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Literature Review

A 10-Year Systematic Review (01 May 2015 to 30 April 2025) on the Effects of Oats Consumption on Gut Microbiome

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Received: October 11, 2025; **Published:** November 12, 2025

Abstract

Oats (*Avena sativa L.*) have gained increasing attention as functional foods due to their bioactive compounds; including β-glucans, resistant starch, arabinoxylan, polyphenols, and avenanthramides; which are known to modulate the gut microbiome and improve host health. Despite promising findings, substantial gaps remain-specific oat fractions are underexplored, few clinical trials span diverse populations, and only limited reviews have synthesized their effects on the gut microbiota and health outcomes. We present a 10-year systematic review to evaluate the effects of oat consumption on gut microbiota modulation and associated health benefits. A PubMed search was conducted, and 199 papers were identified within the past 10 years (1 May 2015 to 30 April 2025). After exclusions, a total of 80 primary research articles were included in this systematic review. The studies were categorised into three main themes: (a) effects of oat composition and processing techniques, (b) potential health benefits of oats mediated by gut microbiota, and (c) potential effects of other dietary ingredients on oats. Future research should focus on whole-food approaches preserving structural complexity, define dose-response relationships for health outcomes, and develop microbiome-based models to optimize personalized oat interventions.

Keywords: *Oats (Avena sativa L.); Gut Microbiome; Host Health; Gastrointestinal Disorders*

Introduction

Chronic metabolic and gastrointestinal disorders represent a growing global health concern. For example, China is projected to have over 867 million obese individuals by 2030 [1]. Meanwhile, colorectal cancer rates remain highest in developed regions such as Australia, Canada and Scandinavia, exceeding 26.3 cases per 100,000 population [2]. Diabetes, a metabolic disease affected an estimated 463 million people worldwide in 2019, with projections of 700 million by 2045 [3]. At the same time, gluten-related disorders, including celiac disease (CeD) and non-celiac gluten sensitivity (NCGS), have continued to rise over the past five decades, posing additional dietary management challenges [4]. Strict adherence to a gluten-free diet (GFD) is the only effective therapy for CeD; however, long-term GFD has been associated with nutritional imbalances such as fibre deficiency, thereby increasing the risk of constipation, obesity and cardiovascular disease [4]. Given that gut microbiota plays a pivotal role in lipid, carbohydrate, and energy metabolism [1], dietary strategies that target microbial modulation have emerged as promising tools to mitigate metabolic risk.

Oats (*Avena sativa L.*), a whole grain cultivated largely in Europe and North America, has gained renewed attention as a functional food owing due to their rich nutrient profile and bioactive compounds [5,6]. Their health-related properties are particularly associated with β-glucans, a soluble fibre recognized by the European Food Safety Authority (EFSA), which advises a daily intake of 3 grams to reduce low density-lipoprotein cholesterol (LDL-C) [7]. Additionally, oats are rich in polyphenols, unique steroidal glycosides such as avenacosides-A and -B, and antioxidants like avenanthramides, which have demonstrated antihypertensive and anti-inflammatory effects [8]. They also provide high-quality protein, complex carbohydrates, and essential minerals including magnesium, zinc, and iron [5]. Accumulating evidence suggests broader systemic benefits, including improvements in liver function, cardiometabolic markers, intestinal microbiota composition, and glycemic control [3,5,9]. In infant gut microbiota models, oats demonstrated distinctive prebiotic properties, significantly enhancing *Veillonellaceae* abundance ($p < 0.05$) above all other tested cereals and increasing propionate production, suggesting specific fermentation patterns that may contribute to their metabolic benefits [10]. The specific effects of oats, however, are influenced by the physicochemical structure of oat fibers, which can be altered by processing methods [11].

The human gut microbiome - an intricate community of microorganisms-plays a central role in host metabolism, immunity, and neurophysiology. Oat β-glucans undergo colonic fermentation, producing short-chain fatty acids (SCFAs) that regulate glucose and lipid metabolism, immune signalling, and gut barrier integrity [9,11,12]. Animal studies have demonstrated that oat-enriched diets promote beneficial taxonomic shifts, elevate SCFA production, and enhance populations of probiotic species such as Lactobacillus [7,13]. In individuals adhering to a GFD, oats have been shown to restore microbial diversity and functionality, mitigating the negative effects of reduced fibre intake [4].

Experimental studies further confirm the diverse metabolic impact of oat constituents. In diabetic rat models, oat β-glucans improved glycemic control, reduced hepatic inflammation, and enhanced microbial richness [3,13]. Oat fibres modulate bile acid metabolism and tryptophan-derived immune pathways, conferring additional benefits for cholesterol regulation and liver health [9]. Supplementation with β-glucans has also shown potential in alleviating metabolic syndrome and reducing uremic toxins [7,12]. Oat flour preparations consistently enhanced *Bifidobacteriaceae* and lactic acid bacteria populations in infant gut fermentation models, with specific varieties increasing *Lactobacillus reuteri* and *Weissella spp.* while suppressing pathogenic *Clostridium perfringens* [14]. These findings, combined with evidence of donor-specific microbial fermentation responses to different oat fractions [11,15], suggest both universal prebiotic benefits and inter-individual variability in outcomes.

Despite promising findings, substantial gaps remain. The biomarker potential of specific oat fractions (e.g. bran, groat, protein isolate) remains underexplored, and relatively few clinical trials have systematically compared their effects across diverse populations [9,11]. Furthermore, the influence of thermal and mechanical processing on β-glucan structure and functional outcomes *in vivo* requires greater clarification [11]. Inter-individual variability in gut microbiome composition also highlights the potential for personalized nutrition approaches to optimize dietary oat interventions [16,17].

Research on their effects on gut microbiota and metabolism has only recently intensified. A preliminary PubMed search shows that, while many studies report microbial endpoints, the evidence remains fragmented. Systematic evidence on how oats influence gut microbiota composition and metabolic function is still limited. This gap hinders evidence-based dietary recommendations and constrains understanding of how processing methods, dosing strategies, and individual microbiome variations shape therapeutic outcomes-knowledge essential for clinical practice, food industry innovation, and metabolic disease prevention. Therefore, this review aims to address these gaps by systematically evaluating experimental studies over the last decade on oat consumption and gut microbial outcomes.

Methods

A literature search was conducted on PubMed to identify articles published between 1 May 2015 and 30 April 2025, focusing on the effects of oats consumption on the gut microbiome. The search string is "(oat OR oatmeal OR oats) AND gut AND (microbiome OR microbiota)", resulting in the following URL: https://pubmed.ncbi.nlm.nih.gov/?term=(oat+OR+oatmeal+OR+oats)+AND+gut+AND+(microbiome+OR+microbiota)&filter=dates.2015/5/1-2025/4/30. The following exclusion criteria were applied: (A) Articles without free full-text availability; (B) Articles not written in English; (C) Articles classified as secondary research, such as systematic reviews, literature reviews, or meta-analyses; (D) Pre-print articles from platforms such as bioRxiv that had not been peer-reviewed at the time of access; and (E) Articles that did not contain any of the following keywords "oat", "gut", "microbiome" and "microbiota" in the title, abstract, or body text, indicating a lack of relevance.

Results

A total of 199 records were retrieved. The final set of 80 primary articles met all inclusion criteria and were included in this systematic review to evaluate the effects of oat consumption on gut microbiome modulation (Figure 1), which were classified into 3 main themes (Table 1).

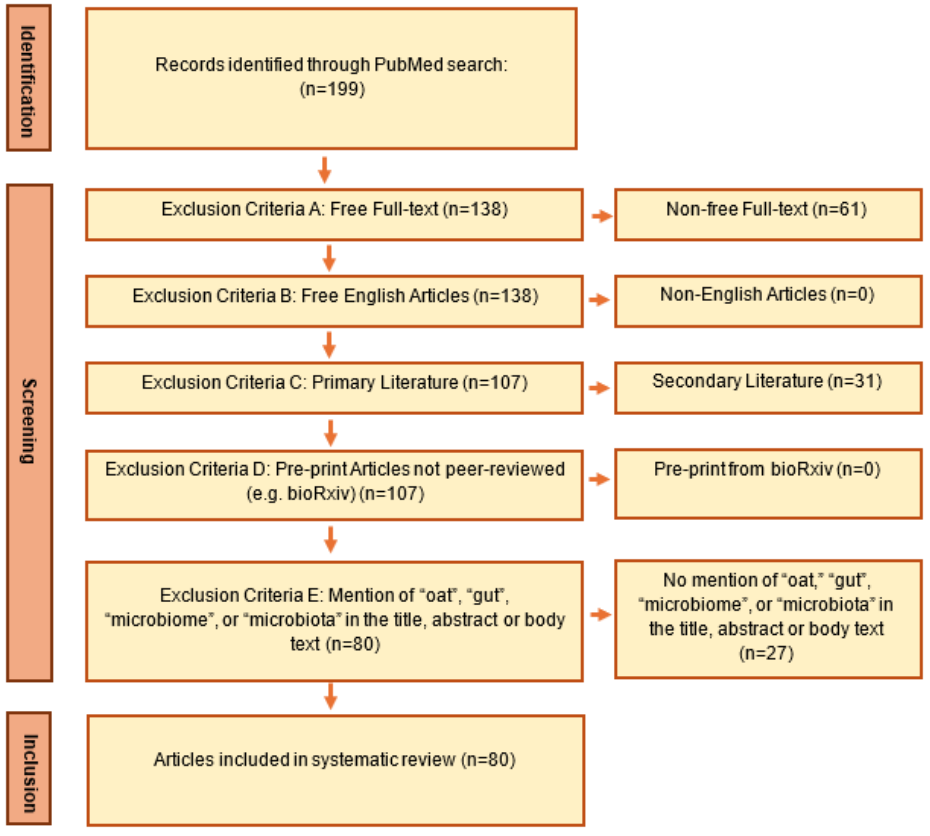

Figure 1: *Flow of process through the 5 exclusion criteria using PRISMA.*

Theme		Number of Articles
1. Effects of Oat Composition and Processing Techniques	1.1 Prebiotic Properties on Gut Microbiota Composition	43 [2, 6, 18-59]
	1.2 Influence of Processing Methods on Functional Outcomes	8 [10, 11, 19, 27, 37, 38, 59, 60]
2. Potential Health Benefits of Oats Mediated by Gut Microbiota	2.1 Anti-inflammatory and Immunomodulatory Effects	12 [3, 13, 18, 21, 23, 49, 57, 61-65]
	2.2 Metabolic and Cardiovascular Effects	16 [1-3, 5, 9, 12, 20, 26, 29, 30, 41, 43, 66-70]
	2.3 Gut Barrier and Intestinal Health	12 [3, 13, 22, 28, 29, 49, 57, 60, 71-74]
3. Potential Effects of Other Ingredients to Oat		12 [6, 30, 31, 41, 64, 72, 73, 75-79]

Table 1: *Thematic classification of studies on the effects of oats consumption on gut microbiome.*

Theme 1.1: Effects of oat composition and processing techniques - prebiotic properties on gut microbiota composition

Oats have emerged as a functional food of significant interest, particularly due to their influence on gut health. The investigation into their prebiotic potential begins with the examination of their diverse fermentable components-β-glucan, resistant starch (RS), arabinoxylan, avenanthramides (AVs), and polyphenols-each imparting unique effects on gut microbiota and the synthesis of short-chain fatty acids (SCFAs).

β-glucan represents the cornerstone prebiotic fibre in oats, with remarkable capacity to reshape gut microbiota architecture. The metabolic machinery for β-glucan utilization appears universally distributed-metagenomic analysis reveals mixed-linkage β-glucan utilization loci in 92.5% of adults worldwide, with over 70% of key Bacteroides species harbouring the genetic capacity for oat β-glucan fermentation [47]. Human trials demonstrate consistent dose-dependent bifidogenic responses across diverse populations. Daily supplementation with 1.3 g β-glucan elevated *Bifidobacterium* by 140% and *Lactobacillus* by 45% (both $p = 0.001$) [41], with Mongolian adults showed significant increases in *Bifidobacterium adolescentis* ($p = 0.0321$), *Bifidobacterium bifidum* ($p = 0.0420$), and *Bifidobacterium catenulatum* ($p = 0.0210$) within one week [42]. Extended supplementation (80g for 45 days) enriched additional taxa including *Roseburia* ($p = 0.02$), *Akkermansia muciniphila* ($p = 0.04$), *Dialister succinatiphilus* ($p = 0.002$), and *Bifidobacterium pseudocatenulatum* ($p = 0.023$) [43], while longer interventions promoted *Prevotella* and *Roseburia* [6,40]. *Ex vivo* studies confirmed a log2 fold change of +1.42 in *Bifidobacterium* ($p < 0.001$) [48], and the SHIME® model demonstrated elevations to 6.2×10^9 and 4.5×10^9 gene copies/mL for *Lactobacillus* and *Bifidobacterium* (both $p < 0.05$), at a concentration of 1.4 g β-glucan/day in the luminal environment of the human gastrointestinal tract [46].

Animal models corroborate oat β-glucan findings across species. High-fat diet (HFD)-fed rats showed enhanced *Lactobacillus*, *Lachnospiraceae* UCG-001 and *Roseburia* (all $p < 0.05$) in 12 weeks [50], pre-weaning calves supplemented with 1.1 g/kg body weight β-glucan enriched *Bacteroides*, *Alloprevotella*, and *Alistipes* (all $p < 0.05$) [52] out of eighty-four genera taxa from the faces, while cats with chronic kidney disease demonstrated clinical improvements including increased body mass and reduced uremic toxins (both $p = 0.004$) [51].

β-glucan consistently elevates SCFA production beyond other fibres. *In vitro* studies with varying purities (22%, 28%, 94%) showed superior fermentability [49,53], with oat β-glucan achieving ~18 mmol/L acetate and 9 mmol/L propionate (both $p < 0.001$) after 72-h [55]. Regardless of whether obtained from commercially packaged 22% oat β-glucan or purified β-glucan, the resulting SCFA profile-

dominated by acetate (~70%) and propionate (~20%)-exceeded most outcomes reported for inulin, whole fibre, and xylooligosaccharides [53]. The 28% purity fraction proved optimal, producing the strongest enhancement in gut barrier tightness, with transepithelial electrical resistance (TEER) increasing by ~123% ($p < 0.001$), likely due to the promotion of propionate and acetate, which exert positive effects via SCFA production [49]. This was accompanied by significant increases, particularly in propionic and valeric acid concentrations ($p < 0.05$) [28]. These findings were consistent with human studies and *in vitro* trials, which reported nearly a threefold increase in butyrate levels [40], alongside significant and consistent increases in total SCFA concentrations [41,48].

Vulnerable populations show pronounced benefits. ICU patients receiving oat-soy fibre exhibited 61% increase in SCFA-producing bacteria vs. 46% decline in the no fibre group ($p = 0.28$), with six-fold higher stool SCFA concentrations ($p = 0.16$) over a three day time span [45]. Under dextran sulfate sodium (DSS) colitis models showed all oat and oat bran interventions restored depleted SCFAs significantly [57], while metabolically challenged states showed consistent enrichment of probiotic (*Lactobacillus plantarum*, *Bacillus subtilis*, and *Saccharomyces cerevisiae*; $p < 0.01$) and gut bacteria genera (*Eubacterium*, *Butyricicoccus*, and Rum*inococcus*; $p < 0.05$) [54,58]. These effects translate to clinical outcomes including total cholesterol (TC) and LDL-C reductions of 3.0-9.1% after forty-five days of intervention was observed [43].

Oat β-glucan demonstrates remarkable stability-during simulated gastric digestion, the molecular weight of oat β-glucan decreased from 469,983 to 388,504 Da, representing about 17.3% degradation after 2-hour of exposure to gastric conditions. This relative resistance to enzymatic hydrolysis and acidic breakdown explains why a substantial proportion of β-glucan transits the upper gastrointestinal tract intact, enabling it to reach the colon where it can serve as a fermentable substrate for microbiota [56]. During the 24-h colonic fermentation, this substrate increased *Lactobacillus* by ~45% while decreasing *Escherichia-Shigella* by ~30% ($p < 0.05$) [56]. Beyond fermentation, oats modulate microbial enzyme activity-daily 60 g oatmeal in one week reduced faecal β-galactosidase by 63.7% ($p = 0.049$) and urease by 17.8% ($p = 0.031$) [44], suggesting selective metabolic reprogramming.

The discovery shows that *Bacteroides uniformis* has specialized genetic machinery (PULs) and a powerful enzyme (BuGH158; Km = 0.27 mg/mL, Kcat = 40.83 s^{-1}) that allow it to efficiently cut β-glucan into smaller pieces. These fragments are then shared with other gut bacteria, especially Gram-positive butyrate producers, amplifying the prebiotic effect of oats [36]. However, substrate preferences vary significantly among gut bacteria, with *Roseburia inulinivorans* decreasing in the oat group ($p < 0.05$) while *Roseburia hominis* increased and the overall *Roseburia* genus rose significantly ($p = 0.02$), demonstrating that even closely related species within the same genus can exhibit opposing responses to β-glucan supplementation [43].

Resistant starch (RS), another significant fermentable carbohydrate in oats, contributes to gut microbiota modulation and SCFA production. *In vitro* fermentation of RS demonstrates butyrogenic potency-48-h incubation yields one-log increase in *Bifidobacterium* ($p < 0.05$), with butyrate reaching 26 μmol/mL and propionate 8.8 μmol/mL, substantially exceeding levels from inulin and other fibers [37]. These selective fermentation patterns translate *in vivo*: HFD-fed mice consuming 40% mixture coarse cereals (containing millet, maize, oat, soybean, and purple potato with various prebiotic components including dietary fibre, β-glucan, and potentially resistant starch) exhibited substantial enrichment of *Bifidobacterium*, *Lactobacillus*, *Ruminococcus*, and *Lactobacillus intestinalis* ($p < 0.05$) [6], demonstrating the prebiotic potential of whole coarse cereals mixtures.

However, achieving these beneficial fermentation outcomes depends critically on processing conditions that govern RS formation and structure. In whole grain oats (WGO), extrusion moisture primarily modulates fermentation kinetics rather than RS yield. While RS content remained relatively stable (2.5-3.6%) across moisture conditions (15 - 21%), the fermentation dynamics varied significantly-15% moisture enhanced butyrate production (0.66 μmol/h) during initial fermentation and elevated *Lactobacillus* counts at 24-h, whereas 18% moisture maximized acetate (2.87 μmol/h) and total SCFA production (4.47 μmol/h) at 8-h ($p < 0.05$) [38].

Arabinoxylan, a hemicellulose present in oat cell walls, is another fermentable fibre that significantly impacts the gut microbiome. *In vitro* fermentation with duck cecal microbiota produced dramatic taxonomic shifts with *Megamonas* surged from 13.9% to 63.8% and *Bifidobacterium* from 1.3% to 18.0% (both $p = 0.003$), while pH dropped from 6.79 to 5.05 ($p < 0.001$), alongside isobutyric acid increasing from 0.47-5.25 mg/mL ($p < 0.05$) [39]. Beyond fermentation-mediated effects, arabinoxylan significantly reduced *Enterococcus faecium* TX16 growth ~four-fold compared to glucose ($p < 0.05$) in an *ex vivo* gut model, an outcome likely attributable to microbiota-mediated interactions rather than direct antimicrobial activity [35].

The molecular basis for arabinoxylan utilization involves sophisticated enzymatic machinery - *Bacteroides xylanisolvens* XB1AT coordinates two gene clusters (PUL43 and rPUL70) encoding specialized enzymes, particularly CBM4-containing GH10 endo-xylanase, to cleave xylan into oligosaccharides [32]. These oligosaccharides serve as cross-feeding substrates that support the growth of other beneficial gut bacteria. Clinical evidence demonstrates this prebiotic cascade effect: 6-week of WGO consumption, containing arabinoxylans among other fibres, significantly increases faecal *Bifidobacterium* (~140%; $p < 0.001$) and *Lactobacillus* (~45%; $p < 0.001$) populations in humans [41]. Hydrolyzed and fermented arabinoxylan-oligosaccharides intake has been shown to modulate the gut microbiome and its metabolic response in the middle-term, suggesting a sustained beneficial effect [59].

Pig models validate arabinoxylan's prebiotic benefits. In controlled porcine studies, 10% arabinoxylan supplementation significantly reduced caecal pH by 11% ($p < 0.0001$) and ammonia by 70% ($p < 0.01$), indicating enhanced carbohydrate over protein fermentation. Distal colon acetate increased by 27% ($p < 0.01$), while beneficial bacteria including *Prevotella*, *Lactobacillus*, and *Lachnospiraceae* (all $p < 0.05$) significantly increased alongside reduction in pathogenic *Clostridium* species [2].

Avenanthramides (AVs) are unique phenolic compounds exclusive to oats, primarily comprising AV-A (2p), -B (2f), and -C (2c), with documented antioxidant and anti-inflammatory properties. While not fermentable fibres, AVs significantly interact with gut microbiota, particularly *Faecalibacterium prausnitzii*, which metabolizes AVs into bioactive dihydro forms. A study identified *F. prausnitzii* as the key bacterium responsible for this transformation through 16S rRNA sequencing ($p < 0.05$) and qPCR validation ($p < 0.05$), revealing distinct metabotypes: 81% of subjects (17/21) were AVs metabolizers while 19% (4/21) were non-metabolizers. The microbial metabolite DH-2c demonstrates enhanced bioactivity compared to its parent compound, including superior anti-cancer effects [34]. This symbiotic relationship between AVs and *F. prausnitzii*, a beneficial bacterium comprising 5-15% of healthy gut microbiota that produces butyrate-represents a critical mechanism for oat's health benefits, with the anti-inflammatory properties potentially enhanced through microbial transformation to improve gut barrier function [34].

Polyphenols demonstrate limited independent prebiotic activity but contribute to synergistic effects within the oat fibre matrix. When isolated polyphenols (1.7 mg mix matching 4.5g digested oats) were tested alone, they failed to increase *Bifidobacterium* abundance and instead promoted *Enterobacteriaceae* family growth, specifically *Hafnia alvei* (21.7% abundance) at 24-h. However, when present within 1% (weight/volume) oat bran, the complete matrix significantly increased SCFA production threefold at 24-h (86-28 mM; $p < 0.05$) and enhanced *Bifidobacterium adolescentis* abundance 7-fold at 10-h and 5-fold at 24-h compared to negative control ($p < 0.02$). This matrix-dependent effect scaled with dose-3% (weight/volume) oat bran yielded 5.4-fold higher total SCFAs at 24-h vs. control (151.5 vs. 28.1 mM; $p < 0.05$), with significant increases in acetic acid at 10 and 24-h ($p < 0.01$) and propionic acid reaching 48 mM at 24-h ($p < 0.01$), effects absent with matched doses of isolated β-glucan (180 mg) or polyphenols [33].

Among the functional components in coarse cereals, polyphenols work synergistically with β-glucan, dietary fibre, and RS to prevent HFD-induced obesity through gut microbiota modulation. WGO flavonoids (50-100 mg/kg/day) significantly reversed HFD-induced dysbiosis by decreasing the *Firmicutes*/*Bacteroidetes* ratio from 145.24 to 28.00-40.62, while dramatically increasing *Akkermansia* abundance - a next-generation probiotic associated with improved intestinal barrier function and lipid metabolism [20]. This polyphenol-

rich fraction specifically decreased pathogenic bacteria including *Lachnoclostridium*, *Blautia*, *Colidextribacter*, and *Desulfovibrio*, which showed strong positive correlations with serum TC, triglyceride (TAE), and hepatic lipogenic gene expression (SREBP-1c, FAS). The flavonoids activated the FXR-dependent pathway, upregulating CYP7A1, NTCP, and BSEP expression while inhibiting intestinal ASBT, thereby promoting bile acid synthesis and faecal excretion-mechanisms that enhanced cholesterol clearance [20].

The impact of oat and oat-derived products on gut microbiota diversity presents a complex, context-dependent landscape with outcomes strongly influenced by host species, health status, intervention duration, and the specific oat fraction utilized. Most human studies in healthy populations demonstrate remarkably consistent null effects on diversity indices. Multiple interventions-including 3 g/day β-glucan for three weeks [30], 12 g/day oat β-glucan for six weeks in elderly subjects [22], and six months of sprouted oat fermented beverage in celiac patients [23] failed to significantly alter Shannon, Chao1, or Simpson indices [22,26-31]. A comprehensive study with 32 participants found alpha diversity remained unchanged (Observed: 846 vs. 836; Chao1: 1456 vs. 1457) [30]. Even in elderly subjects with 85% having suboptimal fibre intake, diversity metrics remained stable [22].

Similar patterns emerged in several animal models. Mealworms fed oat-based diets maintained diversity metrics comparable to controls (Shannon H' = 3.974, Simpson's D = 0.057; $p > 0.05$) [24]. Weaned rabbits receiving 200 mg/kg oat β-glucan showed no diversity changes despite increased beneficial bacteria including *Lactobacillus*, *Prevotellaceae* UCG-001, *Pediococcus*, and *Bacillus* ($p < 0.05$) [28]. One inflammatory bowel disease (IBD) mouse study found no significant differences in ACE and Simpson between treatment groups [57]. In high-fat fibre-deficient (HFFD) mice, while the HFD decreased Shannon ($p = 0.0369$), β-glucan supplementation did not significantly differ from HFFD groups ($p = 0.171$) [18].

Metabolically compromised populations showed more responsive diversity changes. Atherosclerotic mice receiving 0.8% oat fibre for 14 weeks demonstrated significantly improved intestinal microbiota diversity alongside enhanced cognitive function [29]. HFD-fed mice receiving whole-grain oat showed significant increases in Shannon and ACE while Simpson decreased ($p < 0.05$) [20]. Pre-weaning dairy calves supplemented with β-glucan (75 mg/kg for 14 days) demonstrated significantly increased Shannon and Chao1 ($p < 0.05$) [52]. Mixture coarse cereals containing oats significantly increased Shannon index in IBD mice compared to HFD group ($p < 0.05$) with Simpson showing opposite trends [6].

Intriguingly, some studies revealed diversity reductions despite beneficial outcomes. Healthy mice receiving oat β-glucan showed lower Chao1 index and Shannon ($p < 0.001$ and $p = 0.004$, respectively), yet beneficial increases in *Lachnospiraceae* and *Bacteroidales*_S24-7 families occurred [21]. Spodoptera frugiperda (fall armyworm) fed on wild oat diets exhibited the lowest microbial diversity with *Firmicutes* reaching 74.05% and *Enterococcaceae* 67.76 vs. 0.27% on artificial diet ($p < 0.001$) [25].

Different diversity metrics responded variably to identical interventions. Foals fed steam-flaked oats showed higher Chao1 compared to corn or barley groups ($p < 0.05$), yet Shannon and Simpson were significantly higher in the barley group [19], suggesting metric-specific responses to the same intervention.

Importantly, beneficial compositional shifts frequently occurred without diversity changes. HFFD mice receiving β-glucan successfully restored Bacteroidetes and reduced Proteobacteria abundance despite unchanged Shannon [18]. Broiler chickens with 3% oat hull supplementation showed modified caecal microbiota at species level without affecting diversity metrics, with physical form (coarse vs. extruded) influencing outcomes differently [27].

The metabolic consequences consistently outweighed diversity changes. Oat β-glucan supplementation reliably increased colonic SCFA production-particularly propionic and butyric acids-even when bacterial diversity remained unchanged. Rabbits showed increased

intestinal propionic, valeric, and butyric acid concentrations while decreasing lysine and aromatic amino acid derivatives ($p < 0.05$), suggesting metabolic reprogramming rather than simple diversity enhancement [28].

Theme 1.2: Effects of oat composition and processing techniques - influence of processing methods on functional outcomes

Processing technologies fundamentally transform oat fibre architecture, creating distinct physicochemical profiles that dictate downstream fermentability and prebiotic potential. Physical and chemical modifications-through extrusion, hydrolysis, fermentation, thermal treatment, and mechanical disruption-selectively alter solubility, molecular weight distribution, and substrate bio accessibility.

Thermal-mechanical extrusion triggers dual effects on fibre functionality. Processing at 130°C and 300 rpm enhanced water-extractable β-glucan release, achieving 1.8% and 1.7% at 15% and 21% moisture respectively, significantly exceeding the 1.1% baseline in raw oats ($p < 0.05$) [38]. Concurrently, RS content increased to 3.6% at 15% moisture, though protein content remained relatively stable at 22.1% at 18% moisture [38]. Remarkably, extrudates processed at lower moisture demonstrated superior stability, retaining more β-glucan after 8-h of fermentation, suggesting enhanced resistance to degradation and potentially prolonged fermentative activity [38]. Lower-temperature extrusion of oat hulls reveals fibre fraction redistribution. Processing at 100°C decreased neutral detergent fibre (NDF) from 76.2-73.9% and acid detergent fibre (ADF) from 42.3-40.3%, while paradoxically increasing lignin content from 6.62-9.29% [27]. This restructuring coincided with substantial depolymerization-starch declined by 38.2% (from 4.37-2.70%) and simple sugars by 42.7% (from 1.10-0.63%)-likely through Maillard reactions that enhance microbial substrate accessibility [27].

Enzymatic and acid hydrolysis dramatically amplify soluble fibre fractions. Hydrolysis of oat bran fermented with lactic acid bacteria yielded exceptional water-extractable arabinoxylan release at 77.6 g/L, substantially exceeding wheat bran's 1.3 g/L output [59]. This solubilization manifested in enhanced fermentability, with acetic acid production in the proximal colon increasing from 32.3-53.1 mM after two weeks of wash-out ($p < 0.05$), while the formulation maintained high levels of both insoluble fibre (4.6 g/L) and soluble fibre (1.2 g/L) [59]. Similarly, acid hydrolysis using 0.1M citric acid at 85°C for 16-h extracted 9.76g β-glucan per 100g dried material, with subsequent purification yielding 66.98g per 100g extract-a 6.9-fold concentration increase [60]. Enzymatic treatment represents the pinnacle of controlled fibre modification. β-glucanase treatment of oat β-glucan concentrates produced remarkable compositional shifts: soluble fibre reached 52.3% with insoluble fibre at 1.1% and oligosaccharide content at 6.4%, collectively indicating optimized bio accessibility and fermentative potential [37].

Thermal roasting (80-100°C) followed by size reduction (cutting, steaming, pressing) produced varied fermentability profiles. Matrix disruption intensity correlates with fibre release efficiency. Steam-processed oat bran at high moisture (82.8% after cooking) achieved maximum β-glucan (7.62%) and total dietary fibre (14.8%) yields, substantially exceeding steel-cut oats (4.94% β-glucan, 7.9% total dietary fibre) [10]. Conversely, pre-cooked Morrison oat flour processed at low moisture (10.0%) preserved β-glucan at 6.31%, suggesting that structural integrity paradoxically limits solubilization [11]. Steam-flaking differentially affects fibre composition in concentrate supplements containing processed cereals. Analysis of concentrate supplements containing steam-flaked grains revealed distinct fibre profiles: oat-based concentrate supplements showed higher ADF (9.09%) and NDF (27.04%) values compared to corn-based (ADF 5.10%, NDF 24.46%) and barley-based (ADF 5.71%, NDF 24.79%) supplements. These differences suggest that despite steam-flaking processing, oat-based feeds retained higher fibre content, potentially due to the inherent fibre-rich hull structure of oats that is more resistant to thermal-mechanical processing compared to corn and barley [19]. Particle size reduction through milling creates a solubility gradient. Pre-cooked oat flours milled to 50-250 μm demonstrated enhanced dispersibility and hydration, while instant oats with reduced particle dimensions (0.36-0.46 mm) showed consistent solubility improvements [11]. The synergy between milling intensity and cooking moisture emerged as the primary determinant of β-glucan accessibility, with finer particles yielding superior solubilization and fermentation readiness [11].

Theme 2.1: Potential health benefits of oats mediated by gut microbiota - anti-inflammatory and immunomodulatory effects

Cytokine modulation emerges as oats' primary anti-inflammatory mechanism. Clinical evidence demonstrates differential suppression patterns across oat formulations. In lipopolysaccharide (LPS)-stimulated macrophages, raw oat beverages (ROB) significantly inhibited tumour necrosis factor-alpha (TNF)-α production and reduced interleukin (IL)-6 release, with intestinal digests showing greater anti-inflammatory activity than gastric digests [23]. However, population-specific responses exist-elite wheelchair athletes supplemented with 5g daily oat bran for 4 weeks showed no significant alterations in serum inflammatory markers or gut microbiome alpha diversity (all $p > 0.05$), likely due to their baseline low inflammation status and the relatively low prebiotic dose [62].

β-glucan concentration profoundly influences immune activation intensity. Controlled fermentation studies reveal dramatic concentration-dependent responses: 28% oat β-glucan fermentation products increased IFN-γ by 733%, IL-10 by 989%, IL-17 by 1015%, IL-2 by 624%, and IL-9 by 1199% (all $p < 0.05$) [49]. The 94% purified β, -glucan preparation produced even more pronounced effects, with IFN-γ reaching 20,300.0 pg/mL compared to baseline 983.3 pg/mL [49]. Crucially, anti-inflammatory IL-10 emerged exclusively post-fermentation, underscoring microbial metabolism as essential for converting oat substrates into immunoregulatory mediators [49].

Animal models elucidate dose-dependent anti-inflammatory mechanisms. In streptozotocin-induced diabetic rats, oat flakes supplementation significantly reduced IL-1β levels from 74.9-77.94 pg/mL in diabetic controls to 48.03-52.23 pg/mL in treated groups ($p < 0.001$) [13]. Dietary intervention studies using oat and buckwheat compounds demonstrated optimal anti-inflammatory effects at 30% oat inclusion, with significant reductions in both IL-6 and TNF-α levels ($p < 0.05$) [3]. This dose-dependency extends to gastrointestinal inflammation: porcine models revealed significant reductions in inflammatory markers-caecal IL-8 mRNA expression decreased ($p < 0.05$), while colonic IL-8, NF-κB, and TNF-α mRNA expressions were all significantly suppressed ($p < 0.05$) in oat bran-supplemented groups [63].

DSS-induced colitis models revealed optimal therapeutic windows for anti-inflammatory effects. The 30% oat and 10-20% oat bran inclusion groups demonstrated the most effective suppression of inflammatory cytokines, significantly reducing TNF-α and IL-6 while increasing anti-inflammatory IL-10 ($p < 0.05$) [57]. Notably, higher concentrations (45% oats, 30% bran) provided no additional anti-inflammatory benefits and showed mild inflammatory infiltration in epithelial mucosa, suggesting a ceiling effect for therapeutic efficacy [57].

Synergistic antioxidant-anti-inflammatory coupling characterizes oats' protective mechanisms. HFD models consistently demonstrate concurrent effects: oat supplementation significantly reduced serum TNF-α and IL-6 levels while simultaneously increasing antioxidant defences - superoxide dismutase (SOD) activities and total antioxidant capacity (T-AOC) were significantly elevated ($p < 0.05$), while malondialdehyde (MDA) levels decreased ($p < 0.05$) [64]. Ulcerative colitis models further substantiate this synergy: β-glucan treatment increased intestinal glutathione (GSH) by 187%, elevated SOD by 115%, while suppressing NF-κB by 43% (all $p < 0.05$) [65].

Neuroprotective effects manifest through gut-brain axis modulation. β-glucan supplementation demonstrated potent anti-neuroinflammatory effects through gut-brain axis modulation. In the hippocampus, oat β-glucan significantly reduced microglial activation (Iba; $p < 0.05$) and suppressed pro-inflammatory cytokines TNF-α, IL-1β, and IL-6 mRNA (all $p < 0.05$). All three β-glucan types (mushroom, curdlan, oat bran) decreased microglial numbers in both hippocampus and prefrontal cortex (PFC; $p < 0.001$). While hippocampal IL-6 decreased across all groups ($p < 0.001$), the PFC showed differential responses-oat β-glucan increased IL-6 and IL-1β expression ($p = 0.027$ and $p = 0.016$, respectively) [21].

Colonic immune changes strongly correlated with cognitive enhancement. β-glucan promoted M2 macrophage polarization (CD206+ cells; $p = 0.001$) and enhanced anti-inflammatory IL-10 ($p = 0.005$). These changes positively correlated with cognitive performance ($r = 0.55$; $p < 0.01$) and negatively with hippocampal microglial activation ($r = -0.50$; $p = 0.01$) [21].

Synaptic restoration through molecular signaling pathways. β-glucan improved hippocampal PTP1B-IRS-pAKT-pGSK3β-pTau signaling, downregulating PTP1B and enhancing insulin signaling molecules (p-Akt, p-GSK3β; both $p < 0.05$). This translated to structural improvements-increased synaptic proteins (SYN, PSD95), enhanced postsynaptic density thickness, and elevated brain-derived neurotrophic factor (BDNF) in the PFC ($p = 0.011$) [18].

Network pharmacology revealed sophisticated mechanisms targeting PI3K-Akt signalling through synergistic action of oat bioactives and gut microbiota metabolites. Key molecular complexes showed exceptional binding affinities: myricetin-GSK3B (-10.6 kcal/mol), myricetin/quercetin-VEGFA (-8.2 kcal/mol), diosgenin-IL2 (-9.1 kcal/mol), and vestitol-NR4A1 (-9.0 kcal/mol). These compounds, derived from both Avena sativa and beneficial gut bacteria (*Escherichia spp.*, *Enterococcus spp.*, *Bacteroides spp.*, *Bifidobacterium dentium*), passed all drug-likeness parameters, highlighting the essential role of microbial metabolism in therapeutic efficacy [61].

Theme 2.2: Potential health benefits of oats mediated by gut microbiota - metabolic and cardiovascular effects

Oats demonstrate robust cholesterol-lowering effects across diverse populations through complex mechanisms involving gut microbiota modulation and metabolic pathway regulation. WGO (whole grain oats) interventions consistently yield significant reductions in TC and LDL-C, with a 6-week human trial showing TC decreased by 9.7% ($p = 0.0016$) and LDL-C by 5.6% ($p = 0.0055$) compared to non-whole grain (NWG) cereals, where paradoxically TC increased by 9.2% and LDL-C by 9.4% [41].

The dose-dependent relationship between oat consumption and lipid reduction manifests clearly in paediatric populations, where 8-week interventions with 108 g/day WGO and rye reduced LDL-C by ~6% ($p = 0.009$), high-density lipoprotein cholesterol (HDL-C) ratio by 6.7% ($p < 0.001$), and TAE by 17% ($p = 0.048$), with changes correlating to dietary fibre components including β-glucan, fructans, klason lignin, and arabinoxylans [66]. Similarly compelling results emerged from adult Chinese populations with mild hypercholesterolemia, where 80 g/day oat consumption for 45 days reduced TC by 8.7% and LDL-C by 9.1% (both $p < 0.001$) compared to rice controls, establishing oat's superiority over refined grains with TC reduction differences reaching statistical significance between groups ($p = 0.011$) [43].

Animal models provide mechanistic insights, showing oat-based foods containing 65% oat and 25% tartary buckwheat significantly reduced liver TC (13%), cholesteryl esters (16%), and TAE (21%) (all $p < 0.05$) while increasing faecal bile acid excretion by 11% ($p < 0.05$) and total SCFA ($p < 0.05$) by 60% compared to HFD controls [1].

The glycaemic benefits of oats complement their lipid-lowering effects, with a 4-week intervention using gastrointestinal microbiome modulators containing 2.5 g oat β-glucan improving glucose tolerance ($p = 0.008$), though these shorter interventions showed minimal impact on lipid parameters or faecal microbiota composition, suggesting threshold effects for different metabolic outcomes [70]. Composite formulations containing oats demonstrate significant cardiometabolic benefits in diabetic rat models, with buckwheat-oat-pea flour (BOP) at a 6:1:1 ratio exhibiting potent lipid-modulating effects. The low-dose BOP intervention significantly reduced total cholesterol to 1.29 mmol/L ($p < 0.05$) compared to diabetic controls at 1.70 mmol/L, representing a 24% reduction crucial for cardiovascular risk management. HDL-cholesterol levels were also significantly modulated (0.65; $p < 0.05$), while TAE levels showed favourable trends toward reduction. These lipid improvements were accompanied by enhanced hepatic function, with all doses significantly reducing alanine aminotransferase (ALT) levels ($p < 0.05$) and high-dose decreasing AST ($p < 0.05$), suggesting improved hepatic lipid metabolism and reduced fatty liver disease markers as evidenced by lower non-alcoholic steatohepatitis (NASH) scores ($p < 0.05$) [68].

In low-density lipoprotein receptor knock-out (LDLR−/−) atherosclerotic mice, 0.8% oat fibre supplementation for 14 weeks significantly reduced serum TC and LDL-C ($p < 0.05$), while HDL-C and TAE remained unchanged. Circulating lipopolysaccharide decreased ($p < 0.05$), indicating reduced systemic inflammation. Faecal SCFAs increased markedly-acetic acid by 28.9%, propanoic acid by 67.8%,

and butyric acid by 61.0% (all $p < 0.05$)-activating colonic SCFA receptors GPR41, GPR43, and GPR109A ($p < 0.05$) [29]. Oat flavonoids (50-100 mg/kg/day) administered to HFD-fed mice for 4 weeks produced dose-dependent improvements in lipid profiles, reducing serum TC, TAE, and LDL-C while increasing HDL-C (all $p < 0.05$), without affecting energy intake [20].

In healthy volunteers, acute intake of 5.2 g oat β-glucan significantly modulated postprandial metabolic responses and demonstrated that β-glucan-enriched breakfast decreased postprandial glucose area under the curve (AUC) by 9.5% ($p = 0.006$), accompanied by reduced in plasma insulin (18.8%; $p = 0.06$) and C-peptide (7.9%; $p = 0.001$). These changes were associated with decreased plasma ghrelin ($p = 0.030$) and increased gastric inhibitory polypeptide (GIP: 10.4%; $p = 0.035$) and pancreatic polypeptide (PP: 19%; $p = 0.018$), suggesting coordinated modulation of satiety and metabolic hormones [30]. Similarly, an acute oat consumption (40 g) significantly reduced postprandial insulin responses in healthy adults, with decreased AUC (25.6 vs. 42.0 pmol/L × min) and incremental AUC (iAUC: 17.8 vs. 29.6 pmol/L × min). C-peptide responses were also significantly attenuated (AUC: 599 vs. 750 ng/mL × min for control; all $p \leq 0.05$) [69].

In diabetic rat models, the glycaemic benefits of oat supplementation showed interesting dose-dependency patterns. A low-dose oat-buckwheat compound treatment for 11 weeks produced the most significant improvement in oral glucose tolerance test (OGTT-AUC: 28.74 vs. 48.29 mmol/L in control; $p < 0.05$), while higher doses showed diminished effects [3]. The intervention also significantly reduced insulin levels and HOMA-IR index compared to diabetic controls ($p < 0.05$), with treated rats showing markedly lower HOMA-IR than wheat flour-treated controls [3].

In Western diet-fed mice, both oat and rye bran supplementation improved glucose homeostasis, though the specific mechanisms differed. Oat bran supplementation significantly reduced fasting blood glucose levels from significantly and improved glucose tolerance ($p < 0.05$), although insulin sensitivity measured by insulin tolerance test remained unchanged [9]. The circadian disruption model provided additional mechanistic insights. Following 8 weeks of intervention, the oat β-glucan group showed significant overshoot effects in OGTT-AUC, with glucose tolerance improving beyond that of non-shifted controls [12].

Sustained oat consumption yields clinically meaningful blood pressure reductions through gut microbiota-mediated mechanisms. A randomized controlled trial (RCT) demonstrated that daily intake of 30g oat bran (providing 8.9g dietary fibre) combined with dietary guidance produced substantial improvements in hypertensive patients. After 3 months of intervention, office systolic blood pressure (BP) decreased by 15.3 mmHg ($p < 0.001$) and office diastolic BP decreased by 10.2 mmHg ($p = 0.028$) compared to controls [26]. The 24-h ambulatory BP monitoring revealed even more comprehensive benefits: maximum systolic BP decreased by 14.0 mmHg ($p = 0.002$), maximum diastolic BP decreased by 11.1 mmHg ($p = 0.001$), while average 24-h systolic and diastolic BP showed significant improvements ($p = 0.007$ and $p = 0.008$, respectively) [26]. Notably, this intervention also allowed for reduction in antihypertensive medication use ($p = 0.021$), with six patients in the oat bran group decreasing their medications compared to none in the control group [26].

The cardiovascular benefits extend beyond BP reduction. An 8-week intervention with a multifunctional diet containing oats, barley, and rye achieved remarkable improvements in cardiometabolic risk markers in overweight and obese subjects. The diet reduced TC by 26%, LDL-C by 34% (both $p < 0.0001$), and TAE by 16% ($p < 0.05$). Most significantly, this intervention achieved a 36% reduction in Reynold's cardiovascular risk score ($p < 0.05$), while the control diet showed no change in this comprehensive risk assessment metric [67].

Oat consumption's cardiovascular benefits appear mediated through gut microbiota modulation. Oat bran supplementation significantly increased beneficial bacteria including *Bifidobacterium* ($p = 0.019$) and *Spirillum* ($p = 0.006$), while altering β-diversity (Jaccard $p = 0.008$; Bray-Curtis $p = 0.004$). Similarly, oat-containing diets increased *Prevotella copri*, associated with improved glucose

tolerance, while *Faecalibacterium* negatively correlated with blood pressure. These microbial changes likely regulate BP through SCFA production and G protein-coupled receptor activation, mechanistically linking oat consumption to cardiovascular health [26,67].

Theme 2.3: Potential health benefits of oats mediated by gut microbiota - gut barrier and intestinal health

Human studies demonstrate that oat β-glucans strengthen intestinal barrier function through purity-dependent mechanisms. *In vitro* fermentation studies using human microbiota revealed that fermented oat β-glucan supplementation significantly elevated TEER (transepithelial electrical resistance) after 24-h-increasing from 114.9-123.0 for 28% purity oat β-glucan and from 110.7-116.3 for 94% purity (both $p < 0.001$)-with the lower purity variant achieving greater barrier enhancement. Under ethanol-induced basolateral stress conditions, where TEER decreased by ~50%, the 28% β-glucan formulation demonstrated protective effects, increasing TEER to 33.8 compared to 29.0 in stressed controls ($p < 0.01$), while the 94% purity variant showed no significant protective capacity. Similarly, under rhamnolipid-induced apical stress, only the 28% β-glucan effectively reduced Lucifer yellow permeability, suggesting that lower purity oat β-glucan with its accompanying matrix compounds may provide superior barrier protection [49].

Multiple studies confirm coordinated upregulation of barrier proteins across different disease models. In atherosclerotic mice, oat fibre increased the expression of tight junction proteins zonula occludens (ZO)-1 and occludin in the distal colon, while reducing circulating lipopolysaccharide levels [29]. Similarly, across cancer models, 1% oat β-glucan supplementation significantly elevated claudin-3 and claudin-4 expression in the large intestine, though claudin-7 expression was reduced in early-stage cancer. Further gene expression analysis revealed significant downregulation of Claudin-1 in cancer rats ($p < 0.01$), which was notably partially restored by oat supplementation [71]. Extending these barrier-protective effects to inflammatory conditions, DSS-induced colitis models demonstrated that oat and oat bran interventions significantly upregulated colonic epithelial barrier genes Claudin-1 and Claudin-5 ($p < 0.05$), with concurrent metabolic benefits where dietary inclusion of 30% oats significantly elevated acetic, propionic, and butyric acid concentrations compared with controls, thereby reinforcing barrier integrity through SCFA-mediated mechanisms [57].

Intestinal region profoundly influences fibre efficacy, with distinct tissue-specific responses observed across models. For instance, in poultry models, oat hull supplementation induced jejunal-specific morphological improvements: while goblet cell numbers per villus remained unchanged ($p > 0.05$), the total jejunal surface area occupied by goblet cells increased by ~42% ($p = 0.019$) and total villus surface area expanded by 22% ($p = 0.014$). In contrast, no significant effects were observed on ileal villus height, crypt depth, or villus-to-crypt ratio [73]. Similarly demonstrating regional heterogeneity, porcine models revealed dichotomous regional responses to 15% oat bran supplementation. Specifically, ileal tissues showed upregulation of anti-inflammatory IL-10 ($p < 0.05$) with increased NOD2 expression compared to pea-hull fibre, concurrent with suppression of TLR4-pathway mediators IRAK4 and TRAF6 vs. control ($p < 0.05$). Conversely, colonic tissues demonstrated contrasting responses with reduced ZO-1 expression ($p < 0.05$) but increased NF-κB-p65 protein abundance ($p < 0.05$), while barrier-related genes MUC1/2 were nonetheless enhanced. Collectively, these regional-specific effects suggest oat bran's impact on intestinal health operates through NOD-associated rather than TLR-associated pathways, with immune improvements concentrated in the ileum and barrier enhancements in the colon [74].

Oat β-glucans orchestrate comprehensive metabolic reprogramming that underpins barrier resilience through microbiota modulation. Specifically, in weaned rabbit models, oat β-glucan treatment altered 264 intestinal metabolites ($p < 0.1$), notably decreasing lysine derivatives, aromatic amino acid metabolites, and purine metabolites-pathways essential for gut barrier maintenance. This metabolic recalibration correspondingly paralleled significant microbial shifts, including increased abundance of beneficial bacteria (*Lactobacillus*, *Prevotellaceae* UCG-001, *Pediococcus*, *Bacillus*) and decreased pathogenic species [28]. Building on these metabolic benefits, the treatment with protected organic acids-essential oils blends plus 3% oat hulls significantly enhanced intestinal architecture-achieving a 23% higher duodenal villus height-to-crypt depth ratio compared to antibiotic treatment (23.8 vs. 19.4; $p < 0.05$), and 18% shallower crypt depth (97.9 vs. 119 μm; $p < 0.05$). Moreover, this coincided with significant goblet cell proliferation in the duodenum (score 2 vs 0

in controls; p = 0.002). Collectively, these improvements suggest that the combination of organic acids-essential oils with oat hull fibre operates through complementary mechanisms of epithelial repair and enhanced barrier function [72].

Extending to disease-specific applications, oat supplementation consistently demonstrated barrier-protective properties. For instance, DSS-induced colitis models showed that low-to-medium doses of oats (15-30%) and oat bran (10-20%) preserved colon length (7.38 vs. 5.43 cm; $p < 0.05$), maintained crypt architecture, and increased goblet cell populations. These structural improvements were accompanied by molecular changes: increased IL-10 secretion, decreased TNF-α and IL-6 levels ($p < 0.05$), and upregulated claudin gene expression [57]. Furthermore, transgenerational benefits emerged when maternal oat β-glucan supplementation (200 mg/kg bw) enhanced intestinal barrier function in dams through upregulation of tight junction proteins (ZO-1, occludin) and Mucin-2 expression ($p < 0.05$). Remarkably, this gut barrier enhancement was transmitted to offspring, with the soluble oat β-glucan group showing the highest caecal butyrate production and increased abundance of butyrate-producing bacteria (*Firmicutes* phylum, particularly *Lachnospiraceae* family). Consequently, these gut-brain axis modifications correlated with enhanced neurodevelopment, including increased BDNF and post-synaptic density protein 95 expression in 1-week-old pups' brains. Ultimately, the integrated gut barrier-microbiome improvements persisted, supporting superior spatial memory and cognition at 8 weeks compared to controls ($p < 0.05$) [60].

Despite these promising findings, in elderly populations, a 6-week intervention with 12g/day oat β-glucan failed to prevent acute indomethacin-induced intestinal hyperpermeability, despite 85% of participants having fibre intake below recommendations. This contrast highlights that protective mechanisms may be injury-specific rather than universally applicable [22].

Theme 3: Potential effects of other ingredients to oat

The metabolic and prebiotic functions of oats emerge not in isolation but through complex interactions within the dietary matrix, where co-ingredients act as either amplifiers or antagonists of oats' physiological benefits. This context-dependency fundamentally shapes how oats influence both host metabolism and gut microbial ecosystems.

Complementary grains create metabolic synergy beyond additive effects. The most striking demonstration emerges from HFD models, where an oat-buckwheat compound (buckwheat: oat ratio 2.2:1) achieved remarkable metabolic improvements. After 12 weeks of intervention, the compound significantly reduced serum TC and LDL-C, decreased TNF-α and IL-6 levels (all $p < 0.05$), and improved glucose tolerance with the OBC-L group showing significantly lower OGTT-AUC ($p < 0.05$). This amplification likely stems from buckwheat's total flavonoids (1.76g/100g) and resistant starch (0.39g/100g) working synergistically with oat β-glucan (4.19 g/100g), creating an optimized metabolic environment [64]. These synergistic metabolic benefits appear to be mediated through distinct microbiome modulation patterns, as revealed when examining each grain's individual effects. While oats supplementation elevated colonic butyrate 2.16-fold ($p < 0.05$), tartary buckwheat achieved 1.77-fold increase ($p < 0.05$), but foxtail millet showed no significant change ($p < 0.05$). Importantly, oats and tartary buckwheat significantly increased *Lactobacillus* and *Romboutsia* abundance, while foxtail millet promoted potentially unfavorable *Ruminococcaceae* and *Enterobacter* populations [64].

Mixed cereal blends reveal dose-dependent benefits in metabolic syndrome models. When 40% dietary inclusion of a coarse cereal mixture (millet, maize, oat, soybean, and purple potato) was administered to HFD-fed mice for 8 weeks, significant improvements materialized: body weight gain and fat accumulation decreased ($p < 0.05$), blood glucose tolerance improved, serum TC and LDL-C reduced while HDL-C increased (all $p < 0.05$), and the relative abundances of *Lactobacillus* and *Bifidobacterium* increased. The mixture promoted SCFA production, with acetate increasing by 25.56% and total SCFAs by 20.09% ($p < 0.05$) [6].

These beneficial metabolic effects appear consistent across different host species and experimental models. Cross-species fermentation patterns confirm grain-specific modulation. In canine models, rye supplementation significantly increased acetate ($p = 0.044$) and

propionate ($p = 0.018$) concentrations compared to oats alone, with butyrate levels highest after rye consumption [75]. Human *in vitro* fermentation models showed similar patterns, with rye substrate supporting 48% relative abundance of *Subdoligranulum* after 24-h fermentation compared to 1.6% with oats ($p < 0.005$), while maintaining higher butyrate production [31].

Glucose-rich vegetables may complement oat's prebiotic signature through synergistic fermentation pathways. *In vitro* infant fermentation models demonstrated that vegetables with high glucose content-pumpkin (231.0 μg/mg), sweetcorn (185.1 μg/mg), and carrot (96.1 μg/mg glucose)-exhibited low xylose/glucose ratios (0.02-0.07) and drove robust lactate production (5.0-8.7 μM/mL at 10h), with pumpkin achieving the highest lactate (8.7 μM/mL) and acetate formation (13.8 μM/mL) alongside maximal *Bifidobacterium* expansion (32.5%). These glucose-rich vegetables also stimulated beneficial *Clostridiales* families (*Ruminococcaceae* and *Lachnospiraceae*), enhancing overall microbial diversity-a pattern suggesting that combining such vegetables with oats could amplify prebiotic benefits through complementary substrate provision [79].

Xylose-rich fruits demonstrate contrasting fermentation dynamics that may modulate oat's metabolic effects. Apple, blackcurrant, and kiwifruit-characterized by moderate glucose levels (94.0-112.3 μg/mg) but elevated xylose/glucose ratios (0.14-0.17)-shifted metabolism toward propionate production (apple achieving 3.2 μM/mL, the highest among tested foods) while suppressing lactate formation (blackcurrant producing only 0.3 μM/mL, equivalent to control). These fruits preferentially stimulated *Veillonella* (blackcurrant reaching 35.9%) and Bacteroides while reducing bifidobacterial proliferation, suggesting that fruit-oat combinations might redirect fermentation away from oats' typical lactogenic-bifidogenic profile toward alternative metabolic pathways [79].

Human trials confirmed that carefully selected additions enhance rather than diminish oat's dual metabolic-microbiotic benefits. A 6-week (RCT) with WGO containing almonds and dried fruit achieved significant reductions in TC (9.7%; $p = 0.0016$) and LDL-C (5.6%; $p = 0.0055$), while the NWG showed opposite effects with TC and LDL-C increases 9.2% ($p = 0.0016$) and 9.4% ($p = 0.0055$) respectively. The between-treatment differences of 0.94 mmol/L for TC and 0.40 mmol/L for LDL-C were highly significant ($p = 0.0001$ and $p = 0.002$, respectively) [41].

Simultaneously, this nutrient-enriched oat matrix boosted faecal *Bifidobacterium* by 2.4-fold ($p < 0.0001$), *Lactobacillus* by 1.4-fold ($p = 0.001$), and total bacterial counts by 1.3-fold ($p = 0.008$), while the refined corn-based control actually decreased these populations. This demonstrates that strategic combinations-incorporating MUFA-rich almonds (12.5 vs. 10.4%; $p = 0.002$) and polyphenol/fibre-rich dried fruits-can synergistically enhance oats' cholesterol-lowering and prebiotic effects beyond what oats might achieve alone [41].

Antimicrobial interventions demonstrate variable interactions with oat-derived mucosal benefits. In 28-day broiler trials with 25% rapeseed meal challenge, lysozyme supplementation (40 mg/kg) significantly impaired jejunal architecture, reducing villus height from 1,354 μm to 1,076 μm ($p = 0.005$) and decreasing crypt depth from 433 μm to 307 μm ($p = 0.001$). Notably, birds fed oat hulls alone maintained superior villus height (1,558 μm), suggesting that antimicrobial agents may counteract the mucosal improvements typically associated with oat hull consumption. This antagonistic relationship indicates potential interference with oats' prebiotic mechanisms at the tissue level [73].

Protected organic acid-essential oil blends synergistically enhance gut architecture when combined with oat hulls, despite metabolic trade-offs. The combination of protected organic acids (fumaric, sorbic, malic, citric) with essential oils (thymol, vanillin, eugenol) plus 3% oat hulls (OEOH) significantly improved multiple intestinal parameters in 36-day trials. Birds receiving OEOH showed reduced duodenal crypt depth (97.9 vs. 119 μm in OE alone; $p < 0.05$), increased ileal villus height (1,588 vs. 928 μm in OE; $p < 0.001$), and enhanced villus-to-crypt ratio (15.4 vs. 9.8 in OE; $p < 0.001$). Additionally, relative gizzard weight increased substantially from 1.19%-1.67% of body weight ($p < 0.0001$), indicating enhanced mechanical digestion capacity [72].

However, these structural improvements came with energetic costs: birds fed OEOH achieved lower overall body weight gain (2,303 vs. 2,371g in OE treatment; $p = 0.01$), attributed to the ~91-100 kcal/kg reduction in metabolizable energy across feeding phases. This energy deficit suggests that while bioactive blend-oat hull combinations enhance gut morphology and potentially digestive efficiency, the reduced dietary energy density may limit growth performance under standard production conditions [72].

Forage addition accelerates fermentation while compromising ecological resilience. In equine caecal studies, hay/whole oats combinations (high nutrient availability) versus hay alone (low nutrient availability) produced complex fermentation shifts. The oat-supplemented diet reduces levels while increasing total volatile fatty acid concentration compared to hay alone. However, this enhanced fermentation came at an ecological cost: both microbial diversity (Simpson's index; $p < 0.01$) and temporal stability index ($p < 0.01$) were significantly reduced in horses fed the oat-containing diet. Notably, these changes occurred at caecal pH levels far from sub-clinical acidosis (>6.5), suggesting that enhanced fermentation velocity through increased nutrient availability fundamentally sacrifices gut microbiome ecological robustness, independent of pathological stress [76].

Moderate forage inclusion optimizes performance-antioxidant balance. Goat studies showed 30-70% alfalfa with oats maintained daily gain and improved feed conversion rate (FCR) by ~4% ($p = 0.033$) while enhancing antioxidants. However, 100% alfalfa caused 19% lower daily gain (63.84 g/day) and 30-36% poorer FCR ($p = 0.033$) despite unchanged intake ($p = 0.336$). Alfalfa inclusion dose-dependently improved meat antioxidants: T-AOC increased ~144-207% ($p = 0.050$) and MDA decreased 20% ($p = 0.035$). Microbiota analysis linked *Bacteroidales* unclassified with enhanced T-AOC while *Clostridium* (2 fold higher in oats) correlated with MDA, confirming 30-70% alfalfa optimizes oats' benefits whereas complete substitution impairs energy utilization [77].

The synbiotic combination of oat β-glucan (1 g/kg body weight) with probiotics (*Bifidobacterium animalis* and *Lactobacillus paracasei*) demonstrated enhanced metabolic benefits in a 12-week murine study. Compared HFD controls, the synbiotic group achieved superior outcomes: reduced weight gain (55-85%), decreased fasting insulin (1.2-0.55 μg/L), and improved cholesterol (185-155 mg/dL) (all $p < 0.05$). While oat β-glucan alone showed intermediate effects, the synbiotic combination outperformed individual components across multiple markers including HOMA-IR and hepatic steatosis. This enhanced efficacy demonstrates how probiotic bacteria amplify oat β-glucan's prebiotic properties through improved fermentation and SCFA production, maximizing oats' corrective capacity in metabolic dysfunction [78]. In contrast, a study found that a single 5.2g dose of oat β-glucan reduced appetite by 13% and improved glycemic control in healthy adults, but 3g/day for 3 weeks failed to alter gut microbiota. Higher fat and protein in the β-glucan meal may have influenced the increased Glucose-dependent Insulinotropic response [30].

Discussion

This systematic review showed important findings about how oat consumption affects gut bacteria. While the evidence clearly shows oats work as prebiotics, our analysis uncovers key conflicts between studying isolated components vs. whole foods that need to be resolved.

The consistent increase in beneficial bacteria across very different populations, from Mongolian adults [42] to ICU patients [45] to companion animals [51], seems to confirm oats works quite universally. The fact that 92.5% of human gut microbiomes have the genetic machinery to process β-glucan [47] provides a clear explanation. However, this raises an important question: if most people have this ability, why do we see such different clinical results? The answer reveals a basic misunderstanding in the field. The finding that just 1.3 g/day β-glucan-less than half the EFSA's 3 g recommendation-achieves both major bacterial changes (140% increase in Bifidobacterium) and meaningful cholesterol reduction [41] suggests that our dosing guidelines are based on trial and error rather than biological understanding. This gap between official recommendations and what actually works in the body shows a serious problem in how we translate research into practice.

Perhaps the most surprising finding is that medium-purity (28%) β-glucans work better than high-purity (94%) isolates for gut barrier protection [49,53]. This "purity paradox" challenges the common belief that pure compounds are always better. The huge increase in SCFA production when polyphenols stay within the fibre matrix (28 to 86 mmol/L) [33] cannot be explained by simply adding effects together-it shows that components work together in ways we do not fully understand. This finding has major implications for the supplement industry's focus on isolated compounds. The discovery of specialized enzymes like BuGH158 in *Bacteroides uniformis* [36] helps explain the mechanism, but more importantly, it shows that gut bacteria evolved to process whole foods, not pure compounds. The field's focus on isolating single components may actually remove the complex structure that provides health benefits-a warning for precision nutrition approaches.

Our analysis shows processing methods are the most overlooked factor affecting clinical outcomes-a "hidden variable" that explains much of the conflicting research. While heating and extrusion at 130°C increases water-extractable β-glucan (1.8%) and resistant starch (3.6%) [38] and enzyme treatment releases exceptional amounts of arabinoxylan (77.6 g/L) [59], these changes have both positive and negative effects.

The key finding comes from looking at different gut regions: the upper intestine (jejunum) consistently improves while effects in the colon vary [68,73]. This difference between gut regions shows a major flaw in current dietary guidelines that treat the gut as one uniform tube. The paradox-that processing that helps blood sugar control may harm long-term colon health [11,37,38]-suggests we may be focusing on short-term markers while missing long-term health effects. This raises difficult questions about whether food processing truly improves or actually reduces nutritional value.

The strong cardiovascular benefits (TC: 5.7-9.7%, LDL-C: 5.6-9.1% reductions) [41,43,66] contrast sharply with inconsistent blood sugar effects, which mainly appear in people with metabolic problems [3,9,30,69]. This difference suggests separate mechanisms: heart benefits may not depend on gut bacteria (possibly through binding bile acids), while blood sugar improvements need specific bacterial populations.

The link between *Faecalibacterium prausnitzii* levels and improved cholesterol [43] hints at personalized treatment potential, yet we lack tools to predict who will respond based on their gut bacteria. The failure of oats to protect against severe damage like NSAID-induced gut injury [22] further shows they work better for prevention than treatment which a distinction rarely made clear in marketing.

The strong influence of other foods challenges the idea of oats as standalone functional foods. The improved effects with buckwheat (62.5% greater lipid reduction) [3,64] versus the reduced effects with foxtail millet (53% less butyrate production) [3,64] shows that benefits depend on the entire diet, not just oats alone. This context dependency also applies to dosing patterns: a single 5.2g β-glucan dose improves metabolic markers, while 3 g/day for weeks fails to change gut bacteria [37]-suggesting occasional larger doses may work better than daily small amounts. The observation that high-fat diets block benefits [78] questions whether oats work within typical Western diets, suggesting that without broader dietary changes, oats provide limited help.

The discovery that only 80% of people with adequate *F. prausnitzii* can convert avenanthramides to their active form [34] shows the individual variation that undermines one-size-fits-all recommendations. Combined with person-specific fermentation responses [11,15], this variation demands a shift from universal guidelines to personalized approaches based on individual gut bacteria. Yet we lack the tools to implement such personalized nutrition. Without accessible gut microbiome testing and proven prediction methods, personalized oat interventions remain a goal rather than reality-showing the gap between what science knows and what medicine can deliver.

Age adds another layer to this personalization challenge. In suckling piglets, early-life oat β-glucan supplementation had minimal effects on gut microbiota and SCFA levels, which were mainly shaped by natural age-related maturation rather than the supplement itself

[80]. This demonstrates that developmental timing can override dietary interventions during critical growth windows. Just as we need to consider individual microbiomes, we must also recognize that infants, children, adults, and elderly may respond differently to oats based on their developmental stage-suggesting that age-specific dosing and timing strategies may be necessary for optimal benefits.

On the whole, this review reveals fundamental conflicts in nutrition science: between studying components versus whole foods, between processing innovation and maintaining food structure, between population guidelines and individual needs. The consistent finding that whole foods work better than isolated components [49,53] argues for recognizing food structure as being as important as nutrient content. In addition, the emerging evidence on the gut-brain connection [18,21] opens new treatment possibilities but also makes it harder to determine cause and effect. Does improved thinking come from direct brain effects, better metabolism, or stronger gut barriers? These uncertainties require more sophisticated research using multiple measurement techniques and better study designs.

This systematic review, while comprehensive, has important limitations. The search was restricted to PubMed and free-access articles, potentially narrowing the evidence base. Most included studies had small samples, short durations (typically 4-12 weeks), fixed doses, and limited mechanistic analyses-preventing assessment of long-term effects or dose-response relationships. The field also suffers from systematic biases including positive publication bias, potential industry influence, and overreliance on animal models with different gut structures than humans. Most critically, current research studies oats in isolation from real dietary patterns. Future research must embrace complexity by testing oat combinations within normal diets, mapping multi-level interactions, and conducting longer real-world trials. Until we move beyond studying isolated components to understanding how whole foods, gut bacteria, and human health interact as integrated systems, oats will remain incompletely understood as therapeutic tools.

The strengths of this systematic review lie in its methodological transparency and comprehensive thematic structure. The use of a PRISMA diagram ensures clarity, transparency, and accountability in the article selection and exclusion process (Figure 1), providing readers with a clear audit trail of study filtration. This review also organizes evidence from 80 primary studies into well-defined themes (Table 1). This structured synthesis not only supports a comprehensive discussion of oats' nutritional profile and health benefits but also provides practical insights for industry stakeholders and researchers exploring functional food applications. The inclusion of high-quality studies enhances the reliability of the findings. Notably, this review incorporates five RCTs, which represent the gold standard for dietary intervention studies [22,26,43,45,69]. Additionally, two randomized crossover trials provide robust within-subject comparisons, reducing individual variability [62,66].

Conclusion

This systematic review of 80 primary studies provides robust evidence that oat consumption beneficially modulates gut microbiota composition and metabolic function through multiple interconnected mechanisms. The universality of bifidogenic responses-observed across diverse populations from healthy adults to critically ill patients-combined with the widespread presence of mixed-linkage β-glucan utilization loci in 92.5% of human gut microbiomes, establishes oats as exceptionally effective prebiotics.

The key finding that medium-purity (28%) β-glucans outperform high-purity (94%) isolates challenges the prevailing reductionist paradigm, demonstrating that the synergistic architecture of the oat matrix creates emergent properties unattainable through isolated components. Processing methods emerge as critical determinants of clinical outcomes, with thermal-mechanical extrusion and enzymatic hydrolysis dramatically enhancing fermentable substrate availability while potentially compromising distal colonic delivery.

Clinically, oats demonstrate robust cardiovascular benefits with consistent cholesterol reductions (TC: 5.7-9.7%, LDL-C: 5.6-9.1%) and blood pressure improvements. The anti-inflammatory effects through cytokine modulation and gut barrier enhancement via tight junction upregulation position oats as therapeutic adjuncts for metabolic and inflammatory conditions. However, inter-individual variability, exemplified by distinct avenanthramide metabolizer phenotypes, underscores the potential for personalized nutrition approaches.

The profound influence of dietary context-with buckwheat amplifying and millet diminishing oats' benefits-reveals that therapeutic efficacy depends critically on the broader dietary matrix. Future research should prioritize whole-food approaches preserving structural complexity, establish dose-response relationships for specific health outcomes, and develop microbiome-based predictive models to optimize personalized oat interventions for maximum therapeutic benefit.

Supplementary Materials

Supplementary materials can be downloaded from https://bit.ly/Oats_GutMicro.

Conflict of Interest

The authors declare no conflict of interest.

Acknowledgements

The authors wish to thank the institute, Management Development Institute of Singapore, for its support towards this work. The cost of publication fees was borne by the authors.

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