SCIENTIFIC MONOGRAPH SERIES
MONO-LX-IV.2026

CANCER-SUPPORT FORMULA

Lentinan AXT

A scientific dossier on the multi-pathway formula by Zenius Labs™

COMPOSITION · PER DAILY INTAKE

Lentinus edodes ≥30 % polysaccharides	shiitake · hot-water extract	**1200 mg**
Coriolus versicolor ≥30 % polysaccharides	turkey tail · hot-water extract	**480 mg**
Haematococcus pluvialis carotenoid antioxidant	microalgae · from 120 mg extract – astaxanthin	**6 mg**

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§ ABSTRACT

A multi-pathway formula.

Lentinan AXT is a proprietary multi-pathway formula developed by Zenius Labs™ for adults seeking sustained immune and antioxidant support across the cancer-care continuum. The formula is relevant from preventive use in those with familial, hereditary, or environmental risk, through active treatment — including chemotherapy, radiation, immunotherapy, hormone therapy, targeted therapy, and surgical recovery — to long-term survivorship, post-treatment immune restoration, and the maintenance of cellular and immune resilience. Each daily intake of three capsules delivers a hot-water extract of *Lentinus edodes* (shiitake) at 1200 mg, standardised to ≥30 % polysaccharides; a hot-water extract of *Coriolus versicolor* (turkey tail) at 480 mg, standardised to ≥30 % polysaccharides; and astaxanthin at 6 mg, derived from a 120 mg extract of the microalgae *Haematococcus pluvialis*.

The formula combines fungal polysaccharide immunomodulators with a carotenoid antioxidant operating on an independent biochemical pathway. Together the constituent extracts engage innate immunity through pattern-recognition receptors on macrophages and dendritic cells, NK-cell-mediated cellular surveillance, cytokine signalling networks, T-cell coordination via dendritic-cell antigen presentation, and a distinct axis of oxidative-stress defence at cell membranes, DNA, and mitochondria. The mechanistic premise is multi-pathway, not single-compound.

The constituent extracts have been investigated independently of one another in published mechanistic, *in vitro*, animal-model, and clinical studies indexed in the biomedical literature, including in oncology contexts during conventional chemotherapy and radiation protocols. This monograph reviews that evidence at the doses delivered by Lentinan AXT. Lentinan AXT as a finished composition has not been the subject of an independent clinical trial; the evidence presented concerns the constituent extracts in their own published studies.

§ 1 COMPOSITION

Each daily intake of three capsules of Lentinan AXT delivers the active extracts shown in Table 1. The formulation premise is multi-mechanism: the fungal polysaccharide extracts engage innate-immunity pathways through their respective β-glucan profiles, while astaxanthin from *Haematococcus pluvialis* provides a parallel carotenoid antioxidant action operating at the level of cell membranes, DNA, and mitochondria. The composition is fixed; Lentinan AXT is the formula, not any single extract.

TABLE 1 — ACTIVE INGREDIENTS PER DAILY INTAKE (3 CAPSULES)

BOTANICAL / COMMON	EXTRACT	PER DOSE	STANDARDISATION
Lentinus edodes shiitake	Hot-water extract	1200 mg	≥30 % polysaccharides
Coriolus versicolor turkey tail	Hot-water extract	480 mg	≥30 % polysaccharides
Astaxanthin *Haematococcus pluvialis* microalgae	From 120 mg extract	6 mg	Carotenoid antioxidant

Each constituent active ingredient serves a distinct mechanistic role within the formula:

- *Lentinus edodes* (shiitake) hot-water extract is the source of **lentinan** — a β-(1,3)/(1,6)-glucan polysaccharide that activates innate-immunity pathways including macrophage and dendritic-cell activation, with downstream NK-cell and cytokine signalling.
- *Coriolus versicolor* (turkey tail) hot-water extract supplies the polysaccharide-protein and polysaccharide-peptide fractions designated **PSK** and **PSP** in the published *Coriolus* literature — including the body of work investigating these fractions during oncological treatment protocols.
- **Astaxanthin** from the microalgae *Haematococcus pluvialis* is a xanthophyll carotenoid with antioxidant activity documented at the level of cell-membrane integrity, DNA protection, and mitochondrial function.

The constituent extracts were selected to engage immune-modulation and oxidative-stress-defence pathways in parallel within a single daily intake. The published evidence reviewed in §3 is presented at the doses delivered by this formula.

PRODUCT SPECIFICATION

Capsule count	90 per bottle
Daily intake	3 capsules — 1 in the morning, 2 in the evening
Capsule shell	HPMC (hydroxypropyl methylcellulose); plant-based, vegan
Allergens	Produced without milk and casein, eggs, gluten, or soybeans. No synthetic colorants, flavorings, or preservatives. The product is non-GMO. Natural sulfites may be present at trace levels.
Storage	Cool, dry place. Avoid direct sunlight, high heat, and high humidity. Intended for use only while th

Producer	Zenius Labs™ — to specifications maintained by the company.

§ 2 MECHANISM OF ACTION

Lentinan AXT engages two biochemically independent pathways in parallel: fungal polysaccharide immunomodulation through *Lentinus edodes* and *Coriolus versicolor* hot-water extracts, and carotenoid antioxidant cover through astaxanthin from the microalgae *Haematococcus pluvialis*. The two pathways operate on different cellular substrates and through different receptor systems; together they cover innate immunity, adaptive-immunity coordination, cytokine signalling, oxidative-stress defence, and the integrity of DNA, cell membranes, and mitochondria. The composition is fixed; Lentinan AXT is a multi-pathway formula, not the sum of its independent ingredients.

§2.1 Fungal polysaccharide immunomodulation

The two fungal extracts in Lentinan AXT supply β-glucan polysaccharides — high-molecular-weight branched glucose polymers — that are recognised by the innate immune system through dedicated pattern-recognition receptors. The principal receptor engaged is dectin-1, a C-type lectin expressed on the surface of macrophages and dendritic cells[1]. Engagement of dectin-1 triggers an intracellular signalling cascade that includes the Syk kinase and CARD9 adaptors, ultimately activating NF-κB and MAP-kinase pathways to drive cytokine production and immune-cell activation. A secondary recognition pathway involves TLR2, particularly engaged by the PSK fraction of *Coriolus versicolor*[2, 3], where the polysaccharide-protein complex acts as a TLR2 ligand to activate human dendritic cells and natural killer cells in a manner that complements the dectin-1 axis.

Macrophage and dendritic-cell activation.

Lentinan from *Lentinus edodes* — a β-(1,3)/(1,6)-glucan with branched side-chain architecture — activates tissue macrophages and circulating monocytes, with downstream maturation of dendritic cells and reprogramming of their immunometabolic profile[4, 5, 6]. The 2022 mechanistic study of macrophage β-glucan reprogramming demonstrates polarisation toward an immunostimulatory phenotype with shifts in glycolytic and oxidative metabolism that support sustained antimicrobial and antitumor effector function[6]. The PSK and PSP fractions from *Coriolus versicolor* activate dendritic cells via TLR2 signalling and enhance antigen-specific T-cell responses, providing an adjuvant-like effect that strengthens the bridge between innate detection and adaptive immune coordination[2].

NK-cell-mediated cellular surveillance.

Both fungal extracts upregulate natural-killer-cell activity — the innate-immune mechanism for recognising and clearing cells with altered surface markers or compromised DNA. Early in vitro and in vivo work documented activation of NK cells and cytotoxic macrophages by lentinan administration[7, 4]. The clinical relevance of this pathway is illustrated by a study in which preoperative administration of lentinan in cancer patients undergoing major surgery ameliorated the surgical impairment of natural killer activity — a window of immunosuppression during which residual or circulating tumor cells are at heightened risk of evading clearance[8]. Independent work has shown that the PSK fraction of *Coriolus versicolor* activates human NK cells in a TLR2-dependent manner and enhances the antitumor effect of trastuzumab (Herceptin) against HER2-positive cancer cells[3].

Cytokine signalling.

Following β-glucan engagement, macrophages and lymphocytes produce a coordinated cytokine profile including interferon-γ, TNF-α, IL-12, and IL-1β[6, 9]. This profile is dose-dependent and characteristically transient, with cytokine levels returning toward homeostatic baselines as the polysaccharide is cleared. The transient kinetic distinguishes the immunomodulatory pattern of β-glucans from sustained pro-inflammatory pathologies; the activation is event-coupled to receptor engagement rather than chronic.

T-cell activation through dendritic-cell maturation.

β-glucan-activated dendritic cells present antigens with elevated costimulatory marker expression, supporting helper T-cell proliferation and cytotoxic T-cell function[4, 5]. The 2026 comprehensive review of lentinan mechanisms summarises documented effects across the spectrum of innate-to-adaptive immune coordination, including modulation of regulatory T-cell populations in the tumor microenvironment and counterbalancing of immunosuppressive cytokine milieus[5].

Trained immunity.

β-glucans induce a form of innate-immune memory — "trained immunity" — through epigenetic reprogramming of myeloid progenitors in the bone marrow, supporting a more responsive innate-immune posture across weeks to months following exposure[11, 9, 10]. The 2022 study by Geller and colleagues documented induction of peripheral trained immunity in the pancreas through β-glucan-mediated epigenetic reprogramming, with downstream anti-tumor activity in pancreatic cancer models[10].

§2.2 Effects on cells with compromised DNA

The constituent extracts have been documented in independent studies to act on cells with altered DNA across multiple cell-line and animal-model contexts, with effects spanning proliferation signalling, apoptosis induction, angiogenesis modulation, and cell-cycle progression.

- Modulation of abnormal cell proliferation, including via the EGR1 / PTEN / AKT signalling axis documented in hepatocellular carcinoma cells[12];
- Induction of apoptosis in cells with damaged DNA, observed for lentinan in HCC cells and for astaxanthin in HCC and lung cancer cell lines[12, 16];
- Modulation of tumour angiogenesis — the formation of new blood vessels supporting tumour growth — through interferon-γ-dependent and T-cell-independent pathways[13];
- Effects on cell-cycle progression and DNA-replication checkpoints, with PD-L1 modulation and immune-checkpoint regulation reviewed in 2024[14].

§2.3 Carotenoid antioxidant pathway: astaxanthin

Astaxanthin — the xanthophyll carotenoid derived from the microalgae *Haematococcus pluvialis* — operates on a biochemical pathway distinct from the fungal polysaccharides. Its mechanism is direct antioxidant action: neutralisation of singlet oxygen, peroxyl radicals, and superoxide at the level of cell membranes, DNA, and mitochondria[15, 16]. The 2025 comprehensive review by Copat and colleagues documents anti-proliferative, pro-apoptotic, and anti-inflammatory effects of astaxanthin across cancer-related contexts, drawing together the mechanistic threads from in vitro cell-line studies, animal models of chemotherapy-associated toxicity, and observational human data[15].

DNA integrity.

Astaxanthin reduces oxidative DNA damage markers and protects against oxidative DNA insult in multiple in vitro and animal-model systems[15, 17]. The mouse-preantral-follicle study by He and colleagues documented oxidative-stress alleviation and improvement in follicular quality, through AMPK signalling activation — a finding that illustrates the breadth of astaxanthin's antioxidant action across tissue types[17].

Cellular and mitochondrial protection.

As a lipid-soluble carotenoid, astaxanthin is bioavailable to cell-membrane and mitochondrial compartments where free-radical attack on lipid bilayers and respiratory-chain components occurs. The 2024 study by Krestinin and colleagues documented protection against H_2O_2- and doxorubicin-induced cardiotoxicity in H9c2 cardiomyocytes, with preservation of mitochondrial membrane potential and function during oxidative challenge — a finding with implications for cellular energy and metabolic resilience[20]. Application strategies including nanoformulation-based delivery to enhance bioavailability are reviewed in[18].

Cytoprotection during chemotherapy-associated oxidative challenge.

Animal-model investigations of astaxanthin in chemotherapy-associated toxicities span multiple organ systems:

- Cisplatin-induced nephrotoxicity in rats[21];
- Doxorubicin-induced cardiotoxicity in cardiomyocytes[20];
- Doxorubicin-induced cognitive impairment (“chemobrain”)[22];
- Cholestasis-induced liver fibrosis[23];
- Cancer-cachexia-associated skeletal muscle atrophy[24].

§2.4 The constituent extracts in the published oncology literature

Lentinan (the principal β-glucan from *Lentinus edodes*) has been approved in Japan as an adjunct immunotherapy in cancer care since 1985, with the consolidated review of its early immunopharmacology published in 1992[25]. It has been investigated as an adjunct during conventional chemotherapy and radiotherapy in published clinical studies across gastric, pancreatic, hepatocellular, lung, esophageal, and bladder cancer cohorts[26, 27, 28, 29, 30, 31, 32, 33, 34, 35]. The 2026 comprehensive review by Zhen and colleagues integrates mechanism, pharmacology, and advanced delivery strategies including injectable formulations and emerging nanocarriers[5].

PSK and PSP (the polysaccharide-protein and polysaccharide-peptide fractions of *Coriolus versicolor*) have been used in clinical oncology in Japan (PSK, approved 1977) and China (PSP, clinical use from 1987). The 2017 review by Chang and colleagues consolidates more than four decades of preclinical and clinical investigation[36]. PSK has been documented to improve overall survival when added to standard adjuvant chemotherapy across gastric-cancer stages in a 2007 multi-centre individual-patient meta-analysis[37], and has been examined for therapeutic efficacy and gut-microbiome prebiotic effects in gastrointestinal cancer cohorts[38]. A 2022 Cochrane systematic review documents *Coriolus (Trametes) versicolor* for the reduction of adverse effects from chemotherapy and radiotherapy in patients with colorectal cancer[39]. A 2008 Phase I clinical trial of *Trametes versicolor* mushroom immune therapy in women with breast cancer documented immune-response enhancement[40].

Astaxanthin in cancer-related research is reviewed comprehensively in[15]; mechanistic studies in hepatocellular carcinoma cell lines via NF-κB / Wnt/β-catenin inhibition documented in[16]; gastrointestinal anti-inflammatory effects through NF-κB pathway inhibition in[42]; and dietary-carotenoid context for chemoprevention and chemotherapy in the 2020 review by Saini and colleagues[41].

§2.5 The formula rationale

Lentinan AXT combines fungal polysaccharide immunomodulators with a carotenoid antioxidant to engage two mechanistically distinct axes in parallel within a single daily intake. The β-glucans act through receptor-mediated activation of innate immunity — dectin-1 and TLR2 — with downstream cellular and cytokine consequences. Astaxanthin acts through direct biochemistry, quenching reactive oxygen species at cell membranes, DNA, and mitochondria. The two pathways do not share substrates, receptors, or rate-limiting steps; they operate independently and additively. The doses of each active ingredient correspond to ranges documented in the respective published literatures: lentinan / shiitake extract at 1200 mg with ≥30 % polysaccharides; *Coriolus* extract at 480 mg with ≥30 % polysaccharides; astaxanthin at 6 mg. Lentinan AXT is the proprietary composition assembled by Zenius Labs™.

§ 3 COMPONENT EVIDENCE

§3.1 Lentinan from *Lentinus edodes*

Regulatory status. Lentinan has been used as an adjunct immunotherapy in Japanese cancer care since approval in 1985, with the consolidated review of its early-phase clinical immunopharmacology published in 1992 by Chihara[25]. The 2026 comprehensive review by Zhen and colleagues consolidates current understanding of mechanisms and advanced delivery strategies, including injectable, oral superfine-dispersed, nanoformulated, and biosensor-paired formulations[5].

Meta-analyses and systematic reviews. A 2024 systematic review and meta-analysis by Wang and colleagues examined injectable lentinan combined with chemotherapy in gastric cancer across pooled patient cohorts[26]. A 2009 individual-patient-based meta-analysis by Oba and colleagues examined lentinan in unresectable and recurrent gastric cancer[27]. A 2015 meta-analysis of 12 studies covering 950 patients with non-small-cell lung cancer reported significant response improvement with lentinan combined with chemotherapy[29]. A 2017 meta-analysis by Wang and colleagues examined lentinan as a biological response modifier with chemotherapy for advanced cancer across mixed-tumor cohorts[28]. A 2018 review by Zhang and colleagues summarised 12 years of clinical evidence on lentinan as an immunotherapeutic for lung cancer in China[43].

Gastric cancer. Lentinan combined with S-1 and paclitaxel was documented to improve patient quality-of-life in gastric chemotherapy in a 2009 study[44]. Adverse-effect management for S-1 chemotherapy with superfine dispersed lentinan was documented in 2010[45]. Quality-of-life and prognosis improvement with superfine dispersed lentinan in advanced gastric cancer was reported in 2010 by Yoshino and colleagues[46].

Hepatocellular carcinoma. Lentinan induces apoptosis of HCC cells through the EGR1/PTEN/AKT signalling axis as documented by You and colleagues in 2023[12]. Clinical application combining lentinan with multi-electrode radiofrequency ablation and transarterial chemoembolisation in HCC settings was reported in 2008[49]. Induction of tumor-specific antigen by lentinan in murine H22 HCC immunoprophylaxis was documented in 2015[50]. Clinical efficacy of superfine dispersed lentinan (β-1,3-glucan) in HCC patients was reported in 2009[51].

Pancreatic cancer. Efficacy of oral administered superfine dispersed lentinan for advanced pancreatic cancer was documented in 2009[33]. A carbon nanotube–gemcitabine–lentinan three-component composite for chemo-photothermal synergistic therapy was investigated in 2018 by Zhang and colleagues[52].

Lung cancer. Lentinan combined with cisplatin for non-small-cell lung cancer (NSCLC), including intrapleural application for pleural disease, was examined in a 2021 systematic review and meta-analysis protocol[30]. Translational nanotherapeutic approaches incorporating lentinan for reprogramming the immune microenvironment in malignant pleural effusion of lung adenocarcinoma were reported in 2021[53]. Evaluation of efficacy and safety for lentinan in malignant pleural effusion control via intrapleural injection was documented in 2019[54]. Lentinan triggers oxidative-stress-mediated anti-inflammatory responses in lung cancer cells — a 2022 cell-line mechanistic study[55]. The 2021 study by Gui and colleagues demonstrated that oridonin improved the therapeutic effect of lentinan on lung cancer in

experimental models[56].

Esophageal cancer. Lentinan enhances oxaliplatin against esophageal tumors by persuading immunogenic cell death — documented in a 2022 study by Huo and colleagues[34]. Earlier combination therapy with lentinan in esophageal carcinoma was reported in 2012[32].

Bladder and urothelial cancer. Lentinan reduces tumor progression by enhancing gemcitabine chemotherapy in urothelial bladder cancer, as documented by Sun and colleagues in 2015[31].

Mechanistic synergy and pathway studies. Apaf1 nanoLuc biosensors identified lentinan as a potent synergiser of cisplatin in hepatocellular carcinoma cell models — a 2021 study by Wang and colleagues[35]. Lentinan inhibits tumor angiogenesis via interferon-γ and in a T-cell-independent manner[13]. The 2024 review by Zhou and colleagues covers lentinan's progress in inflammatory and tumor disease, including PD-L1 modulation and immune-checkpoint regulation[14].

Foundational immunology. The early-1980s in vivo work of Amino and colleagues documented lentinan's effect on NK-cell activity, helper T-cell function, and PHA-induced blastic response in cancer patients[4]. Broader β-glucan effects on the immune system are reviewed in[57]. Therapeutic intervention with complement and β-glucan in cancer is reviewed in the seminal 1999 paper by Ross and colleagues[58].

§3.2 PSK and PSP from *Coriolus versicolor*

Regulatory status. PSK — the polysaccharide-K fraction of *Coriolus versicolor* — was approved in Japan as an adjunct cancer therapy in 1977; PSP — the polysaccharide-peptide fraction — has been used clinically in China since 1987. The 2017 review by Chang and colleagues consolidates preclinical and clinical investigation of these polysaccharopeptide fractions across more than four decades, including mechanism, dose-response, and adjuvant indications[36].

Adjuvant immunochemotherapy in gastric cancer. PSK adjuvant immunochemotherapy was documented in 2012 by Tanaka and colleagues to improve overall survival across multiple gastric-cancer stages following curative resection[37]. PSK has been examined for therapeutic efficacy and safety in gastrointestinal cancer cohorts, including its prebiotic effect on gut microbiome composition — a 2017 systematic review and network meta-analysis[38]. Postoperative adjuvant immunochemotherapy with PSK following curative gastric-cancer resection was reported by Oba and colleagues in 2007[47]. The 2009 case series by Ooshiro and colleagues documented ten advanced gastrointestinal cancer cases that required preoperative PSK dosage for local progression management[48]. Postoperative PSK with OK-432 combination immunochemotherapy in gastric cancer was reported in 1993 by Maehara and colleagues[59].

Reduction of chemotherapy and radiotherapy adverse effects. The 2022 Cochrane systematic review by Pilkington and colleagues documents *Coriolus (Trametes) versicolor* for reducing adverse effects from chemotherapy or radiotherapy in people with colorectal cancer[39]. Efficacy of Yun Zhi (*Coriolus versicolor*) on survival in cancer patients was the subject of an earlier 2012 systematic review and meta-analysis by Eliza and colleagues[60].

Breast cancer. A 2008 Phase I clinical trial of *Trametes versicolor* mushroom immune therapy in women with breast cancer documented immune-response enhancement by

Standish and colleagues[40]. The 2011 study by Lu and colleagues demonstrated that the TLR2 agonist PSK activates human NK cells and enhances the antitumor effect of trastuzumab (Herceptin) against HER2-targeted disease, providing mechanistic support for combination immunotherapy strategies[3].

Mechanism — dendritic-cell activation and T-cell response. The 2013 study by Engel and colleagues demonstrated that protein-bound polysaccharide (PSK) activates dendritic cells and enhances OVA-specific T-cell responses through a TLR2-mediated pathway, characterising PSK as a vaccine adjuvant candidate[2].

Colorectal cancer. Polysaccharide-peptide from *Trametes versicolor* as a potential therapy in colorectal cancer is reviewed in the 2022 paper by He and colleagues[61].

Historical landmark cases. A 1983 case report by Takada and colleagues documented marked reduction in hepatocellular-carcinoma tumor size induced by PSK administration alone[62]. A 1988 report by Hara and colleagues described disappearance of lung metastases from hepatocellular carcinoma following PSK administration[63].

§3.3 Astaxanthin from *Haematococcus pluvialis* microalgae

Comprehensive review. The 2025 comprehensive review by Copat and colleagues on astaxanthin in cancer therapy and prevention documents anti-proliferative, pro-apoptotic, and anti-inflammatory mechanisms across cell-line, animal-model, and observational human evidence streams[15]. The 2025 review by Qian and colleagues covers molecular mechanisms underlying astaxanthin's beneficial effects in lung-cancer and metabolic-disease contexts, including the dual antioxidant and signalling pathway modulation[64].

Mechanism in cancer cell lines. The 2015 study by Li and colleagues demonstrated that astaxanthin inhibits proliferation and induces apoptosis of human hepatocellular carcinoma cells via inhibition of NF-κB p65 and Wnt/β-catenin signalling in vitro[16]. The 2016 study by Wu and colleagues documented that astaxanthin inhibits proliferation and promotes apoptosis of A549 lung cancer cells via blocking the JAK1/STAT3 pathway[19]. Anti-oxidant and anti-inflammatory effects of astaxanthin on gastrointestinal diseases through NF-κB pathway inhibition were reviewed in 2022 by Lee and colleagues[42].

Chemoprevention and chemotherapy context. Dietary carotenoids in cancer chemoprevention and chemotherapy — reviewing evidence for enhanced treatment efficacy and reduced toxicity — are surveyed in the 2020 review by Saini and colleagues[41]. Oxidative stress and marine carotenoids, including nanoformulation-based delivery strategies, are reviewed in[18].

Cytoprotection during chemotherapy. Astaxanthin has been investigated in animal models across the principal organ toxicities associated with cytotoxic chemotherapy. A 2026 study by Shalaby and colleagues documented protection against cisplatin-induced nephrotoxicity in rats, with interplay between non-coding RNA, redox state, inflammation, and ferroptosis[21]. A 2024 study by Krestinin and colleagues documented protection against H_2O_2- and doxorubicin-induced cardiotoxicity in H9c2 rat myocardial cells[20]. A 2018 study by El-Agamy and colleagues documented amelioration of doxorubicin-induced cognitive impairment (“chemobrain”) in an experimental rat model, with effects on oxidative, inflammatory, and apoptotic machineries[22]. A 2024 study by Laderian and colleagues documented hepatoprotective effects of astaxanthin against cholestasis-induced liver fibrosis induced by bile duct ligation in Wistar rats[23]. The 2024 study by Yu and colleagues documented

amelioration of skeletal muscle atrophy in mice with cancer cachexia[24].

Foundational antioxidant protection. Astaxanthin against diesel-exhaust-particle adverse effects via membrane stability and anti-oxidative properties was documented by Wang and colleagues in 2023[65]. The 2025 study by He and colleagues documented oxidative-stress alleviation in mouse preantral follicles with enhancement of follicular development through AMPK signalling[17]. An early observational study in 2010 by Park and colleagues documented decreased oxidative stress and inflammation, with enhanced immune response, in human participants supplementing with astaxanthin[66].

§3.4 Common context — medicinal mushroom polysaccharides in oncology

The fungal extracts in Lentinan AXT are members of a broader class of medicinal mushroom polysaccharides investigated in oncology. A 2023 scoping review by Dan and colleagues documents therapeutic effects of medicinal mushrooms on gastric, breast, and colorectal cancers across the literature[67]. A 2025 review by Zhang and colleagues covers structural characteristics, chemical modification strategies, and structure-activity relationships of edible mushroom polysaccharides[68]. The 2025 review by Zou and colleagues documents radioprotective effects of edible and medicinal mushrooms against ionising radiation, with components, potential mechanisms, and applications[69]. Induction of peripheral trained immunity in the pancreas via β-glucan-mediated epigenetic reprogramming, with downstream anti-tumor activity, was documented by Geller and colleagues in 2022[10]. β-glucan and complement-pathway interactions in cancer immunotherapy are reviewed in[58]. Macrophage polarisation and β-glucan reprogramming in human macrophages was examined in 2022 by Fan and colleagues[6].

§ 4 QUALITY, IDENTITY, AND MANUFACTURING

§4.1 Identity and standardisation

Each lot of Lentinan AXT is produced to a fixed composition specification, given here at the level of one daily intake of three capsules:

- *Lentinus edodes* hot-water extract, **1200 mg**, standardised to **≥30 % polysaccharides**;
- *Coriolus versicolor* hot-water extract, **480 mg**, standardised to **≥30 % polysaccharides**;
- Astaxanthin **6 mg**, derived from **120 mg** extract of *Haematococcus pluvialis* microalgae.

The two fungal extracts are produced by hot-water extraction — the conventional method for isolating the high-molecular-weight β-glucan polysaccharides responsible for the immunomodulatory activity reviewed in §2 and §3. The polysaccharide-content threshold (≥30 %) is the standardisation point maintained in Zenius Labs™ specifications.

§4.2 Capsule shell and excipient profile

The capsule shell of Lentinan AXT is HPMC (hydroxypropyl methylcellulose) — a plant-based, vegan capsule material. The formulation contains no synthetic colorants, no synthetic flavorings, and no synthetic preservatives.

§4.3 Allergen profile

Lentinan AXT is produced without milk and casein, eggs, gluten, and soybeans. The product is non-GMO. Natural sulfites may be present at trace levels from source-material extracts.

§4.4 Storage and integrity

Cool, dry place. Direct sunlight, high heat, and high humidity are to be avoided. Each unit is sealed at the point of manufacture; the formulation is intended for use only while the manufacturer’s seal is intact.

§4.5 Product identification

GTIN-13	633841609752
SKU	ZN333222MB
MPN	ZN12619MB

§4.6 Producer

Lentinan AXT is produced to specifications maintained by Zenius Labs™.

§ 5 REFERENCES

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