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Original Research Article

Effects of *Mimosa pudica* Extract on Asthma Biomarkers and Fetal Development in Pregnant Rats

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ABSTRACT

Asthma is a chronic disease that affects approximately 12% of pregnant women annually, with the highest prevalence reaching 16%. During pregnancy, asthma can lead to complications such as premature birth, low birth weight, congenital abnormalities, respiratory issues, jaundice, gestational diabetes, intrauterine growth retardation (IUGR), gestational hypertension, premature rupture of membranes, placental abruption, and placenta previa. This study aims to evaluate the potential of ethanol extract of *Mimosa pudica* (EEMP) in pregnant rat models with asthma. The biomarkers assessed included immunoglobulin E (IgE), histamine, superoxide dismutase (SOD), and insulin-like growth factor-1 (IGF-1) through ELISA examination, as well as airway remodeling through lung tissue histopathology and fetal morphometry through measurements of weight and body length. EEMP significantly reduced serum IgE levels ($p < 0.001$), serum histamine levels ($p < 0.001$), serum SOD levels ($p < 0.001$), bronchial epithelial cell proliferation ($p < 0.001$), and lung inflammation ($p < 0.001$). It also significantly increased serum IGF-1 levels ($p < 0.001$), fetal body weight ($p < 0.001$), and fetal body length ($p < 0.001$). While EEMP shows promise as a potential therapy for pregnant women with asthma, further toxicity testing is needed before clinical use.

Keywords: Airway remodeling, Asthmatic pregnancy, Fetal morphometry, *Mimosa pudica Linn*

Introduction

Asthma during pregnancy affects the severity of the condition and its treatment, potentially affecting the fetus as well.[1] In Indonesia, approximately 2-5% of the population suffers from asthma.[2] The Asthma and Allergy foundation of America identifies asthma as the most common disease affecting pregnancy, with a global prevalence of 4-12%. Epidemiological studies have reported that pregnancy with asthma is associated with an increased incidence of fetal growth restriction, which has significant short-term and long-term consequences for the fetus.[3] According to the AAFA, one in three pregnant women with asthma experiences worsening symptoms, another third experiences the same symptoms, and the remaining third experiences improvement of symptoms. Most women experience changes in symptoms from pregnancy to non-pregnancy within three months after giving birth. If asthma is uncontrolled, oxygen levels in the maternal blood decrease, which in turn reduces oxygen levels in the fetal blood, potentially affecting fetal development and survival.

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A constant oxygen supply of oxygen is crucial for the normal growth and development of the fetus .[4]

Asthma in pregnant women is often caused by exposure to allergens, which are processed by dendritic cells called antigen-presenting cells (APCs). These cells stimulate the maturation of dendritic cells and the differentiation of cells into T helper 2 (Th2) cells. Th2 cells produce cytokines that stimulate the maturation of B cells, while mast cells produce IgE and histamine, which are involved in inflammation and the development of asthma. This leads to the thickening of the lung epithelium and reduced smooth contraction. Epithelial cells play a role in the secretion of IL-33, which can bind to mast cells and increase the production of Th2 cytokines.[5] Increased cortisol levels can reduce the function of the 11-β-hydroxysteroid dehydrogenase 2 (11β-HSD2) enzyme, leading to a decrease in insulin-like growth factor 1 (IGF-1) and potentially inhibiting fetal growth, affecting both fetal weight and height. Another cause is the secretion of the IL-33 cytokine induced by Th2 cells due to asthma and increased reactive oxygen species (ROS) during pregnancy.[6]

During pregnancy, asthma can lead to complications such as preterm birth, low birth weight, congenital anomalies, respiratory issues, jaundice, gestational diabetes, intrauterine growth retardation (IUGR), gestational hypertension, premature rupture of membranes, and placental complications such as placental abruption and placenta previa.[7] Compared to the potential risks of palliative and control medications, asthma treatment can have positive effects on both the mother and the child. It is important to ensure that the treatment given includes inhaled corticosteroids (ICS), as asthma exacerbations carry the risk of preterm birth, low birth weight, and fetal death.[8] According to the Global Initiative for Asthma (GINA), the recommended treatment for asthma during pregnancy includes the administration of prednisolone, a corticosteroid that is often used to reduce airway

inflammation. Prednisolone is effective in managing acute exacerbations of asthma and providing long-term control for more severe cases of asthma. Its mechanism of action is to inhibit the inflammatory process by reducing airway inflammation, blocking the release of inflammatory mediators such as histamine and leukotrienes, and reducing capillary permeability. The immunosuppressive effects of prednisolone suppress excessive immune responses that can cause chronic inflammation in asthma.[9]

There are several non-pharmacological or ethnomedicinal plants used to treat asthma. Plants such as *Cassia occidentalis* whole plant (C.O.W.P), *Jatropha curcas* leaves (J.C.L.), *Ximenia americana* leaves (X.A.L.), and *Eucalyptus citriodora* leaves (E.C.L.) have been widely used to treat asthma and other airway-related diseases in Northwestern Nigeria. [10,11] *Mimosa pudica Linn* (MP) is a member of the *Mimosa* family and traditionally used as a remedy for various diseases. *Mimosa pudica* contains compounds such as mimosine (alkaloid), free amino acids, sitosterol, quercetin, tannins, and flavonoid glycosides.[12] The plant has antibacterial, antioxidant, toxic, diuretic, anticancer, antidiabetic, antireproductive, and antihistamine effects. .[13] Its anti-asthma properties include acting as a bronchodilator, antihistamine (H1 antagonist), stabilizing mast cells, and showing potential in the prevention and treatment of asthma.[14] This study aims to evaluate the effects of the ethanol extract of *Mimosa pudica Linn* (EEMP) on immunoglobulin E (IgE) levels, histamine, superoxide dismutase (SOD), IGF-1, fetal weight, fetal length, and airway remodeling in pregnant rat models with asthma

Materials and Methods

Materials

This study used six-week-old *Rattus norvegicus*, weighing 150-200 grams, all of which were healthy and active. *Mimosa pudica Linn* was obtained from Herbal Medika Technical Service Unit (UPT) in Malang, East Java, Indonesia. Ovalbumin (Sigma, St. Louis, MO, USA A5503), 6 mg of aluminum hydroxide [Al (OH)3] (Aluminum hydroxide, 239186-25G Sigma-Aldrich), and 1200 µl of phosphate buffer saline (Biogear, BGPBS-002).

In vivo experiment

The rats were acclimatized for seven days. At seven and eight weeks of age, the rats were induced with 60 µg of ovalbumin (Sigma, St. Louis, MO, USA A5503), 6 mg of aluminum hydroxide [Al (OH)3] (Aluminum hydroxide, 239186-25G Sigma-Aldrich), and 1200 µl of phosphate buffer saline (Biogear, BGPBS-002) intraperitoneally. Subsequently, at nine weeks of age, the rats were mated with male rats overnight at a ratio of 4:2. Successful pregnancy was confirmed by the presence of a vaginal plug, marking day 0 of pregnancy (GD 0). Then, the rats were divided into five groups: N = normal (normal pregnancy) without intervention (n = 7); KN = negative control (asthmatic pregnancy without treatment) (n = 7); KP = positive control (asthmatic pregnancy with 0.9 mg/kg prednisolone intervention) (n = 7); P1 = treatment 1 (asthmatic pregnancy with 125 mg/kg *Mimosa pudica Linn* ethanol extract intervention) (n = 7); and P2 = treatment 2 (asthmatic pregnancy with 250 mg/kg *Mimosa pudica Linn* ethanol extract intervention) (n = 7).

At 10 weeks of age, a pre-test was conducted to check IgE and histamine levels in the 35 rats to confirm asthma. Subsequently, the administration of the ethanol extract of *Mimosa pudica* (EEMP) in two doses and standard therapy for one group commenced and continued for 14 days. To maintain the asthma model, the rats were re-sensitized on the ninth, 12th and 17th days of gestation through aerosol exposure with 1% OVA and PBS for 45 minutes with an interval of two days between exposures. At 19th days of gestation, all rats were euthanized, and the levels of IgE (Bioenzy ELISA Kit IgE No.BZ-22184952-EB), histamine (Bioassay Technology Laboratory ELISA Kit *Histamine* Cat. No. E0788Ra), and superoxide dismutase (SOD) (Bioassay Technology Laboratory ELISA Kit SOD Cat. No. E2269Ra) were measured in the maternal rats using the ELISA method. Insulin-like growth factor-1 (IGF-1) (Bioassay Technology Laboratory ELISA Kit IGF-1 Cat. No. E0709Ra) was measured in the fetuses. Lung histopathology was examined in the maternal rats, while fetal morphometry was assessed by measuring fetal weight and length. This study was conducted at the laboratory of Setia Budi University, Surakarta, Central Java, following the research protocol approved by the Ethics Committee of the Faculty of Medicine, Sebelas Maret University, Indonesia with a certificate number 48/UN27.06.11/KEP/EC/2023.

ELISA Examination, Lung Histopathology, and Fetal Morphometry Measurement

The examination of IgE, histamine, and superoxide dismutase in maternal rats was conducted using the ELISA method. The insulin-like growth factor-1 (IGF-1) was also measured through the ELISA method using rat blood serum. Histopathological examination of lung tissue was conducted at the Anatomy Pathology Laboratory of Sebelas Maret University, Surakarta, Indonesia. Measurements of fetal weight and length were carried out at the laboratory of Setia Budi University, Surakarta, Indonesia.

Statistical Analysis

Data analysis was performed using the Statistical Package for Social Sciences (SPSS) version 27.0. The data on IgE, histamine, SOD, IGF-1, fetal weight, and fetal length were subjected to a paired sample t-test followed by a normality test using the Shapiro-Wilk method. If the data were normally distributed ($p > 0.05$), a repeated measures ANOVA test was used to determine significant differences across repeated measurements of the research variables. Non-normally distributed data were analyzed using the non-parametric Friedman test. If the repeated measures ANOVA test showed significant differences, post-hoc tests were conducted. Lung histopathology data were analyzed using the non-parametric Kruskal-Wallis test. If significant differences were found, the Mann-Whitney test was performed. The significance level was set at 0.05, with statistically significant values considered when p-value was less than 0.05.

Results and Dicussion

Plant Determination

*Mimosa pudica Lin*n was obtained from Herbal Medika Technical Service Unit (UPT) in Malang, East Java, Indonesia. The plant was identified and confirmed as *Mimosa Pudica L* (No. 074/626/102.20-A/2022). The plant extract was prepared at the same facility using the maceration method with 96% ethanol solvent.

Composition of the Ethanol Extract of Mimosa pudica

Qualitative phytochemical testing was conducted at the Herbal Medika Technical Service Unit in Malang, East Java, Indonesia, with the following results: flavonoids (+), alkaloids (+), tannins/phenols (+), steroids (-), triterpenoids (+), saponins (+), carbohydrates (+), proteins (+), and vitamin C (+) (No. 074/122/102-7D/2022). Quantitative testing was performed at LPPT II UGM (No. 00103A.01/III/UN1/LPPT/2023) with the following results: DPPH activity (IC50) = 0.47 (very strong), IKU/7.2/TF-UV-01 (UV-Vis spectrophotometry), quercetin = 327.61 mg/Kg TLC method, total flavonoids = 16.30% (w/w) (UV-Vis spectrophotometry), Zn = 24.04 mg/Kg (Flame SSA), and magnesium = 408.20 mg/Kg (Flame SSA).

The Levels of Immunoglobulin E (IgE)

Immunoglobulin E plays a crucial role in asthma as a primary mediator in allergic reactions. When a person with allergic asthma is exposed to an allergen, IgE binds to receptors on the surface of mast cells and basophils triggers the release of inflammatory mediators, such as histamine, leukotrienes, and cytokines. These mediators cause asthma symptoms, including bronchoconstriction, airway inflammation, and excessive mucus production. IgE also contributes to the amplification of chronic inflammatory responses, which can worsen asthma over time. Figure 1 shows changes in IgE levels in each treatment group. In the normal group without intervention (N), the mean IgE levels were 216.10 ± 23.86 at H0 and 191.20 ± 23.94 at H19, serving as the baseline or comparator for the treatment groups. In the negative control group

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(KN) (asthmatic pregnancy without treatment), the IgE levels increased significantly from 231.00 ± 72.27 at H0 to 277.32 ± 65.38 at H19. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the IgE levels decreased from 234.47 ± 54.40 at H0 to 161.20 ± 18.19 at H19. For treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP), the IgE levels decreased from 271.20 ± 34.20 at H0 to 218.75 ± 39.00 at H19. Similarly, for treatment 2 (P2) (asthmatic pregnancy with 250 mg/kg BW EEMP), the IgE levels decreased from 272.49 ± 44.39 at H0 to 161.20 ± 16.40 at H19. The results of the repeated measures ANOVA test indicated a significant effect of EEMP administration on IgE levels ($p < 0.005$).

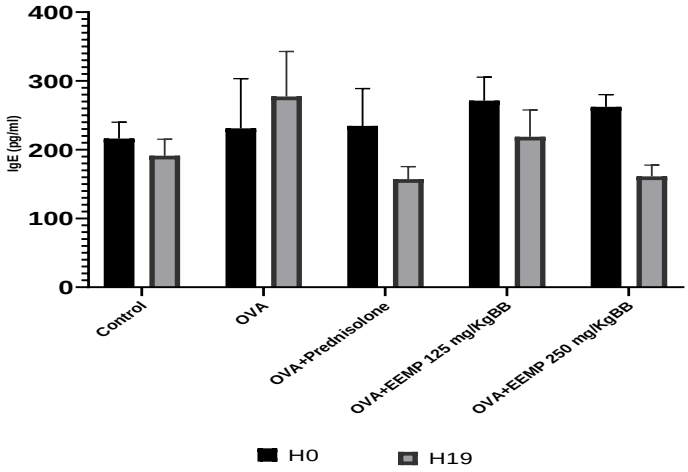

Figure 1: *Immunoglobulin E* level of Before and After Treatment (IgE)

The Levels of Histamine

Figure 2 shows changes in histamine levels in each treatment group. In the normal group without intervention (N), the mean histamine levels were 18.27 ± 2.90 at H0 and 17.97 ± 2.51 at H19, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the histamine levels increased significantly from 22.94 ± 2.39 at H0 to 25.74 ± 1.99 at H19. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the histamine levels decreased from 25.25 ± 2.86 at H0 to 22.03 ± 1.73 at H19. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) showed a decrease in histamine levels from 26.22 ± 3.78 at H0 to 23.06 ± 3.45 at H19. Similarly, treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) showed a decreased in histamine levels from 28.37 ± 3.09 at H0 to 24.11 ± 1.78 at H19. The results of the repeated measures ANOVA indicated a significant effect of EEMP administration on histamine levels ($p < 0.005$).

The Levels of Superoxide Dismutase (SOD)

Figure 3 shows changes in SOD levels in each treatment group. In the normal group without intervention (N), the mean SOD levels were 142.09 ± 32.65 at H0 and 149.30 ± 49.31 at H19, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the SOD levels increased significantly from 416.15 ± 91.96 at H0 to 487.73 ± 39.05 at H19. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the SOD decreased from 378.27 ± 65.05 at H0 to 136.59 ± 12.98 at H19. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) showed a decrease from 362.74 ± 47.95 at H0 to 193.97 ± 36.22 at H19. Oxidative stress plays a role in the pathogenesis of chronic airway inflammation in asthma, with reactive oxygen species (ROS) being key contributors. Increased ROS production can lead to increased levels of malondialdehyde (MDA) and total oxidant status (TOS), as well as a decrease in the activity of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and total antioxidant status (TAS) in asthma patients. Similarly, treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) showed a decrease from 391.67 ± 42.98 at H0 to 194.69 ± 13.84 at H19. The results of the repeated measures ANOVA test indicated a significant effect of EEMP administration on SOD levels ($p < 0.005$).

Histopathological Examination of Airway Remodeling Markers
Epithelial Cell Proliferation

Epithelial cell proliferation is an observed effect of asthma, and the examination was performed using rat lung tissue. Figure 4 shows changes in the epithelial cell proliferation in each treatment group as indicated by mean rank values. In the normal group without intervention (N), the mean rank was 4.00, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean rank mean rank was 26.07, indicating a significant increase. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean rank was 19.07. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean rank of 18.57, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean rank of 20.17. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on epithelial cell proliferation ($p < 0.005$).

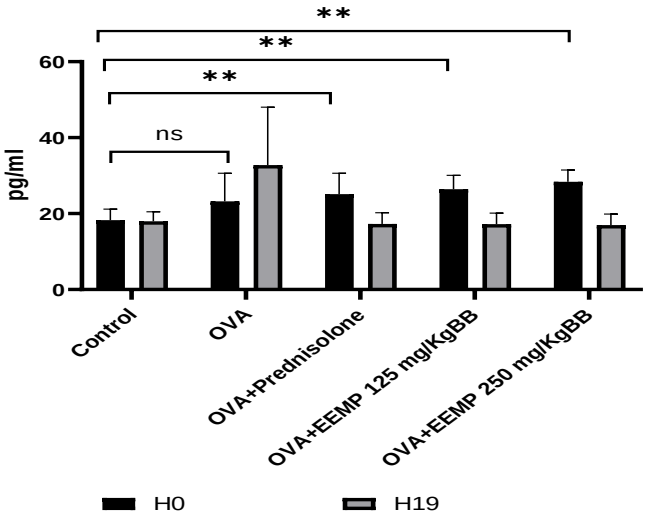

Figure 2: The *Histamine levels* before and after treatment

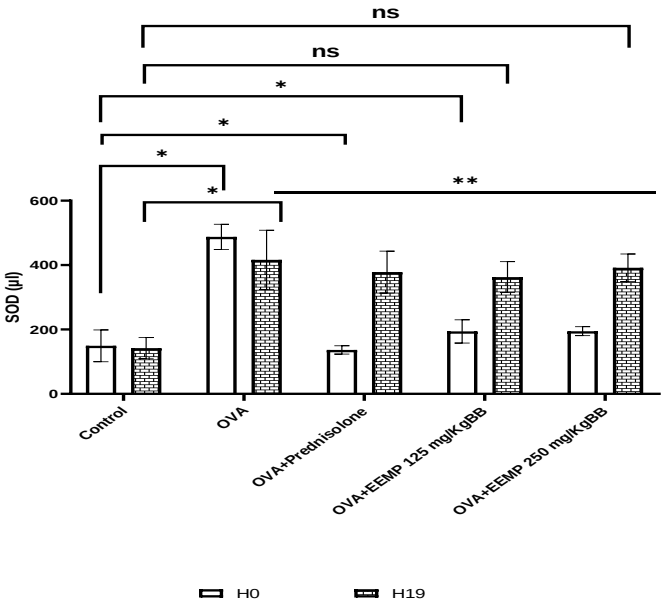

Figure 3: *The Superoxide Dismutase* (SOD) level

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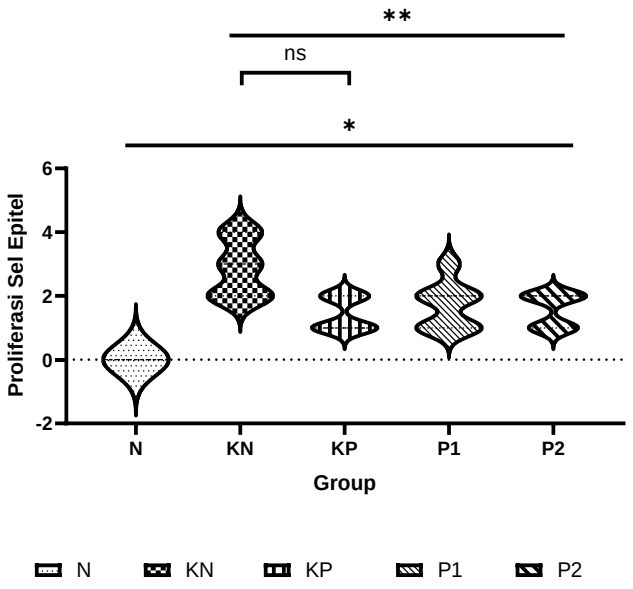

Figure 4: *Epithelial Cell Proliferation*

Muscle Cell Hyperplasia

Muscle cell hyperplasia is another observed effect of asthma, and the examination was performed using rat lung tissue. Figure 5 shows changes in the muscle cell hyperplasia in each treatment group as indicated by mean rank values. In the normal group without intervention (N), the mean rank was 4.00, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean rank was 22.86, indicating a significant increase. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean rank was 15.07. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean rank of 16.07, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean rank of 16.01. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on muscle cell hyperplasia ($p < 0.005$).

Lung Inflammation

Lung inflammation is a common consequence of asthma, and the examination was conducted using rat lung tissue. Figure 6 shows changes in the lung inflammation in each treatment group as indicated by mean rank values. In the normal group without intervention (N), the mean rank was 4.50, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean rank was 27.71, indicating a significant increase. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean rank was 17.79. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean rank of 20.93, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean rank of 19.07. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on lung inflammation ($p < 0.005$).

Insulin-Like Growth Factor-1 Levels

Insulin-like growth factor-1 (IGF-1) is a growth hormone that plays a role in fetal development, and the examination was conducted using fetal blood serum. Figure 7 shows changes in the levels of IGF-1 in each treatment group as indicated by mean values. In the normal group without intervention (N), the mean IGF-1 level was 266.10± 35.86, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean was 231.00 ± 72.27, indicating a significant decrease. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean was 234.47 ± 54.40. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean IGF-1 level of 271.20 ± 34.20, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean of 315.49 ± 44.39. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on IGF-1 ($p < 0.005$).

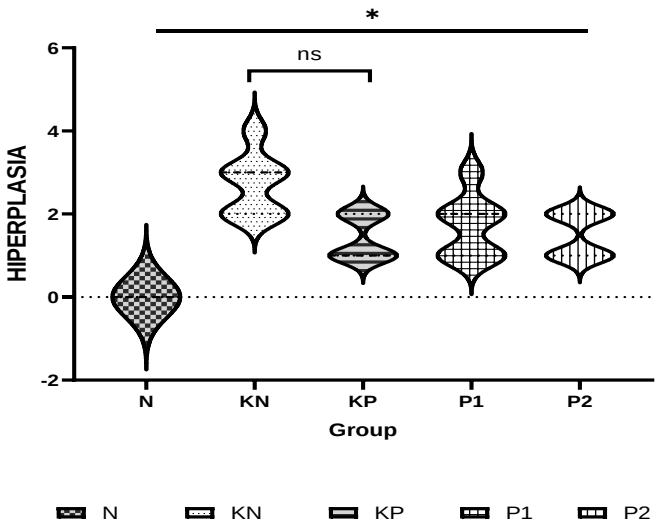

Figure 5: *Muscle Cell Hyperplasia*

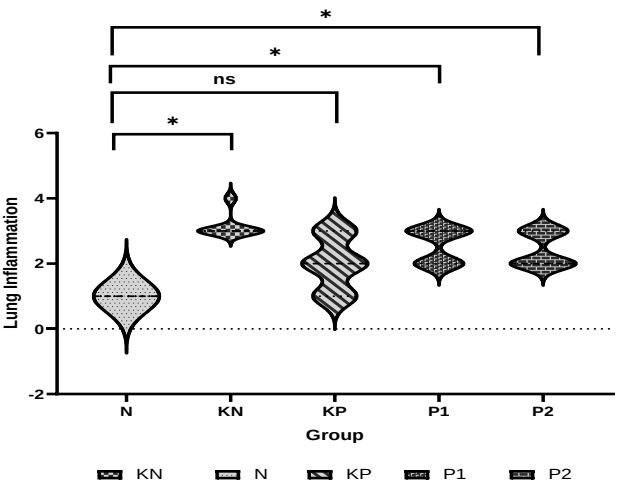

Figure 6: *Lung inflammation*

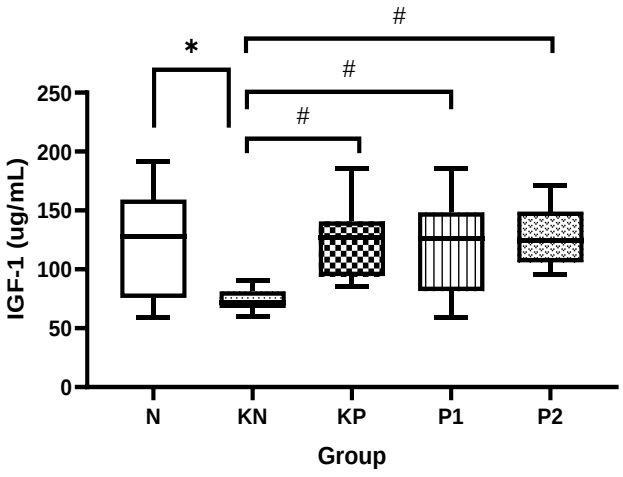

Figure 7: *Insulin Growth Factor-1 Levels*

Fetal Weight

Fetal weight is a critical outcome of asthma, and the measurement was conducted using an Ohaus scale. Figure 8 shows changes in the fetal weight in each treatment group as indicated by mean values. In the normal group without intervention (N), the mean fetal weight was 4.25 ± 0.62, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean was 2.48 ± 0.28, indicating a significant decrease. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean was 4.23 ± 1.04. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean fetal weight of 3.65 ± 0.76, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean of 3.99 ± 1.01. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on fetal weight ($p < 0.005$).

Fetal Body Length

Fetal body length is a critical outcome of asthma, and the measurement was conducted using a ruler. Figure 9 shows changes in the fetal body length in each treatment group as indicated by mean values. In the normal group without intervention (N), the mean fetal body length was 3.73 ± 0.62, serving as the baseline or comparator for the treatment groups. In the negative control group (KN) (asthmatic pregnancy without treatment), the mean was 2.64 ± 0.28, indicating a significant decrease. In the positive control group (KP) (asthmatic pregnancy with prednisolone treatment), the mean was 3.66 ± 1.04. Treatment 1 (P1) (asthmatic pregnancy with 125 mg/kgBW EEMP) yielded a mean fetal body length of 3.38 ± 0.76, while treatment 2 (P2) (asthmatic pregnancy with 250 mg/kgBW EEMP) yielded a mean of 3.65 ± 1.01. The results of the Kruskal-Wallis test indicated a significant effect of EEMP administration on fetal body length ($p < 0.005$).

The determination of research biomarkers in this study aligns with the pathophysiology of asthma and its effects on pregnancy. Immunoglobulin E (IgE) and histamine are early indicators of asthma triggered by exposure to allergens. Superoxide dismutase (SOD) serves as a marker of oxidative stress associated with the disease. Airway remodeling is a consequence of asthma detectable in lung tissue, indicated by smooth muscle cell hyperplasia, epithelial cell proliferation, and lung inflammation. Furthermore, insulin-like growth factor-1 (IGF-1) plays a role in fetal growth, with fetal weight and length being key measures to assess the effects of asthma on fetal morphometry.

This study provides evidence that EEMP is a potentially effective alternative treatment for pregnant rat models with asthma. The data suggested that EEMP is equivalent or even has the same effect as prednisolone in improving asthma symptoms during pregnancy. These findings offer a new and effective strategy for managing asthma during pregnancy. *Mimosa pudica*, a plant commonly found in Indonesia, can be an innovative solution for treating asthma in pregnant women. With effective management, the prevalence of asthma during pregnancy can be mitigated, thereby reducing its negative impacts on both mothers and babies.[15]

The ethanol extract of *Mimosa pudica* (EEMP) had a notable impact on asthma biomarkers, namely immunoglobulin E (IgE), histamine, and superoxide dismutase (SOD). EEMP alone could significantly improve asthma symptoms in pregnant rats. The examination of lung tissue revealed significant changes in smooth muscle cell hyperplasia, epithelial cell proliferation, and lung inflammation as confirmed by statistical analysis. Additionally, fetal morphometry in terms of fetal weight and length showed an increase, which was supported by an increase in the insulin-like growth factor-1 (IGF-1), a key marker of fetal development.

Mimosa pudica is a plant known for its biological activities, including antimicrobial, antioxidant, antivenom, diuretic, anticancer, antidiabetic, antifertility, and antihistamine properties.[13] The anti-asthma effects of *Mimosa pudica* include bronchodilation, antihistamine (H1 antagonist), mast cell stabilization, showing its potential in asthma prevention and management.[16] The quercetin content in EEMP contributes to its anti-allergic effects by inhibiting histamine production and pro-inflammatory mediators. Quercetin also plays a role in regulating Th1/Th2 stability and reducing the release of specific IgE antibodies by B cells. As a natural polyphenolic flavonoid found in many fruits, vegetables, and nuts, quercetin is recognized for its anti-inflammatory properties, which have been associated with potential benefits for managing asthma, especially in patients who do not responded well to inhaled beta-agonist and corticosteroid treatments.[17]

Immunoglobulin E (IgE) plays a crucial role in determining a disease. As an initial marker, IgE reflects an individual's immune response, with variations depending on their immune response. Clinical symptoms of type I hypersensitivity reactions involve chemical mediators such as histamine, leukotrienes, and cytokines. These mediators can increase blood vessel permeability, cause smooth muscle constriction, stimulate mucus secretion, and promote inflammation. IgE levels are associated with asthma, where IgE antibodies are sensitized by plasma cells. Several studies have demonstrated a correlation between IgE levels and asthma.[18]

Histamine is a central mediator released from mast cells during allergic reactions. Histamine contributes to airway obstruction by causing smooth muscle contraction, bronchial secretion, and mucosal airway edema. Histamine induces bronchial contraction.[10] Histamine released by mast cells in response to allergens is a major mediator in allergic reactions, including asthma, and its effects can be exacerbated by pregnancy.[19] During asthma attacks, histamine causes narrowing of the airways, resulting in difficulty breathing, excessive mucus production, and increased blood flow to the airway walls, leading to inflammation and swelling. This process causes asthma symptoms such as wheezing, coughing, shortness of breath, and chest tightness. Histamine affects the airways through H1 receptors. When histamine binds to these receptors, it triggers various events that lead to inflammation and bronchoconstriction.[20]

Increased superoxide dismutase (SOD) activity in asthma indicates a complex relationship between inflammation, oxidative stress, and cell function in the respiratory tissues. Research shows that SOD activity is reduced in the respiratory tissues of individuals with asthma, which is an indicator of cell damage and serious inflammation.[21]Severe inflammation can lead to structural changes in the respiratory tissues, known as airway remodeling. This remodeling involves morphological changes in the respiratory tissues, including changes in the bronchial epithelium, which can exacerbate asthma conditions.[22]

Insulin-like growth factor-1 (IGF-1) plays a crucial role in the growth and development of embryos, significantly influencing fetal morphometry, especially the physical size and shape of the fetus. IGF-1 has been shown to influence several aspects of embryo development, including cell proliferation and morphology. In rats, IGF-1 stimulates preimplantation embryo growth in vitro by increasing the number of cells in the inner cell mass (ICM). This indicates that IGF-1 can influence the cellular composition of developing embryos.[23]

The clinical manifestations of asthma, such as airway hyperresponsiveness, airflow limitation, and persistent symptoms, result from these structural changes. Airway remodeling is associated with worse clinical outcomes in asthma patients. The mechanisms responsible for airway remodeling in asthma patients are not yet fully understood, but chronic inflammation is believed to play a significant role in initiating and perpetuating the remodeling process. During the inflammatory response, inflammatory mediators, growth factors, and cytokines can activate various cell types in the airway wall, leading to structural changes.[24]

The components of the ethanol extract of *Mimosa pudica* (EEMP) for asthma management function as anti-inflammatory agents by inhibiting various inflammatory pathways, including the activities of lipoxygenase and cyclooxygenase enzymes, and reduce inflammatory cytokines. Its antioxidant mechanism involves scavenging free radicals and reducing oxidative stress, which plays a role in the pathogenesis of asthma. The immunomodulatory function modulates the immune response, reducing the activity of mast cells and eosinophils involved in allergic reactions and respiratory tract inflammation

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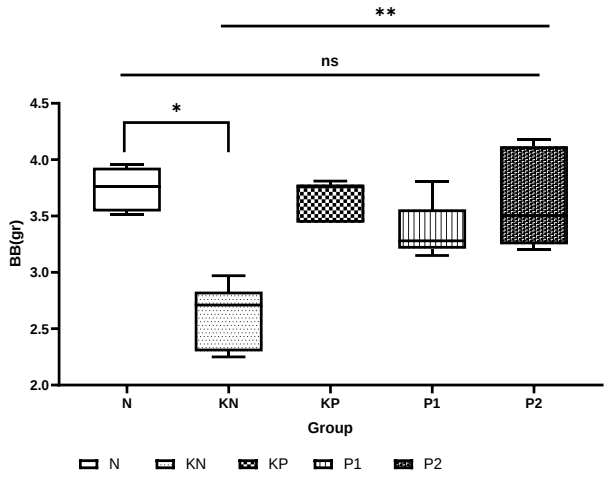

Figure 8: *Fetal Weight*

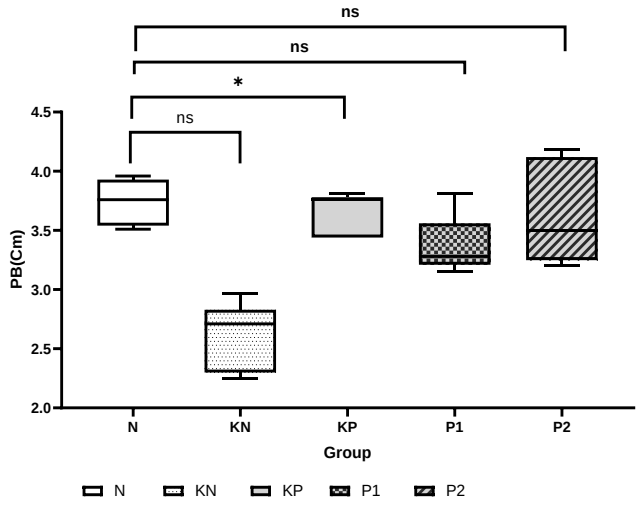

Figure 9: *Fetal Body Length*

Conclusion

The effect of EEMP in both mothers and babies had a similar performance to that of prednisolone. However, further studies on the toxicity and safety of consuming EEMP are necessary before administering it to pregnant women despite its effective results in animal experiments.

Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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