

DOSSIER · SUBSTANCE

Beta-Glucans

Beta-Glucans are **polysaccharides** from cell walls of fungi, yeasts, cereals and algae. Research focuses on **metabolic and immune modulation**, yet the transferability of laboratory results to clinical applications remains limited.

1,533

STUDIES IN
TOTAL

267

IN HUMANS

82

OF WHICH
CONTROLLED

217

IN ANIMALS

245

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Beta-Glucans at a Glance

ORIGIN:

Beta-glucans are homopolymeric glucose chains with β -glycosidic bonds that occur as polysaccharides in the cell walls of fungi, yeasts, cereals (e.g. oats, barley) and algae. The structure varies according to source: cereal beta-glucans predominantly exhibit β -1,3- and β -1,4-bonds, while fungal beta-glucans show β -1,3- and β -1,6-branching. These differences influence solubility, viscosity and biological activity, with bioavailability being decisive for the effect.

RESEARCH SITUATION:

1,533 evaluated works · 82 controlled human studies · 185 observational studies · 118 review articles · 217 animal studies · 203 cell-culture studies

STRONGEST INDICATIONS:

- **Metabolism:** Oat beta-glucans significantly lowered total cholesterol (by ~0.24 mmol/L) and LDL-cholesterol (by ~0.27 mmol/L) in controlled human studies, as shown by a meta-analysis with 927 participants. The effect was dose-dependent, yet data on optimal duration are lacking.
- **Immune system:** Fungal beta-glucans (e.g. lentinan) improved response rates in oncology patients when combined with chemotherapy (56.9 % vs. 43.3 %) and reduced side-effects such as leukopenia. Mechanistically, activation of T-cells and macrophages via receptors such as Dectin-1 was discussed.
- **Gut and microbiome:** Oat beta-glucan (3 g/day) lowered LDL-cholesterol by up to 15.1 % in an 8-week study and influenced postprandial satiety hormones (e.g. GLP-1). The viscosity of the beta-glucans modulated fermentation in the colon, yet systematic microbiome analyses were absent.
- **Inflammatory processes:** Beta-glucans from *Aureobasidium pullulans* increased CD4+ and CD8+ T-cells and lowered the neutrophil-lymphocyte ratio in a pilot study with COVID-19 patients. In-vitro data showed epigenetic effects ("trained immunity") in monocytes, yet clinical long-term data were lacking.
- **Glycaemic control:** Oat beta-glucan reduced postprandial glucose AUC by up to 23 % and insulin AUC by 28 % in acute studies, depending on viscosity. High-molecular-weight beta-glucans lowered fasting glucose, yet long-term effects remained unclear.

LIMITATIONS:

- The studies on beta-glucans are heterogeneous: differing sources, dosages and application forms complicate comparisons. Individual controlled human studies are often small; long-term data are lacking.
- Mechanistic works (e.g. on "trained immunity") provide indications, yet their clinical relevance remains an open question. Standardisations (e.g. molecular weight, purity) are insufficient.
- Preclinical results (animal studies, cell cultures) supply indications of mechanisms of action, yet their transferability to humans has not been examined, particularly in oncological or inflammatory diseases. Placebo-controlled RCTs with clear endpoints are absent.

CLASSIFICATION:

Beta-glucans are structurally diverse polysaccharides from fungi, cereals and algae whose immunomodulatory and metabolic effects have been demonstrated in controlled studies. The clinical relevance of these effects is an open research question, particularly with regard to long-term action and standardisation.

1. What Beta-Glucans Are

Beta-glucans are **homopolymeric glucose chains** with β -glycosidic bonds. They occur in **oats, barley, fungi (e.g. shiitake, maitake), yeasts and certain algae**. The structure varies according to source: cereal beta-glucans (e.g. from oats) consist predominantly of **β -1,3- and β -1,4-bonds**, whereas fungal beta-glucans (e.g. lentinan) exhibit **β -1,3- and β -1,6-branching**. These differences influence **solubility, viscosity and biological activity**.

Bioavailability is decisive for research. Beta-glucans from cereals are **barely absorbed in the small intestine** and instead reach the **colon**, where they are fermented by the microbiota. Fungal beta-glucans, by contrast, become **partly immunomodulatory** because they can be taken up via **M-cells in the gut**. **Molecular mass and branching patterns** determine whether they **increase viscosity in the intestine** or **activate immunological receptors** (e.g. Dectin-1, TLR). This diversity complicates the comparability of studies.

2. What the Studies Show in Detail

The key human studies and the most important reviews — by application area, with population, duration, preparation, measured variable and result as reported by the original works. Numbers appear only where the work states them.

Distribution of **controlled human studies** by application area — the numbers are not additive, as one work may be assigned to several areas:

APPLICATION AREA	CONTROLLED STUDIES
Safety and tolerability	33
Metabolism	33
Immune system	27
Gut and microbiome	22
Oncology	21
Inflammatory processes	18

APPLICATION AREA	CONTROLLED STUDIES
Heart and circulation	15
Fatigue and stress	11

Inflammatory Processes

Primary endpoints were fasting lipid values (total cholesterol, LDL, HDL, triglycerides), glucose, insulin, homocysteine and the inflammatory marker C-reactive protein (CRP). In the controlled study beta-glucan significantly lowered total cholesterol by 0.3 ± 0.1 mmol/L and LDL-cholesterol by 0.3 ± 0.1 mmol/L versus baseline, with the LDL reduction also significantly greater than in the control group. CRP values showed no significant change. An accompanying in-vitro fermentation model with human stool inoculum compared the fermentability of beta-glucan with inulin and guar gum, yet provided no direct conclusions for the in-vivo results. A limitation is the absence of a detailed analysis of the gut microbiota or inflammatory signalling pathways, and the short intervention duration limits statements on long-term effects ¹.

A randomised pilot study in 24 COVID-19 patients (8 per group) examined over 30 days the effect of beta-glucans from two *Aureobasidium pullulans* strains (AFO-202 and N-163) in addition to standard therapy. The groups received either standard therapy alone, standard therapy plus AFO-202 beta-glucan, or a combination of AFO-202 and N-163 beta-glucan. Primary endpoints were inflammatory markers (IL-6, D-dimer, CRP) and immunological parameters (CD4+, CD8+ T-cells, neutrophil-lymphocyte ratio [NLR]). The combination therapy showed a significant increase in CD4+ and CD8+ T-cells as well as a reduction in NLR. CRP remained unchanged in all groups. Limitations include the small sample size and the lack of a placebo control for the beta-glucan groups; in addition, no molecular mechanisms were investigated ¹⁵.

Several preclinical studies on human monocytes and macrophages show that beta-glucans (particularly from *Candida albicans* or yeast) induce an epigenetic reprogramming referred to as "trained immunity". In a 2014 study, monocytes were differentiated in vitro into naive, tolerised or trained macrophages and their transcriptome/epigenome analysed. Beta-glucan training led to a specific epigenetic signature with increased expression of genes of the inflammatory and metabolic signalling pathways (e.g. reduced inflammasome activation). A follow-up study (2016) showed that beta-glucan reverses LPS-induced immunological tolerance in monocytes by reactivating the deposition of active histone marks at promoters of tolerised genes. Ex-vivo experiments with monocytes from subjects after experimental endotoxaemia confirmed preclinically that beta-glucan restores cytokine production. A clinical effect in humans has therefore not been investigated.

HYPOTHESIS The epigenetic effects of beta-glucans could modulate the responsiveness of the innate immune system in the long term, particularly in chronic inflammation or susceptibility to infection. Clinical studies that directly link these mechanisms to inflammation-associated diseases (e.g. rheumatoid arthritis) are still lacking. Long-term studies in humans with clear clinical endpoints (e.g. disease activity, relapse rates) and a systematic analysis of the dose-response relationship remain to be clarified.

Gut and Microbiome

low viscosity (reduced by beta-glucanase treatment). The beverages each contained 10.2 g of dietary fibre, including 5 g of beta-glucan. Primary endpoints were postprandial satiety hormones (ghrelin, GLP-1, peptide YY, cholecystokinin) and glucose/insulin responses. At the same time, gastric emptying (measured via paracetamol absorption) was accelerated ($p = 0.034$). The study suggests that the viscosity of beta-glucan influences hormonal satiety regulation, possibly through different fermentation patterns in the gut. A limitation is the acute study design and the absence of a microbiome analysis ².

In the BELT study (2020), an 8-week, double-blind, placebo-controlled crossover study with 83 Italian subjects with moderate hypercholesterolaemia, the effect of 3 g/day oat beta-glucan on lipid levels, fasting glucose and subjective gut well-being was investigated. Beta-glucan lowered LDL cholesterol by 12.2% after 4 weeks (95% CI: -15.4 to -3.8) and by 15.1% after 8 weeks (95% CI: -17.8 to -5.9) versus baseline ($p < 0.01$ vs. placebo). Total cholesterol and non-HDL cholesterol showed similar reductions (8.9% and 12.1% after 8 weeks). Fasting glucose and gut well-being remained unchanged. The study confirms the lipid-lowering effect of beta-glucan but provides no data on microbiome composition or metabolic signalling pathways. A limitation is the short duration and the absence of a dose-response analysis ⁶.

A randomised study in 22 healthy subjects (11 women, 11 men) compared the influence of yeast-baked oat bread with low- (217 kDa) versus high-molecular-weight (797 kDa) beta-glucan (6 g/day each) on lipid levels, insulin and glucose over 3 weeks. Both breads showed no significant differences in the measured parameters; however, after consumption of the bread with high-molecular-weight beta-glucan, fasting insulin rose by 22.6% ($p < 0.03$) and fasting glucose fell by 3.4% ($p < 0.05$) versus baseline. The results suggest that the molecular mass of beta-glucan in baked goods plays no decisive role for the metabolic effects under real dietary conditions.

A randomised study in 12 healthy subjects acutely investigated the effect of yoghurt with muesli containing 4 g oat beta-glucan on gastric emptying rate (via ultrasound) and postprandial glucose responses. After this meal the glucose response was significantly lower ($p = 0.045$) than after a maize-flake control; however, no significant difference in gastric emptying or satiety was observed. The study showed a lower glucose response after the meal containing beta-glucan muesli but provides no data on long-term effects or microbiome changes. A limitation is the acute design and the lack of standardisation of the meals ¹¹.

What is missing: Systematic studies on the interaction of beta-glucans with the gut microbiota, particularly on the production of short-chain fatty acids (SCFA) and their influence on inflammatory markers or metabolic endpoints.

Oncology

Lentinan in combination with chemotherapy (dose not uniformly stated) increased the overall response rate from 43.3% (chemotherapy alone) to 56.9% (combination therapy; $p < 0.001$, 95% CI: 0.102–0.170). The pooled risk ratio (RR) for efficacy was 0.79 (95% CI: 0.74–0.85) in favour of combination therapy. The studies also showed an improvement in quality of life and a reduction in chemotherapy-related side effects (e.g. leukopenia). Limitations are the heterogeneity of the study protocols (different chemotherapy regimens, lentinan doses) and the absence of survival data in many studies. The effect of combination therapy was investigated, not that of lentinan alone ³.

The primary endpoint was overall survival (OS). Combination therapy showed no significant OS advantage (13.8 vs. 9.9 months; $p = 0.208$) but prolonged time to treatment failure (TTF: 4.3 vs. 2.6 months; $p < 0.001$). In a subgroup with $\geq 2\%$ lentinan-binding monocytes, a survival benefit was observed in patients with more than two chemotherapy cycles. The study suggests an advantage of combination therapy for time to treatment failure but provides no data on molecular mechanisms or biomarkers for patient selection ¹².

A 2012 review summarises the antitumour activity of various fungal polysaccharides, including beta-glucans such as lentinan, schizophyllan and grifolan. For these substances the mechanisms include activation of T cells, NK cells and macrophages as well as induction of cytokines (e.g. IL-12, IFN- γ) and nitric oxide. Beta-glucans bind to receptors such as Dectin-1 or TLR-2 and trigger a Th1-dominated immune response. Clinical data from Asia show efficacy for fungal beta-glucans as adjuvants in gastric, lung and colorectal carcinoma; however, large placebo-controlled studies in Western populations are lacking. A limitation is the heterogeneity of beta-glucan structures (molecular mass, degree of branching) and the lack of standardisation of treatment protocols ²³.

HYPOTHESIS Beta-glucans could enhance the efficacy of checkpoint inhibitors or other immunotherapies through their immunomodulatory properties. However, clinical studies that systematically investigate this hypothesis are still lacking. What is missing: Placebo-controlled RCTs with clear immunological endpoints (e.g. T-cell activation, cytokine profiles) and standardisation of beta-glucan dose and preparation.

Oxidative Stress

B. oxidised LDL, 8-isoprostanes). Beta-glucan significantly reduced the postprandial insulin rise ($p = 0.037$) and the insulin AUC ($p = 0.034$) after a standard breakfast but had no influence on oxidative stress markers. In a subgroup with BMI ≥ 31.5 kg/m², beta-glucan lowered systolic blood pressure by 8.3 mmHg ($p = 0.008$) and diastolic by 3.9 mmHg ($p = 0.018$) versus placebo. The study suggests metabolic effects of beta-glucan but provides no evidence of an antioxidant effect. A limitation is the absence of a detailed analysis of beta-glucan dose and preparation ⁴.

A 2021 review summarises the immunomodulatory and antioxidant effects of fungal beta-glucans in preclinical models. In-vitro studies and animal models show that beta-glucans (e.g. from *Ganoderma lucidum* or *Lentinula edodes*) reduce the production of reactive oxygen species (ROS) in macrophages and increase the activity of antioxidant enzymes (e.g. superoxide dismutase). These preclinical findings suggest an antioxidant potential; however, an effect in humans has not been investigated. A limitation is the heterogeneity of beta-glucan sources and the lack of standardisation of extraction methods.

What is missing: Clinical studies in humans that systematically investigate the influence of beta-glucans on oxidative stress markers (e.g. malondialdehyde, glutathione) and their association with inflammatory or metabolic diseases.

Metabolism

A randomised, double-blind study in 11 healthy subjects acutely compared the effect of oat beta-glucan with high (Oat-A, enzymatically processed) versus low viscosity (Oat-B, aqueous processed) on postprandial glycaemia. Both preparations contained 4 g beta-glucan, but viscosity differed significantly ($p < 0.001$). Oat-A reduced glucose AUC by 23% ($p < 0.05$) and insulin AUC by 28% ($p < 0.05$) versus Oat-B. The study shows differences in postprandial glucose and insulin response between the differently viscous beta-glucan preparations but provides no data on long-term effects or microbiome changes. A limitation is the acute design and the small sample ⁵.

A randomised study in 43 hyperlipidaemic subjects investigated the effect of 4 g oat beta-glucan (in a ready meal) on lipid levels and postprandial glucose/insulin responses over 5 weeks. Beta-glucan lowered total cholesterol by 0.22 ± 0.41 mmol/L and LDL cholesterol by 0.16 ± 0.36 mmol/L versus baseline, but the differences versus the control group were not significant. Postprandial glucose and insulin values showed no significant changes. The study suggests a lipid-lowering effect but provides no data on the dose-response relationship or molecular mechanisms ¹⁰.

A meta-analysis of 13 RCTs with 927 hypercholesterolaemic subjects showed that oat beta-glucan lowers total cholesterol by 0.24 mmol/L (95% CI: -0.28 to -0.20) and LDL cholesterol by 0.27 mmol/L (95% CI: -0.35 to -0.20). Triglycerides and HDL cholesterol remained unchanged. The analysis confirms the lipid-lowering effect but provides no data on the optimal dose or duration of the intervention. A limitation is the heterogeneity of the included studies (different beta-glucan sources, doses, preparations) ¹³.

Beta-glucan-containing rye bread led to a significantly lower glucose AUC ($p < 0.05$) and insulin AUC ($p < 0.05$) versus white bread, but not versus the other whole-grain products. The study shows that beta-glucan-containing rye bread improves the glycaemic response versus white bread but provides no data on long-term effects or microbiome changes. A limitation is the lack of standardisation of the meals ²².

What is missing: Long-term studies on the effect of beta-glucans on glucose metabolism (e.g. HbA1c, insulin resistance) and a systematic analysis of the interaction with the gut microbiota.

Immune System

A randomised, placebo-controlled study in 162 healthy subjects with recurrent infections investigated the effect of 900 mg/day insoluble yeast beta-glucan (1,3-1,6-beta-glucan) on the frequency of cold episodes over 16 weeks. Primary endpoints were the number of symptomatic colds and a symptom score (based on 10 predefined symptoms). Beta-glucan reduced the number of cold episodes by 25% versus placebo ($p = 0.041$) and improved subjective sleep quality ($p = 0.028$). The symptom score was 15% lower but not significant ($p = 0.125$). The study suggests a lower frequency of symptomatic colds but provides no data on immunological mechanisms (e.g. cytokine profiles, T-cell activation). A limitation is the absence of a dose-response analysis ⁹.

A 2014 review summarises the immunomodulatory effects of yeast beta-glucan (1,3-1,6-beta-glucan). Preclinical studies show that beta-glucan activates macrophages, NK cells and dendritic cells and stimulates the production of cytokines (e.g. IL-1, IL-6, TNF- α). Clinical studies confirm a reduction in upper respiratory tract infections, yet data on specific immune parameters or long-term immunological effects in humans are absent. The preclinical findings on cell activation and cytokine production are not transferable to humans and have not been investigated there.

What is missing: clinical studies with clear immunological endpoints (e.g. antibody response after vaccination, cytokine profiles) within the existing controlled human studies and a systematic analysis of the dose-response relationship.

Overall Picture

These effects are dose-dependent (usually 3–6 g/day) and appear to be linked to the viscosity of the beta-glucans, with high-molecular-weight preparations tending to show stronger effects. The data on anti-inflammatory or antioxidant activity are less clear: while preclinical studies (in-vitro and animal models) suggest immunomodulatory effects (e.g. “trained immunity”), these mechanisms have not yet been investigated in clinical human studies with clear endpoints (e.g. CRP, cytokine profiles).

In oncology, beta-glucans (particularly lentinan) as an adjuvant to chemotherapy show promising results in Asian populations, with improvements in response rates and quality of life. However, the studies are heterogeneous and large, placebo-controlled RCTs in Western countries are lacking. The immunomodulatory effects of beta-glucans (e.g. reduction of infections) have been investigated in controlled studies, yet the underlying molecular mechanisms and the optimal dose for different applications (e.g. vaccine support, autoimmune diseases) remain unclear.

Methodology of the evidence base

The supporting works comprise randomised controlled trials (RCTs) in humans (n = 10), observational studies (n = 5) and reviews (n = 6). The RCTs typically last 4–12 weeks and primarily examine metabolic endpoints (lipids, glucose, insulin) or immunological parameters (infection frequency, cytokines). Oncological studies (mostly from Asia) are often longer (up to 12 months) and combine beta-glucans with chemotherapy. Systematic gaps exist in the standardisation of beta-glucan preparations (molecular mass, viscosity, purity), the lack of microbiota analysis in most studies and the absence of long-term studies (>6 months) with clinically relevant endpoints (e.g. cardiovascular events, diabetes incidence). In addition, dose-response studies and investigations into interactions with medications (e.g. statins, immunosuppressants) are missing.

Trained Immunity: Laboratory Phenomenon or Clinical Reality?

The MOST-CITED WORKS (e.g. on *mTOR*, *HIF-1α* or *epigenetic reprogramming of monocytes*) originate from **basic research** and **cell cultures**. They show that beta-glucans can induce **immunological memory effects** (*trained immunity*) – a **laboratory phenomenon** based on **metabolic and epigenetic changes** in immune cells. These findings are often interpreted as **evidence of clinical efficacy**, yet **controlled human studies** are largely absent. The **reviews** (e.g. on *antitumour polysaccharides*) rely on **animal models or in-vitro data**, not on **clinical endpoints**.

Even in ONCOLOGICAL STUDIES (e.g. on lentinan) these are mostly **observational studies or meta-analyses** that do not permit **causal conclusions**. The **most-cited clinical work** on lentinan is a **meta-analysis of individual patient data** that did not include **placebo control**. The **safety data** are **consistent**, yet **long-term effects** or **interactions** with medications (e.g. immunosuppressants) have been **barely investigated**. Research on beta-glucans therefore remains **theoretically strong** but **clinically weakly supported**.

3. How Robust the Research Is

The **research landscape** on beta-glucans is **heterogeneous**. A substantial portion of the works falls under **basic research**, followed by **animal studies** and **cell cultures**. Controlled studies have been conducted in humans, with application areas distributed across **safety, metabolism, immune system and gut health**. Oncology and inflammatory processes follow. The most-cited human studies examine **cholesterol-lowering effects** (oat beta-glucans) and **immunotherapeutic approaches** (lentinan).

The **robustness** of the data is **inconsistent**. Some controlled human studies exist, but they are often **small**. The **mechanistic works** (e.g. on *trained immunity*) are numerous, yet their **clinical relevance** is unclear. Many studies use **different beta-glucan sources, doses and application forms**, which **complicates comparisons**. In addition, **long-term data** or **standardisations** (e.g. molecular weight, purity) are frequently missing. The **reviews** emphasise **immunomodulatory potential**, yet the **transfer to humans** remains speculative.

4. What Is Frequently Claimed — and What the Data Support

STATEMENT	EVIDENCE BASE
Beta-glucans and cholesterol levels	Controlled studies show associations between oat beta-glucans and reduction of LDL cholesterol in the dose ranges studied. However, the effects are moderate .
Beta-glucans and immune system	Beta-glucans have been studied to strengthen the immune system; the controlled results are inconsistent. Laboratory studies show activation of immune cells (e.g. macrophages, neutrophils, dendritic cells).

STATEMENT	EVIDENCE BASE
Beta-glucans and antitumour effect	Fungal beta-glucans (e.g. Lentinan) have been studied to support cancer therapy. The study situation consists mainly of observational studies or animal models .
Beta-glucans and gut health	Beta-glucans have been investigated in relation to gut health. Studies show associations with gut microbiota or inflammatory markers within the dose ranges studied.

5. The Most Robust Works

Ordered by strength of evidence, not by familiarity. The complete evaluation comprises considerably more works; the most robust ones are listed here.

Controlled Human Studies

- ¹ Concentrated oat beta-glucan, a fermentable fiber, lowers serum cholesterol in hypercholesterolemic adults in a randomized controlled trial (2007)
- ² Viscosity of oat bran-enriched beverages influences gastrointestinal hormonal responses in healthy humans (2009)
- ³ Lentinan as an immunotherapeutic for treating lung cancer: a review of 12 years clinical studies in China (2018)
- ⁴ Effects of consuming foods containing oat beta-glucan on blood pressure, carbohydrate metabolism and biomarkers of oxidative stress in men and women (2007)
- ⁵ Beta-glucan from two sources of oat concentrates affect postprandial glycemia in relation to the level of viscosity (2007)
- ⁶ A randomized Placebo-Controlled Clinical Trial to Evaluate the Medium-Term Effects of Oat Fibers on Human Health: The Beta-Glucan Effects on Lipid Profile (2020)
- ⁷ Simultaneous intake of beta-glucan and plant stanol esters affects lipid metabolism in slightly hypercholesterolemic subjects (2007)
- ⁸ Yeast-leavened oat breads with high or low molecular weight beta-glucan do not differ in their effects on blood concentrations of lipids, insulin, or (2004)
- Mushrooms and immunity (2021)
- ⁹ Yeast (1,3)-(1,6)-beta-glucan helps to maintain the body's defence against pathogens: a double-blind, randomized, placebo-controlled, multicentric study (2013)
- ¹⁰ Serum lipids and postprandial glucose and insulin levels in hyperlipidemic subjects after consumption of an oat beta-glucan-containing ready meal (2008)
- Comparative efficacy and safety of Chinese herbal injections combined with the FOLFOX regimen for treating gastric cancer in China: a network meta-analysis (2017)
- Fiber mixture-specific effect on distal colonic fermentation and metabolic health in lean but not in prediabetic men (2022)
- ¹¹ Effect of muesli with 4 g oat beta-glucan on postprandial blood glucose, gastric emptying and satiety in healthy subjects: a randomized crossover trial (2008)
- ¹² Randomised phase III study of S-1 alone versus S-1 plus lentinan for unresectable or recurrent gastric cancer (JFMC36-0701) (2016)
- ¹³ Effects of Oat Beta-Glucan Intake on Lipid Profiles in Hypercholesterolemic Adults: A Systematic Review and Meta-Analysis of Randomized Controlled Trials (2022)
- ¹⁴ The effect of fibre amount, energy level and viscosity of beverages containing oat fibre supplement on perceived satiety (2010)
- ¹⁵ Beneficial effects of novel aureobasidium pullulans strains produced beta-1,3-1,6 glucans on interleukin-6 and D-dimer levels in COVID-19 patients; results (2022)
- ¹⁶ Effects of Yeast (1,3)-(1,6)-Beta-Glucan on Severity of Upper Respiratory Tract Infections: A Double-Blind, Randomized, Placebo-Controlled Study in Healthy Subjects (2019)
- ¹⁷ Effect and Tolerability of a Nutritional Supplement Based on a Synergistic Combination of β -Glucans and Selenium- and Zinc-Enriched *Saccharomyces cerevisiae* (2021)
- ¹⁸ An Oat β -Glucan Beverage Reduces LDL Cholesterol and Cardiovascular Disease Risk in Men and Women with Borderline High Cholesterol: A Double-Blind, Randomized, Controlled Trial (2021)
- ¹⁹ Effect of oat supplementation interventions on cardiovascular disease risk markers: a systematic review and meta-analysis of randomized controlled trials (2022)
- ²⁰ Effects after inhalation of (1 $\rightarrow$ 3)-beta-D-glucan in healthy humans (2001)
- ²¹ Purified β -glucans from the Shiitake mushroom ameliorates antibiotic-resistant *Klebsiella pneumoniae*-induced pulmonary sepsis (2020)

6. Summary

What is proven: Beta-glucans from oats demonstrably lower LDL cholesterol and improve glycaemic control, with the viscosity of the molecules playing a central role. Fungal beta-glucans such as lentinan have been studied in combination with chemotherapy; the combination showed immunomodulatory effects and increased response rates in cancer patients in studies. Human studies also confirm an influence on satiety hormones and a lipid-lowering effect, but the effects are dose-dependent and short-term. The data on inflammatory markers (e.g. CRP) are contradictory, and systematic analyses of the microbiome are largely lacking.

Fungal beta-glucans could, according to preclinical data, enhance the efficacy of immunotherapies (e.g. checkpoint inhibitors) by triggering a Th1-dominated immune response. The interaction with the gut microbiota and the production of short-chain fatty acids (SCFA) could mediate further metabolic and anti-inflammatory effects. However, these assumptions are based on preclinical data, and clinical studies with clear endpoints in humans are still outstanding.

What a study would need to clarify: Placebo-controlled, randomised long-term studies with standardised beta-glucan sources (e.g. defined molecular weight, branching pattern) and clear clinical endpoints are needed. Systematic analyses of the dose-response relationship, microbiome composition and epigenetic effects in humans are essential to examine the transferability of mechanistic findings. In addition, studies should investigate the interaction with immunotherapies or inflammation-associated diseases (e.g. rheumatoid arthritis) and identify biomarkers for patient selection. The standardisation of extraction methods and application forms is a prerequisite for comparable results.

7. Broader Context

Beta-glucans from oats and barley have been shown to be viscosity-increasing dietary fibres that are fermented in the colon, thereby promoting the production of short-chain fatty acids (SCFA) such as butyrate. Preclinical studies show that this fermentation could stabilise the intestinal barrier by increasing the expression of *Tight-Junction* proteins (e.g. Occludin, Claudin) and modulating the mucosal immune response. The interaction with the microbiome is central here: beta-glucans select butyrate-producing bacteria such as *Faecalibacterium prausnitzii*, which are considered opponents of silent inflammation. In parallel, oat beta-glucans demonstrably lower LDL cholesterol. The chain from the gut to metabolic regulation is directly measurable here, yet the transferability to other beta-glucan sources (e.g. fungal) remains unclear, as their bioavailability and receptor interactions (e.g. Dectin-1) proceed differently.

Fungal beta-glucans with β -1,3/1,6 branchings activate immune receptors such as Dectin-1 and TLR2/4. Preclinical studies show that they lead to *trained immunity* in animal models and cell cultures – an epigenetic reprogramming of myeloid cells that strengthens defence against pathogens in the long term. This mechanism could bridge the intestinal barrier and systemic immunomodulation: in preclinical models, fungal beta-glucans interact after uptake via M cells in the gut with dendritic cells, which in turn influence T helper cells and regulatory T cells (Tregs). Preclinical findings also suggest that beta-glucans could reduce intestinal permeability by stimulating the secretion of IgA and antimicrobial peptides. It remains unresolved whether these effects are dose-dependent in humans, whether they vary with the structural heterogeneity of the beta-glucans and to what extent the preclinical observations are transferable to humans.

In preclinical models it was investigated whether the combination of beta-glucans with zeolite could act synergistically by addressing two central links of the intestinal barrier-inflammation axis: animal and cell studies suggested that beta-glucans could support the barrier via butyrate and microbiome modulation, while zeolite would potentially bind harmful metabolites (e.g. LPS). It would need to be examined whether this combination strengthens *trained immunity* without triggering excessive inflammatory reactions. Long-term studies would have to clarify whether such synergies have clinically relevant effects on silent inflammation or metabolic endpoints and to what extent the observed effects are attributable to the combination or to the individual components.

8. What Remains Open

- **Standardisation:** Which **molecular structure** (mass, branching) is decisive for which **biological effect**?
- **Long-term effects:** Are there **clinically relevant benefits** (e.g. cardiovascular) or **risks** (e.g. immune overactivation)?
- **Combination therapies:** Can beta-glucans be used **adjuvantly** (e.g. in oncology) **safely and effectively**?

9. Further Paths

Topics, mechanisms of action and chains in which this substance occurs – for all those who wish to look beyond the substance.

- From Exclusion to Inflammation: Stress, Cortisol, Gut
 - Intestinal Barrier and Binding: Zeolite, Butyrate, Probiotics
-

Frequently Asked Questions

Are all beta-glucans the same?

No. **Structure and origin** determine the **biological activity**. Cereal beta-glucans (e.g. from oats) act primarily **metabolically modulating** (e.g. cholesterol-lowering), while fungal beta-glucans (e.g. lentinan) are **immunomodulatory** active. The **molecular mass and branching patterns** influence **solubility, viscosity and receptor interaction**. Studies are therefore **not directly comparable**.

Can beta-glucans be used in autoimmune diseases?

The **evidence base** is **insufficient**. Laboratory studies show **immunomodulatory effects**, yet **controlled human studies** are lacking. Theoretically, beta-glucans could **enhance proinflammatory processes**. **Safety data** for this patient group do not exist. An application is **currently not evidence-based**.

How are beta-glucans dosed in studies?

The **dosages** vary **widely** and are **not standardised**. In controlled human studies **different amounts** (e.g. from oats or fungi) are used, often as **food supplements or fortified foods**. **Bioavailability** depends on **source, processing and application form**. **Comparisons between studies** are therefore **difficult**.

Sources

- 1 Concentrated oat beta-glucan, a fermentable fiber, lowers serum cholesterol in hypercholesterolemic adults in a randomized controlled trial
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