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EVALUATION OF ANTI-ASTHMATIC ACTIVITY OF *MENISAMOOLA PARPAM* IN HISTAMINE-INDUCED BRONCHIAL ASTHMA MODEL IN WISTAR RATS

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ABSTRACT

Background: Bronchial asthma is a chronic inflammatory disorder of the respiratory system characterized by airway hyperresponsiveness, eosinophilia, and elevated immunoglobulin E (IgE) levels. Although conventional therapies are effective, their long-term use may be associated with adverse effects. Traditional Siddha formulations offer a potential alternative approach for the management of respiratory diseases.

Objective: To evaluate the anti-asthmatic activity of Menisamoola Parpam (MSP) using a histamine-induced bronchial asthma model in Wistar rats.

Materials and Methods: Wistar rats were divided into five groups: normal control, disease control, standard (mepyramine-treated), and MSP-treated groups receiving low and high doses. Bronchial asthma was induced by exposure to 0.5% histamine aerosol, and the pre-convulsion time was recorded. Haematological parameters, including total leukocyte count and eosinophil count, were evaluated. Serum IgE levels and the concentrations of inflammatory cytokines (TNF-α, IL-4, and IL-5) in bronchoalveolar lavage fluid were determined using enzyme-linked immunosorbent assay (ELISA). Mast cell stabilization activity and histopathological examination of lung tissues were also performed. Statistical analysis was conducted using one-way analysis of variance (ANOVA) followed by Dunnett's post hoc test.

Results: MSP significantly reduced total leukocyte and eosinophil counts, serum IgE levels, and the concentrations of pro-inflammatory cytokines compared with the disease control group. MSP also exhibited dose-dependent inhibition of mast cell degranulation. Histopathological examination revealed reduced inflammatory cell infiltration and restoration of normal lung architecture in the MSP-treated groups.

Conclusion: Menisamoola Parpam demonstrated significant anti-asthmatic activity through its anti-inflammatory, immunomodulatory, and mast cell-stabilizing effects. These findings suggest that MSP has potential as a therapeutic agent for the management of bronchial asthma.

Keywords: *Bronchial asthma, Menisamoola Parpam, Siddha, IgE, Cytokines, Mast cell stabilization.*

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1. INTRODUCTION

Bronchial asthma is a chronic respiratory disorder characterized by intermittent airway obstruction, increased bronchial sensitivity, and persistent inflammation of the airways. It continues to be a major global health concern, affecting individuals across all age groups and contributing significantly to morbidity**.** The underlying mechanism of asthma involves a complex network of inflammatory cells and mediators. Among these, eosinophils play a pivotal role by releasing toxic granule proteins and inflammatory substances that damage airway tissues and enhance bronchial reactivity. Elevated levels of immunoglobulin E (IgE) are frequently associated with allergic asthma and contribute to mast cell activation, leading to the release of histamine and other inflammatory mediators. Cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin-4 (IL-4), and interleukin-5 (IL-5) are key contributors to the inflammatory

cascade in asthma. These mediators regulate eosinophil activation, IgE synthesis, and the persistence of airway inflammation. Increased concentrations of these cytokines are commonly observed in asthmatic conditions and are considered important biomarkers for disease severity. Experimental animal models, particularly histamine-induced bronchospasm, are widely utilized to study the pathogenesis of asthma and to evaluate potential therapeutic agents. Histamine is a potent bronchoconstrictor that mimics many features of human asthma, making it a reliable model for pharmacological studies. Conventional anti-asthmatic therapies, including corticosteroids and antihistamines, are effective but may lead to adverse effects with prolonged use. This has encouraged the exploration of traditional medicinal systems such as Siddha medicine for safer and alternative treatment options. *Menisamoola Parpam* (MSP) is a traditional Siddha formulation that has been used in the management of respiratory ailments. However, there is limited scientific evidence supporting its efficacy in asthma. Therefore, the present study was undertaken to investigate the anti-asthmatic activity of MSP using a histamine-induced experimental model, focusing on its effects on inflammatory markers, immune response, and lung histopathology.

MATERIALS AND METHODS

Animals

Healthy Wistar rats weighing 200–250 g was used for the study. Animals were maintained under controlled environmental conditions of temperature (22 ± 2°C), relative humidity (55 ± 5%), and a 12 h light/dark cycle. They were provided with a standard pellet diet (Pranav Argo, Baroda) and water ad libitum.

Experimental Design

Animals were randomly divided into five groups (n = 6):

- ✓ Group I: Normal control (Distilled water)
- ✓ Group II: Disease control (0.5% Histamine HCl aerosol)
- ✓ Group III: Standard (Mepyramine 8 mg/kg, p.o. + Histamine aerosol)
- ✓ Group IV: Test drug low dose (MSP + Histamine aerosol)
- ✓ Group V: Test drug high dose (MSP + Histamine aerosol)

Induction of Asthma

Asthma was induced by exposing animals to 0.5% histamine dihydrochloride aerosol in a closed chamber. Pre-convulsion time (PCT) was recorded as an index of bronchoconstriction.

Collection of Blood and BALF

On day 14, blood samples were collected from the retro-orbital plexus and serum was separated for analysis of IgE and haematological parameters.

Bronchoalveolar lavage fluid (BALF) was collected by instilling 1 mL of cold phosphate-buffered saline (PBS, pH 7.4) through a tracheal cannula. The fluid was centrifuged, and the supernatant was used for cytokine estimation (TNF-α, IL-4, IL-5).

Estimation of IgE and Cytokines

Serum IgE and BALF cytokines (TNF-α, IL-4, IL-5) were quantified using ELISA kits following standard protocols. Results were expressed in ng/mL and pg./mL respectively.

Histopathological Studies

Lung tissues were excised and fixed in 10% formalin. Paraffin-embedded sections (5 µm) were stained with haematoxylin and eosin (H&E) and examined for inflammatory changes and structural alterations.

Mast Cell Stabilization Activity

Mesenteric tissues were isolated and incubated in Ringer-Locke solution. They were treated with standard (ketotifen) and MSP (low and high doses), followed by exposure to compound 48/80 to induce degranulation.Mast cells were stained with toluidine blue and examined microscopically.% Inhibition of Mast Cell Degranulation:%Inhibition=(1−Total mast cellsDegranulated cells)×100

Biochemical Analysis

The mice were killed and a tracheal catheter was implanted at the end of the experiment (24 hours after sensitisation). Bronchoalveolar lavage fluid (BALF) was obtained by savaging the lung with two aliquots of five millilitres of 0.9% sodium chloride solution. The recovery volume for each mouse was roughly 5 millilitres. Leukocytes, neutrophils, and eosinophils were all counted under a microscope.

Statistical Analysis

Data were expressed as mean ± SEM (n = 6). Statistical analysis was performed using one-way ANOVA followed by Dunnett's multiple comparison test. Values were considered significant at $P < 0.05$.

RESULTS

Evaluation of serum, TNF-α, IL-4AND IL-5 FROM BALF in MSP

Table 01:

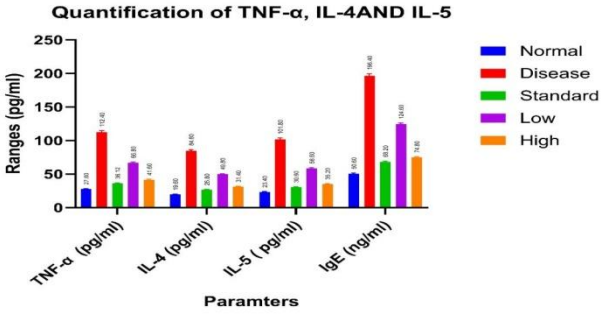
PARAMETERS	NORMAL CONTROL	DISEASE CONTROL	STANDARD CONTROL	LOW DOSE	HIGH DOSE
Leukocytes (cells/mm³)	1106.25 ± 6.18	1492.80 ± 9.64	1068.40 ± 7.32	1162.60 ± 8.41	1124.20 ± 6.95
Neutrophils (×10⁶/µl)	4.12 ± 0.09	16.82 ± 0.18	5.86 ± 0.07	8.24 ± 0.21	6.74 ± 0.11
Eosinophils (cells/mm³)	45.20 ± 0.84	192.60 ± 2.54	58.40 ± 0.66	124.80 ± 1.12	60.30 ± 0.72
TNF-α (pg/ml)	27.8 ± 0.58	112.4 ± 2.36	36.2 ± 0.81	66.8 ± 1.24	41.6 ± 0.88

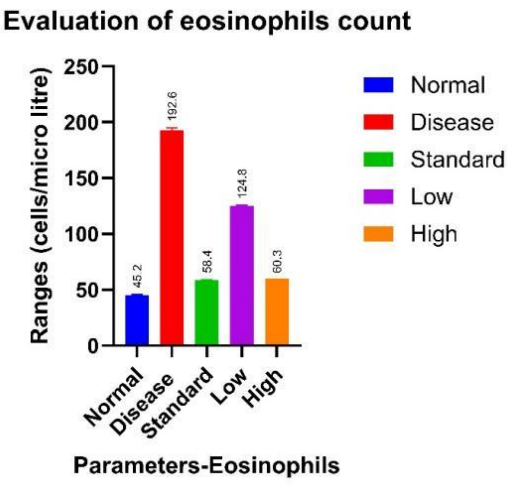
IL-4 (pg/ml)	19.6 ± 0.52	84.6 ± 1.94	26.8 ± 0.69	49.8 ± 0.97	31.4 ± 0.64
IL-5 (pg/ml)	23.4 ± 0.63	101.8 ± 2.12	30.6 ± 0.74	58.6 ± 1.08	35.2 ± 0.71
IgE (ng/ml)	50.6 ± 0.94	196.4 ± 3.18	68.2 ± 1.26	124.6 ± 1.72	74.8 ± 1.05

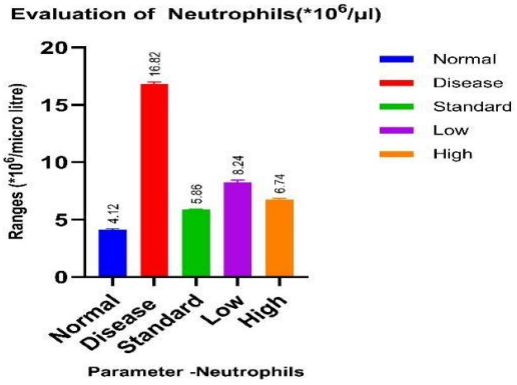
All values were expressed as mean ± SEM, n = 6, * P < 0.05, **P < 0.01, ***P < 0.001 as compared to the disease control group. Results were done by one-way ANOVA followed by Dunnett's test.

Table 02: Effect of *Menisamoola Parpam* (MSP) on mast cell stabilization in experimental bronchial asthma.

Group	Granulated Mast Cells (%)	Degranulated Mast Cells (%)
Normal Control	–	–
Disease Control	26.4	73.6
Standard Control	78.2	21.8
Low Dose (MSP)	56.8	43.2
High Dose (MSP)	71.6	28.4







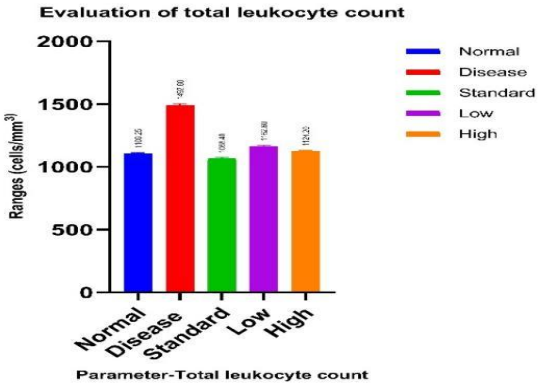


Figure 01:
% Inhibition of Mast Cell Degranulation of MSP in Rats

Histopathological Studies:

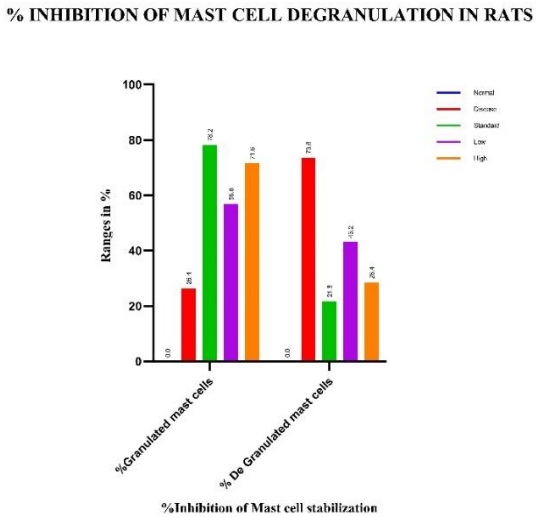
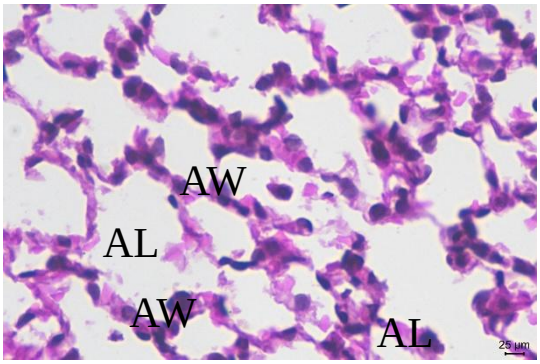
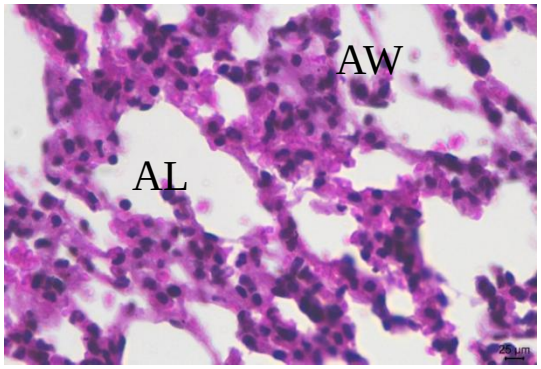


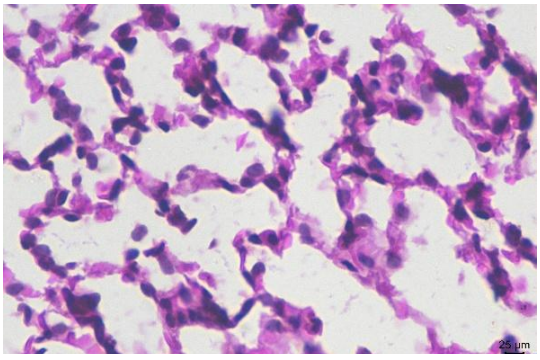
Figure 02: % Inhibition of Mast Cell Degranulation of MSP in Rats



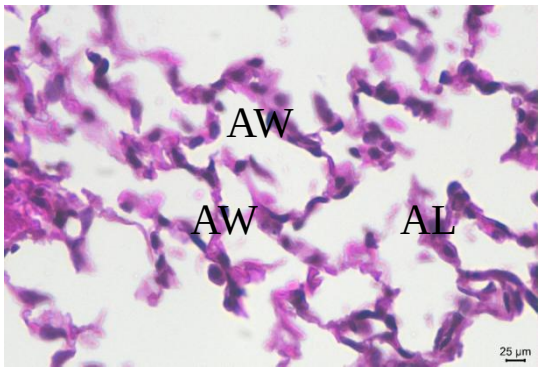
Lung section from control group showing normal alveoli wall (Aw) and alveolar lumen (Al). H&E.



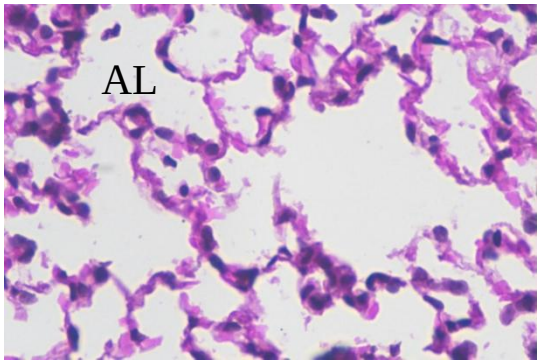
Lung section from Group II serves as Toxic Control GrouponlyReceived ALUM showing Infiltrates, mononuclear, interstitial, multifocal, mild.H&E



Lung section from Group III served as Standard receivedonly ALUM + Dexamethazone showing Infiltrates, mononuclear, interstitial, multifocal, minimal.H&E.



Lung section from MSP low dose – received p.o.) + ALUM showing Infiltrates, mononuclear, interstitial, multifocal, minimal.H&E.



Lung section from MSP high dose – received p.o.) + ALUM showing Infiltrates, mononuclear, interstitial, multifocal, minimal.H&E.

Effect of MSP on Haematological and Cytokine Parameters

MSP treatment significantly reduced elevated inflammatory markers compared to the disease control group.

- Leukocytes & Neutrophils: Reduced dose-dependently
- Eosinophils: Markedly decreased, especially at high dose
- Cytokines (TNF-α, IL-4, IL-5): Significantly lowered
- IgE Levels: Reduced towards normal levels
- High dose MSP showed results closer to standard drug

Mast Cell Stabilization Activity

Disease control showed maximum degranulation (73.6%)

MSP showed dose-dependent protection

High dose MSP showed 71.6% granulated cells, comparable to standard

Indicates strong mast cell stabilizing activity.

Histopathological Findings

Normal Control: Normal alveolar structure

Disease Control: Inflammatory infiltration and airway damage

Standard & MSP Groups: Markedly reduced inflammation and near-normal lung architecture

Histopathological Findings

Normal Control: Normal alveolar structure

Disease Control: Inflammatory infiltration and airway damage

Standard & MSP Groups: Markedly reduced inflammation and near-normal lung architecture

DISCUSSION

Bronchial asthma is associated with airway inflammation, characterized by increased leukocyte and eosinophil infiltration. Eosinophils play a crucial role in disease progression by releasing cytotoxic proteins and inflammatory mediators, leading to bronchial hyper responsiveness. In the present study, histamine exposure significantly elevated leukocyte count, eosinophils, IgE levels, and cytokines, confirming the induction of an asthmatic condition. MSP treatment effectively reduced these parameters, indicating its anti-inflammatory and anti-allergic properties. The reduction in eosinophil count suggests suppression of airway inflammation, while decreased IgE levels indicate modulation of allergic immune responses. Additionally, inhibition of cytokines such as TNF-α, IL-4, and IL-5 further supports the immunomodulatory action of MSP.Mast cell stabilization observed in this study highlights the ability of MSP to prevent the release of histamine and other mediators, thereby reducing bronchoconstriction. Histopathological findings also confirmed the protective effect of MSP on lung tissue.Overall, the results demonstrate that MSP exerts its anti-asthmatic activity through multiple mechanisms, including anti-inflammatory, immunomodulatory, and mast cell stabilizing effects.

CONCLUSION

The present study demonstrated that ***Menisamoola Parpam*** (MSP) possesses significant anti-asthmatic activity in a histamine-induced bronchial asthma model in Wistar rats. Administration of MSP resulted in a marked reduction in leukocyte, neutrophil, and eosinophil counts, along with significant suppression of serum IgE levels and pro-inflammatory cytokines, including TNF-α, IL-4, and IL-5. The formulation also exhibited notable mast cell stabilizing activity by inhibiting mast cell degranulation in a dose-dependent manner. Histopathological examination of lung tissues revealed reduced inflammatory cell infiltration and restoration of normal pulmonary architecture in MSP-treated animals. These findings indicate that MSP effectively attenuates airway inflammation and allergic responses associated with bronchial asthma.The observed therapeutic effects may be attributed to the anti-inflammatory, immunomodulatory, and mast cell stabilizing properties of MSP. Overall, the study provides scientific evidence supporting the traditional use of ***Menisamoola Parpam*** in respiratory disorders and suggests its potential as a promising Siddha therapeutic intervention for the management of bronchial asthma. Further pharmacological investigations and clinical studies are warranted to establish its safety, efficacy, and mechanism of action in humans.

ACKNOWLEDGEMENT

Not Applicable

CONFLICT OF INTEREST

Not Declared

AUTHOR CONTRIBUTION

All authors are contributed equally

ETHICAL STATEMENT AND INFORM CONSENT

Not Applicable

REFERENCE

1. Government of India, Ministry of Health and Family Welfare, Department of Health. *The Siddha Formulary of India*. Part I. New Delhi: Controller of Publications; 1992.
2. Shimizu T. Role of eosinophils in airway inflammation. Allergol Int. 2003;52(1):1-6.
3. Gould HJ, Sutton BJ. IgE-mediated mechanisms in allergic diseases. Nat Rev Immunol. 2008;8(3):205-217.
4. Walsh ER. Eosinophils in allergic airway disease. Curr Opin Immunol. 2010;22(6):781-788.
5. Limbasiya KK, et al. Experimental evaluation of anti-asthmatic activity. Int J Pharm Sci Res. 2012;3(7):2105-2110.
6. Barnes PJ. Mechanisms of asthma and airway inflammation. Br J Pharmacol. 2006;147(Suppl 1):S297-S303.
7. Global Initiative for Asthma. *Global strategy for asthma management and prevention*. Fontana (WI): Global Initiative for Asthma; 2023.
8. Kumar V, Abbas AK, Aster JC. *Robbins and Cotran Pathologic Basis of Disease*. 10th ed. Philadelphia (PA): Elsevier; 2020.