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Health Benefits of Oats – a Review of Cardiovascular, Glycemic, and Gut Effects, and more

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ABSTRACT

Oat (Avena sativa L.), particularly as oatmeal, is distinguished by its high β-glucan content, a soluble dietary fiber with proven therapeutic potential. Meta-analyses and randomized controlled trials confirm that regular intake of oats or β-glucan-enriched products lowers total and LDL-cholesterol without significantly affecting HDL-cholesterol or triglycerides, while also improving anthropometric measures in individuals with mild metabolic disturbances. Additionally, β-glucan slows glucose absorption, reducing postprandial glycemia and long-term glycemic control markers like HbA1c and HOMA-IR in type 2 diabetes patients, as well as stabilizing glucose variability in type 1 diabetes. Oats favourably modulate gut microbiota, boosting short-chain fatty acid (SCFA) production, beneficial bacterial abundance, and intestinal barrier integrity, thereby supporting the gut-metabolism axis. Preliminary data also suggest benefits for satiety, inflammation reduction, and cognitive function. Despite promising results, heterogeneity in β-glucan dose, form, and intervention duration highlights the need for further high-quality studies, including long-term trials with well-characterized oat preparations.

Keywords: Oat, β-glucan, oatmeal, cardiovascular health, LDL-cholesterol, glycemic control, postprandial glucose, type 2 diabetes, gut microbiota, short-chain fatty acids (SCFAs), cardiometabolic risk, intestinal barrier

1. Introduction

Oat (Avena sativa L.) is a unique cereal crop distinguished by its multifunctional nutritional profile, particularly its high content of β-glucan, a soluble dietary fiber with a significant therapeutic potential. Oat is distinct among the cereals due to its multifunctional characteristics and nutritional profile. It is a good source of dietary fiber, especially β-glucan, minerals and other nutrients, while whole oat groats contain high amounts of valuable nutrients such as soluble fibers, proteins, unsaturated fatty acids, vitamins and phytochemicals. Oat bran in particular is a good source of B-complex vitamins, protein, fat, minerals and heart-healthy soluble fiber β-glucan, and the dietary fiber–phytochemical complex [1]. Oats are regarded as a cardioprotective food because their soluble fiber, particularly β-glucan, lowers total and LDL cholesterol while leaving HDL cholesterol and triglycerides largely unchanged. This lipid-modifying effect is reflected in international dietary guidelines and authorized health claims that emphasize oats as a useful component of strategies to improve the lipid profile and reduce cardiovascular risk [2]. Beyond lipid lowering, oats appear to influence glycemic control

and type 2 diabetes (T2D) risk, largely through the viscosity-forming properties of oat β-glucan that slow glucose absorption and attenuate postprandial glycemic and insulinemic responses. This glycemic effect supports the use of oat-based foods as a dietary component in strategies aimed at improving glucose homeostasis and reducing long-term T2D risk [3]. Oats also contribute to gut health, adding a third dimension to their cardiometabolic benefits beyond lipid and glycemic effects. Regular consumption of oat-based foods has been associated with modest improvements in blood lipids accompanied by favorable shifts in gut microbiota and increased production of short-chain fatty acids, supporting the concept that oatmeal acts as a complex food matrix that beneficially influences the gut–metabolism axis [4].

Taken together, these properties position oatmeal as a convenient, evidence-based dietary option for the prevention and adjunctive management of cardiometabolic diseases. This narrative review summarizes current evidence on the cardiovascular, glycemic, and gut-related effects of oatmeal.

2. Cardiometabolic Effects of Oats: Lipids, Blood Pressure, and Inflammatory Markers

Long-term intake of oats or oat bran has been most consistently linked with improvements in blood lipids, particularly total and LDL cholesterol. In a systematic review of sixty-nine intervention studies, just over half of the trials reported significant reductions in total cholesterol and LDL-cholesterol, whereas effects on HDL-cholesterol and triglycerides were infrequent. By contrast, only a small minority of studies showed any lowering of blood pressure, and the few trials that assessed insulin sensitivity or inflammatory markers did not detect meaningful changes with long-term oat consumption [5]. These food-based findings have been refined by more recent quantitative syntheses of oat supplementation trials, which confirm that oat-based interventions in adults with mild metabolic disturbances lower total and LDL cholesterol and modestly improve anthropometric indices such as BMI, body weight and waist circumference. In this meta-analysis, oat supplementation compared with non-oat control diets reduced total cholesterol by about 0.42 mmol/L and LDL-cholesterol by about 0.29 mmol/L, alongside small but significant decreases in body weight, BMI and waist circumference [6]. To better quantify the lipid-lowering contribution of the key active component, β-glucan, recent analyses have focused on isolated oat β-glucan in hypercholesterolemic adults. A meta-analysis of thirteen randomized controlled trials demonstrated that dietary oat β-glucan significantly reduces total and LDL-cholesterol without consistently altering HDL-cholesterol or triglycerides, and that the magnitude of this effect depends on disease severity, daily β-glucan dose, source (foods vs added β-glucan) and intervention duration [7]. Beyond effects on circulating lipids, some clinical

data suggest that oat β-glucan may also modulate nitric oxide metabolism. In a randomized controlled trial in hypercholesterolemic patients, 4-week consumption of β-glucan-rich oat bread led to a significant increase in serum nitric oxide concentration within the oat group, whereas no significant change was observed with a wheat-fiber control bread. Flow-mediated dilation did not differ between diets, but the authors noted that nitric oxide itself can be considered an indicator of endothelial function and called for further studies to clarify the vascular relevance of this response [8]. Beyond classical lipid endpoints, recent work has examined whether oats can modulate age-related systemic chronic inflammation. In a double-blind, placebo-controlled trial in adults at risk for cardiovascular disease, a β-glucan–containing oat product lowered a composite biomarker of systemic chronic inflammation, the Inflammatory Age (iAge), but only in participants who had elevated iAge and LDL cholesterol at baseline. In this high-risk subgroup, reductions in iAge were detectable as early as two weeks and were not observed in the rice-based control group, and appeared to be largely driven by a decrease in circulating CCL11 (Eotaxin-1), an aging-related chemokine, suggesting that oat constituents may specifically target this inflammatory axis independently of sex, BMI or chronological age [9].

3. Glycemic Effects of Oats: Postprandial Response and Long-Term Glycemic Control

Oat β-glucan intake consistently lowers blood glucose and insulin spikes after carb-rich meals. A meta-analysis of 103 trials found 23% drops in glucose area-under-curve, 28% in glucose peaks, and similar insulin reductions, working in healthy patients and those with diabetes. Each extra gram of β-glucan per 30g carbs cuts the glucose response by about 8-9%, especially with high-molecular-weight forms. Solid evidence backs using 3g+ β-glucan per 30g carbs for meaningful 20%+ glucose reductions [10]. While acute effects of oat β-glucan on postprandial responses are well-established, longer-term benefits in diabetes management are also emerging. A meta-analysis of 8 RCTs in 407 adults with type 2 diabetes showed that eating oats, oat bran, or β-glucan-enriched foods (like porridge, cereal, bread, median 3.25 g β-glucan over 4.5 weeks) reduced HbA1c by 0.47%, fasting glucose by 0.75 mmol/L, 2h-postprandial glucose by 0.42 mmol/L, and HOMA-IR by 0.88, with non-significant insulin reductions. Participants were mostly middle-aged, overweight, on meds or insulin, with moderate diabetes control. A linear dose-response cut fasting glucose by 0.39 mmol/L per gram β-glucan [11]. An earlier meta-analysis in T2DM patients confirms similar benefits. A meta-analysis of 4 RCTs found 2.5-3.5 g/day oat β-glucan for 3-8 weeks reduced fasting glucose by 0.52 mmol/L and HbA1c by 0.21%. No significant effect on fasting insulin concentrations was observed. Benefits were

linked to β-glucan's viscosity, which depends on dose and molecular weight [12]. Oat β-glucan also benefits type 1 diabetes (T1D) patients by stabilising blood glucose swings. In a crossover study of thirty adolescents with type 1 diabetes using CGM (Continuous Glucose Monitoring), adding 6 g/day via oat flakes to meals lowered mean glucose to 137 mg/dL (from 152), peaks to 243 mg/dL (from 275), and nighttime/daytime levels, while raising minimums to 65 mg/dL (reducing hypoglycemic risk). Peaks were blunted and delayed, with lower variability. 3 g/day showed minimal change, confirming dose-dependency even in T1D [13]. Oat processing influences glycemic and insulinaemic responses, with less processed forms eliciting lower blood glucose peaks and overall exposure. In a randomised crossover trial showed steel-cut oats (minimally processed) producing the lowest glucose peak-rise and iAUC (incremental area under the curve) compared with instant oats, Honey Nut Cheerios and Cream of Rice control. Old-fashioned large-flake oats showed intermediate effects, outperforming highly processed options but not matching steel-cut. Insulin responses followed a similar pattern, with iAUC significantly lower after steel-cut oats than instant oats, though peak insulin rises were comparable across meals. These differences arise because minimal processing preserves oat structure and β-glucan viscosity, slowing gastric emptying, starch digestion and glucose absorption in the gut [14].

4. Gut Health Effects of Oatmeal: Modulation of Microbiota, Short-Chain Fatty Acids, and Intestinal Barrier Function

Long-term consumption of oats or oat products has been consistently associated with favorable modulation of the gut microbiota, increased production of short-chain fatty acids (SCFAs), and enhanced intestinal barrier integrity. In an integrative review of 16 studies, oat intake promoted the abundance of beneficial taxa such as *Akkermansia muciniphila*, *Roseburia, Lactobacillus*, *Bifidobacterium*, and *Faecalibacterium prausnitzii* across models, while β-glucan fermentation yielded acetate, propionate, and butyrate as key metabolites. Human RCTs, including one with 210 mildly hypercholesterolemic subjects consuming 80 g/day oats for 45 days, demonstrated shifts in microbiota composition correlating with elevated plasma SCFAs and improved lipid profiles. Animal studies on high-fat diets further revealed oat bran or fiber increasing tight junction expression, mucin production, and reductions in inflammatory markers like TNF-α and IL-6 [15]. Similar findings were reported in a microbiota analysis from another RCT (n=28 hypercholesterolemic subjects, 80 g/day oatmeal for 45 days), which observed a characteristic 9.8% increase in Firmicutes (the dominant bacterial phylum encompassing SCFA-producing genera like *Roseburia*, *Faecalibacterium prausnitzii*, and *Lactobacillus*) along with *Blautia*

genus enrichment, both correlated with reductions in TC, LDL-C, and apoB, without changes in diversity. This highlights how specific taxonomic shifts within Firmicutes drive oatmeal's lipid-lowering effects via enhanced β-glucan fermentation to butyrate. These data complement the integrative review of 16 studies, positioning Firmicutes/Blautia alongside *Akkermansia muciniphila* and Bifidobacterium as key mediators in the microbiota-lipid axis with oat consumption [16]. In a small study of ten healthy adults consuming 60 g oatmeal porridge daily for 1 week, microbial fermentation capacity remained unchanged, as evidenced by stable lactulose-induced gas production and faecal SCFA excretion. However, faecal β-galactosidase activity significantly decreased from 14,6 to 5,3 mU/ml, and urease levels dropped from 4,5 to 3,7 mg/ml, suggesting adaptive shifts in microbial enzymatic functions toward reduced lactose and urea breakdown. Rectal PGE_2 levels also trended downward, hinting at potential anti-inflammatory effects on the host mucosa. These findings indicate that oatmeal modulates gut microflora-associated characteristics (MAC) beyond compositional changes, potentially conferring prebiotic benefits by suppressing urease-related ammonia production [17]. In atherosclerosis mice fed high-fat diet, oat fiber boosted faecal SCFAs (acetate, propionate, butyrate) and increased gut microbiota diversity. It shifted composition toward more *Actinobacteria*, *Coriobacteriaceae*, *Eisenbergiell*a, and *Romboutsia* with less *Rikenellaceae*, *Anaerotruncus*, and *Parabacteroides*. Enhanced tight junctions and SCFA receptors reduced serum lipopolysaccharide levels, attenuating brain inflammation and improving cognition [18]. Complementing these observations, supplementation with oat bran in mice for 17 weeks reduced body weight gain, hepatic inflammation, and impaired glucose tolerance while restoring cecal SCFAs to levels comparable to the standard diet control group. Gut microbiota shifted with higher Bacteroidetes and Lactobacillus abundance and lower Firmicutes and Proteobacteria proportions, enhancing tight junction expression. Oat bran increased ileal FXR-antagonistic bile acids, promoting hepatic bile acids synthesis and faecal excretion while shifting tryptophan metabolism toward anti-inflammatory indoles. These microbiota-mediated changes underscore oat bran's gut-liver axis protection against Western diet metabolic damage[19].

5. Additional Health Outcomes of Oats

Oatmeal demonstrates satiety-enhancing properties that support weight management beyond its cardiometabolic benefits. In a randomized trial with 48 healthy adults, instant oatmeal (higher β-glucan viscosity) significantly increased fullness, reduced hunger, desire to eat, and prospective intake compared to an oat-based ready-to-eat cereal. These effects persisted up to

240 minutes post-meal, translating to 81 kcal lower ad libitum lunch energy intake, primarily via reduced fat and protein consumption. Mechanistically, oatmeal's superior β-glucan molecular weight, radius of gyration, and gastric viscosity delayed digestion and prolonged nutrient exposure. Such acute appetite suppression positions hydrated oatmeal as a practical dietary tool for sustained energy balance in free-living settings [20].

Beyond metabolic and appetite-related effects, oat β-glucans show promise in the management of gastrointestinal inflammation, particularly chronic gastritis. In a randomized trial involving 48 patients with histologically confirmed chronic gastritis, 30-day oral supplementation with purified oat β-glucan induced molar mass-dependent improvements across immunological, antioxidant, and gut microbial parameters. Notably, the high molar mass fraction reduced mucosal lymphocyte and neutrophil infiltration scores, indicating attenuation of the inflammatory cascade at the tissue level. Both β-glucan fractions significantly decreased glutathione peroxidase and reductase activities and elevated fecal concentrations of acetic, propionic, and hydroxybutyric acids, reflecting enhanced colonic fermentation and shifts in short-chain fatty acid profiles. Importantly, no adverse hematological, biochemical, or clinical events were recorded. These findings position oat β-glucans not only as a cardioprotective dietary fiber but also as a potential adjunctive nutritional therapy in chronic inflammatory conditions of the upper gastrointestinal tract [21].

Oat extracts exhibit potential cognitive-enhancing effects in healthy adults. A systematic review of six RCTs involving 287 participants found that acute ingestion of oat extracts positively influenced accuracy and speed of cognitive performance across attention, memory, and executive function tasks. Four studies consistently showed improvements in global cognitive speed and accuracy. Short-term chronic supplementation significantly enhanced overall cognitive function, though long-term effects were inconsistent. Evidence quality was rated average, supporting acute benefits but calling for more high-quality, long-term trials due to dose variability [22].

Oat β-glucan supplementation alleviates non-gastrointestinal symptoms in healthy adults. In a double-blind RCT with 191 adults, consuming 3 g/day oat β-glucan daily for 4 weeks reduced the prevalence of exhaustion and fatigue versus baseline. Headache severity was significantly lower on oat β-glucan than control at 2 weeks, with anxiety trending lower and feeling cold reduced at 4 weeks. Fatigue and hot flashes severity decreased at 4 weeks, limb/joint pain at 2 weeks, and difficulty concentrating at both timepoints on oat β-glucan versus baseline. These

preliminary findings suggest oat β-glucan modulates affective and physical states potentially via gut-brain axis influences on inflammation and oxidative stress [23].

6. Conclusion

Oatmeal emerges as a convenient dietary vehicle for delivering oat β-glucan and other bioactives that jointly improve key cardiometabolic risk markers. Across randomized trials and meta-analyses, regular consumption of oats, oat bran, and β-glucan-enriched foods lowers total and LDL cholesterol, often with modest concurrent reductions in body weight, BMI, and waist circumference, while leaving HDL cholesterol and triglycerides largely unchanged. Acute and short-term interventions further demonstrate clinically relevant attenuation of postprandial glycemic and insulinemic excursions when sufficient doses of high–molecular-weight β-glucan are provided, with emerging evidence that longer-term intake can modestly improve HbA1c, fasting glucose, and insulin resistance indices in type 2 diabetes and stabilize glycemic variability in type 1 diabetes.

In parallel, both human and animal studies indicate that oatmeal and oat β-glucan modulate the gut ecosystem, promoting SCFA-producing taxa, increasing acetate, propionate and butyrate production, and enhancing epithelial barrier function and tight-junction expression. These microbiota-mediated effects align with observed improvements in lipid profiles, inflammatory tone, and host metabolic resilience, supporting the concept of a gut–liver–brain axis through which oats exert part of their health impact. Beyond classical cardiometabolic outcomes, preliminary trials suggest that oat-based interventions may enhance satiety and reduce subsequent energy intake, alleviate symptoms in chronic gastritis, and acutely improve selected cognitive performance domains, with additional signals for reduced fatigue, headache severity, and other non-specific somatic complaints, potentially via anti-inflammatory and gut–brain pathways.

Despite these encouraging findings, several limitations temper the strength and generalizability of the current evidence. Many trials are small, of short duration, and heterogeneous in terms of β-glucan dose, molecular weight, food matrix, and background diet, which complicates precise dose–response modelling and identification of minimal effective intakes for different outcomes. Cardiovascular and glycemic benefits are better established than effects on blood pressure, low-grade systemic inflammation, body weight regulation, cognitive function, and clinical endpoints such as incident CVD, T2D, and mortality, where results remain mixed or insufficient. Future research should prioritize longer, adequately powered randomized

controlled trials using well-characterized oat preparations, standardized reporting of β-glucan physicochemical properties, and integrated cardiometabolic, microbiome, and patient-reported outcomes, as well as large prospective cohorts and mechanistic studies to clarify causal pathways. Collectively, current data support recommending oatmeal as an evidence-based component of cardiometabolic risk reduction and gut-health strategies, while underscoring that more rigorous and mechanistically oriented studies are needed to fully define its therapeutic potential.

DISCLOSURE

Author's contribution

Conceptualization: B.Wróbel; methodology: B.Wróbel; check: J.Klonowska; formal analysis: L.Wójcik; investigation: M.Filipski; resources: M.Filipski; data curation: S.Kosek; writing - rough preparation: K.Ptaszkiewicz; writing - review and editing: K.Ptaszkiewicz; visualization: L.Wójcik; supervision: S.Kosek; project administration: J.Klonowska; receiving funding- no specific funding.

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