Mini-Review

Acupuncture and Herbal Medicine

# The role of ginseng as an anti-asthmatic agent

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## Introduction

Asthma is an inflammatory obstructive respiratory lung disease characterized by limited airflow. It is estimated that more than 300 million people worldwide are affected by asthma, and this number is expected to increase over the next few years[1]. Uncontrolled asthma is associated with a severely impaired quality of life and increased mortality rates[2]. Other adverse effects of poorly controlled asthma include obesity, pneumonia, impaired concentration, and depression[3]. Despite the wide availability of diagnostic methods and therapies, asthma is currently underdiagnosed and undertreated[4]. The current mainstay therapy for mild to moderate persistent asthma is a daily low-dose inhaled corticosteroid (ICS) and a combination of a daily ICS and long-acting β-agonist (LABA), respectively. The side effects of using long-term ICS include oropharyngeal candidiasis, vocal cord changes, nasal bleeding, and mucosal irritation. Bronchodilators such as β-agonists are widely considered safe and effective, with tremors and tachycardia being the most frequently reported side effects[5]. However, certain individuals may be at an increased risk of arrhythmias and cardiomyopathy owing to the positive ionotropic and chronotropic effects of β-agonists[6].

The therapeutic potential of ginseng has grown in recent years, likely due to increased interest in alternative and complementary medicines. The anti-inflammatory properties of ginseng have been demonstrated in the treatment of various diseases, including cardiovascular diseases, diabetes, and cancer[7]. Ginseng is metabolized by the gut flora in stepwise deglycosylation reactions, resulting in the absorption of ginsenoside metabolites into the systemic circulation[8]. Airflow limitations in lung diseases such as asthma, chronic obstructive pulmonary disease (COPD), lung fibrosis, and respiratory tract infections are caused by excessive inflammatory response to irritants, and ginsenosides have been shown to downregulate pro-inflammatory cytokines and transcription factors such as nuclear factor kappa B (NF-κB)[9]. Moreover, naturally occurring ginsenosides have been shown to play a role in preventing and ameliorating asthma symptoms in mouse models. This mini-review summarizes current studies on ginseng and its role as an anti-asthmatic agent in murine models. The goal of this mini-review is to discuss the potential therapeutic role of ginseng as an alternative therapy to ameliorate the symptoms of asthma.

## Methods

### *Search strategy, search selection, inclusion and exclusion criteria*

The PubMed, EMBASE, and Cochrane databases were used to conduct a systematic search of published literature until March 29, 2023. The following search terms were used: (((ginseng[Title/Abstract]) OR (ginsenosides[Title/Abstract])) AND ((asthma[Title/Abstract]) OR (airway inflammation[Title/Abstract]))). A PubMed search yielded 23 papers. No additional papers were identified by searching the Cochrane and EMBASE databases after filtering duplicates. After reading the abstracts, eight full-text articles were reviewed. Eight studies were excluded because they were not specific to asthma. Five studies were excluded because they were not specific to ginseng or asthma. Two studies were excluded because they were not specific to ginseng. Studies examining the effects of ginseng on the development or amelioration of asthma symptoms were included. A flowchart summarizing the study identification and selection process is shown in Figure 1.

### *Data extraction*

The following variables were extracted from each study: first author information, year of publication, materials studied, animal model, treatment modality, and proposed mechanism of action. The following outcome measures were extracted: mucosal hypersecretion, epithelial hyperplasia, granulocyte infiltration/expression, lymphocyte infiltration/expression, cytokine infiltration/expression,

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reactive oxygen species (ROS) generation/reduction, goblet cell proliferation, hematological parameters, and morphological changes. These outcome measures represent pathophysiological changes that occur in response to asthma. Two authors independently extracted the information for each study and any discrepancies were resolved by a third author.

## Results

Eight animal studies were identified. The most frequently administered asthmatic-inducing agent was ovalbumin (OVA) with and without the adjuvant, aluminum chloride. One study used lambda-cyhalothrin (LTC), a synthetic pyrethroid, as an inflammatory agent. Five studies followed OVA sensitization by administration of Korean red ginseng (KRG) and/or Korean white ginseng (KWG) as anti-asthmatic agents. The remaining studies used derivatives of ginseng, including fermented and aged ginseng sprouts (FAGS) and the ginsenosides Rg3 and Rh2. Seven studies analyzed inflammatory cell counts in bronchoalveolar lavage fluid (BALF), six studies examined pathological changes in airway histology through mucus hypersecretion, epithelial hyperplasia, and/or inflammatory cell infiltration, four studies measured changes in airway hyperresponsiveness (AHR), two studies measured ROS production, and three studies analyzed NF-κB activity through western blotting or immunohistochemical staining (IHC).

Results showed that treatment in OVA-sensitized mice with KRG, KWG, or ginsenoside metabolites significantly reduced BALF levels of interleukin (IL)-4, IL-5, IL-13, tumor necrosis factor-alpha (TNF-α), neutrophils, and eosinophils. Hematoxylin and eosin staining of lung tissue also revealed significantly decreased levels of epithelial hyperplasia and goblet cell mucus secretion in the KRG-, KWG-, and ginsenoside Rh3–treated groups. However, in one study, KRG treatment resulted in a mild increase in bronchial epithelial hyperplasia. KRG, KWG, ginsenoside Rh2, and ginsenoside Rg3 significantly reduced the AHR in five studies. KRG and ginsenoside Rg3 significantly reduced ROS levels. Furthermore, KRG, KWG, and ginsenoside Rh2 decreased NF-κB phosphorylation and nuclear translocation in three studies. Serum IgE expression also reduced in ginseng-treated groups in seven studies. One study showed that KRG or KWG treatment did not mediate changes in OVA-induced IgG1 or IgG2 expression, whereas another showed that ginsenoside Rg3 significantly decreased IgG1, but increased IgG2 expression. Finally, KRG demonstrated greater efficacy in controlling inflammatory cell infiltration and mucosal thickening than KWG. A comprehensive summary of these results is presented in Table 1.

## Discussion

### *The role of regulatory T cells (Tregs) and the T-helper cell type 1 (Th1)/T-helper cell type 2 (Th2) imbalance*

Immune regulation involves homeostasis of T helper cells, particularly Th1 and Th2 cells. Th1 cytokines mediate the cellular immunity pathway involved in protection against viruses and intracellular pathogens, whereas Th2 cytokines mediate the humoral immunity pathway to upregulate antibody production against extracellular pathogens. Overactivation of either pathway can result in disease and downregulation of other pathways. Tregs regulated the responses of both groups by maintaining the responses of Th1 and Th2 cells within the normal range to limit excessive inflammation and mediate peripheral tolerance. Uncontrolled inflammation and disease occur when Tregs are dysfunctional and fail to suppress the excessive Th1 or Th2 responses[18].

Th2 cytokines such as IL-4, IL-5, and IL-13 play crucial roles in the pathophysiology of allergic diseases such as asthma. Tregs prevent the immune system from overreacting to inhaled allergens *via* Th2 cytokines. In asthma, reduced number or impaired function of Tregs can cause uncontrolled Th2 cell activation and cytokine proliferation, ultimately inhibiting Th1 cells and disrupting the Th1/Th2 balance[19]. This accelerates eosinophil migration and maturation, further increasing the production of IgE and pro-inflammatory cytokines. These molecules lead to airway obstruction in asthma attacks *via* increased smooth muscle constriction, airway remodeling in response to ROS, inflammatory cell migration, and mucus hypersecretion in the airways[10]. In our analysis, ginseng and its derivatives reduced the number of inflammatory cells such as eosinophils, ultimately decreasing the production of Th2 cytokines

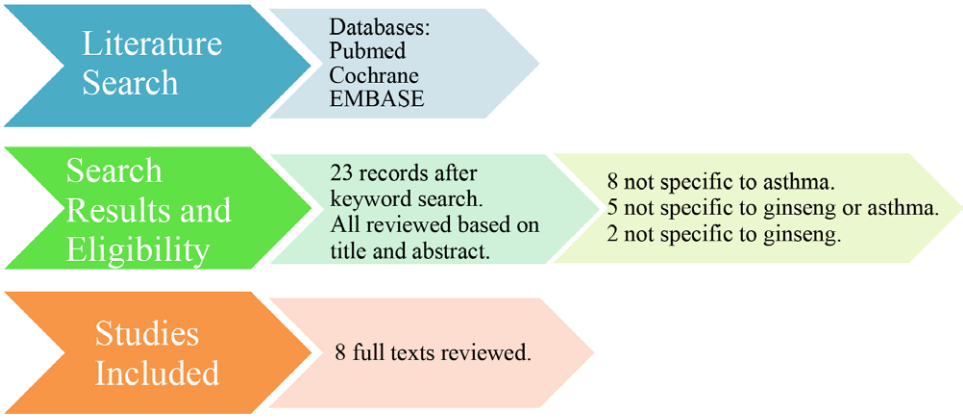


**Figure 1.** Flowchart summarizing study identification and selection process.

**Table 1**

**Summary of studies on ginseng and asthma**

| First author, year published | Material | Animal model | Treatment | Outcome measures | Main findings | Proposed mechanism |
|---|---|---|---|---|---|---|
| Kim, 2022[10] | 1) OVA<br>2) KRG<br>3) DEX | Female BALB/c mice | 1) Normal control<br>2) OVA treatment only<br>3) 100 mg/kg KRG days 18–23 post-OVA<br>4) 300 mg/kg KRG days 18–23 post-OVA<br>5) 3 mg/kg DEX days 18–13 post-OVA | 1) AHR<br>2) Serum IgE<br>3) BALF evaluation of Th2 cytokines and ROS activity<br>4) Mucus secretion on lung histology<br>5) Western blot protein levels of NF-κB, phosphor-NF-κB, iNOS, HO-1, NRF2, lamin-B1, and B-actin | 1) AHR values significantly decreased in KRG groups.<br>2) Serum IgE was significantly lower in the KRG-treated groups.<br>3) BALF levels of IL-4, 5, and 13 were significantly lower in the KRG-treated groups.<br>4) Mucus production significantly decreased in the KRG groups.<br>5) ROS, HO-1, NRF2, and iNOS decreased significantly in the KRG groups | By reducing Th2 cytokines, KRG combats asthma progression by significantly reducing inflammatory factors and mucus hypersecretion. KRG also exerts an anti-asthmatic effect by reducing the production of reactive oxidative species |
| Ryu, 2022[11] | 1) OVA<br>2) FAGS<br>3) Ginsenoside CK | Female C57BL/6 mice | 1) OVA + low-dose FAGS (LFAGS): 300 mg/day for 39 days.<br>2) OVA + high dose FAGS (HFAGS): 600 mg/day for 39 days.<br>3) OVA + CK: 50 μM/kg/day for 39 days | 1) AHR<br>2) BALF inflammatory cell, Th2 cytokine, and histamine levels<br>3) Total and OVA-specific plasma IgE<br>4) Lung inflammation, mucus production, and collagen deposition on lung histology<br>5) Iron in lung tissue<br>6) MDA concentration, ER stress, and apoptotic markers | 1) AHR decreased in the FAGS and CK group.<br>2) Inflammatory cell count, Th2 cytokines, histamine, and tryptase levels decreased in the FAGS and CK group.<br>3) Total and OVA-specific IgE plasma significantly decreased in the FAGS and CK group.<br>4) Inflammatory cell infiltration and goblet cell count significantly decreased in the FAGS and CK group. The number of collagen fibers decreased slightly in the FAGS and CK group.<br>5) Iron deposition decreased in the HFAGS and CK groups.<br>6) ROS, MDA, ER stress markers, and apoptotic levels reduced in the HFAGS and CK group | Compared to ginseng, FAGS has been uniquely fermented and aged to increase its ginsenoside contents. CK is a secondary ginsenoside produced by primary ginsenoside fermentation and intestinal microbiota.<br>FAGS and CK inhibit inflammation and ferroptosis by preventing iron accumulation, GSH depletion, and lipid peroxidation. CK also exerts an anti-asthmatic effect by inhibiting airway resistance, IgE plasma levels, airway inflammation, and mucus secretion due to its higher bioavailability and solubility compared to its parent ginsenosides |

(*Continued*)

**Table 1**
(Continued)

| First author, year published | Material | Animal model | Treatment | Outcome measures | Main findings | Proposed mechanism |
|---|---|---|---|---|---|---|
| Lee, 2020[12] | 1) OVA<br>2) KRGWE<br>3) DEX | Female BALB/c mice | 1) OVA + DEX 1 mg/kg/day for 5 days<br>2) OVA + KRGWE 5 mg/kg/day for 5 days<br>3) OVA + KRGWE 25 mg/kg/day for 5 days<br>4) OVA + KRGWE 50 mg/kg/day for 5 days | 1) Serum IgE<br>2) BALF inflammatory cell count<br>3) Mucous secretion, inflammatory cell inflammation, and epithelial cell hyperplasia on lung histology<br>3) IL-12, Il-4, and IL-6 in lung tissue<br>4) Activation of T helper cell transcription factors and NF-κB/COX-2 pathways | 1) There was a dose-dependent decrease in serum IgE concentrations in the KRGWE groups.<br>2) WBC and neutrophil levels in BALF significantly reduced in the KRGWE groups.<br>3) Mucous hypersecretion, epithelial cell hyperplasia, and inflammatory cell infiltration reduced in all KRGWE groups.<br>3) KRGWE suppressed IL-4, IL-6, and IL-12 levels at all levels of treatment.<br>4) NF-κB translocation and COX-2 expression decreased in the 50 mg/kg/day KRGWE group | KRG reduces inflammation through the reduction of IL-12, IL-4, and IL-6 cytokines, and NF-κB/COX-2 and PGE2 pathways. This limits epithelial and goblet cell hyperplasia and mucous hypersecretion, thus reducing airway blockage associated with asthma |
| Lim, 2015[13] | 1) OVA<br>2) KRG<br>3) KWG | Female BALB/c mice | 1) 30, 90, and 300 mg/kg KRG 15-24 days post-OVA treatment<br>2) 30, 90, and 300 mg/kg KWG 15–24 days post-OVA treatment<br>3) 30 µL PBS containing 25 g OVA on days 22, 23, and 24 intranasally | 1) AHR<br>2) Inflammatory cell count in BALF<br>3) OVA-specific IgE, IgG1, and IgG2a counts in serum<br>4) Mucosal thickening and inflammatory cell infiltration on lung histology<br>5) IL-4, IL-5, IL-6, and TGF-β in peribronchial lymph nodes | 1) AHR decreased in the KRG and KWG groups.<br>2) Inflammatory cell count significantly decreased in the KRG and KWG-treated groups.<br>3) Serum IgE counts were lower in the KRG and KWG groups. However, there was no effect on OVA-specific IgG1 and IgG2a serum levels.<br>4) Inflammatory cell infiltration and mucosal thickening decreased in KWG and KRG groups compared to PBS group, and KRG had a greater effect than KWG.<br>5) Cytokine levels decreased in the KWG and KRG groups, and KRG had a greater effect than KWG | Both KRG and KWG suppress the hyperresponsive Th2 cytokine production associated with the inflammatory effects of asthma. While KRG and KWG both show anti-asthmatic effects in OVA-induced mice, the effects of KRG appear greater than those of KWG. KWG is produced by sun drying fresh ginseng, whereas KRG is produced by steaming fresh ginseng then drying to obtain a moisture content of <15%. The steaming process may increase the bioactivity of KRG compared to KWG |

(*Continued*)

**Table 1**

**(Continued)**

| First author, year published | Material | Animal model | Treatment | Outcome measures | Main findings | Proposed mechanism |
|---|---|---|---|---|---|---|
| Huang, 2021[14] | 1) OVA<br>2) Ginsenoside Rg3<br>3) Prednisolone | Female BALB/c mice | 1) Normal control<br>2) OVA treatment only<br>3) Normal mice + 5 mg/kg prednisolone 1 h prior challenge<br>4) OVA + 5 mg/kg prednisolone 1 h prior challenge<br>5) OVA + 5 or 10 mg/kg ginsenoside Rg3 1 h prior to challenge<br>6) BEAS cells + 0-30 μM ginsenoside Rg3 1 h prior to challenge | 1) Inflammatory cell count in BALF<br>2) AHR<br>3) Eosinophil and goblet cell infiltration in lung tissue using the H&E stain<br>4) Serum IgG1, IgG2a, and IgE<br>5) ROS expression | 1) Eosinophils, goblet cells CCL11, CCL24, TNF-α, IL-5, IL-4, IL-13, and IL-6 in BALF were reduced in mice treated with ginsenoside Rg3 and prednisolone.<br>2) AHR significantly reduced in mice treated with ginsenoside Rg3 and prednisolone.<br>3) Ginsenoside Rg3 reduced OVA-induced goblet cell hyperplasia and eosinophil infiltration in lungs.<br>4) Ginsenoside Rg3 significantly decreased IgG1 and IgE and increased IgG2 expression.<br>5) ROS expression decreased in ginsenoside Rg3-treated BEAS cells | Ginsenoside Rg3 activates PI3K/AKT/mTOR pathways and inhibits the NF-κB pathway to reduce inflammatory mediators in lung epithelial cells. Ginsenoside Rg3 also inhibits COX-2 expression in IL-1β–activated lung epithelial cells, and decreases the levels of eotaxin, IL-13, IL-9, IL-4, and IL-6 in human asthmatic airway epithelial tissue |
| Mohi El-Din, 2014[15] | 1) LTC<br>2) KRG<br>3) Garlic extract | Female albino rats | 1) 9.34 mg/kg LTC daily for 21 days<br>2) 200 mg/kg KRG + 9.34 mg/kg LTC daily for 21 days<br>3) 100 mg/kg garlic + 9.34 mg/kg LTC daily for 21 days | 1) Hematological parameters including RBC count, WBC count, hemoglobin, PCV%<br>2) Evaluation of lung histology | 1) No significant differences were found in any parameters at day 15. However, the LTC + KRG group showed significant differences in RBC, Hb, and PCV% at day 21 compared to LTC alone.<br>2) The KRG + LTC group demonstrated mild bronchiole hyperplasia and decreased neutrophil count compared to the TLC-only group | KRG is protective against the asthmatic and toxic effects of LTC. KRG enhances phagocytosis, NK cell activity, and interferon production to ameliorate the inflammatory effects of exogenous stress factors |

(*Continued*)

**Table 1**
**(Continued)**

| First author, year published | Material | Animal model | Treatment | Outcome measures | Main findings | Proposed mechanism |
|---|---|---|---|---|---|---|
| Lee, 2019[16] | 1) OVA<br>2) KGC3P (1:3 mixture of KRG and S. plebeia)<br>3) Nepetin<br>4) Montelukast | Male BALB/c mice | 1) Normal control<br>2) OVA control<br>3) OVA + montelukast 10 mg/kg daily for 4 weeks<br>4) OVA + nepetin 20 mg/kg daily for 4 weeks<br>5) OVA + KGC3P 200 mg/kg daily for 4 weeks<br>6) OVA + KGC3P 100 mg/kg daily for 4 weeks | 1) AHR<br>2) BALF, lung, and MLN inflammatory cell count<br>3) Serum IgE, IL-4, IL-5, IL-13, and IFN-γ levels in BALF and splenocytes<br>4) Inflammatory cell infiltration and collagen deposition in lung and tissue<br>5) PPARγ mRNA, p-Akt, p-PTEN, and β-actin expression *via* western blot | 1) KGC3P, nepetin, and montelukast groups demonstrated significantly decreased AHR.<br>2) Number of total lung cells decreased in KGC3P, nepetin, and montelukast groups. Total MLN cell count decreased in KGC3P 200 mg/kg group.<br>3) IL-4, IL-5, and IL-13 levels in BALF and splenocytes were significantly lower in the KGC3P, nepetin, and montelukast groups. There was no difference in IFN-γ levels.<br>4) KGC3P and montelukast significantly reduced IL-4, IL-13, IL-17, and TNF-α levels in lung tissue.<br>5) PPARγ mRNA significantly increased in KGC3P and nepetin groups however decreased in the montelukast group. KGC3P and montelukast reduced phosphorylation levels of AKT and PTEN | KRG, S. plebeia, and nepetin induce anti-asthmatic effects comparable to that of montelukast through decreased eosinophilic and granulocyte infiltration in the lungs, BALF, and MLN |
| Li, 2015[17] | 1) OVA<br>2) G-Rh2<br>3) DXM | Female BALB/c mice | 1) Normal control<br>2) OVA + G-Rh2 50 mg/kg at 24-h intervals from days 17 to 19<br>2) OVA + G-Rh2 100 mg/k at 24-h intervals from days 17 to 19<br>3) OVA + DEX 0.5 mg/kg at 24-h intervals from days 17 to 19 | 1) BALF inflammatory cell count<br>2) Serum total IgE and OVA-specific IgE<br>3) Evaluation of lung histology<br>4) Nuclear protein extraction of NF-κB<br>5) Western blot of inflammatory proteins<br>6) AHR | 1) BALF eosinophil, lymphocyte, and neutrophil count significantly decreased in the G-Rh2 groups.<br>2) Total and OVA-specific IgE serum levels significantly reduced in the G-Rh2 groups.<br>3) G-Rh2 reduced inflammatory cytokines in lung tissue.<br>4) G-Rh2 prevents OVA-induced NF-κB translocation and p38 MAPK phosphorylation.<br>5) OVA-induced increase in IL-4, IL-5, and IL-13 significantly reduced in the G-Rh2 groups<br>6) G-Rh2 decreased AHR | G-Rh2 prevents the activation of inflammatory cells, cytokines, and inflammatory pathways NF-κB and MAPK that induce symptoms of asthma |

AHR: Airway hyperresponsiveness; AKT: Protein kinase B; BALF: Bronchoalveolar lavage fluid; BEAS: Human bronchial epithelial cell; CK: Compound K; COX-2: Cyclooxygenase-2; DEX: Dexamethasone; ER: Endoplasmic reticulum; FAGS: Fermented-aged ginseng sprouts; G-Rh2: Ginsenoside Rh2; GSH: Glutathione; Hb: Hemoglobin; HFAGS: High dose fermented and aged ginseng sprouts; HO-1: Heme oxygenase-1; IFN-γ: Interferon-gamma; IL: Interleukin; iNOS: inducible nitric oxide synthase; KRG: Korean red ginseng; KRGWE: Korean red ginseng water extract; LFAGS: Low dose fermented and aged ginseng sprouts; LTC: Lambda-cyhalothrin; MAPK: Mitogen-activated protein kinase; MDA: Malondialdehyde: MLN: Mesenteric lymph node; mTOR: Mammilian target of rapamycin; NF-κB: Nuclear factor kappa B; NK: Natural killer; NRF2: Nuclear factor erythroid 2-related factor 2; OVA: Ovalbumin; PBS: Phosphate-buffered saline; PCV: Packed cell volume; PGE2: Prostaglandin E2; PI3K: Phosphoinositide 3-kinase; PPARγ: Peroxisome proliferator-activated receptor gamma; p-PTEN: Phosphorylated phosphatase and tensin homolog deleted on chromosome 10; RBC: Red blood cell; ROS: Reactive oxygen species; TGF-β: Transforming growth factor-beta; Th2: T-helper cell type 2; TNF-α: Tumor necrosis factor-alpha; WBC: White blood cell.

and IgE in the serum. Ginseng inhibits the inflammatory effects of asthma by inhibiting Th2 cytokines and restoring Th1/Th2 balance.

### *The role of NF-κB and cyclooxygenase-2 (COX-2) inflammatory pathways*

Asthma progression is well characterized by the activation of various inflammatory signaling pathways mediated by intracellular proteins, particularly NF-κB and COX-2. NF-κB is a key transcription factor that upregulates inducible nitric oxide synthase (iNOS), an enzyme that increases nitric oxide production. This results in oxidative stress and inflammation in asthma. The results of these studies show that ginseng may decrease the phosphorylation of NF-κB and subsequent activation of iNOS[10].

COX-2 is an enzyme activated in a pro-inflammatory state. In asthma, this pathway is responsible for the production of prostaglandins, which alter mucosal blood flow, resulting in bronchoconstriction and excessive mucus production. In our analysis, ginseng and its metabolites inhibited COX-2 expression in lung epithelial cells. However, the qualitative effects on COX-2 vary based on the type of ginseng extract and preparation used[14].

### *Role of ginseng in the pathological mechanism of asthma*

Ginsenosides, particularly Rg3 and Rh2, are the primary pharmacologically active constituents of ginseng. The results of this study suggest that these agents inhibit pro-inflammatory NF-κB and COX-2 pathways, both of which are pivotal in the pathogenesis and progression of asthma. The resulting downstream effects include attenuation of oxidative stress, inflammation, bronchoconstriction, and mucus production, thereby alleviating asthmatic symptoms. The results of this study also showed that ginsenosides reduced the production of Th2 cytokines, possibly through the inhibition of pro-inflammatory pathways, although the exact mechanisms are unknown. Figure 2 summarizes the role of ginseng in the pathogenesis of asthma.

### *Limitations*

Studies cited in this review were conducted using murine models. Although these studies provide valuable insights

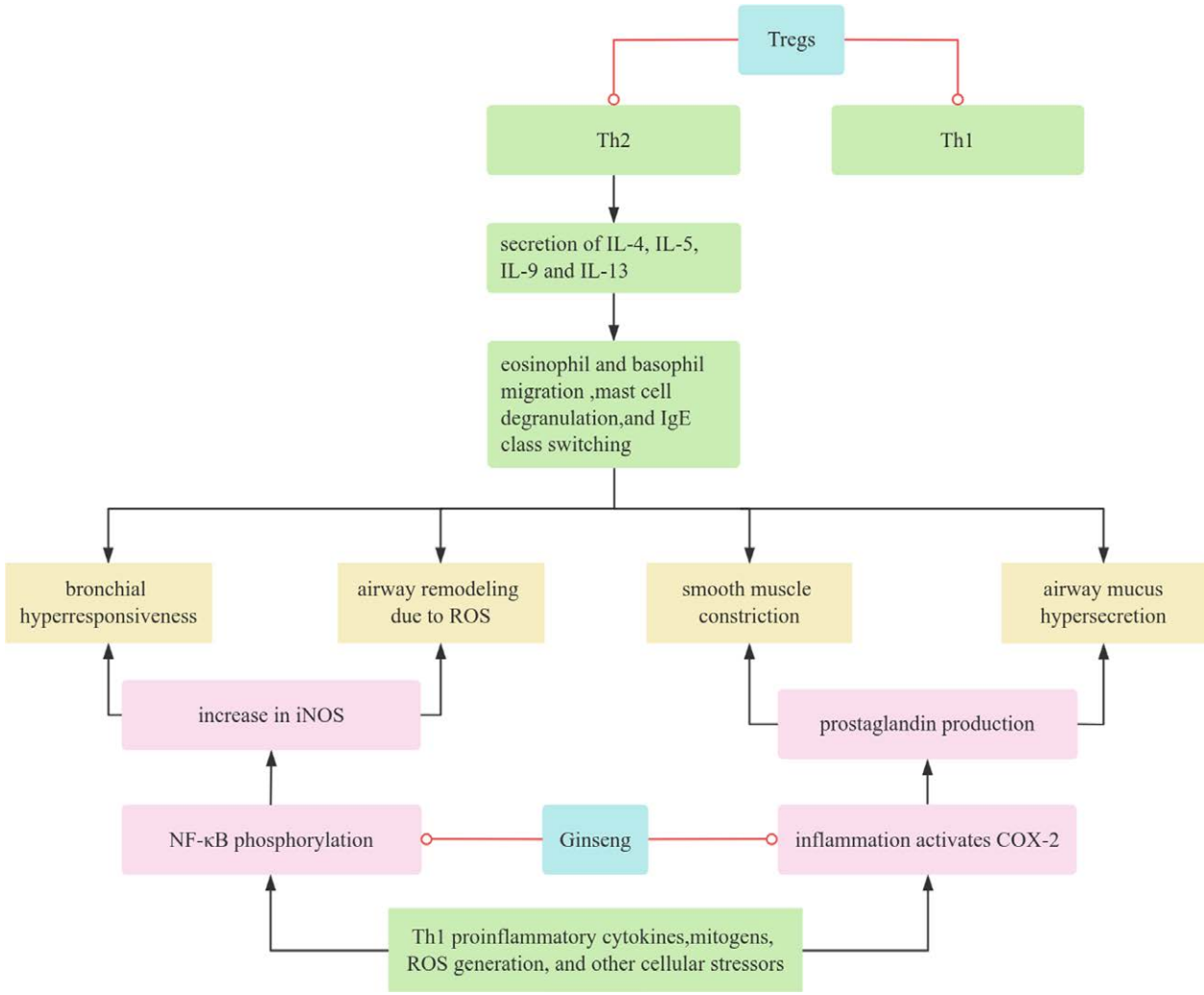


**Figure 2.** Summary of the role of ginseng on the pathophysiology of asthma. COX-2: Cyclooxygenase-2; IL: Interleukin; iNOS: inducible nitric oxide synthase; NFkB: Nuclear factor kappa B; ROS: Reactive oxygen species; Th1: T-helper cell type 1; Th2: T-helper cell type 2; Tregs: Regulatory T cells.

into the potential mechanisms of ginseng in asthma, their applicability in human patients with asthma may be limited. Further clinical studies are required to establish the efficacy and safety of ginseng and its derivatives in humans.

These studies included different forms of ginseng such as KRG, KWG, and FAGS. Variations in ginseng type, dosage, and preparation may lead to differences in its effects on asthma outcomes, which makes it challenging to draw consistent conclusions. Many of these studies have relatively short observational periods. Asthma is a chronic condition and the long-term effects and safety of ginseng as an anti-asthmatic agent requires further investigation.

Although the reviewed sources suggest the potential benefits of ginseng in alleviating asthma symptoms through anti-inflammatory mechanisms, the limitations highlighted above must be considered when the findings are interpreted. Since this study is a mini-review and not a systematic review or meta-analysis, the absence of a quality assessment is a limitation. Therefore, selection, implementation, and measurement biases are possible. Future studies should address these limitations and provide more robust evidence regarding the efficacy and safety of ginseng as an adjunct treatment for asthma.

## Conclusion

The findings of this study demonstrate that KRG, KWG, and ginsenosides exert an anti-asthmatic effect primarily through a reduction in mucus cell hypersecretion, inflammatory cell infiltration, Th2 cytokines, and inhibition of inflammatory pathways NF-κB and COX-2. We suggest that ginseng has therapeutic potential for the treatment of asthma. Future studies should investigate the long-term safety and efficacy of ginseng as an anti-asthmatic agent. Future studies should explore the mechanisms by which ginseng and its derivatives decrease Th2 cytokine production. As this analysis utilized murine models, the anti-asthmatic effects of ginseng must be further supported by laboratory studies and clinical trials.

## Conflict of interest statement

The authors declare no conflict of interest.

## Funding

None.

## Author contributions

Research design: Kailynn J. Yang; methodology: Kailynn J. Yang; formal analysis: Kailynn J. Yang, Liana Y.A. Bautista, Danielle G. Iben, and Dana H. Tran; investigation: Kailynn J. Yang, Liana Y.A. Bautista, Danielle G. Iben, and Dana H. Tran; data curation: Kailynn J. Yang, Liana Y.A. Bautista, Danielle G. Iben, and Dana H. Tran; writing-original draft preparation: Kailynn J. Yang, Liana Y.A. Bautista, Danielle G. Iben, and Dana H. Tran; writing-review and editing: Kailynn J. Yang, Liana Y.A. Bautista, Danielle G. Iben, and Dana H. Tran; and project administration: Kailynn J. Yang. All authors have read and agreed to the published version of the manuscript.

## Ethical approval of studies and informed consent

Not applicable.

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None.

## Data availability

All data generated or analyzed during this study are included in this published article.

## References

[1] Braman SS. The global burden of asthma. *Chest* 2006;130(1 Suppl):4S–12S.
[2] Lemmetyinen RE, Karjalainen JV, But A, et al. Higher mortality of adults with asthma: a 15-year follow-up of a population-based cohort. *Allergy* 2018;73(7):1479–1488.
[3] O'Byrne PM, Pedersen S, Schatz M, et al. The poorly explored impact of uncontrolled asthma. *Chest* 2013;143(2):511–523.
[4] Stibolt T. Asthma. *Perm J* 2007;11(1):56–61.
[5] Lulich KM, Goldie RG, Ryan G, et al. Adverse reactions to beta 2-agonist bronchodilators. *Med Toxicol* 1986;1(4):286–299.
[6] Gupta P, O'Mahony MS. Potential adverse effects of bronchodilators in the treatment of airways obstruction in older people: recommendations for prescribing. *Drugs Aging* 2008;25(5):415–443.
[7] Wee JJ, Park KM, Chung AS. Biological activities of ginseng and its application to human health. In: Benzie IFF, Wachtel-Galor S, editors. *Herbal Medicine: Biomolecular and Clinical Aspects*. 2nd ed. Boca Raton, FL: CRC Press/Taylor & Francis; 2011:157–174.
[8] Qi LW, Wang CZ, Du GJ, et al. Metabolism of ginseng and its interactions with drugs. *Curr Drug Metab* 2011;12(9):818–822.
[9] Jang WY, Hwang JY, Cho JY. Ginsenosides from Panax ginseng as key modulators of NF-κB signaling are powerful anti-inflammatory and anticancer agents. *Int J Mol Sci* 2023;24(7):6119.
[10] Kim JH, Kim JW, Kim CY, et al. Korean Red Ginseng ameliorates allergic asthma through reduction of lung inflammation and oxidation. *Antioxidants (Basel)* 2022;11(8):1422.
[11] Ryu JH, Woo MS, Cao DL, et al. Fermented and aged ginseng sprouts (Panax ginseng) and their main component, compound K, alleviate asthma parameters in a mouse model of allergic asthma through suppression of inflammation, apoptosis, ER stress, and ferroptosis. *Antioxidants (Basel)* 2022;11(10):2052.
[12] Lee SY, Kim MH, Kim SH, et al. Korean Red Ginseng affects ovalbumin-induced asthma by modulating IL-12, IL-4, and IL-6 levels and the NF-κB/COX-2 and PGE2 pathways. *J Ginseng Res* 2021;45(4):482–489.
[13] Lim CY, Moon JM, Kim BY, et al. Comparative study of Korean white ginseng and Korean Red Ginseng on efficacies of ova-induced asthma model in mice. *J Ginseng Res* 2015;39(1):38–45.
[14] Huang WC, Huang TH, Yeh KW, et al. Ginsenoside Rg3 ameliorates allergic airway inflammation and oxidative stress in mice. *J Ginseng Res* 2021;45(6):654–664.
[15] Mohi El-Din MM, Mostafa AM, Abd-Elkader A. Experimental studies on the effect of (Lambda-Cyhalothrin) insecticide on lungs and the ameliorating effect of plant extracts (Ginseng (Panax Ginseng) and garlic (Allium sativum L.) on asthma development in albino rats. *BMC Res Notes* 2014;7(1):243.
[16] Lee YS, Yang WK, Yee SM, et al. KGC3P attenuates ovalbumin-induced airway inflammation through downregulation of p-PTEN in asthmatic mice. *Phytomedicine* 2019;62:152942.

[17] Li LC, Piao HM, Zheng MY, et al. Ginsenoside Rh2 attenuates allergic airway inflammation by modulating nuclear factor-κB activation in a murine model of asthma. *Mol Med Rep* 2015;12(5):6946–6954.
[18] Pinto RA, Arredondo SM, Bono MR, et al. T helper 1/T helper 2 cytokine imbalance in respiratory syncytial virus infection is associated with increased endogenous plasma cortisol. *Pediatrics* 2006;117(5):e878–e886.
[19] Yang YL, Pan YQ, He BS, et al. Regulatory T cells and Th1/Th2 in peripheral blood and their roles in asthmatic children. *Transl Pediatr* 2013;2(1):27–33.