



Ethanol Extract of *Jatropha curcas* (Euphorbiaceae) Leaves Ameliorates Allergic Airway Inflammatory Responses in Wistar Rats

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ABSTRACT

Jatropha curcas (*J. curcas*), a member of the Euphorbiaceae family, is a tropical medicinal plant that has been used in African Traditional Medicine for the treatment of asthma and related allergic airway diseases. This study investigated the ameliorative effects of *Jatropha curcas* leaf extract on ovalbumin-induced allergic airway inflammation in Wistar rats. Preliminary phytochemical screening of the extract was carried out using standard procedures, and oral median lethal dose (LD₅₀) was determined using Lorke's method. Allergic airway inflammation was induced in Wistar rats via ovalbumin sensitization and challenge over 28 days. Rats were divided into six groups: normal control, ovalbumin control, three groups treated with *J. curcas* extract at 35, 70, and 140 mg/kg, and a positive control receiving prednisolone (10 mg/kg). Post-treatment, bronchoalveolar lavage fluid (BALF) was analyzed for leukocytes and cytokines, and lung tissues were examined histologically. Phytochemical analysis identified flavonoids, alkaloids, terpenes, saponins, glycosides, steroids, and anthraquinones. Acute oral toxicity test revealed an LD₅₀ exceeding 5000 mg/kg, indicating that the extract is relatively safe. Anti-inflammatory activity evaluation showed that *J. curcas* at 140 mg/kg dose significantly ($p \leq 0.05$) reduced eosinophil counts and IL-13 levels, which was comparable to that of prednisolone group. The extract also showed a dose-dependent reduction in IL-4 and IL-5 levels. Histopathological examination revealed protection against alveolar necrosis and tracheal hyperplasia at higher doses. Overall, the extract demonstrated notable anti-inflammatory and airway-protective effects, suggesting its potential as a complementary therapy for allergic airway diseases. Further clinical studies are warranted to confirm these findings.

Keywords: *Jatropha curcas*, Allergic Airway Inflammation, Cytokines, Eosinophilia, Phytochemicals, Bronchoalveolar Lavage Fluid

Introduction

Allergic airway inflammatory diseases, including allergic rhinitis and asthma, are chronic conditions in humans, with increasing global prevalence, particularly in children and adolescents.¹ These conditions are characterized by excessive mucus secretion, hyperresponsiveness of airways, and eosinophilic infiltration, which contribute to significant morbidity and healthcare burdens.² According to the 2022 global asthma report, asthma alone affected about 262 million people (3416 cases per 100,000 population) and caused 461,000 deaths worldwide (more than 1000 persons per day), resulting in substantial clinical and economic burdens.³

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The prevalence of allergic rhinitis varies widely across the globe, affecting 10 - 40% of the population, with a significant impact on quality of life and overall cognitive functioning.¹ Asthma and allergic rhinitis often coexist with other atopic diseases, such as atopic dermatitis, forming part of an "atopic march".² The pathophysiology of airway allergic inflammatory conditions involves the recruitment of different inflammatory cells, including eosinophils and neutrophils, which release inflammatory mediators such as interleukin-4 (IL-4), interleukin-5 (IL-5), and interleukin-13 (IL-13), leading to airway remodeling and hyperresponsiveness. IL-4 promotes the differentiation of T-helper type 2 (Th2) cells, which drive the allergic inflammatory response,⁴ while IL-5 enhances eosinophil survival and recruitment.⁵ IL-13 significantly induces excessive secretion of mucus and airway fibrosis.⁶ Agents capable of modulating these cytokines and reducing immune cell infiltration could serve as promising therapeutic alternatives. The conventional management of allergic airway diseases primarily relies on antihistamines, glucocorticoids, and leukotriene receptor antagonists.⁷ However, these pharmacological agents are associated with adverse effects such as impaired growth in children, decreased bone mineral density, cataracts, skin thinning and bruising, altered glucose metabolism, and hypothalamic-pituitary-adrenal (HPA) axis suppression (corticosteroids), Tremor and hypokalemia (β_2 agonists), high recurrence rates, and variable patient responses, necessitating the search for alternative treatments.⁸

Jatropha curcas (*J. curcas*), a member of the Euphorbiaceae family, is

a tropical medicinal plant, abundantly distributed across Africa, Asia, and Latin America.⁹ It is a small tree or large shrub which can reach a height of up to 5 m,¹⁰ and possess 5 to 7 shallow lobed leaves with a length and width of 6 to 15 cm, which are arranged alternately. The plant is monoecious and flowers are unisexual; occasionally hermaphrodite flowers occur.¹¹ The plant has a rich ethnomedicinal history, particularly in Nigeria, where the leaf of the plant is mentioned by the locals to treat asthma. Decoction of the leaves is traditionally employed to treat edema, and cough,^{12,13} and latex for otorrhoea, cold, and cough.¹⁴ Previous studies have demonstrated that *J. curcas* leaf possesses airway smooth muscle relaxant and mast cell stabilizing properties, supporting its potential use in managing allergic airway inflammation.¹⁵ The present study aimed to provide pharmacological validation for the traditional use of *J. curcas* in the treatment of allergic airway inflammation. Specifically, it investigated the effects of ethanol leaf extract of *J. curcas* on ovalbumin-induced airway inflammatory responses in Wistar rats.

Materials and Methods

Drugs and chemicals

Prednisolone (Hovid Pharmaceuticals LTD), Ovalbumin from freshly laid chicken egg, Aluminium hydroxide (Al(OH)₃) (BDH Laboratory Division, POOLE, England), Heparin (American Remedies Healthcare pvt. Ltd.), staining solutions [Toluidine blue, (Enzo Life Science), Haematoxylin Eosin (Abbey Color)], ketamine, Ketotifen (Novartis Pharmaceuticals), ELISA kits for interleukines-4 (IL-4), IL-5 and IL-13 (Biosource International, Camarillo, CA, USA), Absolute ethanol (JHD, Sci-Tech Co Ltd., China), Biorad protein assay reagent (Bio-Rad Laboratories, Hercules, CA, USA), and normal saline (Juhel Pharmaceuticals).

Plant material collection and authentication

Jatropha curcas leaves were collected from Zangon Shanu area of Samaru, Zaria, in May 2019. The plant was identified and authenticated by a plant taxonomist at the Herbarium Section, Department of Biological Sciences, Ahmadu Bello University, Zaria, Kaduna State, Nigeria. Authentication was performed by comparing the specimen with existing herbarium samples (Voucher Specimen No. 1531).

Preparation of plant extract

The collected healthy, matured, and clean leaves were shade-dried in a single layer on a clean and dry surface with intermittent turning and weighing in a well-ventilated space until a constant weight was obtained. The leaves were protected from dust, insects, and environmental contaminants, and were regularly checked for mold, pests, and signs of deterioration throughout the drying period. The dried leaves were comminuted into a fine powder using a mortar and pestle. A total of 750 g of powdered *J. curcas* leaves was extracted with 7.5 L of 70% ethanol by cold maceration at room temperature for 72 hours with intermittent shaking. The extract was filtered, concentrated on a water bath at 40 - 60°C, and stored in a desiccator until required. Fresh solutions of the extract were prepared by reconstitution in distilled water for each experiment.

Phytochemical screening

Preliminary phytochemical screening of the ethanol leaf extract of *J. curcas* was carried out using standard procedures.¹⁶ The screening was conducted to detect the presence or absence of alkaloids, flavonoids, tannins, saponins, steroids, terpenoids, anthraquinones, glycosides, and carbohydrates.

Experimental animals

Adult Wistar rats (180 - 200 g) of either sex were obtained from the Animal House Facility of the Department of Pharmacology and Therapeutics, Ahmadu Bello University, Zaria, Kaduna State, Nigeria. The animals were acclimatized under standard laboratory conditions of a normal phase light-dark cycle for two weeks prior to the experiment. They were maintained on a standard laboratory animal diet with water *ad libitum*. All experimental procedures adhered to ethical guidelines for laboratory animal care, as outlined by the Sa'adu Zungur University

Research Policy (Revised 2010) and internationally accepted guidelines (National Institutes of Health, 1985; Publication No. 85-25, Revised 1996). Ethical approval (Approval No: SAZU/FBMS/REC/ VOL. 61/23/00101) for the study was obtained from the Committee on Animal Use and Care, Sa'adu Zungur University, Gadau, Bauchi State, Nigeria.

Acute toxicity study (Determination of LD₅₀)

The oral median lethal dose (LD₅₀) of *J. curcas* leaf extract was determined using Lorke's method.¹⁷ The study was conducted in two phases; phases I and II. In phase I, three groups of three rats each received the extract orally at single doses of 10, 100, and 1000 mg/kg. The animals were observed for 24 hours for signs of toxicity and mortality. In phase II, since no mortality was recorded in Phase 1, three additional groups (one rat per group) received single doses of 1600 mg/kg, 2900 mg/kg, and 5000 mg/kg of the extract. The LD₅₀ was calculated as the geometric mean of the lowest lethal dose and the highest non-lethal dose.

Pharmacological studies

Induction of allergic airway inflammation

Allergic airway inflammation was induced using an ovalbumin sensitization and challenge model, as described by Casaro *et al.* (2019).¹⁸ Thirty (30) Wistar rats were divided into six groups of five rats each (n = 5):

Group 1 (Normal control/Treatment naïve) animals received distilled water (1 mL/kg) without sensitization.

Group 2 (Ovalbumin control) was sensitized with ovalbumin (20 mg/kg, intraperitoneally) and received phosphate-buffered saline (PBS, 10 mL/kg orally).

Group 3 was sensitized with ovalbumin and treated with *J. curcas* leaf extract at a dose of 35 mg/kg, orally.

Group 4 was sensitized with ovalbumin and treated with *J. curcas* leaf extract at a dose of 70 mg/kg.

Group 5 was sensitized with ovalbumin and treated with *J. curcas* leaf extract at a dose of 140 mg/kg, orally.

Group 6 (Positive control) was sensitized with ovalbumin and treated with prednisolone at a dose of 10 mg/kg.

Sensitization was performed on days 0, 7, and 14 by intraperitoneal injection of ovalbumin (20 mg/kg) adsorbed on alum (100 mg/kg) in 1 mL of saline. On days 21–26, rats were sedated with ketamine (60 mg/kg) and challenged twice daily with intranasal 0.1 mL of 1% (100 mg/mL) ovalbumin. Treatments were administered orally 1 hour before each challenge daily from day 19 to 27. Rats were euthanized using ketamine anesthesia at a dose of 100 mg/kg on day 28 for sample collection.

Bronchoalveolar lavage fluid (BALF) analysis

After euthanasia, the trachea was cannulated, and the lungs were lavaged with 1 mL of phosphate-buffered saline. The retrieved fluid was centrifuged at 3000 rpm for 10 minutes. The supernatant was collected for cytokine analysis, while the cell pellet was resuspended for total and differential leukocyte counts using a hemocytometer and Diff-Quik staining. Cells were differentiated into eosinophils, neutrophils, and mononuclear cells using the cell counter mentioned above, and the absolute number and percentage of each cell type were determined. Multiple inflammatory mediators were analyzed in the cell-free supernatant of BALF by using a multiplex flow cytometry-based assay according to the manufacturer's recommendations (Bio-Rad Laboratories; Hercules, CA) and as previously described.¹⁹

Measurement of pro-inflammatory cytokines in BALF

The concentrations of IL-4, IL-5, and IL-13 in BALF were determined using enzyme-linked immunosorbent assay (ELISA) kits with a detection limit of <10 pg/mL for IL-2, <5 pg/mL for IL-4, <10 pg/mL for IL-10, and <10 pg/mL for IFN- γ .²⁰ A microplate reader was used to measure absorbance at 450 nm. BALF total protein was measured using Bio-Rad Protein Assay Reagent.²⁰

Histopathological examination

Lung and tracheal tissues were excised, fixed in 10% formalin, and embedded in paraffin. Sections (5 µm thick) were stained with hematoxylin and eosin (H&E) and examined under a light microscope.²¹ The severity of inflammation was scored by two independent pathologists on a scale of 0 - 3:

Where;

0 = Normal histology.

1 = Infiltration in <25% of the section.

2 = Infiltration in 25 - 50% of the section.

3 = Infiltration in >50% of the section.

Statistical analysis

Data were expressed as mean ± standard error of the mean (SEM) and analyzed using one-way analysis of variance (ANOVA) followed by Dunnett's post hoc test. Statistical significance was set at $p \leq 0.05$. SPSS version 20 was used for all analyses.

Results and Discussion

Yield of ethanol soluble extract of *Jatropha curcas*

The extraction of 750 g of powdered *Jatropha curcas* leaves yielded 75.91 g of ethanol-soluble extract, corresponding to a percentage yield of 10.12% (w/w) based on air-dried plant material.

Physicochemical properties of the extract

The ethanol leaf extract of *J. curcas* was dark green in colour, had a bitter taste, and was odorless. The pH was recorded at 7.16 at 30.8°C, and the specific gravity was determined to be 1.20.

Phytochemical constituents of *Jatropha curcas* leaf ethanol extract

Phytochemical screening of the ethanol extract of *Jatropha curcas* leaves revealed the presence of flavonoids, alkaloids, terpenes, saponins, glycosides, steroids, and anthraquinones, while tannins were absent (Table 1). Previous studies reported similar findings.²² These bioactive compounds are known for their pharmacological effects, including immunomodulatory properties, anti-inflammatory, and antioxidant activities.^{22,23}

Table 1: Phytoconstituents of ethanol extract of *Jatropha curcas* leaves

Phytoconstituent	Test	Result
Carbohydrates	Molisch's test	+
Steroids and terpenoids	Liebermann-Burchard's test	+
Anthracene (free)	Bontrager's test	+
Cardiac glycoside	Keller-killiani test	+
	Frothing test	+
Saponins	Hemolysis test	-
Tannins	Lead acetate test	-
	Shinoda's test	-
Flavonoids	Sodium hydroxide test	+
	Mayer's test	+
Alkaloids		+

Inference: + = Present; - = Absent

Oral median lethal dose (LD₅₀) of *Jatropha curcas* leaf ethanol extract

No sign of toxicity and no death was recorded after 24 hours following the oral administration of *Jatropha curcas* leaf ethanol extract even at the highest dose of 5000 mg/kg. This indicate that the LD₅₀ of the extract is greater than 5000 mg/kg. The high oral LD₅₀ (>5000 mg/kg) suggests that the extract has a favorable safety profile, supporting its potential use in therapeutic applications.

Effect of *Jatropha curcas* leaf ethanol extract on ovalbumin-induced airway leukocytosis

The present study investigated the ameliorative effects of ethanol leaf extract of *J. curcas* on ovalbumin-induced allergic airway inflammation in Wistar rats.

Table 3 presents the effect of *J. curcas* ethanol leaf extract on ovalbumin-induced airway leukocytosis in bronchoalveolar lavage fluid (BALf) of Wistar rats. The findings demonstrated that *J. curcas* extract possesses significant anti-inflammatory properties, as evidenced by its ability to inhibit airway leukocytosis, reduce pro-inflammatory cytokine levels, and protect against airway tissue damage. Neutrophil, eosinophil, monocyte, and lymphocyte counts increased following ovalbumin sensitization. However, ovalbumin-induced eosinophilia was significantly ($p \leq 0.05$) reduced after treatment with the highest dose of *J. curcas* (140 mg/kg) and prednisolone (10 mg/kg), while the neutrophil, monocyte, and lymphocyte counts were also reduced following treatment with *J. curcas* leaf extract. Although, these reductions were dose-dependent but not statistically significant (Table 3).

Table 2: Oral median lethal dose (LD₅₀) of ethanol extract of *Jatropha curcas* leaves

Parameter	Result
Sign(s) of Toxicity	Nil
LD ₅₀ Value (mg/kg)	> 5000

Table 3: Effect of *Jatropha curcas* leaf ethanol extract on ovalbumin-induced airway leukocytosis in bronchoalveolar lavage fluid of Wistar rats

Treatment Group	Leukocyte Count (Cells/µL)						
	Neutrophils		Lymphocytes		Eosinophils		Monocytes
Normal control/Treatment Naïve	23.80 ± 1.53		57.20 ± 3.67		1.20 ± 0.37		1.00 ± 0.32
Ovalbumin sensitized	31.40 ± 2.25		66.80 ± 2.08		2.40 ± 0.24		1.60 ± 0.40
Sensitized + <i>J. curcas</i> 35 mg/kg	29.60 ± 3.00		62.20 ± 5.84		1.40 ± 0.51		1.20 ± 0.58
Sensitized + <i>J. curcas</i> 70 mg/kg	32.60 ± 2.80		62.80 ± 2.31		1.20 ± 0.37		1.40 ± 0.40
Sensitized + <i>J. curcas</i> 140 mg/kg	32.40 ± 3.49		59.40 ± 2.06		1.00 ± 0.45 ^a		1.20 ± 0.37
Sensitized + Prednisolone (10 mg/kg)	32.40 ± 3.40		57.40 ± 2.46		0.80 ± 0.58 ^a		1.00 ± 0.32

Values are mean ± SEM (n = 5). "a" indicates statistically significant difference compared to the control (Distilled Water) group at $p \leq 0.05$.

Allergic airway inflammation, a key feature of asthma, is characterized by complex cellular and molecular mechanisms resulting in airway tissue infiltration by both eosinophil and neutrophil, mainly dependent on Th2 and Th17 cell differentiation and activation, respectively, leading to airway remodeling and hyperresponsiveness.^{2,24} Dendritic cells, which direct the commitment of different Th cell lineages (Th1, Th2, and Th17), are responsible for activation and recruitment of eosinophils and neutrophils into the airways.²⁵ Generally, these phenomena occur in asthma as a result of the defective function of specific regulatory T-lymphocytes (Treg cells), because Treg cells directly or indirectly inhibit proinflammatory dendritic cells and promote the generation of tolerogenic dendritic cells, consequently preventing the activation of effector Th1, Th2, and Th17 lymphocytes.^{26,27} This functional defect of Treg cells-mediated

immunomodulation makes asthmatic patients susceptible to the development of eosinophilic and neutrophilic bronchial inflammation. The potential of ethanol leaf extract of *J. curcas* to significantly inhibit airway eosinophilia at a dose of 140 mg/kg which was comparable to prednisolone (10 mg/kg) suggests that the extract may exert its anti-inflammatory effect by modulating immune cell recruitment and activation.

Effect of *Jatropha curcas* leaf ethanol extract on pro-inflammatory cytokines in BALF

Table 4 presents the effect of *J. curcas* leaf ethanol extract on the levels of key pro-inflammatory cytokines (IL-4, IL-5, and IL-13) in BALF of Wistar rats. The results revealed the antigenic activity of ovalbumin by its ability to increase cytokine levels (IL-4, IL-5, and IL-13) in the ovalbumin-sensitized control group, whereas *J. curcas* treatment, particularly at 140 mg/kg, significantly ($p \leq 0.05$) reduced IL-13 levels (Table 4) in comparison with the normal control.

Table 4: Effect of *Jatropha curcas* leaf ethanol extract on ovalbumin-induced pro-inflammatory cytokine release in Wistar rats

Treatment Group		Cytokine level (pg/mL)			
		IL-4	IL-5	IL-13	
Normal	control/Treatment	26.13 ± 0.68	375.90 ± 20.68	9.81 ± 0.33	
Naïve					
Ovalbumin sensitized		31.61 ± 2.84	488.57 ± 33.87	11.54 ± 0.56	
Sensitized +. <i>curcas</i>	35 mg/kg	24.05 ± 0.84	351.85 ± 17.56	8.99 ± 0.52	
Sensitized +. <i>curcas</i>	70 mg/kg	26.84 ± 0.50	388.67 ± 41.23	9.42 ± 0.20	
Sensitized +. <i>curcas</i>	140 mg/kg	24.87 ± 1.39	253.70 ± 13.60 ^a	7.12 ± 0.50 ^a	
Sensitized + Prednisolone	(10 mg/kg)	27.04 ± 1.55	338.11 ± 23.47	9.04 ± 0.27	

Values are mean ± SEM (n = 5). "a" indicates statistically significant difference compared to the control (Distilled Water) group at $p \leq 0.05$.

Cytokines such as IL-4, IL-5, and IL-13 play critical roles in the pathogenesis of allergic airway inflammation by promoting Th2 cell differentiation, eosinophil survival, and mucus hypersecretion.²⁸ The lymphocytes that participate in asthma pathophysiology are biased toward the T-helper type 2 (Th2) phenotype, leading to increases in production of interleukin 4 (IL-4), IL-5, and IL-13.²⁹ The IL-4 from Th2 cells and basophils provides help for IgE synthesis in B-cells, IL-4 also plays a role in the differentiation of Th2, in allergic responses, and in the switching of antibody types.^{30,31} IL-5 is known to affect eosinophil differentiation, maturation, and prolongation of survival, and promote eosinophilic inflammation in the airways.³² IL-13 is very sensitive to monocytes and B-cells. IL-13 does not act on T-cells but inhibits the proliferation of leukemic pro-B-cells.³³ The ability of *J. curcas* extract to significantly reduce IL-13 levels at 140 mg/kg suggests a potential mechanism for its therapeutic action. Although, the reductions in IL-4 and IL-5 were not statistically significant, the observed trend indicates a possible dose-dependent effect that warrants further scientific investigation.

Effect of ethanol leaf extract of *J. curcas* on ovalbumin-induced lung and tracheal tissues inflammatory response in rats

Histopathological analysis of lung tissues revealed that *J. curcas* extract did not elicit any visible protection against ovalbumin-induced congestion and necrosis of lung tissues at 35 mg/kg. However, only a slight protection was observed at a 140 mg/kg dose. In addition, the extract protected the lungs against ovalbumin-induced alveoli hyperplasia and necrosis at a dose of 70 mg/kg, meaning the extract could exhibit hormesis (Figure 1). The extract also protected against ovalbumin-induced tracheal inflammation and necrosis at the 140

mg/kg dose (Figure 2).

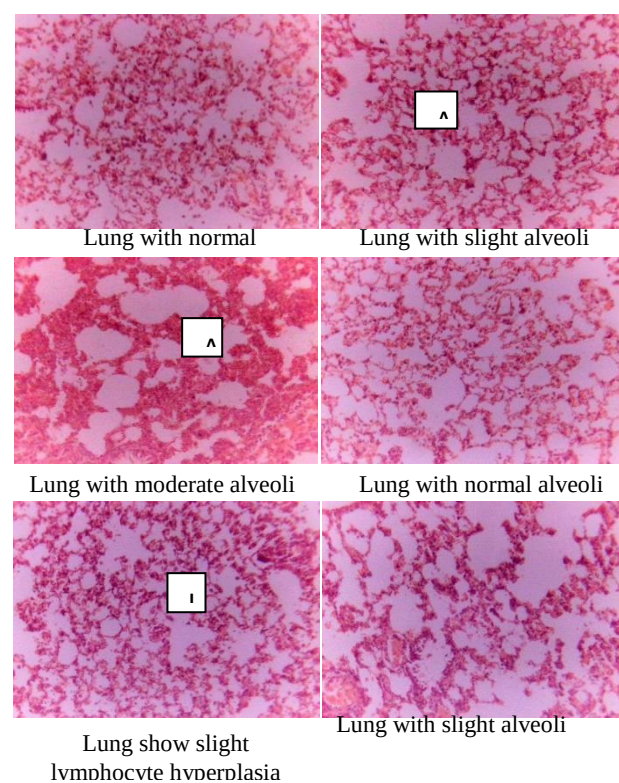


Figure 1: Effect of ethanol extract of *Jatropha curcas* leaves on ovalbumin-induced lung tissue inflammatory response in rats (Mag. x1000, H & E stain)

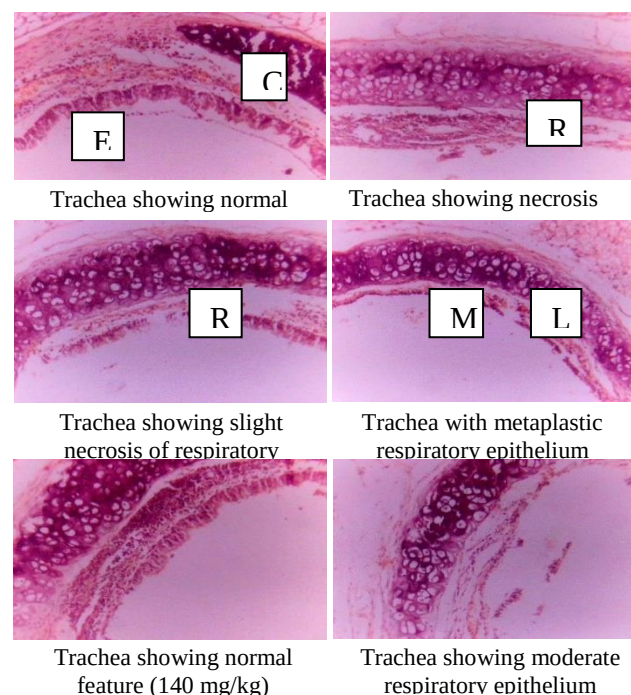


Figure 2: Effect of ethanol extract of *Jatropha curcas* Leaves on ovalbumin-induced tracheal tissue inflammatory response in rats (Mag. x1000, H & E stain)

The hormetic response to the ethanol leaf extract of *J. curcas* that was observed on the lung alveolar tissue could be due to toxicity, receptor desensitization, or other pharmacological factors. The promise of *J.*

curcas extract to protect against ovalbumin-induced alveolar necrosis and tracheal hyperplasia at doses of 70 and 140 mg/kg, respectively is crucial, as airway remodeling contributes to disease progression and treatment resistance in chronic allergic conditions.³⁴ The current theory that describes the origins of airway remodeling was based on the hypothesis that chronic allergic inflammation is the initiating event of remodeling and the main reason behind most of its features. This hypothesis was confirmed by studies using transgenic mice that overexpressed interleukin (IL)-4, IL-5, and IL-9 in the lung, and wherein marked mucus metaplasia, thickening of the reticular basement membrane, and induction of airway hyperresponsiveness (AHR) were observed.^{35,36} IL-4 and IL-13 also caused fibroblast differentiation into myofibroblasts and enhancement of type III collagen release, whilst blockade of IL-13 reversed AHR,³⁷ and IL-13 administration enhanced AHR, eosinophilic inflammation, and mucus cell hyperplasia.³⁸ The observed protective effects of *J. curcas* extract may be attributed to the extract's ability to modulate inflammatory responses and prevent structural lung damage. Prednisolone, a standard corticosteroid therapy, exhibited similar anti-inflammatory and airway-protective effects as *J. curcas* extract. However, corticosteroid use is associated with significant adverse effects, including immunosuppression and metabolic disturbances.^{39,40} The findings of this study suggest that *J. curcas* could serve as a complementary or alternative therapy with a potentially better safety profile.

Study limitations

Some limitations of this study include the use of Wistar rat model, which may not completely replicate the human allergic airway inflammation. The effect of chronic administration and/or different routes of administration was not evaluated. Lack of *in vitro* studies to complement the *in vivo* to evaluate the effect of *J. curcas* on cells or other macromolecules implicated in allergic airway inflammation limits the translation of the results.

Conclusion

The ethanol leaf extract of *Jatropha curcas* demonstrated significant anti-inflammatory and airway-protective properties in a rodent model of ovalbumin-induced allergic airway inflammation. These effects were likely mediated via inhibition of leukocyte infiltration, reduction of pro-inflammatory cytokines, and prevention of airway tissue remodeling. These findings suggest the potential use of *Jatropha curcas* as a complementary therapeutic agent for allergic airway diseases, pending further clinical validation. Based on the findings of this study, additional research is recommended to elucidate the precise molecular pathways through which *Jatropha curcas* exerts its anti-inflammatory effects. Also, chronic toxicity studies should be conducted to ensure the long-term safety of *Jatropha curcas* extract for therapeutic use.

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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