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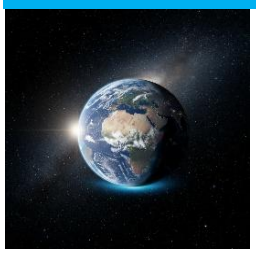

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Therapeutic potential of ginseng in the treatment of allergic respiratory diseases

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Abstract / Summary

Introduction and Purpose: Ginseng (*Panax spp*), known as the king of herbs, is one of the most important medicinal plants in East Asia. Its several thousand years of history in medicine is due to its various therapeutic properties, including immunomodulatory and anticancer properties. The aim of this paper is to present the effects of ginseng use on reducing the symptoms of allergic respiratory diseases, which are becoming increasingly common in the population, as described in scientific studies

State of knowledge: Animal studies have shown that *Panax ginseng* reduces inflammation of the respiratory tract through its anti-inflammatory and immunomodulatory effects on immune system cells. The effectiveness of black ginseng extract in studies on mice was comparable to that of dexamethasone. Korean red ginseng extract showed similar biological activity, alleviating asthma symptoms and reducing allergic inflammatory reactions in the nose, as well as acting as an antioxidant to reduce inflammation. Human studies have shown that red ginseng extract acts similarly to antihistamines, relieving itching of the nose and eyes, reducing nasal discharge, and reducing nasal congestion. Important medicinal components of the plant are ginsenosides, which can, among other things, reduce the number of inflammatory response cells and suppress cytokine expression, thereby reducing allergy symptoms.

Conclusions: Ginseng shows promising results in the treatment of allergic respiratory diseases by reducing the severity of clinical symptoms and decreasing inflammatory response indicators. It is worth noting that despite good results, further research on a larger group of patients is still needed to confirm its efficacy and safety.

Keywords:
Ginseng; asthma; allergic rhinitis; allergic respiratory diseases;

Introduction

In recent years, there has been a significant increase in the incidence of allergic respiratory diseases. Epidemiological studies show a higher incidence of conditions such as asthma and allergic rhinitis [1]. The World Health Organization (WHO) recognizes allergic diseases as one of the three major diseases that require significant effort to control and prevent in the 21st century. According to the ECAP (Epidemiology of Allergic Diseases in Poland) study, 40% of the Polish population suffers from allergic diseases, of which 25% have allergic rhinitis and 10% have asthma [2]. The pathogenesis of allergic diseases is complex, involving many environmental, genetic, epigenetic, and individual immune function factors [3]. Symptoms include watery rhinitis, itching, coughing, wheezing, and shortness of breath. Pharmacological treatment is based, among other things, on the use of antihistamines, immunosuppressants, and, in asthma, also long-acting beta-agonists and glucocorticosteroids [4]. Scientific studies also provide evidence of the effectiveness of ginseng (*Panax* species) in the treatment of allergic respiratory diseases due to its anti-inflammatory and immunomodulatory properties, which it owes to various chemical substances contained in the plant, e.g., ginsenosides Rb1, Rg1, Rg3 [5]. Among scientific papers, we can read about animal model studies showing that black ginseng extract alleviates allergic inflammation of the respiratory tract by inhibiting the signaling molecule protein kinase C, which has similar efficacy and mechanism of action as dexamethasone used in treatment. [6]. Red ginseng extract also showed similar biological activity in a mouse model, reducing airway hyperresponsiveness and inflammation, as well as mucus secretion in asthma, by inhibiting the production of ROS (reactive oxygen species) [7]. Human studies have shown that oral administration of a fermented red ginseng preparation reduces nasal obstruction in patients with perennial allergic rhinitis and decreases allergic reactions in skin prick tests [8]. This article aims to present the effects of ginseng use on reducing the symptoms of allergic respiratory diseases, as described in various scientific publications.

Materials and Methods

An analysis of the available literature for the purpose of selecting appropriate publications was conducted in May 2025 using the Google Scholar and Pubmed databases. Most of the scientific papers used in the review were published after 2019, and both English and Polish articles were included. The search strategy included the following keywords: “ginseng,” “asthma,” “allergic rhinitis,” and “allergic respiratory diseases.” The study included research conducted on both animal models and humans. The review selected publications that show the effect of ginseng use, including black and red ginseng extract, on reducing the symptoms of allergic respiratory diseases such as asthma and allergic rhinitis, and described the most important effects of ginsenosides contained in the plant in the treatment of these diseases.

Description of the state of knowledge

ASTHMA – pathomechanism,

Allergic respiratory diseases are caused by immune system disorders and are chronic in nature with an inflammatory basis. The primary diseases include allergic asthma (AAS) and allergic rhinitis (AR). They occur globally, with over 272 million people suffering from AAS alone [9]. The main pathomechanism of allergic asthma is based on an abnormal response of Th2 lymphocytes as a result of contact with allergens. In the mechanism of asthma development, we distinguish between Th2-dependent, Th2-independent, and mixed asthma. In Th2-type inflammation, allergens entering, for example, the respiratory tract are processed by antigen-presenting cells (APCs) and presented to Th2 lymphocytes. These lymphocytes secrete cytokines IL-4, IL-5, and IL-13, which activate B lymphocytes, which in turn produce IgE. When allergens re-enter the body, they bind to IgE, which activates mast cells via the FcεRI receptor, and these release mediators: leukotrienes (LT), histamine, and interleukins (IL). Under the influence of allergens, acetylcholine is also released, which together with inflammatory mediators causes bronchoconstriction [10]. The cytokine IL-5 leads to the migration of eosinophils to the lungs, releasing, among other things, the major basic protein MBP, which increases the release of acetylcholine and stimulates mast cells to release histamine and leukotrienes. The cytokine IL-13 is also responsible for inducing fibrosis of the airways. In response to damaged airway epithelium, e.g. under the influence of allergens, innate type 2 lymphoid cells (ILC2) are activated, leading to the production of IL-5 and IL-13. In asthma with low Th2 concentrations, IL-17, produced under the influence of CD4+ Th17 cells or CD8+

T cells, plays a major role. IL-17 leads to the migration of neutrophils to the lungs and hyperresponsiveness and remodeling of the airways [11]. Another substance that affects asthma is tumor necrosis factor alpha (TNF-alpha), which also leads to neutrophil migration and increases the concentration of cytokines such as IL-4, IL-5, and IL-13 [12, 13]. The pathomechanism of allergic rhinitis is similar to that described above for asthma, in which ILC2 cells and Th2 lymphocytes stimulating cytokine production, B lymphocytes responsible for IgE production, and dendritic cells (DC) presenting antigen play a decisive role [14]. Pharmacotherapy for asthma involves controlling the course of the disease through the use of glucocorticosteroids (GCS), inhaled beta2-mimetics, anticholinergic drugs, and anti-leukotriene drugs [15]. GCS, among other things, inhibit the activity of antigen-presenting cells and reduce the production of proinflammatory cytokines and TNF-alpha [16]. Beta2-mimetics lead to the widening of narrowed airways [17]. Anticholinergic drugs block muscarinic receptors, limiting bronchoconstriction, and also inhibit eosinophilia and tissue remodeling associated with inflammation in the airways [18]. Anti-leukotriene drugs block the leukotriene receptor CysLTR1 responsible for pro-inflammatory effects, limiting vascular permeability, bronchial mucus production, bronchoconstriction, eosinophilia, and bronchial remodeling [19]. H1 antihistamines, often used in AR, block the pro-inflammatory action of histamine, reducing edema, bronchoconstriction, limiting eosinophilia, neutrophil migration, and reducing the release of cytokines IL-4, IL-6, IL-8, and IL-13 [20].

Ginseng - characteristics

The *Panax* genus belongs to the *Araliaceae* family and includes several species, the most well-known of which are: *Panax ginseng* (Korean ginseng), *Panax quinquefolius* (American ginseng) and *Panax notoginseng* (Chinese ginseng) [21]. These plants occur naturally in East Asia and North America. Ginseng, also known as the king of herbs, is one of the most important medicinal plants in the Orient. This is evidenced by the presence of six herbs of this genus in the Chinese Pharmacopoeia (2015 edition) [22]. Ginseng owes its several thousand years of history in medicine to its various properties, including immunomodulatory, neuroprotective, anticancer, and body regeneration support properties. The main medicinal part of the plant is its root, but other parts, including leaves, flowers, and rhizomes, can also be used for therapeutic purposes. The most important chemical compounds found in Panax spp. are: triterpene saponins (ginsenosides), flavonoids, polysaccharides, sterols, phenolic acids, amino acids, fatty acids, and coumarins [23]. A detailed breakdown of the chemical components of ginseng is shown in the diagram (Fig. 1).

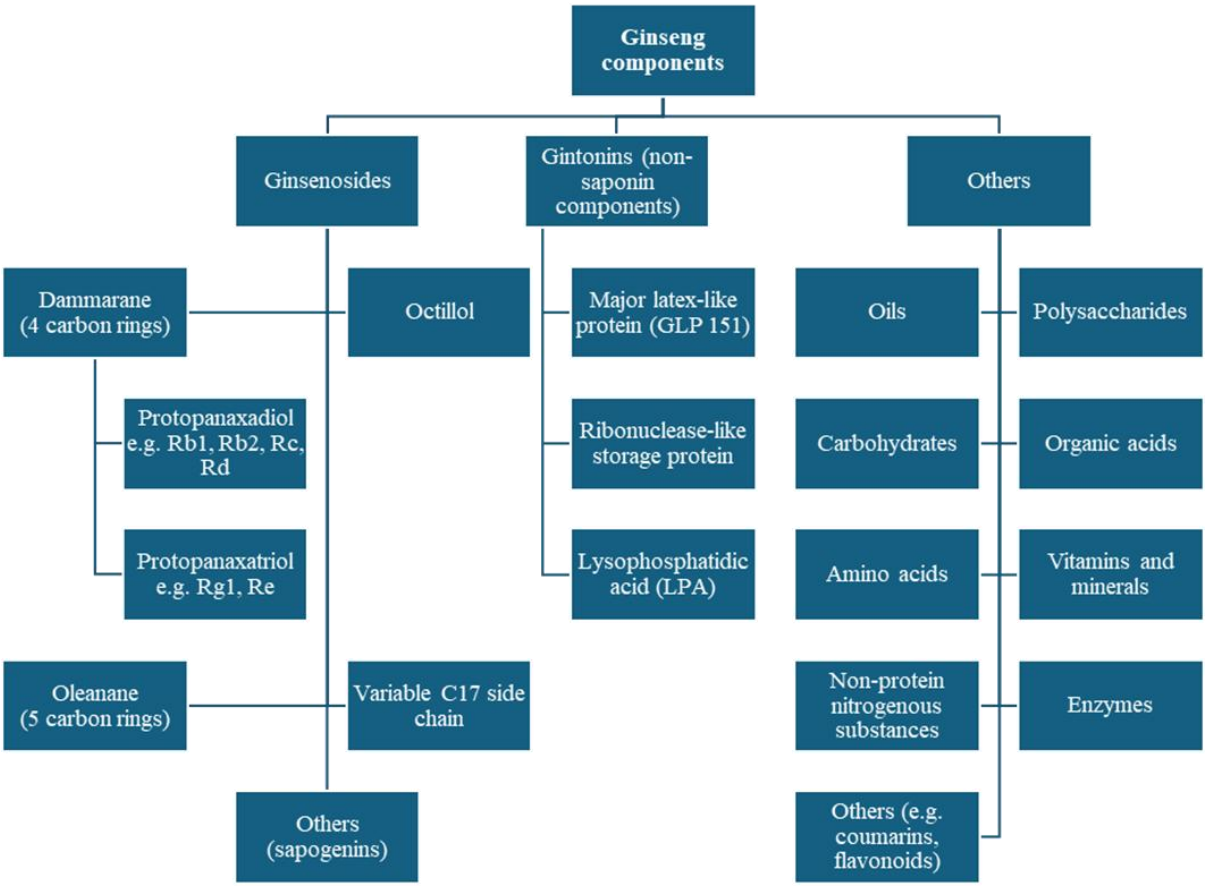

Figure 1. Chemical components of ginseng [based on 23, 24].

Ginsenosides, such as Rg1 and Rb1, exhibit antioxidant properties – they neutralize free radicals, increase the activity of antioxidant enzymes, and reduce lipid peroxidation and DNA damage [25]. Triterpene saponins, such as Rg1 and Rg6, are also responsible for regulating the immune response. They affect the activity of lymphocytes, macrophages, and cytokine production (e.g., TNF-α, IL-6) by modulating signaling pathways, such as NF-κB and MAPK [26]. Ginseng components, especially Rb1, have a cardioprotective effect – they improve the functioning of the vascular endothelium, have anti-inflammatory and anti-aggregatory effects, and lower blood pressure. In addition, they participate in the modulation of calcium channels, improve lipid metabolism, and regulate blood glucose levels [27]. It has also been shown that some ginsenosides, such as Rg3, Rh2, or Rd, may have anticancer effects because they have the ability to arrest the cell cycle, induce apoptosis, and inhibit tumor proliferation and associated angiogenesis. In addition, ginseng components may be chemosensitizing agents, increasing the sensitivity of cells to anticancer drugs such as gefitinib [23]. The beneficial

neuroprotective effects of ginsenosides are also often highlighted. Triterpene saponins, especially Rg1 and Rd, have been shown to improve cognitive function by modulating signaling pathways associated with neurogenesis, reducing oxidative stress, and inhibiting neuronal apoptosis. Ginsenosides may also limit brain damage after a stroke through their anti-inflammatory and antioxidant effects. Some of these compounds regulate the levels of neurotransmitters such as serotonin and dopamine, which may have a beneficial effect on mood disorders [28].

Ginseng in allergic respiratory diseases

The effect of selected types of ginseng

Panax ginseng

It has been shown that PG (*Panax ginseng*) has a beneficial effect on airway inflammation in mice by regulating the involvement of CD40 lymphocytes, cytokines, and MAP kinase signaling (29). The anti-inflammatory effects of PG on epithelial cells, mast cells, and basophils, as well as its immunomodulatory effects on dendritic cells, enhancing Treg or Th1 responses, also appear promising [5]. The types and methods of ginseng processing are presented in Fig. 2.

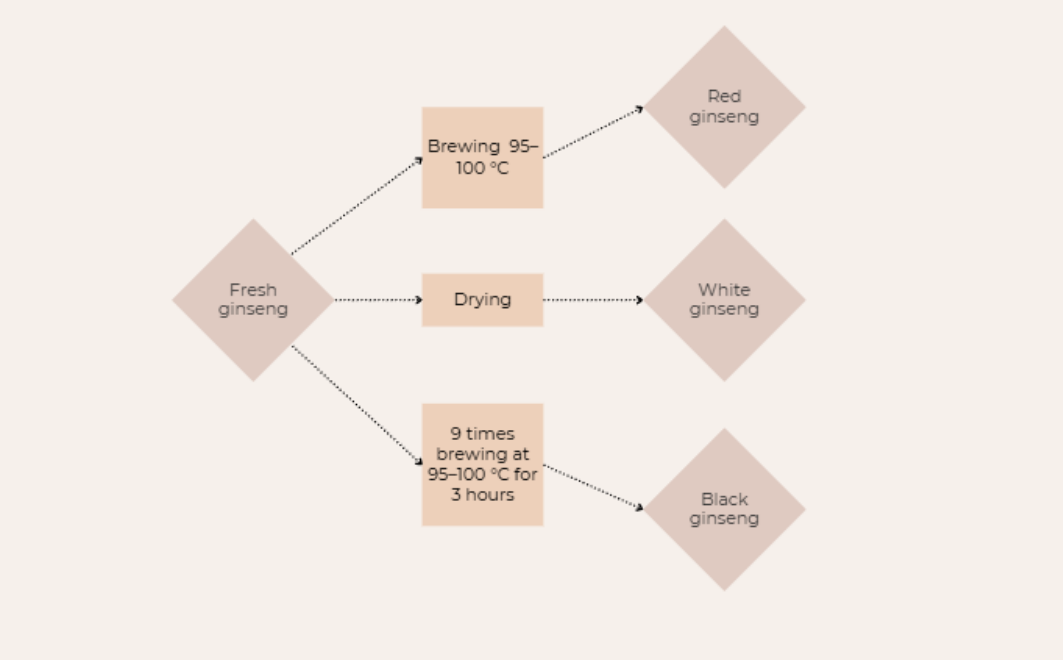

Figure 2. Types and methods of ginseng processing [30].

Black ginseng

The use of black ginseng extract (BGE) may alleviate allergic airway inflammation in mice. It has been found that BGE inhibits protein kinase C-mediated signaling molecules, thus having similar efficacy and mechanism of action to dexamethasone. Ginsenosides Rg3, Rg5, and Rk1 are responsible for this activity. These components, through phosphorylation of PKCθ and STAT6, have a suppressive effect on IL-4 expression. BGE may therefore prove to be a safer substitute for dexamethasone, the use of which is associated with many side effects [31]. A study conducted by a team of scientists from China has shown that the active substances contained in black ginseng can influence the occurrence of inflammatory responses by regulating the TLR signaling pathway. The results of this study assign particular importance to ginsenoside Rb1. This factor can inhibit the expression of TLR3 and TRIF mRNA, subsequently suppressing the activation of the TLR3/TRIF signaling pathway, thereby reducing the inflammatory response in the lungs of rats with asthma [6]. It is therefore an interesting therapeutic option for the treatment of allergic asthma (AAS), although its efficacy should be proven in humans.

Korean red ginseng

KRG (Korean red ginseng) was also studied, whose biological activity, according to research, is similar to that of black ginseng: the results were similar in terms of immunomodulation, regulation of fatigue and blood pressure, improvement of cognitive function, and antioxidant activity [32]. In a study on mice, KRG alleviated asthma symptoms, including airway hyperresponsiveness, airway inflammation, and mucus secretion. These effects resulted from KRG's inhibition of ROS (reactive oxygen species) production, which prevented the depletion of oxidative proteins, i.e., NRF2 and HO-1. This effect was attributed to ginsenosides Rb1 and

Rg3. In addition, as a result of its antioxidant activity, it reduced the inflammatory response by suppressing NF-κB phosphorylation and, as a result, reducing iNOS expression [7].

A Korean study using a mouse model, in which mice previously sensitised to ovalbumin were used, demonstrated the positive effect of KRG in allergic rhinitis (AR). It was found that serum IgE antibody levels were significantly lower in the KRG-treated group than in the group not receiving KRG. Similarly, the number of eosinophils was lower. Immunohistochemical analysis showed that there were fewer IL-4, IL-5 and MUC5AC-positive cells in the KRG-treated group compared to the group that did not receive KRG. These results suggest that KRG reduces the allergic inflammatory response in the nose in an allergic mouse model by reducing Th2 cytokines [33].

A study of 60 patients found that KRG works similarly to antihistamines in reducing nasal discharge, relieving nasal and eye itching, and improving sleep disturbances in patients with AR [34]. Another study involving 59 patients yielded similar results, confirming that oral administration of a fermented KRG preparation reduces nasal congestion in patients with perennial AR. In addition, patients who used KRG for more than 4 weeks showed a reduction in allergic reactions to histamine and inhaled allergens in skin prick tests [8]. As already demonstrated in a previous study in a mouse model, this effect is associated with a reduction in total IgE antibody levels in patients with AR compared to those receiving placebo, as well as with the inhibition of Th2 cytokines and eosinophil recruitment. It was found that serum IL-4 levels and eosinophil counts in nasal swabs were significantly reduced in both the antihistamine and CRG groups [34].

Table 1. Mechanism of action of selected types of ginseng in allergic respiratory diseases.

Ginseng type	Effect	Source
PG – *Panax ginseng*	In studies on mice: – Reduction of airway inflammation through regulation of CD40 lymphocyte involvement, cytokines, and MAP kinase signaling – Anti-inflammatory effect on epithelial cells, mast cells, and basophils – Immunomodulatory effect on dendritic cells, enhancing Treg or Th1 responses	[5] [26]
BG – black ginseng	In studies on mice: – Inhibition of signaling molecules mediating protein kinase C – Reduction of IL-4 expression through phosphorylation of PKCθ and STAT6 signaling pathways – Inhibition of TLR3/TRIF signaling pathway activation	[28] [30] [29]
KRG – Korean red ginseng	In studies on mice: – Alleviation of asthma symptoms, including airway hyperresponsiveness, inflammation, and excessive mucus secretion by inhibiting ROS production – Reduction in serum IgE antibody levels – Reduction in eosinophil counts and IL-4, IL-5, and MUC5AC-positive cell counts In clinical studies involving humans – Reduction in serum IL-4 cell levels and eosinophil counts in nasal smears	[31] [32] [33]

Abbreviations: MAP kinase - Mitogen-activated protein kinase; IL – Interleukin; PKCθ - Protein kinase C Theta; STAT6 - Signal transducer and activator of transcription 6; TLR3 - Toll-like receptor 3; TRIF - Toll/IL-1R domain-containing adaptor inducing IFN-β; MUC5AC - Mucin-5AC

The effect of ginsenosides contained in ginseng

Ginsenosides Rb1, Rg1, Rg3

Ginsenosides can modulate DC cell function and generate anti-inflammatory responses. In experiments with an 80% ethanol extract of dried P. ginseng roots consisting of ginsenosides Rb1, Rd, and Rg3, the expression of CD11c, CD40, CD80, and CD86 decreased during the differentiation of human CD14 monocytes into DC cells. Ginsenoside Rg1 was found to have the strongest effect on inducing antigen-specific splenocyte proliferation, IFN-γ and IL-5 production, as well as antigen-specific IgG, IgG1, and IgG2a antibody levels. Oral

administration of ginsenoside Rb1 to mice with ovalbumin-induced AAS can reduce airway resistance and decrease immune cell infiltration in BALF, as well as lower IL-4 levels and ovalbumin-specific IgE antibody levels associated with inhibited GATA-3 expression, while increasing T-bet expression and IFN-γ levels in BALF [5].

Rg3, a rare saponin with strong anti-inflammatory properties, has also been studied by a team of scientists from Taiwan, who demonstrated that it can inhibit the hypertrophy of airway goblet cells, thereby reducing mucus production in asthma, as well as reducing collagen expression, eosinophil infiltration, and airway hyperresponsiveness. These effects were observed in the lungs of mice [35]. In in vitro studies on human asthmatic respiratory epithelial cells and A549 cells, ginsenoside Rg3 was shown to be effective in inhibiting the activation of the NF-κB pathway, thereby reducing COX-2 production. The levels of secreted IL-4, TNF-α, and eotaxin were also reduced [36].

Ginsenoside Rd

Another component of ginseng that alleviates AR symptoms is ginsenoside Rd. This compound reduced the secretion of IgE, IL-4, IL-5, and IL-13 antibodies in mice and in RBL-2H3 cell cultures in vitro. In addition, it contributed to a reduction in the number of mast cells and eosinophils in BALF [37]. The therapeutic effect of ginsenoside Rd may also be related to its effect on restoring the composition of the intestinal microbiota disturbed by ovalbumin, including bacteria from the Bacteroidetes, Actinobacteria, and Firmicutes groups. This suggests that the mechanisms of action of natural compounds in the treatment of AR may go beyond the anti-inflammatory effect and include modulation of the microbiota to achieve an anti-allergic effect [38].

Notoginsenoside R1

In mouse models, intravenous injection of notoginsenoside R1 (PNR1) reduced airway hyperresponsiveness, immune cell infiltration, mucus production, and IgE production in

ovalbumin-induced asthma by inhibiting the pIKK–NF–κB signaling pathway, leading to decreased levels of IL-4, IL-5, IL-8, IL-13, and TNF-α [5].

Several studies have compared the effects of PNR1 with those of GCS. Although steroids are the basis for asthma treatment, long-term use can inhibit the repair process of damaged airway epithelium, which is destroyed in the course of this disease [39]. The simultaneous administration of PNR1 and dexamethasone in asthmatic mice and in model human airway epithelial cells (16HBE line) prevented this adverse effect. The use of PNR1 in asthmatic mice led to a reduction in the number of eosinophils, neutrophils, and lymphocytes in bronchial secretions [40]. However, the decrease in the number of neutrophils and lymphocytes was smaller than after the use of dexamethasone, which may indicate a less broad anti-inflammatory effect compared to GCS. Nevertheless, symptoms such as bronchial hyperresponsiveness, mucus hypersecretion, and airway inflammation were reduced as effectively as in the dexamethasone-treated group [41]. PNR1 has also been shown to be effective in alleviating AR in mice. Acting through AMPK (AMP-activated protein kinase) signaling pathways, it preserved mitochondrial integrity by influencing the activation of Drp1 genes and inhibiting ROS production [42].

Table 2. The effect of selected ginsenosides in allergic respiratory diseases.

Ginsenoside	Effect	Source
PNR1	− Supporting the repair of damaged respiratory tract epithelium − Reducing the number of eosinophils, neutrophils, and lymphocytes in bronchial secretions − Inhibiting ROS production − Increasing mitochondrial stability − Reducing bronchial hyperresponsiveness − Reduction in bronchial mucus hypersecretion − Reduction in IgE antibody production − Reduction in IL-4, IL-5, IL-8, IL-13, and TNF-α production (through inhibition of the pIKK–NF–κB pathway)	[5] [40] [41] [42]
Rd	− Reduction in IgE, IL-4, IL-5, and IL-13 secretion − Reduction in the number of mast cells and eosinophils in BALF − Modulation of the gut microbiota to achieve an anti-allergic effect	[37] [38]

Rb1	– Reduction of airway hyperresponsiveness, airway inflammation, and mucus secretion in the airways – Reduction of airway resistance, immune cell infiltration in BALF – Inhibition of ROS production – Reduction of IL-4 and IgE levels – Reduction of inflammatory response by inhibiting activation of the TLR3/TRIF signaling pathway	[5] [6] [7]
Rg1	– Induction of splenocyte proliferation, IFN-γ and IL-5 production, as well as antigen-specific IgG, IgG1, and IgG2a levels – Suppressive effect on IL-4 expression – Reduction of tracheal goblet cell hyperplasia	[5]
Rg3	– Reduction of airway hyperresponsiveness, airway inflammation, and airway mucus secretion – Inhibition of ROS production – Reduction of collagen expression – Reduction of COX-2 expression by inhibiting NF-*κ*B pathway activation	[7] [32] [35] [36]
Rg5 Rk1	– Suppressive effect on IL-4 expression through phosphorylation of PKCθ and STAT6 pathways	[32]

Abbreviations: PNR1 - notoginsenoside R1; ROS - reactive oxygen species; TNF-α tumor necrosis factor α; pIKK - phosphoinositide 3 kinase-associated kinase; NF–κB - nuclear factor kappa B; BALF - bronchoalveolar lavage fluid; IFN-γ - interferon γ; COX-2 – cyclooxygenase 2

Conclusion

The latest sources show that the use of ginseng in medicine has a number of positive effects. Studies conducted on animal models confirm the alleviation of symptoms of both allergic rhinitis and asthma. A reduction in Th2 cytokine levels, a decrease in the number of eosinophils, and a decrease in IgE antibodies in serum have been demonstrated. In individuals with allergic rhinitis, clinical symptoms were reduced, including decreased nasal secretion, less itching of the eyes and nose, and reduced nasal congestion. Studies have shown that ginsenosides, which are found in ginseng, affect the body's immune response by regulating, among other things, the number of eosinophils, neutrophils, and lymphocytes in bronchial secretions, reducing the expression of collagen and the secretion of IgE antibodies, as well as IL-4, IL-5, and IL-13. This results in a reduction in the severity of clinical symptoms and a decrease in inflammatory response markers. The use of ginseng in the treatment of allergic respiratory diseases requires further research involving humans, but the studies that have already been conducted show promising results and a wide range of effects of Panax spp.

Disclosure

Author contributions

Conceptualization: WS

Methodology: WS, GŻ, FS, MD, JD, KM

Formal analysis: WS, GŻ, FS, MD, JD, KM

Investigation: WS, GŻ, FS, MD, JD, KM

Resources: WS, GŻ, FS, MD, JD, KM

Check: WS, KG

Data curation:

Writing - rough preparation: WS, GŻ, FS, MD, JD, KM

Writing - review and editing: WS, GŻ, FS, MD, JD, KM, KG

Supervision: WS, KG

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