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In Vitro Evaluating Anti-Asthmatic and Anti-Inflammatory Activities of *Allium ampeloprasum* Ethanolic Extract in Airway Smooth Muscle Cells and T Lymphocytes

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ABSTRACT

Background & Objective: The incidence of asthma is rising, incurring considerable healthcare expenses. Although corticosteroids are the most effective anti-inflammatory drugs for improving lung function in asthma, they have a limited influence on airway remodeling and can cause adverse effects. Therefore, interest in developing natural medications with fewer side effects is growing. The increasing use of traditional herbal medicine demands more scientific evidence for its efficacy. *Allium ampeloprasum*, which is suggested for use in traditional medicine to treat asthma, was chosen for this investigation due to its traditional applications.

Materials & Methods: A hydroalcoholic extract of the plant was prepared. Its effect on the viability and proliferation of airway smooth muscle cells was determined using the MTT assay. The production of nitric oxide and the levels of inflammatory proteins in T lymphocyte cells were also investigated.

Results: *A. ampeloprasum* extract significantly reduced the proliferation of airway smooth muscle cells in a dose- and time-dependent manner ($p \leq 0.05$). Platelet-derived growth factor-BB (PDGF-BB) enhanced proliferation, whereas the extract significantly suppressed this PDGF-BB-induced proliferation ($p \leq 0.05$). The extract did not influence basal cell viability or proliferation, regardless of LPS stimulation. In LPS-stimulated cells, the extract significantly decreased nitric oxide production ($p \leq 0.05$). The expression and production of LPS-induced inflammatory markers were also significantly reduced ($p \leq 0.05$).

Conclusion: *A. ampeloprasum* has anti-inflammatory effects and can prevent the proliferation of airway smooth muscle cells. It can be proposed as a possible corticosteroid substitute.

Keywords: Asthma, Inflammation, *A. Ampeloprasum*, T Lymphocytes

1. Introduction

Asthma is a chronic inflammatory condition of the airways that affects over 300 million individuals worldwide. It is still a significant cause of illness and a worse quality of life (1). Even with the availability of several pharmaceutical therapies, such as leukotriene receptor antagonists, β-agonists, and inhaled corticosteroids (ICS), many patients still suffer from poor disease management, frequent flare-ups, and negative medication side effects (2, 3). The identified limitations underscore the necessity for safer, cost-effective, and more targeted therapeutic strategies.

Type 2 helper T-cell (Th2)-driven inflammation, characterized by elevated cytokine levels including interleukin (IL)-3, IL-4, and IL-13, is a hallmark of asthma pathophysiology (4). These cytokines are involved in subepithelial fibrosis, mucus overproduction, and airway eosinophilia. Simultaneously, airway smooth muscle (ASM) hyperplasia and remodeling worsen airflow restriction and increase the severity and chronicity of the condition (5).

Due to their immunomodulatory properties, affordability, and fewer adverse effects, plant-based

medicines have garnered interest as complementary or alternative therapies (6, 7). One of them, *Allium ampeloprasum* (wild leek), has been used in herbal treatment for a long time in both Mediterranean and Iranian traditions. It has antibacterial, anti-inflammatory, and antioxidant properties, mediated by its flavonoids and organosulfur compounds (8-10). Recent in vitro studies suggest its potential to modulate inflammatory pathways and inhibit cellular proliferation (11); however, scientific evidence supporting its specific efficacy in asthma remains limited.

This research aims to investigate the anti-asthmatic and anti-inflammatory properties of the ethanolic extract of *A. ampeloprasum* in vitro, focusing on its effects on ASM cell proliferation and the regulation of Th2-associated cytokines in T lymphocytes. By elucidating its underlying cellular mechanisms, this research seeks to provide a scientific rationale for considering *A. ampeloprasum* as a safe, affordable, and complementary therapeutic option in asthma management.

2. Materials and Methods

2.1 Preparation of Extract

In the spring, *A. ampeloprasum* was gathered from the area around Kermanshah, recognized by a botanist, and then cleaned and dried. 200 milliliters of 70% ethanol were mixed with 20 grams of dried root, stem, and leaf powder, and the mixture was left on a magnetic stirrer at room temperature overnight. Centrifugation was then used to extract the supernatant, which was then filtered through Whatman paper and lyophilized using a freeze dryer. The extracted material was kept at 2 - 8 °C. The extract was dissolved in the cell culture medium to create the stock solution just before use (12). Every experimental method was conducted in accordance with global ethical standards for the use of biological materials in laboratories.

2.2 Cell Culture

EL4 mouse cells, which are derived from a T-cell lymphoma cell line in mice, along with smooth muscle cells from the airways, were cultured in RPMI-1640 medium (Gibco, Thermo Fisher Scientific, USA) supplemented with 10% fetal bovine serum (FBS, Sigma-Aldrich, Germany) and 1% penicillin-streptomycin (Gibco, USA). The cells were maintained under standard conditions of 37°C and 5% CO_2 (13).

2.3 MTT Test

The MTT test kit (Sigma-Aldrich, Germany) was used to assess cell viability according to the kit protocol. Cell survival was measured as a percentage of control cells that had not been treated. Nagative controls (cells without extract) and blank wells (medium only) were included in each trial (14).

2.4 Nitric Oxide Test

EL4 T lymphocytes were used to assess whether the product could reduce inflammation. For an entire day, the extract was applied to the cells. Equal volumes of Griess solution (Promega, USA) and cell culture supernatant were mixed. After 15 minutes, a microplate reader (BioTek, USA) measured nitrite levels, a marker of nitric oxide production. To find the nitrite amounts, a sodium nitrite (NaNO2) reference curve was used. The cells that were used as negative standards had not been treated (15).

2.5 Immunofluorescence Assay

0.1% Triton X-100 was used to make the cells permeable. 5% BSA (Sigma-Aldrich, Germany) was used to stop the cells, and 4% paraformaldehyde was used to fix them for 15 minutes. The cells were incubated overnight at 4 °C with primary antibodies targeting IL-13, IL-5, and IL-4 (Abcam, UK). Then, they were treated with fluorophore-conjugated secondary antibodies (Invitrogen, USA) for an hour. The nuclei were stained with DAPI (Sigma-Aldrich, Germany), and then images were captured using a Zeiss fluorescent microscope. There were also negative controls that lacked key antibodies.

2.6 Gene Expression Test

The RNeasy Mini Kit (Qiagen, Germany) was used to isolate total RNA after treating cells with extract and LPS for 24 and 48 hours, as directed by the manufacturer. A NanoDrop spectrophotometer (Thermo Fisher Scientific, USA) was used to measure the A260/A280 ratio and determine the quantity and purity of the RNA (16). To visualize the 28S and 18S rRNA bands, agarose gel electrophoresis (1% gel with ethidium bromide) was performed to assess RNA stability. Additional tests were conducted on samples that exhibited clear bands and no smearing. The RevertAid First Strand cDNA Synthesis Kit from Thermo Fisher Scientific, USA, was used to make the first strand of cDNA (17). SYBR Green Real-Time PCR Master Mix (Applied Biosystems, Thermo Fisher, USA) was used to measure gene expression using the StepOnePlus Real-Time PCR System (Applied Biosystems). The reference gene was GAPDH (18). Primer sequences are as follows:

IL-3 TGGCTTCTGTACTCGGTTTC AGCATCTGTTGTTGTGAGGT, IL-4 ACAGGAGAAGGGACGCCAT GAAGCCCTACAGACGAGCTCA, IL-13 CCTGGCTCTTGCTTGCCTT GGTCTTGTGTGATGTTGCTCA, and GAPDH GAAGGTGAAGGTCGGAGTCA GAAGATGGTGATGGGATTTC.

2.7 Statistical Analysis

Statistical analyses will be performed using SPSS software version 16. The results were expressed as mean ± standard deviation and analyzed using one-way ANOVA, followed by the Tukey HSD post hoc test. Each experiment was conducted in triplicate. A P-value of less than 0.05 will be considered statistically significant.

3. Result

3.1 Investigation of the effect of A. *ampeloprasum* extract on the proliferation of airway smooth muscle cells

This study employed the MTT test to investigate the effect of *A. ampeloprasum* extract on the growth of airway smooth muscle cells at rest and in the presence of PDGF. The extract significantly slowed down the growth of cells, with the effect dependent on both dose and time ($p \leq 0.05$) (Figure 1). Subsequently, this study investigated whether the extract could inhibit PDGF-BB-induced growth. PDGF-BB increased cell division; however, the extract significantly inhibited PDGF-BB-induced cell division in HASMCs ($p \leq 0.05$), resulting in fewer cells alive than in the control group (Figure 2).

3.2 Investigation of the effect of A. *ampeloprasum* extract on the proliferation of T lymphocyte cells

The effect of *A. ampeloprasum* extract on T lymphocyte proliferation was investigated using the MTT assay. The *A. ampeloprasum* extract did not affect cell viability or proliferation, regardless of its presence or absent in the absence of LPS (Figures 3 and 4).

The effect of A. ampeloprasum extract on nitric oxide production in T lymphocyte cells was examined.

The *A. ampeloprasum* extract significantly reduced the production of nitric oxide in cells when LPS was added ($p \leq 0.05$) (Figure 5).

3.3 The effect of A. *ampeloprasum* extract on the production of inflammatory markers in T lymphocyte cells was investigated

We used a fluorescence experiment to investigate how *A. ampeloprasum* extract affected the production of IL-13, IL-3, and IL-4 in T-cell lymphoma cells. The results are shown in Figure 6. *A. ampeloprasum* extract significantly decreased the production of inflammation markers in cells that were activated by LPS ($p \leq 0.05$).

3.4 The effect of A. *ampeloprasum* extract on the expression of inflammatory markers in T lymphocytes was investigated

The effects of *A. ampeloprasum* extract on the expression of IL-13, IL-3, and IL-4 in T-cell lymphoma cells were investigated using real-time PCR and are shown in Figure 7. *A. ampeloprasum* extract significantly reduced the expression of LPS-stimulated inflammatory markers in the cells ($p \leq 0.05$).

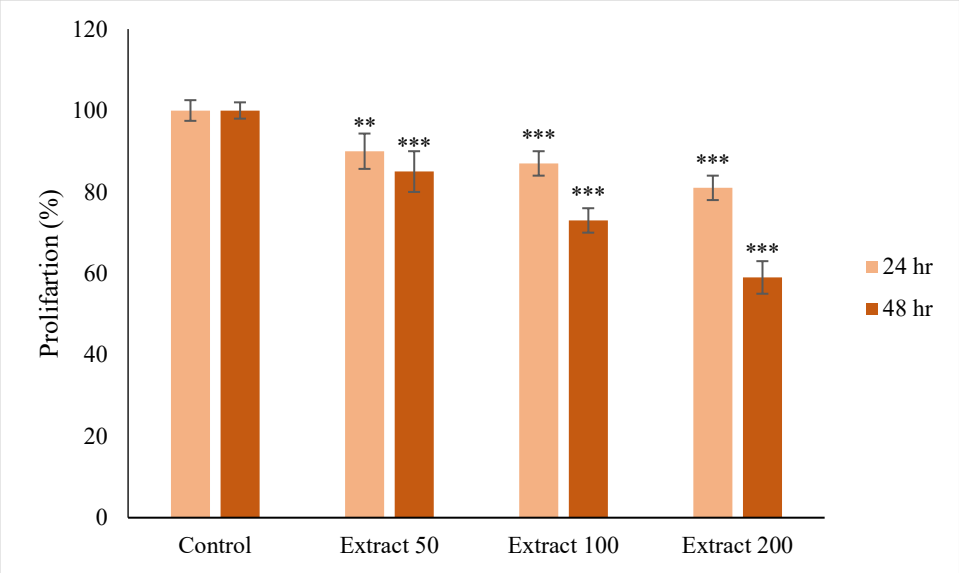


Figure 1. Effect of *A. ampeloprasum* extract on the proliferation of airway smooth muscle cells. ** indicates $p < 0.05$ and *** indicates $p < 0.001$ compared to control (**Prepared by Authors, 2025**).

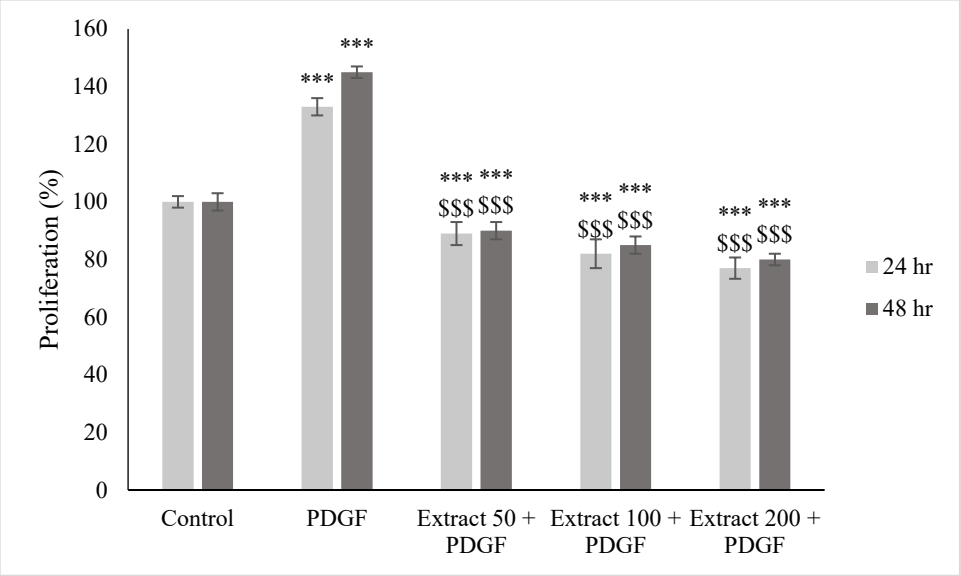


Figure 2. PDGF makes lung smooth muscle cells grow, and *A. ampeloprasum* extract affects this. *** indicates $p < 0.001$ compared to the control, and $$$ indicates $p < 0.001$ compared to PDGF (**Prepared by Authors, 2025**).

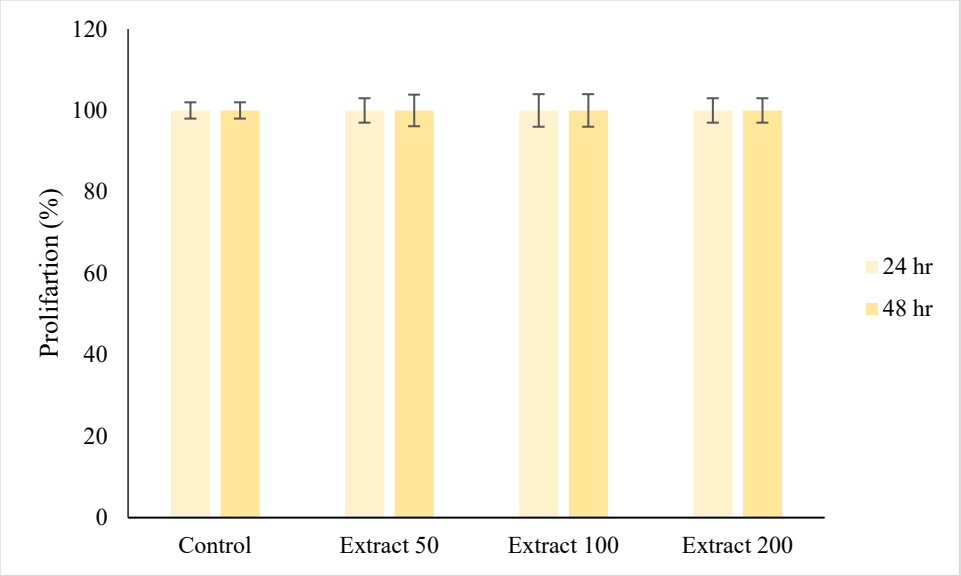


Figure 3. Effect of *A. ampeloprasum* extract on T lymphocyte proliferation (**Prepared by Authors, 2025**).

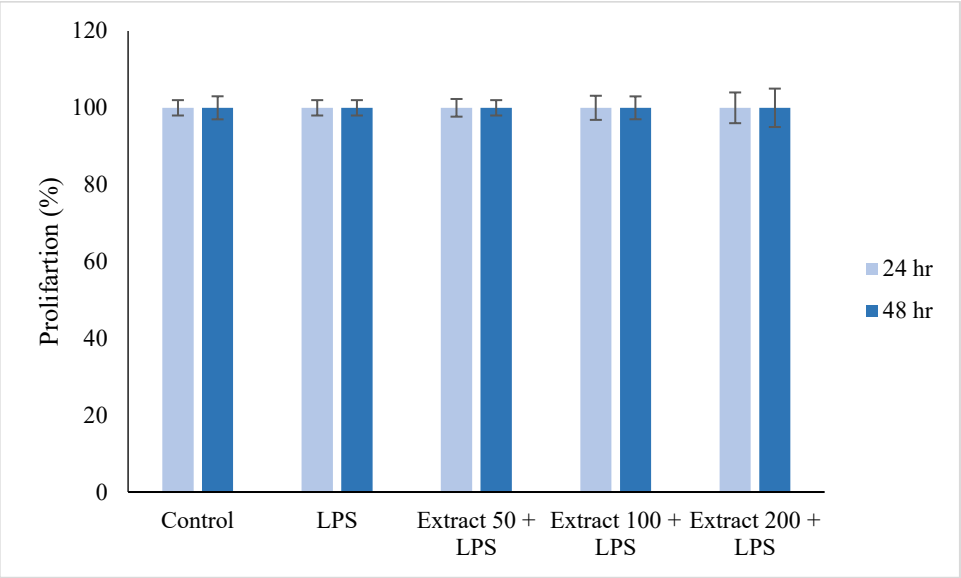


Figure 4. Effect of *A. ampeloprasum* extract on the viability of T-cell lymphoma cells in the presence of LPS (**Prepared by Authors, 2025**).

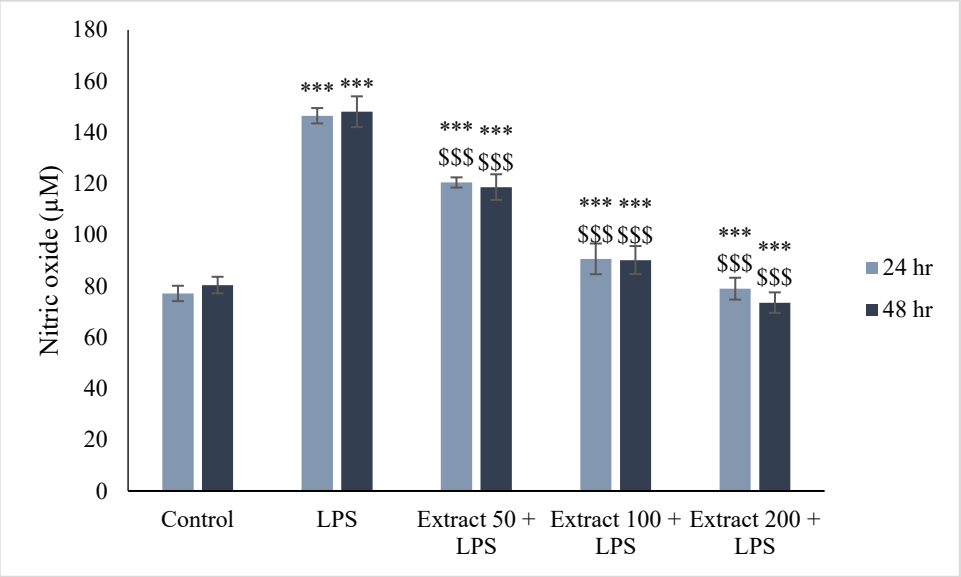
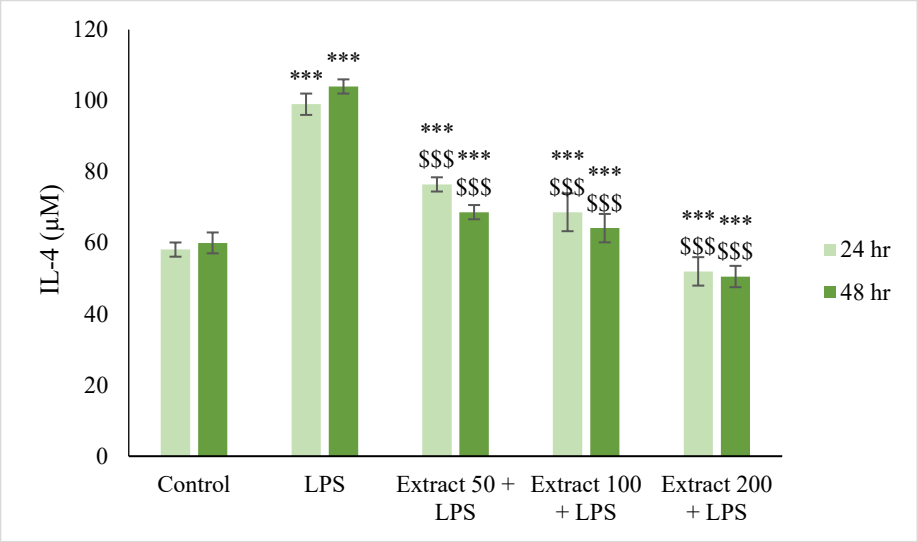
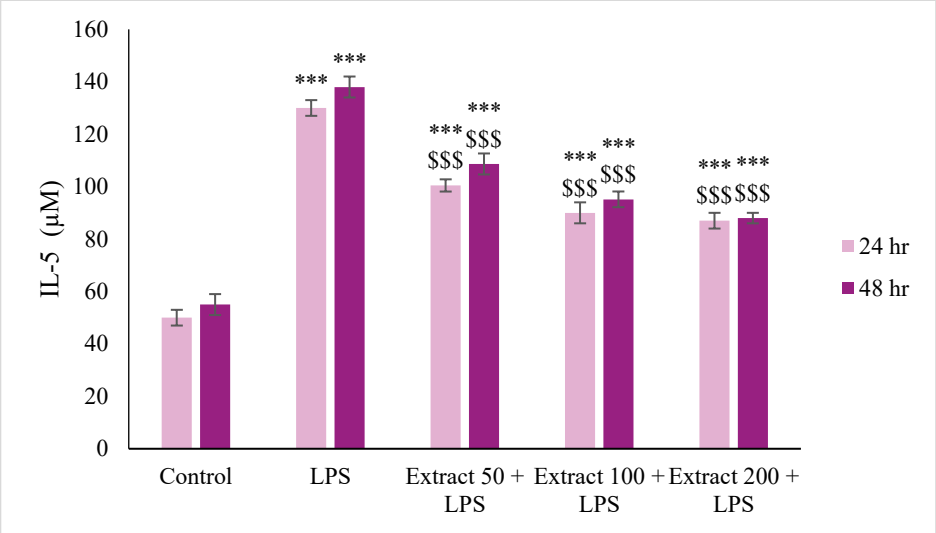


Figure 5. Effect of *A. ampeloprasum* extract on nitric oxide production in T lymphocyte cells. *** indicates $p < 0.001$ compared to control and $$$ indicates $p < 0.001$ compared to LPS (**Prepared by Authors, 2025**).

A



B



C

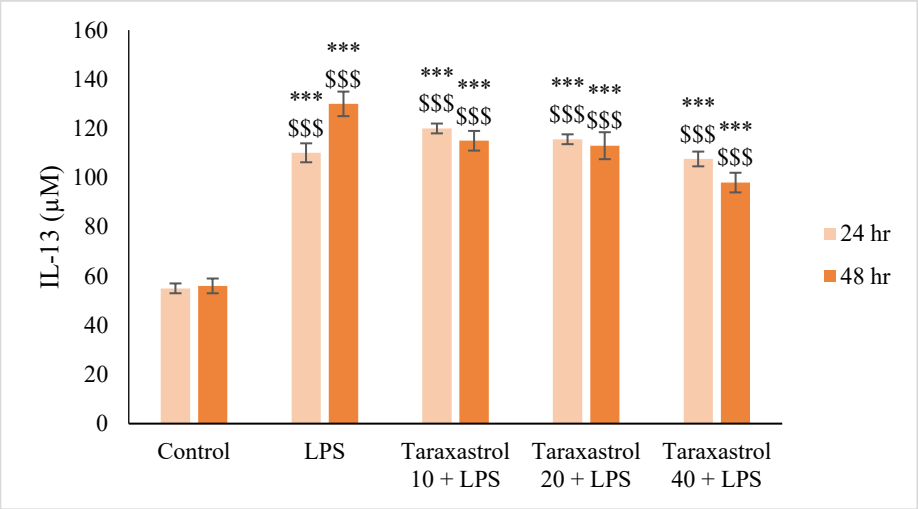
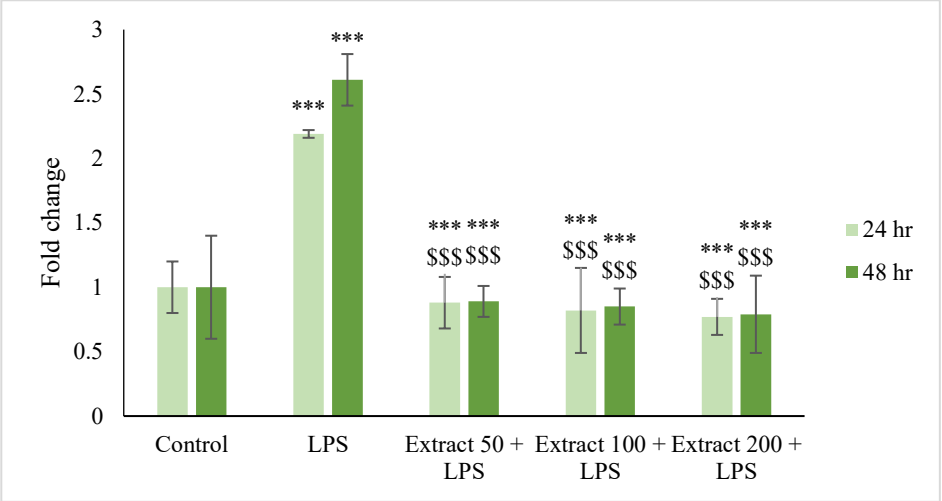
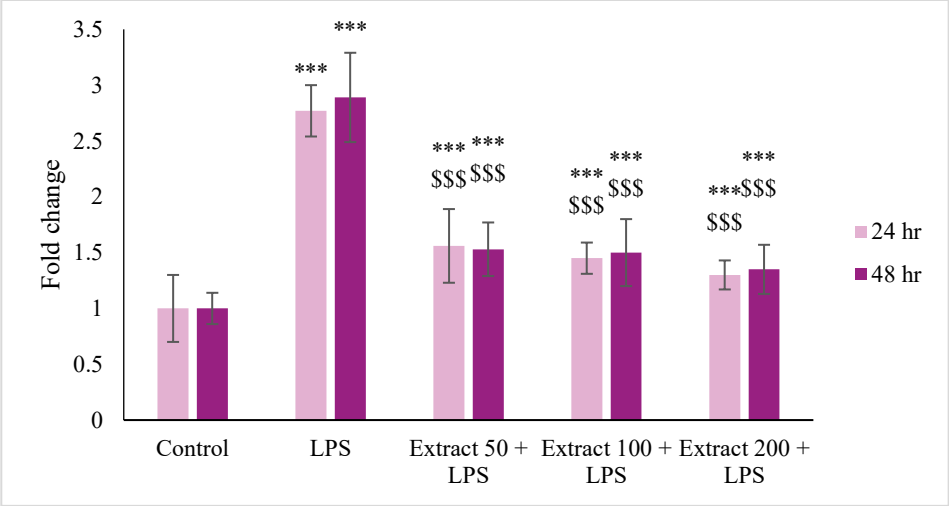


Figure 6. Effect of A. ampeloprasum extract on the levels of inflammatory factors IL-3 (A), IL-4 (B), and IL-13 (C) in T lymphocyte cells. *** indicates $p < 0.001$ compared to control and $$$ indicates $p < 0.001$ compared to LPS (**Prepared by Authors, 2025**).

A



B



C

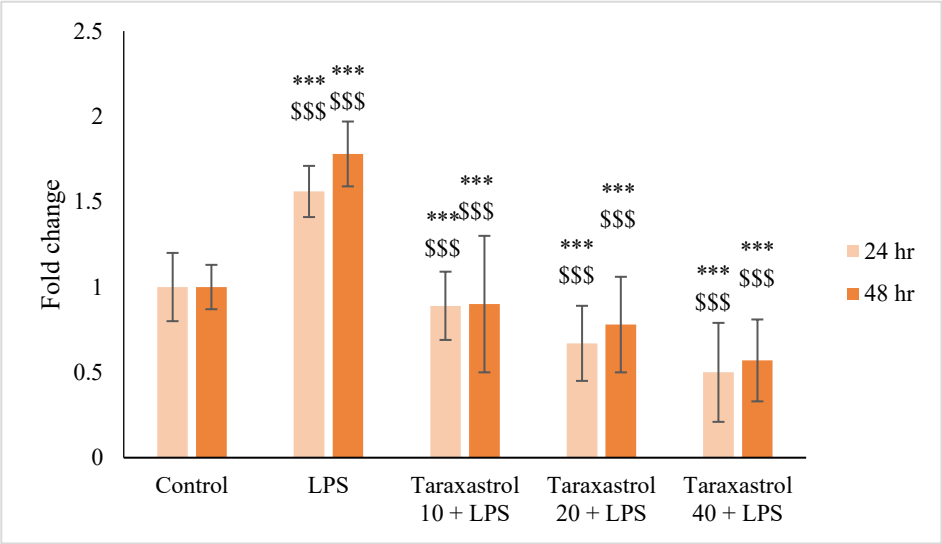


Figure 7. Effect of *A. ampeloprasum* extract on the expression of inflammatory factors IL-3 (A), IL-4 (B), and IL-13 (C) in T lymphocyte cells. *** indicates $p < 0.001$ compared to control and $$$ indicates $p < 0.001$ compared to LPS (**Prepared by Authors, 2025**).

4. Discussions

Airway hyperresponsiveness (AHR), inflammation, and airway remodeling are characteristics of asthma, a chronic inflammatory illness (19). The increase in airway smooth muscle bulk is a key structural alteration (20, 21). ASM cells are not only contractile elements but also active participants in inflammation and tissue remodeling. In this study, we simulated asthmatic conditions by inducing ASM cell proliferation using platelet-derived growth factor and found that *Allium ampeloprasum* extract effectively inhibited PDGF-induced ASM proliferation. This suggests the extract may interfere with remodeling-related signaling pathways involved in ASM hyperplasia.

In parallel, the anti-inflammatory potential of the extract was confirmed in a lipopolysaccharide (LPS)-induced T cell model, where it significantly suppressed the expression of key pro-inflammatory mediators. This is relevant since immune cell activation and cytokine overproduction especially IL-4, IL-5, and IL-13 are central to Th2-driven asthma pathogenesis (22-24). IL-4 promotes IgE production, IL-5 recruits eosinophils, and IL-13 enhances mucus secretion and airway hyperresponsiveness, while also upregulating inducible nitric oxide synthase (iNOS), contributing to inflammation (24).

Although corticosteroids are effective in reducing inflammation, prolonged use is associated with negative outcomes, including osteoporosis and an increased risk of infections (25). Furthermore, corticosteroid resistance in certain asthma phenotypes underlines the need for alternative strategies (26). In this context, *A. ampeloprasum* is a promising plant-based candidate, offering anti-inflammatory and anti-remodeling effects that may be achieved by downregulating Th2 cytokines and iNOS expression.

While the extract has been previously studied for its antioxidant and metabolic benefits in diabetic models (27-29), and for antimicrobial and analgesic properties (30, 31), our study narrows the focus to asthma-related mechanisms. The extract's impact on both inflammatory mediators and smooth muscle proliferation indicates its potential as a multi-target therapeutic agent for asthma, particularly in phenotypes less responsive to conventional therapies.

Future studies should assess the efficacy of the extract in animal models of allergic asthma and identify the active constituents responsible for its biological effects. Investigating specific signaling pathways, such as NF-κB, STAT6, or PI3K/Akt, will help clarify the mechanisms underlying the observed effects. Moreover, exploring potential synergies with current asthma medications could open avenues for adjunctive therapies.

This study was conducted under in vitro conditions using cell lines, which may not fully replicate the complex immune and inflammatory responses observed in vivo. Additionally, the effects of *A. ampeloprasum* extract were not tested in co-culture systems or animal models, limiting the translation of the findings to clinical settings. The extract composition was not standardized or characterized by HPLC or LC-MS, which may affect reproducibility. Furthermore, although the viability assay indicated non-toxic concentrations, further studies using live/dead cell staining and mechanistic pathway analysis (e.g., NF-κB, MAPK) are needed to confirm the observed anti-inflammatory effects. Finally, the absence of positive control treatments (such as dexamethasone or known inhibitors) limits the comparative evaluation of efficacy.

5. Conclusion

This study provides the first experimental evidence that the ethanolic extract of *Allium ampeloprasum* exhibits dual anti-asthmatic effects by targeting two key pathological features of asthma: airway remodeling and Th2-driven inflammation. The extract effectively inhibited PDGF-induced airway smooth muscle cell proliferation. It suppressed LPS-stimulated expression of pro-inflammatory mediators, including IL-4, IL-3, IL-13, and inducible nitric oxide synthase (iNOS), positioning it as a promising modulator of both structural and immune components of asthma. In contrast to current pharmacotherapies many of which are limited by corticosteroid resistance, high cost, or adverse effects *A. ampeloprasum* offers a cost-effective and potentially safer alternative. These findings not only support its traditional use but also establish a foundation for further research. Future work should focus on isolating active compounds, validating the extract's efficacy in vivo, and exploring its synergistic potential with existing therapies. If confirmed, this plant-based intervention may contribute meaningfully to the development of novel, accessible strategies for asthma management.

6. Declarations

6.1 Acknowledgments

We sincerely appreciate the assistance provided by Kermanshah University of Medical Sciences in financing this project.

6.2 Ethical Considerations

The study has been approved by the Ethical Committee of North Khorasan University of Medical Sciences (Ethical approval ID: IR.NKUMS.REC.1398.038). Relevant guidelines and regulations are followed for all methods. Furthermore, informed consent was obtained from all subjects.

6.3 Authors' Contributions

Conceptualization, I.R.; methodology, M.P. and M.N.A. and A.G; software, S.H.A.; validation, I.R., M.N.A.Y. and A.G.; formal analysis, M.P.; investigation, S.H.A.; resources, I.R.; data curation, M.P.; writing—

original draft preparation, M.N.A.; writing—review and editing, I.R.; visualization, A.G.; supervision, I.R.; project administration, I.R.; funding acquisition, A.G.

6.4 Conflict of Interest

The authors have no conflict of interest.

6.5 Fund or Financial Support

This research was financially supported by Kermanshah University of Medical Sciences.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

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