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Fermentable Resistant Dextrin Links In-Vitro Short-Chain Fatty Acid Production to Human Microbiota Modulation

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Abstract: Fermentable resistant dextrin has to survive upper-gastrointestinal digestion before it can act as a colonic substrate. We examined two high-fiber BALIMONT preparations using simulated digestion and an anaerobic fecal-microbiota model, then compared the formulation findings with randomized human microbiota studies. RD-1 and RD-Clear retained 83.6% and 85.2% resistant components during simulated digestion. Relative to ordinary maltodextrin (1.00), RD-1 produced 1.46 total SCFA, 1.39 acetate, 1.31 propionate and 1.22 butyrate; RD-Clear produced 1.58, 1.51, 1.42 and 1.34. Human studies of resistant dextrin/resistant maltodextrin have reported changes in Bacteroides, bifidobacteria, Fusicatenibacter and Parabacteroides, together with stool or metabolic-function changes that depend on dose and host microbiota. A 2026 randomized trial in 124 healthy men increased Parabacteroides to approximately 5.5% versus 1.4% with placebo but did not increase fecal SCFAs significantly. The formulation data and human findings therefore support a two-step research model: resistant substrate reaches the colon, then microbial composition and metabolic output are measured separately rather than assuming that fecal SCFAs must rise whenever fermentation changes.

Keywords: resistant dextrin; gut microbiota; prebiotic; short-chain fatty acids; Parabacteroides; fermentation; soluble fiber.

Introduction

A fermentable fiber first has to escape enough host digestion to reach the colon. Once there, microbial access and community composition determine which organisms use the substrate and which metabolites appear. SCFAs are useful products to measure, but fecal concentrations represent what remains after microbial cross-feeding, epithelial uptake and transit. They are therefore not a direct readout of total colonic production.

The BALIMONT resistant-dextrin platform was selected because its upper-gastrointestinal profile is defined before fermentation testing. RD-1 and RD-Clear contain 89.2% and 90.6% total dietary fiber, 5.2% and 4.6% combined glucose/maltose/maltotriose, and 64.8% and 66.5% DP4-DP12. Their simulated-digestion retention values are 83.6% and 85.2%. These measurements describe the substrate presented to the fecal-microbiota model.

Human work has moved from targeted bacterial counts to sequencing and metagenomics. Lefranc-Millot et al. reported higher *Bacteroides* and lower *Clostridium perfringens* during resistant-dextrin supplementation [1]. Baer et al. showed that resistant maltodextrin altered fecal microbiota and fecal output in adult men [2]. Burns et al. found that 25 g/day increased fecal bifidobacteria and stool wet weight [3], while targeted analysis from the same feeding study identified an approximately twofold increase in *Fusicatenibacter saccharivorans* at 15 and 25 g/day [4]. Thirion et al. linked a *Parabacteroides distasonis* response to strain-level carbohydrate-utilization capacity [5]. Barber et al. followed gas handling, metagenomic features and metabolomics during 14 g/day supplementation [6]. Perreau et al. then tested 15 g/day in 124 healthy men and found a marked *Parabacteroides* response across both habitual-fiber strata without a significant fecal-SCFA increase [7]. This progression is useful for deciding which endpoints should be separated in a formulation-specific trial.

Materials and Methods

Formulation characteristics relevant to colonic delivery

RD-1 and RD-Clear contained 89.2% and 90.6% total dietary fiber, 5.2% and 4.6% combined glucose/maltose/maltotriose, and 64.8% and 66.5% DP4-DP12, respectively. Simulated digestion retained 83.6% and 85.2% resistant components. These values defined the substrate used for fermentation testing.

In-vitro anaerobic fermentation

Fecal microbiota were used as the inoculum in an anaerobic fermentation model. Resistant dextrin was the carbon source. Total SCFAs, acetate, propionate and butyrate were measured in the supernatant after fermentation. Ordinary maltodextrin was assigned a relative value of 1.00 for each product, and RD-1/RD-Clear values were expressed relative to that reference.

Human-evidence search

Randomized, placebo-controlled and crossover human studies through August 2026 were selected when they reported microbiota composition, targeted bacterial counts, stool output, fecal pH, SCFAs, functional genes, gas production or gastrointestinal outcomes after resistant dextrin/resistant maltodextrin. The 2026 trial NCT05105425 was retained because it tested whether habitual fiber intake modified the response [7].

Evidence interpretation

The in-vitro fermentation values were used as evidence of substrate fermentability, not as predicted human fecal concentrations. Human microbiota and SCFA endpoints were kept separate because absorption, cross-feeding and transit can change fecal SCFA values even when production differs.

Table 1. In-vitro fermentation profile and upstream digestive-resistance characteristics.

Sample	Total SCFA	Acetate	Propionate	Butyrate	Digestion retention (%)
RD-1	1.46	1.39	1.31	1.22	83.6
RD-Clear	1.58	1.51	1.42	1.34	85.2
Maltodextrin	1.00	1.00	1.00	1.00	14.7

Results

In-vitro fermentation phenotype

RD-1 produced relative values of 1.46 for total SCFA, 1.39 for acetate, 1.31 for propionate and 1.22 for butyrate versus maltodextrin at 1.00. RD-Clear produced 1.58, 1.51, 1.42 and 1.34. Relative total SCFA was therefore 46% above reference for RD-1 and 58% above reference for RD-Clear; the corresponding acetate increases were 39% and 51%, propionate 31% and 42%, and butyrate 22% and 34%.

Early randomized human studies

In two randomized placebo-controlled studies involving 48 and 40 healthy volunteers, resistant dextrin at 8-20 g/day increased *Bacteroides* and reduced *Clostridium perfringens* [1]. Beta-glucosidase activity changed at selected doses and colonic pH decreased at 20 g/day, whereas the SCFA increase was not statistically significant. The result already separated an ecological response from a uniform fecal-SCFA response.

Controlled crossover evidence

Baer et al. used 0, 25 and 50 g/day resistant maltodextrin in a double-blind crossover design and reported changes in fecal mass and microbial taxa [2]. Burns et al. tested 0, 15 and 25 g/day in 51 healthy adults; 25 g/day increased fecal bifidobacteria and stool wet weight [3]. Targeted qPCR on samples from that study identified an approximately twofold increase in *Fusicatenibacter saccharivorans* at both 15 and 25 g/day [4].

Metagenomic evidence

Thirion et al. found increased *Parabacteroides distasonis* after six weeks of resistant dextrin in 50 healthy women, with response linked to carbohydrate-utilization genes in the resident strains [5]. Barber et al. administered 14 g/day for four weeks and documented adaptation in intestinal gas handling together with metagenomic and metabolomic changes [6]. These studies make the host-community background part of the exposure rather than a nuisance variable.

2026 randomized trial and habitual-fiber context

Perreau et al. randomized 124 healthy men to 15 g/day resistant dextrin or maltodextrin placebo for four weeks and stratified participants by habitual fiber intake [7]. *Parabacteroides* abundance reached approximately 5.5% with resistant dextrin versus 1.4% with placebo, and *P. distasonis* reached 2.6% versus 0.5%. The response was present in both habitual-fiber strata. Fecal pH, SCFAs, secretory IgA and stool frequency/consistency did not differ significantly between groups.

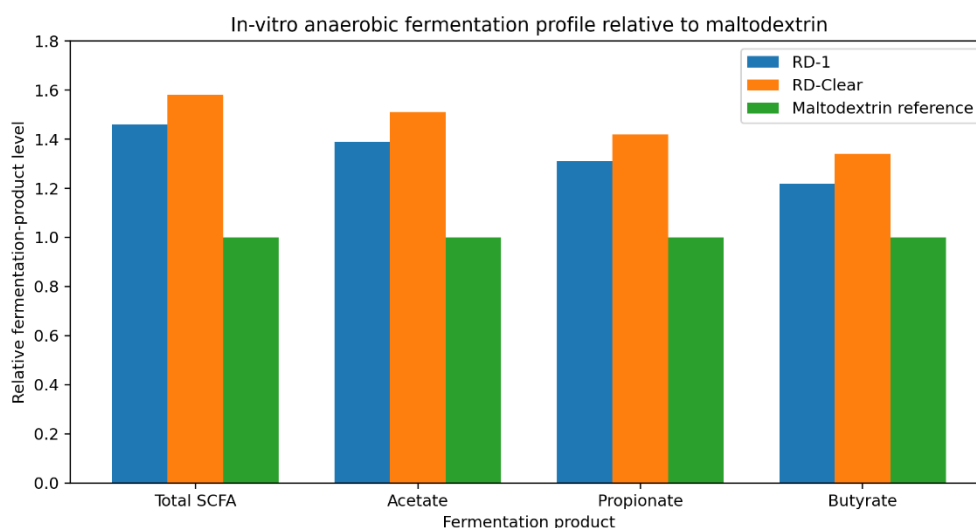


Figure 1. In-vitro anaerobic fermentation products relative to ordinary maltodextrin (reference = 1.00)

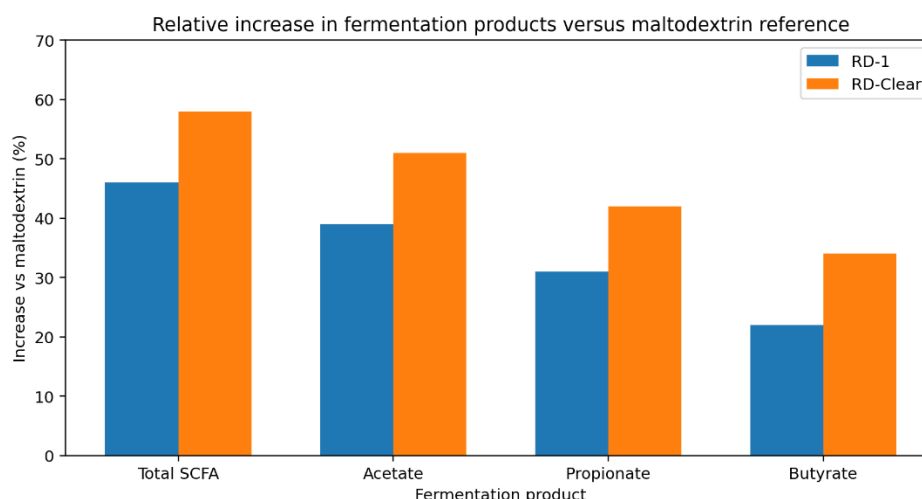


Figure 2. Percentage increase in fermentation products relative to the maltodextrin reference

Table 2. Selected human microbiota studies of resistant dextrin/resistant maltodextrin

Study	Participants / design	Dose & duration	Microbiota-related observation
Lefranc-Millot et al. [1]	48 + 40 healthy volunteers; randomized placebo-controlled	8-20 g/day; 14 d	Bacteroides increased; C. perfringens decreased
Burns et al. [3]	51 healthy adults; double-blind crossover	0/15/25 g/day; 3 wk	25 g/day increased bifidobacteria and stool wet weight
Thirion et al. [5]	50 healthy women; controlled supplementation	RD for 6 wk	P. distasonis increased; response linked to substrate-utilization genes
Perreau et al. [7]	124 healthy men; randomized double-blind placebo-controlled	15 g/day; 4 wk	Parabacteroides ~5.5% vs 1.4%; no significant fecal SCFA change

Discussion

Fermentability is supported by both substrate survival and fermentation products

The formulation experiments show the two steps required for a prebiotic-type mechanism. RD-1 and RD-Clear first preserved 83.6% and 85.2% resistant material through the simulated upper-gastrointestinal sequence. In the separate fecal-microbiota model, both were then fermented to higher relative total SCFA, acetate, propionate and butyrate than ordinary maltodextrin. The data establish substrate survival and microbial accessibility under the tested conditions.

Why fecal SCFAs need not mirror in-vitro production

The human studies warn against using fecal SCFA concentration as the sole translation endpoint. Fasting et al. observed changes in fecal microbiota and stool characteristics during resistant-maltodextrin supplementation in healthy adults [8]. A short randomized crossover trial in young healthy participants also showed that several fiber substrates, including resistant maltodextrin, can shift selected genera over only a few days [9]. Perreau et al. [7] provides the clearest contrast for this paper: a strong Parabacteroides signal occurred without a significant fecal-SCFA difference. Production, absorption and cross-feeding are all part of that mismatch.

Taxonomic effects appear dose- and host-dependent

Fiber structure and resident microbial capacity determine which organisms respond. The broader literature describes this interaction as a substrate-selection problem rather than a universal 'good-bacteria' effect [10]. SCFAs then connect microbial fermentation to host physiology through several tissue-specific routes [11]. Their measured concentration depends on formation, uptake and further microbial use [12], and the balance among acetate, propionate and butyrate depends on the metabolic

pathways represented in the community [13]. These mechanisms explain why the in-vitro product profile and the human taxonomic response should be measured in parallel, not forced into a one-to-one relationship.

Habitual fiber intake and precision prebiotic research

Habitual fiber intake did not abolish the Parabacteroides response in the 2026 trial [7], but that does not eliminate interindividual variation. Baseline community composition, carbohydrate-utilization genes and habitual diet remain plausible response modifiers [5,10]. A BALIMONT trial should therefore record baseline fiber intake and metagenomic functional capacity before supplementation rather than using only post-treatment relative abundance.

Translational relevance of low viscosity and processing stability

The same resistant-dextrin class also has downstream metabolic evidence. Twelve-week supplementation in overweight men improved insulin-resistance-related variables [14], and an eight-week trial in women with type 2 diabetes reported lower fasting insulin and HOMA-IR [15]. Those results do not show that microbiota changes caused the metabolic effects. They do, however, justify measuring glycemic markers as secondary host outcomes in a microbiota-focused study if microbial and metabolite data are collected at the same time.

Research Scope and Future Directions

A formulation-specific trial should preserve the sequence already used here: verify upper-gastrointestinal resistance, document dose and batch composition, then sample the microbiota repeatedly during exposure. Shotgun metagenomics, targeted Parabacteroides/bifidobacteria quantification, fecal and plasma metabolomics, breath hydrogen and stool measures can distinguish community change from metabolite handling. Early repeat sampling is especially useful because gas production and substrate utilization can adapt during the first days of supplementation.

Conclusion

RD-1 and RD-Clear retained 83.6% and 85.2% resistant components during simulated digestion and produced higher relative total SCFA, acetate, propionate and butyrate than ordinary maltodextrin in the fecal-microbiota model. Randomized human studies independently show changes in Bacteroides, bifidobacteria, Fusicatenibacter and Parabacteroides after resistant-dextrin/resistant-maltodextrin exposure, while fecal SCFAs do not always rise in parallel. This evidence supports direct human evaluation of the BALIMONT material with substrate delivery, microbiota composition and microbial/host metabolites treated as separate linked measurements.

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