic, probiotic, and synbiotic supplementation: effects on luminal, inflammatory, epigenetic, and epithelial biomarkers of colorectal cancer. The American journal of clinical nutrition. 2009: 90; 578-586.

78. Grubben M,Braak Cvd,Essenberg Mv, et al. Effect of resistant starch on potential biomarkers for colonic cancer risk in patients with colonic adenomas. Digestive diseases and sciences. 2001: 46; 750-756.
79. Hughes RL,Horn WH,Finnegan P, et al. Resistant starch type 2 from wheat reduces postprandial glycemic response with concurrent alterations in gut microbiota composition. Nutrients. 2021: 13; 645.
80. Salonen A,Lahti L,Salojärvi J, et al. Impact of diet and individual variation on intestinal microbiota composition and fermentation products in obese men. The ISME journal. 2014: 8; 2218-2230.
81. Nogal A,Asnicar F,Vijay A, et al. Genetic and gut microbiome determinants of SCFA circulating and fecal levels, postprandial responses and links to chronic and acute inflammation. Gut Microbes. 2023: 15; 2240050.
82. Mayo-Martínez L,Paz Lorenzo M,Martos-Moreno GÁ, et al. Short-chain fatty acids in plasma and feces: An optimized and validated LC-QqQ-MS method applied to study anorexia nervosa. Microchemical Journal. 2024: 200; 110255.
83. Higueras C,Rey AI,Escudero R, et al. Short-Chain and Total Fatty Acid Profile of Faeces or Plasma as Predictors of Food-Responsive Enteropathy in Dogs: A Preliminary Study. Animals. 2022: 12; 89.
84. Chen SJ,Chen CC,Liao HY, et al. Association of Fecal and Plasma Levels of Short-Chain Fatty Acids With Gut Microbiota and Clinical Severity in Patients With Parkinson Disease. Neurology. 2022: 98; e848-e858.
85. Deroover L,Boets E,Tie Y,Vandermeulen G,Verbeke K Quantification of Plasma or Serum Short-chain Fatty Acids: Choosing the Correct Blood Tube. Journal of Nutritional Health & Food Science. 2017: 5; 1-6.
86. Fung KY,Cosgrove L,Lockett T,Head R,Topping DL A review of the potential mechanisms for the lowering of colorectal oncogenesis by butyrate. Br J Nutr. 2012: 108; 820-831.
87. Topping DL,Fukushima M,Bird AR Resistant starch as a prebiotic and synbiotic: state of the art. Proc Nutr Soc. 2003: 62; 171-176.
88. Litvak Y,Byndloss MX,Bäumler AJ Colonocyte metabolism shapes the gut microbiota. Science. 2018: 362; eaat9076.
89. Deleu S,Arnauts K,Deprez L, et al. High Acetate Concentration Protects Intestinal Barrier and Exerts Anti-Inflammatory Effects in Organoid-Derived Epithelial Monolayer Cultures from Patients with Ulcerative Colitis. Int J Mol Sci. 2023: 24.
90. Gao Y,Davis B,Zhu W, et al. Short-chain fatty acid butyrate, a breast milk metabolite, enhances immature intestinal barrier function genes in response to inflammation in vitro and in vivo. Am J Physiol Gastrointest Liver Physiol. 2021: 320; G521-g530.
91. Huang XZ,Li ZR,Zhu LB, et al. Inhibition of p38 mitogen‐activated protein kinase attenuates butyrate‐induced intestinal barrier impairment in a Caco‐2 cell monolayer model. Journal of pediatric Gastroenterology and Nutrition. 2014: 59; 264-269.
92. Chen T,Kim CY,Kaur A, et al. Dietary fibre-based SCFA mixtures promote both protection and repair of intestinal epithelial barrier function in a Caco-2 cell model. Food & Function. 2017: 8; 1166-1173.

93. Johnston K,Thomas EL,Bell JD,Frost G,Robertson MD Resistant starch improves insulin sensitivity in metabolic syndrome. Diabetic Medicine. 2010: 27; 391-397.
94. Johnstone AM,Kelly J,Ryan S, et al. Nondigestible Carbohydrates Affect Metabolic Health and Gut Microbiota in Overweight Adults after Weight Loss. The Journal of Nutrition. 2020: 150; 1859-1870.
95. Bush JR,Baisley J,Harding SV,Alfa MJ Consumption of Solnul(™) Resistant Potato Starch Produces a Prebiotic Effect in a Randomized, Placebo-Controlled Clinical Trial. Nutrients. 2023: 15.
96. Bush JR,Iwuamadi I,Han J, et al. Resistant Potato Starch Supplementation Reduces Serum Free Fatty Acid Levels and Influences Bile Acid Metabolism. Metabolites. 2024: 14.
97. Hanes D,Nowinski B,Lamb JJ, et al. The gastrointestinal and microbiome impact of a resistant starch blend from potato, banana, and apple fibers: A randomized clinical trial using smart caps. Front Nutr. 2022: 9; 987216.
98. Peterson CM,Beyl RA,Marlatt KL, et al. Effect of 12 wk of resistant starch supplementation on cardiometabolic risk factors in adults with prediabetes: a randomized controlled trial. Am J Clin Nutr. 2018: 108; 492-501.
99. Esgalhado M,Kemp JA,Azevedo R, et al. Could resistant starch supplementation improve inflammatory and oxidative stress biomarkers and uremic toxins levels in hemodialysis patients? A pilot randomized controlled trial. Food Funct. 2018: 9; 6508-6516.
100. 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.
101. Wang Y,Chen J,Song Y-H, et al. Effects of the resistant starch on glucose, insulin, insulin resistance, and lipid parameters in overweight or obese adults: a systematic review and meta-analysis. Nutrition & Diabetes. 2019: 9; 19.
102. Xiong K,Wang J,Kang T,Xu F,Ma A Effects of resistant starch on glycaemic control: a systematic review and meta-analysis. Br J Nutr. 2021: 125; 1260-1269.
103. Rashed AA,Saparuddin F,Rathi DG,Nasir NNM,Lokman EF Effects of Resistant Starch Interventions on Metabolic Biomarkers in Pre-Diabetes and Diabetes Adults. Front Nutr. 2021: 8; 793414.
104. Vahdat M,Hosseini SA,Khalatbari Mohseni G,Heshmati J,Rahimlou M Effects of resistant starch interventions on circulating inflammatory biomarkers: a systematic review and meta-analysis of randomized controlled trials. Nutrition Journal. 2020: 19; 33.
105. Lu J,Ma B,Qiu X,Sun Z,Xiong K Effects of resistant starch supplementation on oxidative stress and inflammation biomarkers: A systematic review and meta-analysis of randomized controlled trials. Asia Pac J Clin Nutr. 2021: 30; 614-623.
106. So D,Yao CK,Gibson PR,Muir JG Evaluating tolerability of resistant starch 2, alone and in combination with minimally fermented fibre for patients with irritable bowel syndrome: a pilot randomised controlled cross-over trial. J Nutr Sci. 2022: 11; e15.

107. Sobh M,Montroy J,Daham Z, et al. Tolerability and SCFA production after resistant starch supplementation in humans: a systematic review of randomized controlled studies. The American Journal of Clinical Nutrition. 2022: 115; 608-618.

108. Baxter Nielson T,Schmidt Alexander W,Venkataraman A, et al. Dynamics of Human Gut Microbiota and Short-Chain Fatty Acids in Response to Dietary Interventions with Three Fermentable Fibers. mBio. 2019: 10; 10.1128/mbio.02566-02518.

109. Devarakonda SLS,Superdock DK,Ren J, et al. Gut microbial features and dietary fiber intake predict gut microbiota response to resistant starch supplementation. Gut Microbes. 2024: 16; 2367301.

110. Bindels LB,Walter J,Ramer-Tait AE Resistant starches for the management of metabolic diseases. Current Opinion in Clinical Nutrition & Metabolic Care. 2015: 18; 559-565.

111. Kang M-S,Jang K-A,Kim H-R,Song S Association of Dietary Resistant Starch Intake with Obesity and Metabolic Syndrome in Korean Adults. Nutrients. 2024: 16; 158.

112. Jang K-A,Kim HA,Kang M-S, et al. Development of a database to estimate dietary intake of resistant starch in Koreans. Journal of Food Composition and Analysis. 2023: 120; 105283.

113. Salis S,Virmani A,Priyambada L, et al. 'Old Is Gold': How Traditional Indian Dietary Practices Can Support Pediatric Diabetes Management. Nutrients. 2021: 13.

114. Srichamnong W,Lasukhang W Structural and chemical analysis of resistant starch effected by pre-treatments, cooking methods, reheating and storage condition in parboiled germinated brown rice (Oryza sativa). International Journal of Food Science & Technology. 2022: 57; 2060-2068.

115. Fabbri ADT,Schacht RW,Crosby GA Evaluation of resistant starch content of cooked black beans, pinto beans, and chickpeas. NFS Journal. 2016: 3; 8-12.

116. Simsek S,El SN Production of resistant starch from taro (Colocasia esculenta L. Schott) corm and determination of its effects on health by in vitro methods. Carbohydr Polym. 2012: 90; 1204-1209.

117. Tribess T,Hernández-Uribe J,Méndez-Montealvo M, et al. Thermal properties and resistant starch content of green banana flour (Musa cavendishii) produced at different drying conditions. LWT - Food Science and Technology. 2009: 42; 1022–1025.

118. Khoza M,Kayitesi E,Dlamini BC Physicochemical Characteristics, Microstructure and Health Promoting Properties of Green Banana Flour. Foods. 2021: 10.

119. Bashmil YM,Dunshea FR,Appels R,Suleria HAR Bioaccessibility of Phenolic Compounds, Resistant Starch, and Dietary Fibers from Australian Green Banana during In Vitro Digestion and Colonic Fermentation. Molecules. 2024: 29; 1535.

120. Song S,Lee JE,Song WO,Paik HY,Song Y Carbohydrate intake and refined-grain consumption are associated with metabolic syndrome in the Korean adult population. J Acad Nutr Diet. 2014: 114; 54-62.

121. Lim CGY,Whitton C,Rebello SA,van Dam RM Diet Quality and Lower Refined Grain Consumption are Associated With Less Weight Gain in a Multi-Ethnic Asian Adult Population. J Nutr. 2021: 151; 2372-2382.

122. Pingali P Westernization of Asian diets and the transformation of food systems: Implications for research and policy. Food Policy. 2007: 32; 281-298.

123. Kisuse J,La-Ongkham O,Nakphaichit M, et al. Urban Diets Linked to Gut Microbiome and Metabolome Alterations in Children: A Comparative Cross-Sectional Study in Thailand. Front Microbiol. 2018: 9; 1345.

124. Raethong N,Nakphaichit M,Suratannon N, et al. Analysis of Human Gut Microbiome: Taxonomy and Metabolic Functions in Thai Adults. In Genes). 2021: 331.

125. Lu J,Zhang L,Zhai Q, et al. Chinese gut microbiota and its associations with staple food type, ethnicity, and urbanization. npj Biofilms and Microbiomes. 2021: 7; 71.

126. Healey G,Murphy R,Butts C, et al. Habitual dietary fibre intake influences gut microbiota response to an inulin-type fructan prebiotic: a randomised, double-blind, placebo-controlled, cross-over, human intervention study. Br J Nutr. 2018: 119; 176-189.

127. Thaiss CA,Itav S,Rothschild D, et al. Persistent microbiome alterations modulate the rate of post-dieting weight regain. Nature. 2016: 540; 544-551.

128. Hinte LC,Castellano-Castillo D,Ghosh A, et al. Adipose tissue retains an epigenetic memory of obesity after weight loss. Nature. 2024: 636; 457-465.

129. Schmitz J,Evers N,Awazawa M, et al. Obesogenic memory can confer long-term increases in adipose tissue but not liver inflammation and insulin resistance after weight loss. Mol Metab. 2016: 5; 328-339.

130. Siersbæk M,Varticovski L,Yang S, et al. High fat diet-induced changes of mouse hepatic transcription and enhancer activity can be reversed by subsequent weight loss. Scientific Reports. 2017: 7; 40220.

131. McCleary BV,Monaghan DA Measurement of resistant starch. J AOAC Int. 2002: 85; 665-675.

132. McCleary BV,McLoughlin C,Charmier LMJ,McGeough P Measurement of available carbohydrates, digestible, and resistant starch in food ingredients and products. Cereal Chemistry. 2020: 97; 114-137.

133. Nakayoshi Y,Nakamura S,Kameo Y, et al. Measurement of resistant starch content in cooked rice and analysis of gelatinization and retrogradation characteristics. Biosci Biotechnol Biochem. 2015: 79; 1860-1866.

134. Gao M, Jia, Jianhui, Zhou, Xun, Zhang, Chujia, Dou, Boxin, Liu, Ying, Zhang, Na, Optimization of Englyst Method for Determination of Starch Digestibility of Japonica Rice Based on AOAC Method. Science and Technology of Food Industry. 2023: 44.

135. Monro J,Mishra S In Vitro Digestive Analysis of Digestible and Resistant Starch Fractions, with Concurrent Glycemic Index Determination, in Whole Grain Wheat Products Minimally Processed for Reduced Glycaemic Impact. Foods. 2022: 11.

136. Riwes MM,Golob JL,Magenau J, et al. Feasibility of a dietary intervention to modify gut microbial metabolism in patients with hematopoietic stem cell transplantation. Nat Med. 2023: 29; 2805-2813.

137. Luk-In S,Leepiyasakulchai C,Saelee C, et al. Impact of resistant starch type 3 on fecal microbiota and stool frequency in Thai adults with chronic constipation randomized clinical trial. Sci Rep. 2024: 14; 27944.

138. Hanes D,Nowinski B,Lamb JJ, et al. The gastrointestinal and microbiome impact of a resistant starch blend from potato, banana, and apple fibers: A randomized clinical trial using smart caps. Frontiers in Nutrition. 2022: Volume 9 - 2022.

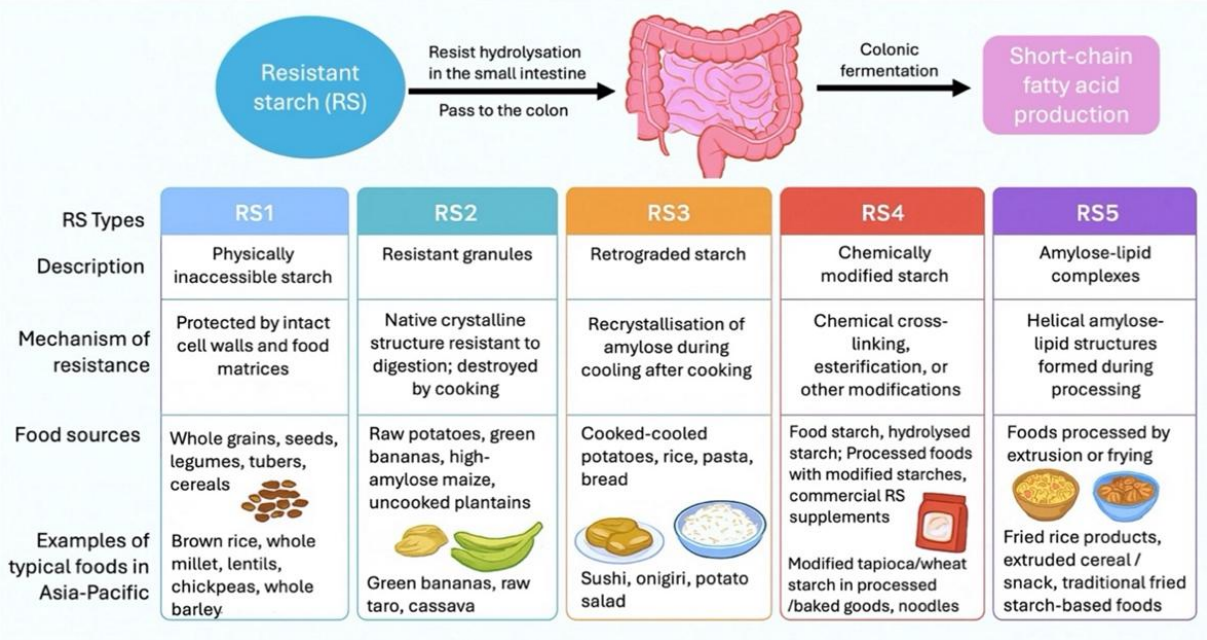

Figure 1. Classification of resistant starch

Table 1. Major beneficial effects of resistant starch supplementation in humans

	Beneficial effect	RS type / dose	Population	Key findings	References
1	Improved insulin sensitivity	RS2 15–40 g/day × 4–12 weeks	Males with overweight/obesity or metabolic syndrome	RS improved insulin sensitivity in men with overweight and metabolic syndrome, with effect magnitudes comparable to moderate weight loss. No significant change observed in women.	56, 93
2	Reduced postprandial glucose	RS2 40 g/day × 12 weeks	Well-controlled T2DM (HbA1c ~47 mmol/mol)	RS lowered postprandial glucose and enhanced postprandial GLP-1 concentrations.	57
3	Reduced fasting glucose and HOMA-IR	RS2/RS3 10–45 g/day (>28 g/day optimal; >8 weeks)	Overweight/obese adults; T2DM	RS reduced fasting glucose, fasting insulin, and HOMA-IR, with greater effects at higher doses and longer durations. Insulin sensitivity and HbA1c were improved in diabetic population. Dose threshold: >28 g/day and >8 weeks for optimal glycaemic benefit.	101, 102
4	Reduced intrahepatic triglycerides	RS2 40 g/day × 4 months	MASLD patients	RS reduced intrahepatic triglyceride content and liver enzyme levels in MASLD patients, with benefits partially independent of weight loss.	58
5	Reduced visceral adiposity and body weight	RS2 40 g/day × 8 weeks	Overweight/obese T2DM adults	RS reduced body weight and visceral fat in adults with overweight/obesity and T2DM. Weight loss was not maintained after RS discontinuation.	48, 100
6	Lower metabolic syndrome prevalence (observational)	Habitual dietary RS (estimated from 24-h recall; no supplementation)	Korean adults with/without obesity and metabolic syndrome (KNHANES 2016–2018)	Higher habitual RS intake was inversely associated with metabolic syndrome and elevated triglycerides in men. No significant association was found in women.	111
7	Enrichment of SCFA-producing microbiome	RS2/RS3 25–40 g/day × 3–8 weeks	Overweight/obese adults	RS consistently enriched *R. bromii*, Roseburia, *F. prausnitzii*, *B. adolescentis* and Akkermansia. Enrichment of B. adolescentis was associated with increased weight loss.	48, 76
8	Alterations in gut microbiota composition; high inter-individual variability	RS2 3.5–25 g/day × 3–4 weeks	Healthy adults; adults with obesity or chronic constipation	RS-rich diet increased the abundance of Bifidobacterium, Akkermansia, Prevotella, Megamonas, Roseburia, *R. bromii*, *F. prausnitzii*,, and induced distinct compositional changes compared to non-starch polysaccharide and weight-loss diets. However, diet accounted for only 10% of the total variance in microbiota composition, 90% attributable to inter-individual differences in baseline microbiota.	76, 95, 137, 138
9	Increased faecal SCFA production	RS2 (hi-maize vs potato) 20–40 g/day × 2–4 weeks; RS4 10–35 g/day	Healthy adults	RS increased butyrate and total SCFAs compared to habitual diet and non-RS fibre. The source of RS strongly influenced SCFA production — potato RS2 produced the greatest butyrate increase, while maize RS2 did not. Different RS4 structures directed either butyrate or propionate production, with effects plateauing at 35 g/day.	50, 69, 70

RS: Resistant starch; T2DM: Type 2 diabetes mellitus; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; HbA1c: Glycated haemoglobin; GLP-1: Glucagon-like peptide-1; SCFA: Short-chain fatty acid; MASLD: Metabolic dysfunction-associated steatotic liver disease; IL-6: Interleukin-6; TNF-α: Tumour necrosis factor-alpha; CRP: C-reactive protein; IS: Indoxyl sulfate; TBARS: Thiobarbituric acid reactive substances; KNHANES: Korea National Health and Nutrition Examination Survey.

Table 1. Major beneficial effects of resistant starch supplementation in humans (cont.)

	Beneficial effect	RS type / dose	Population	Key findings	References
10	Improved bowel function and stool regularity	RS2 (potato RS or RS blend of potato + green + apple) 3.5–30 g/day × 2–6 weeks; RS3 9 g/day × 12 weeks	Healthy adults Or adults with chronic constipation	RS improved stool consistency and frequency, and normalised stool form (reducing both diarrhea- and constipation-associated bowel movements)	95, 137, 138
11	Reduced systemic inflammation	Varied RS types, 10–45 g/day × 4–14 weeks	Healthy adults; or adults with, overweight, prediabetes, diabetes, or haemodialysis	RS reduced circulating and inflammatory biomarkers (IL-6 and TNF-α) across diverse populations, reduced CRP in T2DM patients.	98, 104, 105
12	Reduced uraemic toxins and oxidative stress	RS2 16 g/day × 4 weeks	Haemodialysis patients	RS reduced uraemic toxin (IS), oxidative stress marker (TBARS), and inflammatory biomarker (IL-6)	99

RS: Resistant starch; T2DM: Type 2 diabetes mellitus; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; HbA1c: Glycated haemoglobin; GLP-1: Glucagon-like peptide-1; SCFA: Short-chain fatty acid; MASLD: Metabolic dysfunction-associated steatotic liver disease; IL-6: Interleukin-6; TNF-α: Tumour necrosis factor-alpha; CRP: C-reactive protein; IS: Indoxyl sulfate; TBARS: Thiobarbituric acid reactive substances; KNHANES: Korea National Health and Nutrition Examination Survey.