International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

The Dose-Dependent Anti-Asthmatic Effects of *Zingiber Officinale*: Evidence from Lung Histopathology and Immunoglobulin Profiles in Wistar Rats

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Date of Submission: 08-01-2026 Date of Acceptance: 20-01-2026

ABSTRACT

Background and Aim: Asthma, one of the most common chronic and noncommunicable diseases, is a heterogeneous airway illness that affects millions of people globally. This study investigated the anti-asthmatic and immunomodulatory properties of *Z. Officinale* (ginger) in a Wistar rat asthma model, focusing on pulmonary histopathology and systemic immunoglobulin responses.

Materials and Methods: Asthma was induced with ground peanut protein (20 mg/mL), and animals were randomly assigned to six groups: normal, negative, positive (dexamethasone), and three *Z. Officinale* treatment groups (250mg/kg, 500mg/kg and 1000mg/kg). Animals were acclimatised for two weeks before induction, and treatments were administered over two weeks including both pre- and post-induction phases. Immunological evaluation involved quantitative assessment of serum IgG, IgE, IgA, IgM, and IgD levels before and after treatment, alongside histopathological examination of lung tissues to assess airway inflammation, mucus hypersecretion, and bronchial architecture. Data were expressed as mean ± SEM, standard deviation, and variance, and statistical comparisons were performed using ANOVA and Dunnett's post hoc test, with significance set at $p < 0.05$.

Results: The induction of asthma into the experimental animals resulted in inflammation, thickening of the alveolar wall and clogged blood vessels. Dexamethasone-treated rats demonstrated restoration of the normal pulmonary structure, confirming its efficacy as an anti-asthma. Animals administered *Z. Officinale* had dose-dependent effects with significant improvements at 1000mg/kg. Immunological analysis revealed a reduction in serum IgE following the administration of the standard drug and ginger extract. These findings demonstrate that ginger extract exhibits significant anti-inflammatory and immunomodulatory effects, as evidenced by altered immunoglobulin profiles and improved airway histology.

Conclusion: The results suggest that *Zingiber Officinale* has promising therapeutic potential for asthma control. However, the observed dose-dependent variability emphasises the necessity of optimal dosing, and the data indicate the potential utility of combination therapy with conventional corticosteroids such as dexamethasone for improved asthma control.

Keywords: asthma, respiratory diseases, *Zingiber officinale*, dexamethasone, airway obstruction, anti-inflammatory, immunoglobulin.

I. INTRODUCTION

Diseases are conditions that impair the normal functioning of the human body and can be either communicable or noncommunicable. Asthma is a noncommunicable disease that has long been a common disease that affects the bronchi in living organisms by constricting the bronchial mucosa, causing difficulties in breathing. The word "Asthma" originated from the Greek language, which also means "breathing difficulty" [24, 32]. Asthma is a condition characterised by inflammation of the airways, causing intermittent airflow obstruction, inflammation, epithelial cells sloughing, lung remodelling and bronchial hyperresponsiveness. These characteristics lead to recurrent episodes of dyspnea, chest tightness, coughing and wheezing [4, 13, 14, 29]. The World Health Organisation [36] defines asthma as a chronic lung disease that affects people of all ages and also, the most common chronic disease among children. Asthma can be referred to as

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

a complex disease that requires the interaction of several cells, such as mast cells, eosinophils, T-lymphocytes, dendritic cells, B-lymphocytes, macrophages, cytokines [32]. Asthma is caused by a complex combination of genetic, environmental, and immunological factors that results in immune dysregulation and structural changes in the airways, all of which contribute to asthma's clinical symptoms [4, 7].

1.1. Epidemiology of Asthma

Asthma prevalence has risen considerably in low- and middle-income nations, owing primarily to underdiagnosis and undertreatment (World Health Organisation, 2024). It affects around 339million people worldwide and is anticipated to increase by 2025, with the low-and middle-income countries accounting for the majority of fatality cases [9]. According to Shinde et al. [32], Bronchial Asthma affects roughly 310million individuals globally, and the number is increasing as air quality and environmental factors deteriorate. Dharmage [10] report that children have a higher number of asthma prevalence and incidence, yet the morbidity and mortality associated with asthma attacks are higher in adults. Asthma has been shown to affect more boys in childhood, while it is more common in women in adulthood. Various studies have found a link between changes in sex hormones and the occurrence and severity of asthma [12, 37]. A study conducted by Ozoh et al. [27] indicated that Nigeria has a high prevalence of asthma, with approximately 13million people suffering from this disease, making it one of the highest in Africa. A recent study that examined the prevalence of asthma across 17 nations revealed diverse results ranging from 3.4%-6% for adults and children in India, Taiwan, Kosovo, Nigeria, Russia and an estimated higher rate of 17%-33% for Honduras, Costa Rica, Brazil and New Zealand [24]. Despite the worrisome numbers, the issue with diagnosis remains persistent. The World Health Survey showed that only 4.3% of asthma illnesses were detected globally, with significant differences of up to 21-fold between nations [34].

1.2. Pathophysiology of Asthma

The mast cells, which play an important role in the pathophysiology of asthma, are found in abundance in the lungs of an asthmatic patient. Chronic airway inflammation is the primary pathological characteristic of asthma. Most individuals with asthma, particularly those with allergic illnesses, have airway inflammation caused by Type-2 (T2-high) immunological mechanisms [11, 17]. Airway epithelium injury induced by allergens, pollutants, or respiratory virus infections triggers the release of epithelial-derived cytokines such as thymic stromal lymphopoietin, interleukin (IL)-25, and IL-33 [17]. These cytokines activate dendritic cells, causing T-helper 2 cells to differentiate and secrete IL-4, IL-5, and IL-13 [6, 17]. IL-4 and IL-13 stimulate B lymphocytes to produce IgE, whereas IL-5 is required for eosinophil maturation, recruitment, and survival in the airways [6, 11].

Activated eosinophils and mast cells produce inflammatory mediators such as histamine, cysteinyl leukotrienes, and prostaglandins, which cause bronchial smooth muscle contraction, increased vascular permeability, and mucosal oedema, resulting in acute airflow limitation and clinical symptoms such as wheezing and dyspnea [11, 28]. Chronic inflammation also causes goblet cell hyperplasia and hypertrophy of submucosal glands, which leads to increased mucus production and airway clogging [17, 28].

Non–Type 2 (T2-low) asthma is characterised by neutrophilic or paucigranulocytic airway inflammation involving cytokines such as IL-17 and interferon-γ. It is often associated with reduced responsiveness to corticosteroid therapy [11, 35]. Persistent airway inflammation also causes airway remodelling, such as subepithelial fibrosis, thickening of the reticular basement membrane, increased smooth muscle mass, angiogenesis, and epithelial dysfunction [17, 28]. These structural changes increase disease severity and, in some cases, cause irreversible damage [28].

1.3. Classification of Asthma

a. Based on aetiology: Asthma can be classified into allergic atopic (also called extrinsic or atopic asthma), non-allergic (also called intrinsic or idiosyncratic asthma), asthma with persistent airflow limitation, asthma with obesity, adult-onset asthma and exercise-induced asthma [4, 23].

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

Table 1: Types of Asthma

Features	Extrinsic or allergic asthma	Intrinsic or non-allergic asthma
age at onset	common in childhood	common in adulthood
personal/family history	commonly present	absent
preceding allergic illness	present (example includes rhinitis, urticaria and eczema)	absence of a preceding allergic illness
allergens	presence of allergens such as pollens and dust	absence of allergens
drug hypersensitivity	none	present (mostly with aspirin)
serum IgE level	elevated	normal
associated chronic bronchitis, nasal polyps	absent	present
emphysema	unusual	common

b. Based on severity: Following the degree of occurrence of asthmatic attacks, it can be classified into; mild intermittent, mild persistent, moderate persistent and severe persistent [4, 22, 23].

Table 2: Types of asthma based on severity

Severity*		**Mild Intermittent**	**Mild Persistent**	**Moderate Persistent**	**Severe Persistent**
Features of asthma symptoms	effects on breathing	difficulty breathing less than twice a week	difficulty breathing 3 -6 times a week	difficulty breathing daily	difficulty breathing that are continual
	frequency of occurrence	occurs less than once in a week	occurs more than once or more in a week but not everyday	occurs everyday	occurs everyday
	intensity of attacks	mild and brief	disturbs daily life or sleep at least once in a month	disturbs daily life or sleep at least once in a week. It worsens frequently	restricts daily life. It worsens frequently
	duration and effects of flare-ups	flare-ups are usually brief and no symptoms in between	flare-ups can last for minutes to hours and also, may affect activity level	flare-ups can last for hours to days and also, may affect activity level	flare-ups could last from minutes to days and also, it severely affects activity level.
	symptoms at night	occurs less that twice a month	occurs about twice or more in a month	it occurs once or more a week	it occurs frequently
PEF, FEVI**	%FEVI, %PEF. diurnal variation of PEF	≥80% 20%	≥80% 20-30%	≥60%, <80% >30%	<60% >30%

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1.4. Causes of Asthma

Despite the occurrence of asthma not having a definite cause, some risk factors could contribute to the development of asthma. These include: family history, bronchiolitis in childhood, occupational exposure, exposure to tobacco smoke, particularly during pregnancy, low birth weight, premature birth, exposure to air pollutants, and obesity and overweight children.

1.5. Triggers of Asthma

These include anything that could either induce the symptoms of asthma or exacerbate the symptoms. Some of the common triggers of asthma include: pollen, cold air, exercise, dust mites, mould, pet dander, occupational exposure, respiratory infections, smoking (either actively or passively), physical or emotional stress, and strong chemicals such as perfumes and toxins in the air.

1.6. Management and Treatment of Asthma

The severity and frequency of asthma symptoms determines how the condition is managed. The goal of asthma treatment is to control the symptoms of asthma and reduce the risk of further exacerbations [26]. Although there is currently no cure for asthma, effective therapeutic options exist to manage symptoms and prevent severe episodes. The most commonly used treatments are inhalers, which are of two major categories.;

- Bronchodilators, such as β_2-adrenergic agonists (e.g., salbutamol, formoterol, albuterol, and vilanterol), help to relax airway muscles and improve airflow.
- Inhaled corticosteroids, including beclomethasone, mometasone, and triamcinolone, reduce airway inflammation, improve symptom control, and lower the risk of asthma exacerbations and asthma-related mortality.

In addition to inhalers, other medications may be used depending on disease severity and patient response. These include;

- Leukotriene modifiers (e.g., montelukast and zileuton)
- Phosphodiesterase inhibitors such as theophylline, muscarinic antagonists (e.g., ipratropium, aclidinium and umeclidinium) and
- Biologic therapies such as anti-IgE antibodies (e.g., omalizumab).
- Antihistamines may be beneficial in patients with asthma associated with allergic conditions.

Together, these therapies allow for individualised and effective asthma management.

1.7. Ginger (Zingiber officinale)

Ginger (Zingiber officinale) is part of the Zingiberaceae family. They are widely used as spices throughout the world, particularly in most Asian nations [8, 19]. Ginger contains a high concentration of active substances such as phenolic and terpene compounds. Gingerols, shogaols, and paradols account for the majority of the phenolic chemicals found in ginger. The primary polyphenols in fresh ginger are known as gingerols, which include 6-gingerol, 8-gingerol, and 10-gingerol. Gingerols can be transformed into matching shogaols through heat treatment or long-term storage. Shoaols can be converted into paradols through hydrogenation. The unique odour and flavour of ginger are created by a mixture of zingerone, shogaols and gingerols, volatile oils that make up to three percent of the weight of fresh ginger. Ginger contains a wide range of other phenolic chemicals, including 6-dehydrogingerdione, zingerone, quercetin, and gingerenone-A. Ginger essential oils are mostly composed of terpenes such as β-bisabolene, α-curcumene, zingiberene, α-farnesene, and β-sesquiphellandrene. In addition, ginger includes lipids, organic acids, polysaccharides, and raw fibres. Chemical research of ginger revealed that it contains over 400 distinct chemicals. Ginger rhizomes are mostly composed of carbohydrates, lipids, terpenes, and phenolic compounds [1, 16, 18]. Also, there are amino acids, minerals, ash, protein, phytosterols, and vitamins (such as vitamin A and nicotinic acid). In laboratory animals, the gingerols improve gastrointestinal motility and have analgesic, sedative, antipyretic, anti-inflammatory, antioxidant and antibacterial effects. Ginger also makes swallowing easier due to its sialagogue action, stimulating saliva production [30, 33].

Over time, herbal medicines, often known as plant-based medications, have provided several therapeutic benefits in the treatment of a variety of acute and chronic diseases [3].

1.8. Anti-Inflammatory and Anti-oxidative Effects of Ginger

Ginger has been shown to inhibit the expression of several proinflammatory cytokines and inflammatory mediators produced by various cells, such as nitric oxide, IL-1, TNF-α and IL-8 [25]. It also

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

affects T-helper type 1 cells derived responses [18, 31]. According to research, ginger lowers lipid peroxidation [15, 22, 30]. Ginger's significant antioxidant capabilities can be ascribed to the existence of gingerol, which has a variety of bioactivities, including anti-inflammatory, antioxidant, growth-promoting, and antimicrobial actions. Heeba and Abd-Elghany [20] conducted an earlier study to establish the antioxidant properties of *Z. officinale*. Another review conducted by Ali [2] highlighted the pharmacological properties of ginger including immune-modulatory, anti-inflammatory, anti-hyperglycemic actions.

II. MATERIALS AND METHODS

2.1. Procurement and Acclamatisation of Experimental Animals

Fifty-two adult female Wistar rats, each weighing 180g, were obtained from the animal house of the Department of Pharmacology at the University of Port-Harcourt, which is well-known for its ethical breeding and care standards. They were housed in a controlled environment, maintaining specific conditions such as a temperature range of 22-24°C, humidity levels ranging between 40-60%, and a 12-hour light and dark cycle. The housing was made of a polypropylene cage and had a sawdust bedding that was changed regularly to keep it clean. A standard rat diet and water were provided every day. The animals were acclamatised for at least one week before the experiment commenced to allow them to adjust to their new environment.

2.2. Collection of Samples

The plant, *Z. officinale,* was sourced from the farm at the University of Port Harcourt, and then it was sent to the Department of Plant Science and Biotechnology at the university. At the laboratory, the leaves were identified, their morphological characteristics examined and assigned an herbarium number by a qualified and well-trained botanist. Afterwards, the authenticated leaves were thoroughly cleansed to remove dust and other contaminants, processed and then stored appropriately for further use. Dexamethasone was gotten from the Alpha Pharmacy and Stores, located at NTA road, Port Harcourt, Rivers State, Nigeria.

2.3. Preparation of Peanut Protein (the inducing agent)

For efficient extraction, a 70% ethanol was prepared from 99% ethanol by diluting 70.7mL of ethanol with 29.3mL of distilled water to a final volume of 100mL. Ground peanut powder (2g) was extracted with 100mL of 70% ethanol by continuously stirring. The mixture was filtered using a sterile handkerchief to remove particulate matter and filtrate concentrated using an autoclave. The resulting extract had a final concentration of 20mg/mL, which is the required concentration for asthma induction.

2.4. Extraction of the Plant (Ginger extract)

Fresh ginger (*Z. officinale)* rhizomes were washed, peeled and ground into a fine paste using a laboratory blender. 500g of the ground ginger was extracted with 1,250mL of distilled water by boiling for 30 minutes while stirring continuously. The mixture was allowed to cool to room temperature before filtering using a sterile handkerchief. The resulting extract was collected using clean and washed reagent bottles and placed in a refrigerator for preservation.

2.5. Animal Dosing

For two weeks, the experimental animals were given varying doses of ginger extract and the conventional medicine (dexamethasone). The extract and standard drug were both administered orally via gavage. Gavage dosage was done with a curved, ball-tipped metal needle attached to a 1mL syringe.

2.6. Tissue, Blood Collection and Sacrifice

After the final treatment on days 7 and 14 of the experimental period, the animals were fasted (with access to water) overnight. On days 8 and 15, the rats were anaesthetised under deep anaesthesia using an appropriate anaesthetic agent. Following the confirmation of adequate anaesthesia, blood samples were collected via a cardiac puncture into appropriate sample bottles. The animals were subsequently euthanised, and in each the abdominal cavity was carefully opened. Lung tissues were carefully excised, rinsed in cold normal saline to remove excess blood, and processed for further analysis.

2.7. Histopathological Examination

Cross-sections of lung tissues were processed using standard histological procedures. After deep anaesthesia, the animals were euthanised, and then, portions of the lung were fixed in 10% formal saline for adequate preservation. The fixed tissues were then dehydrated in progressively higher grades of alcohol, cleared in xylene and then immersed in paraffin wax. The paraffin-embedded tissues were sectioned at 4-5µm thickness using a rotary microtome. The sections were then mounted on glass slides and then, stained using Hematoxylin and Eosin (H&E). Stained sections were examined under a light

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

microscope and photomicrographs were captured using a digital camera attached to the microscope at a magnification of 100x (oil immersion).

2.8. Immunological Analysis

After sacrifice, about 5mL of blood sample was taken from each rat and placed into appropriate and properly labelled sample bottles. The sample was left to clot at room temperature for about 20-30 minutes and serum formed. Immunoglobulin assay was carried out using the Enzyme-Linked Immunosorbent Assay (ELISA).

2.9. Determination of Ginger Bulb Extract and its LD_{50}

In this technique, a clean, dry crucible (an evaporating dish) was weighed at room temperature and recorded as 52.97g. One (1mL) of the aqueous ginger bulb extract was transferred into the crucible, and recorded as 53.16g. The crucible was then placed over a beaker of hot water on a hot plate set at 40°C to allow for evaporation. The contents of the dish were heated and allowed to dry completely, after which the crucible was removed and allowed to cool to room temperature. The weight of the crucible and the dried extract residue were measured again and recorded as 53.02g. The weight of the extract residue was calculated by subtracting the weight of the empty crucible from the combined weight of the crucible and dried extract, as indicated below.

Table 3: The determination of the concentration of the extract

Weight determination	Ginger extract
Weight of dish +liquid extract	53.16g
Weight of dish + dry extract	53.20g
Weight of dish	52.97g
Weight difference	0.05g
Weight in mg	0.05 ×1000=50mg

Since the volume of the extract used was 1 ml, it implies that the concentration of the extract is 50mg/mL.

2.10. Oral Toxicity Testing (LD_{50} Determination) for "You and I Formula"

In this study, the Bruce method of 1985[5] was utilised to determine the LD_{50}, with all the animals used weighing 180g. With this method, a nulliparous and non-pregnant female Wister rat, fasted overnight (food but not water was withheld) before being orally given a single dose by gavage using a suitable stomach intubation cannula, beginning with a dose of 120.5mg/kg of aqueous Ginger bulb Extract (i.e. 21.69mg/180g or 0.43mL/180g animal). The female species was chosen to limit variability and minimise the number of animals used. Food was withheld for a further 3-4 hours following the administration of aqueous Ginger bulb extract. The animal was monitored for death for 48 hours. This dosage was chosen because there was no understanding of the extract's potential toxicity. At this dosage, no deaths were reported. Since no death was observed, the dose for the next animal was increased by a factor of one-half log times the initial dose. This was calculated to be 385.6mg/kg or 69.41mg/180g, corresponding to 1.39mL of extract per 180g animal. The animals were carefully examined for up to 48 hours before deciding whether and how much to inject the next animal, and no deaths occurred. The process of gradual escalation was continued with the following doses: 1233.92mg/kg or 222.106mg/180g, i.e. (4.4mL/180g of animal extract). Another animal was treated with 3948.48mg/kg or 710.73mg/180g or (14.2mL/180g) of the aqueous Ginger bulb extract and watched for 48 hours; no death was recorded. Since there was no observed death, a factor that when employed, would result in 5000mg/kg was required, as it is scientifically acknowledged that a substance is non-toxic at a level of 5000mg/kg. Thus, 5000mg/kg or 900mg/180kg (18mL/180g animal), was provided, and no animals died, even when the final dose was given to three Wistar rats. It therefore implies that the aqueous Ginger bulb extract is considered safe using this LD_{50} approach. When death occurs, the LD_{50} is commonly calculated using the geometric mean of the last two doses, the highest dose that did not cause death and the lowest dose that caused death, as follows;

The LD_{50} is determined from the formula

$LD_{50}=\sqrt{D1}\times\sqrt{D2}$; where D1 is the highest dose that did not cause death and, D2 is the lowest dose that caused death in the test animals.

2.11. Pilot Study

Two Wistar rats were purchased from the University of Port Harcourt (Uniport) animal house and used to perform the pilot study. Following the pilot study, fifty-two albino rats were also obtained from the Uniport animal house and used to conduct the experiment. The pilot investigation was conducted using two animals, one of which was experimentally

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

induced with asthma, while the other served as a normal control with normal physiological and biological parameters.

2.12. Study Design

The first animal was experimentally induced with asthma over a period of seven days before being sacrificed. Biological parameters associated with asthma, including lung histology and the levels of immunoglobulin E and immunoglobulin A, are then compared to the normal control (animal that was not induced with asthma), to see the difference between their levels of IgE and IgA and their various lung histology.

2.13. Research Design

In this study, fifty-two albino rats were used, sixteen of which were used in the LD_{50} calculation, while the remaining 36 animals were divided into six groups of six animals each after an acclimatisation period. The groups consisted of the normal control, negative control, positive control, low dose, medium dose and high dose. Using peanut-ethanol, asthma was induced for a period of seven days. Following this, the animals were administered either ginger extract at varying doses or a conventional drug (positive control) for a period of two weeks (14 days). After the first week of treatment (which was for 7days long), one animal from each group was sacrificed to assess biological parameters. At the end of the second week, the remaining animals were sacrificed, and lung histology as well as the serum level of immunoglobulin A and E were assessed and then recorded.

Table 4: Test animal groups and treatment protocols

Treatment group	Identity	No. of rats	Treatment protocol
group 1	low dosage group	6	250mg/kg of ginger extract after being induced with asthma using peanut- ethanol extract for seven days
group 2	medium dose group	6	500mg/ kg of ginger extract after being induced with asthma using peanut-ethanol extract for seven days
group 3	high dose group	6	1000mg/kg of ginger extract, after being induced with asthma using peanut-ethanol extract for seven days
group 4	positive control group	6	received the standard drug, Dexamethasone
group 5	negative control group	6	induced with asthma but not treated
group 6	normal control group	6	maintained normal biological parameters and was not induced with asthma

2.14. Statistical Analysis

The collected data was expressed as mean ± standard error of the mean (SEM). Descriptive analysis, including the mean, standard deviation, were used to analyse the data obtained from the experimental groups. Inferential statistics was conducted using the one-way analysis of variance (ANOVA). Dunnett's multiple comparison post hoc test was applied to determine statistically significant differences between control and test groups, with significance set at $p<0.05$. All statistical analysis was performed with the statistical package for the social sciences (IBM SPSS version 26.0 software).

3. RESULTS

The results presented below show histological parameters tested on the Wistar rats, such as lung histology results and the haematological parameters showing the IgE and IgA levels, as well as comparing them with other immunoglobulin levels as well as their effects during pre- and post-treatment with ginger extract and the standard drug, Dexamethasone.

3.1. Immunological Results

3.1.1. Immunoglobulin Levels in Post-Treated Wistar Rats

Table 5 shows that the negative control group exhibited a lower mean **IgG** level compared to the normal control. Similarly, administration of the standard drug, dexamethasone, and the extract at doses of 250, 500, and 1000 mg/kg resulted in a significant reduction in IgG levels when compared with the normal control group ($p < 0.05$). In contrast, the negative control group demonstrated a significantly higher mean **IgE** level relative to the normal control ($p < 0.05$). Treatment with the extract

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at low, medium, and high doses produced a reduction in IgE levels compared to the normal control, indicating a suppressive effect of ginger extract on IgE-mediated responses.

The mean **IgA** level in the negative control group was lower than that observed in the normal control. However, treatment with *Zingiber Officinale* at all administered doses (250, 500, and 1000 mg/kg) resulted in a significant increase in IgA levels compared to the normal control ($p < 0.05$). Similarly, the negative control group showed a lower mean **IgM** level relative to the normal control. Treatment with plant extract at low, medium, and high doses did not result in a significant increase in IgM levels when compared with the normal control group ($p > 0.05$). The negative control group also exhibited a lower mean **IgD** level compared to the normal control. Administration of *Zingiber Officinale* at all tested doses did not produce a significant change in IgD levels relative to the normal control ($p > 0.05$).

In summary, the negative control group demonstrated suppression of IgG, IgA, IgM, and IgD levels, alongside a significant elevation in IgE when compared with the normal control ($p < 0.05$), indicative of an altered humoral immune response. Treatment with *Zingiber Officinale* at 250, 500, and 1000 mg/kg produced a significant reduction in IgG and IgE levels relative to the normal control ($p < 0.05$), reflecting its immunosuppressive and anti-inflammatory activity. Conversely, *Zingiber Officinale* significantly increased IgA levels across all doses ($p < 0.05$), suggesting a modulatory effect on mucosal or protective immunity. No statistically significant alterations were observed in IgM or IgD levels following *Zingiber Officinale* treatment compared with the normal control ($p > 0.05$).

Table 5: Effect of ginger extract on immunoglobulins levels in post-treated Wistar rats

Group	IgG(g/l)	IgE(ul/ml)	IgA(g/l)	IgM(g/l)	IgD(mg/l)
Neg. count	5.17±0.04	12.00±0.58	0.80±0.12	.98±0.05	1.22±0.01
Normal	10.58±0.05	4.77±0.04	3.13±0.02	3.42±0.04	3.64± 0.02
Dexamethasone	4.20±0.47	2.11 ±0.03	3.09±1.65	2.17±0.12	3.00±0.06
250mg/kg	3.63±0.09	4.14 ±0.02	3.89±0.03	1.90±0.20	2.43±0.23
500mg/kg	4.97±0.09	3.20 ± 0.04	3.15±0.02	3.13±0.09	3.03±0.22
1000mg/kg	5.83±0.27	2.64 ±0.03	2.27±0.12	3.60±0.12	3.53±0.18

Values are expressed as mean± std error of the mean *significant at P≤0.005 when compared with normal group.

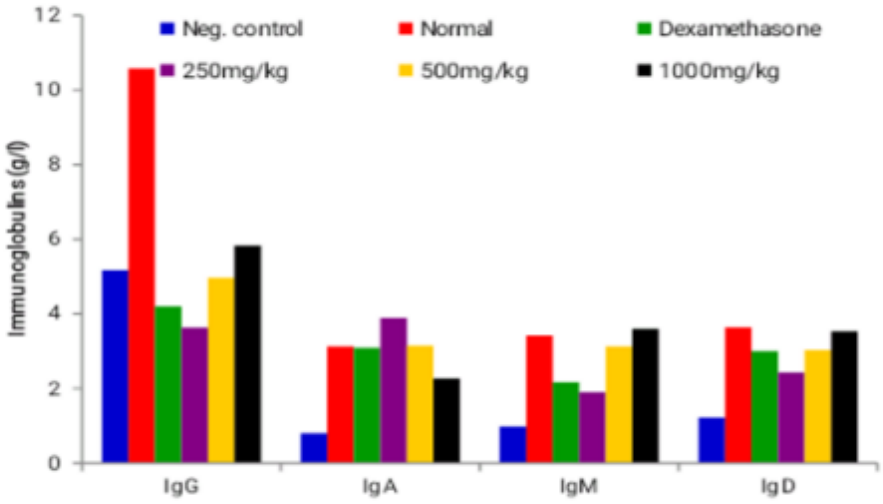

Figure 1: Effect of ginger leaf extrac on immunoglobulin levels in post-treated Wistar rats

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 1, Jan - Feb 2026, pp:154-173 www.ijprajournal.com ISSN: 2456-4494

3.1.2. Immunoglobulin-E Levels in Post-Treated Wistar Rats

As illustrated in the bar chart below **(Figure 2)**, IgE levels were significantly higher in the negative control group compared with the normal control ($p < 0.05$). In comparison to dexamethasone, administration of the extract at low (250 mg/kg), medium (500 mg/kg), and high (1000 mg/kg) doses resulted in a significant reduction in IgE levels relative to the negative control ($p < 0.05$), with values approaching those observed in the normal control group. No clear dose-dependent trend was evident across the plant extract-treated groups.

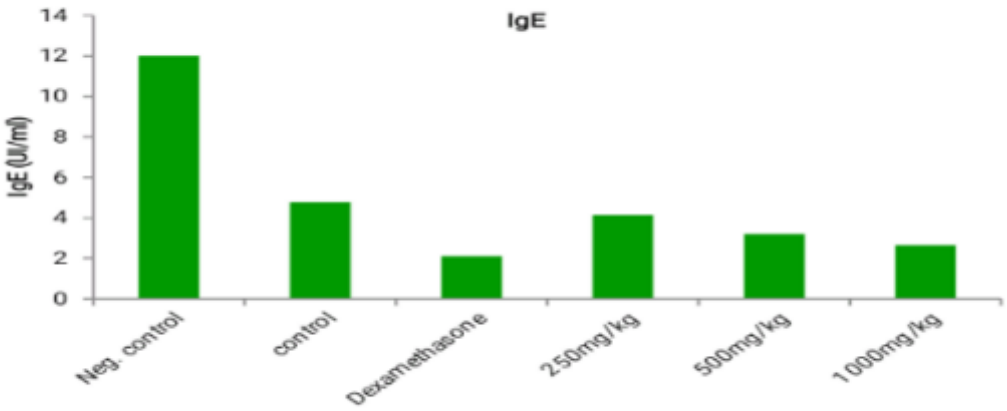

Figure 2: Effect of ginger leaf extract on immunoglobulin E level in post-treated Wistar rats

3.1.3. Immunoglobulin Levels in Pre-Treated Wistar Rats

As presented in **Table 6**, comparison between the normal and negative control groups revealed a reduction in **IgG** levels in the negative control, alongside a significant elevation in **IgE** ($p < 0.05$), indicating an altered humoral immune profile. Relative to the normal control, administration of ginger extract at 250, 500, and 1000 mg/kg produced a significant decrease in IgG levels ($p < 0.05$). Similarly, IgE levels were reduced following treatment across all doses when compared with the normal control, suggesting suppression of IgE-associated immune responses.

In contrast, **IgA** levels were significantly lower in the negative control compared with the normal control, whereas treatment using the extract at all doses resulted in a significant increase in IgA levels relative to the normal control ($p < 0.05$). Although **IgM** and **IgD** levels were reduced in the negative control group compared with the normal control, administration of ginger extract at low, medium, and high doses produced only non-significant increases in both immunoglobulins when compared with the normal control ($p > 0.05$).

Collectively, these findings demonstrate that *Zingiber Officinale* extract exerts dose-independent immunomodulatory effects on humoral immunity, characterised by suppression of IgG and IgE, selective enhancement of IgA, and minimal impact on IgM and IgD. This immunoglobulin-specific modulation provides mechanistic insight into the therapeutic efficacy of dexamethasone in inflammatory and immune-mediated conditions.

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Table 6: Effect of ginger extract on immunoglobulin levels in pre-treated animals

Group	IgG(g/l)	IgE(ul/ml)	IgA(g/l)	IgM(g/l)	IgD(mg/l)
Neg. count	5.17±0.04	12.00±0.58	.80 ± 0.12	.98±0.04	1.22±0.01
Normal	10.58±0.05	4.77±0.04	3.13±0.02	3.42±0.04	3.640±0.02
Dexamethasone	4.70±0.26	2.09±0.03	1.52±0.04	2.13±0.09	3.07±0.12
250mg/kg	4.27±0.37	3.42±0.03	3.25±0.02	2.53±0.22	2.83±0.15
500mg/kg	5.43±0.23	2.64±0.02	2.85±0.02	3.10±0.10	2.97±0.17
1000mg/kg	6.167±0.22	2.12±0.02	1.93±0.01	3.63±0.15	3.57±0.19

Values are expressed as mean± std error of the mean, *significant at p≤0.005 when compared with the normal group.

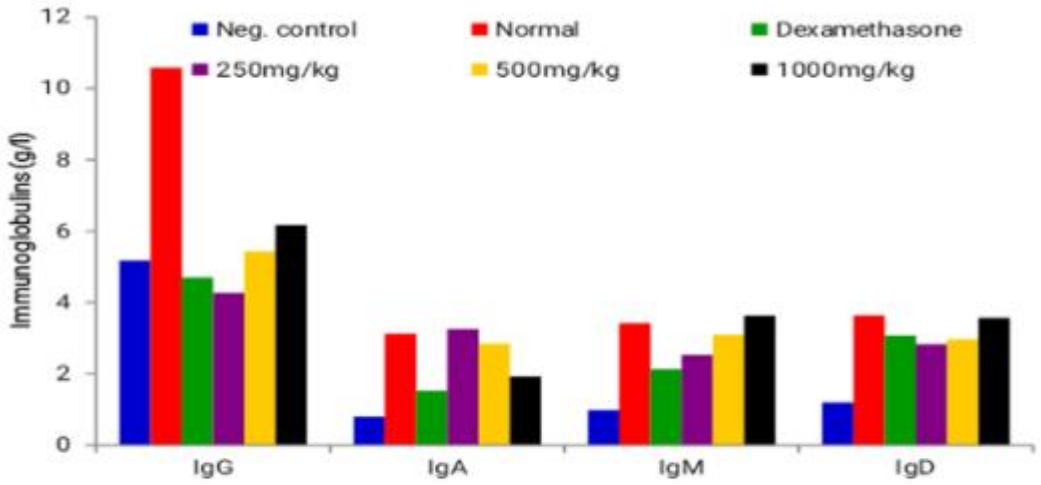

Figure 3: Effect of ginger leaf extrac on immunoglobulin levels in pre-treated Wistar rats

3.1.4. Immunoglobulin-E Levels in Post-Treated Wistar Rats

Figure 4 shows that IgE levels were significantly elevated in the negative control group compared with the normal control ($p < 0.05$). Treatment at 250mg/kg, 500mg/kg, and 1000 mg/kg significantly reduced IgE levels relative to the negative control ($p < 0.05$), bringing them closer to the levels observed in the normal control. While reductions were observed at all doses, the changes did not indicate a clear dose-dependent trend.

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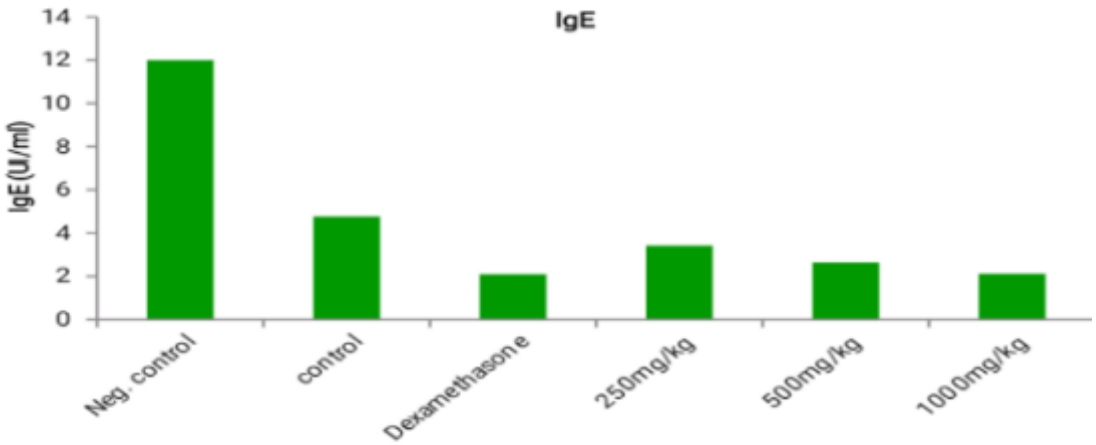

Figure 4: Effect of ginger leaf extract on immunoglobullin E level in pre-treated Wistar rats

3.2. Histopathological Result - Photomicrograph of the Lungs of the Test Animals and their Interpretations

3.2.1. Effect of the extract on the histology of the lungs

In the **first week** of the experimental study (Figure 5), the lung sections of the normal control group appeared rosy pink with regular borders, demonstrating the normal pulmonary architecture. The terminal bronchiole was lined with smooth pseudostratified columnar epithelial cells, structured smooth muscles without evidence of hypertrophy. The alveolar septa were thin and delicate, facilitating rapid gas exchange. The lamina propria was well vascularised, and the mucosa folds and adventitia were all intact. Collectively, these features serve as the baseline for comparing pathological findings in the treated group.

In contrast, the lung section from the negative control group (Figure 7) appeared to be red and swollen. The lung tissue appeared to be congested and oedematous with thick walls of the alveolar sac and inter-alveolar septa, diffuse hyperaemia, which are all hallmarks of asthmatic lung injury.

Asthma-induced animals treated with dexamethasone (Figure 6) exhibited appreciable improvement, with the lungs appearing close to normal. Other changes, including the formerly thick walls of the alveolar sac appeared to be thin, a decreased number of mononuclear cells usually present during inflammation. Reduced inflammation of the lung tissues is indicated, and also, validating the anti-inflammatory efficacy of dexamethasone.

The 250mg/kg of ginger group (Figure 9) portrayed engorged blood vessels within the alveolar sac, glassy and proteinaceous material consistent with hyalinisation and accumulation of mononuclear cells, all signifying an ongoing inflammatory activity. All these suggest limited protection against asthma-induced lung injury by the ginger extract given at this dose.

The lung section of the group dosed with 500mg/kg of extract (Figure 10) demonstrated moderate inflammation of the lung tissues. Although some level of capillary engorgement persisted, mononuclear cell activity reduced. Notably, the protein called hemosiderin suggests previous episodes of severe lung injury. Overall, there was a progressive improvement in the pulmonary architecture.

Lung sections dosed with 1000mg/kg (Figure 8) revealed considerable protection against asthma-induced lung injury. Inflammatory cell infiltration was minimal, and the bronchiolar epithelium and smooth muscle layers appeared largely intact. Alveolar septa were thin, with only mild evidence of vascular congestion. These findings indicate that the high-dose ginger extract had an obvious anti-inflammatory and tissue-protective effect, which is similar to the effects observed with dexamethasone treatment.

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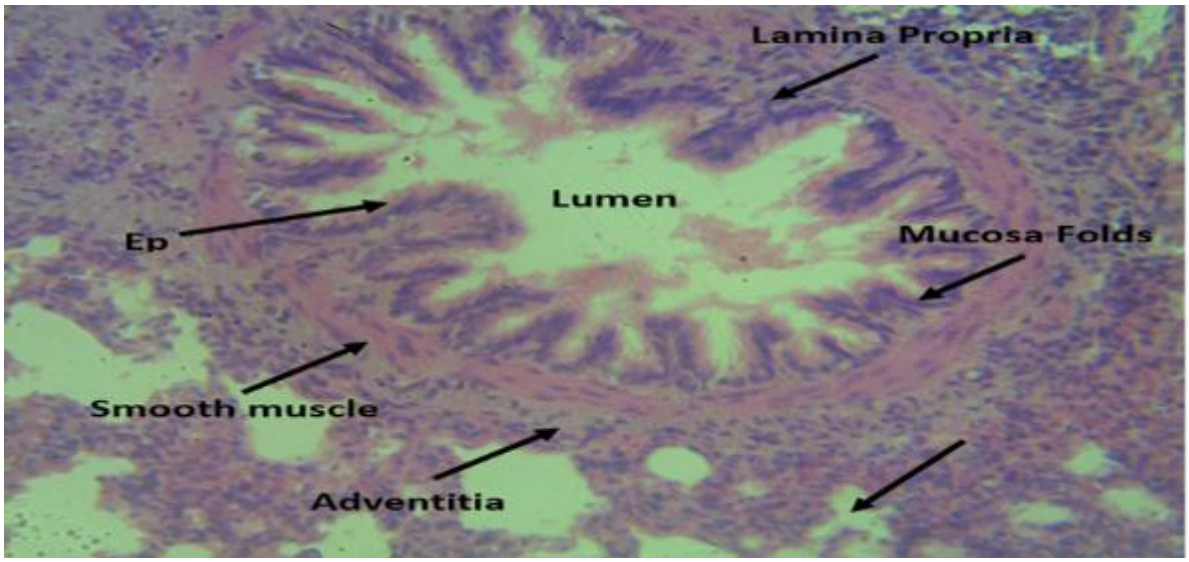

Figure 5: Photomicrograph (H&E X400) of the lungs of normal control animals showing normal architecture of the terminal bronchioles with smooth columnar epithelial lining (Ep), smooth muscle, adventitia, lamina propria and mucosa fold.
Diagnosis: Lung tissue with normal terminal bronchioles.

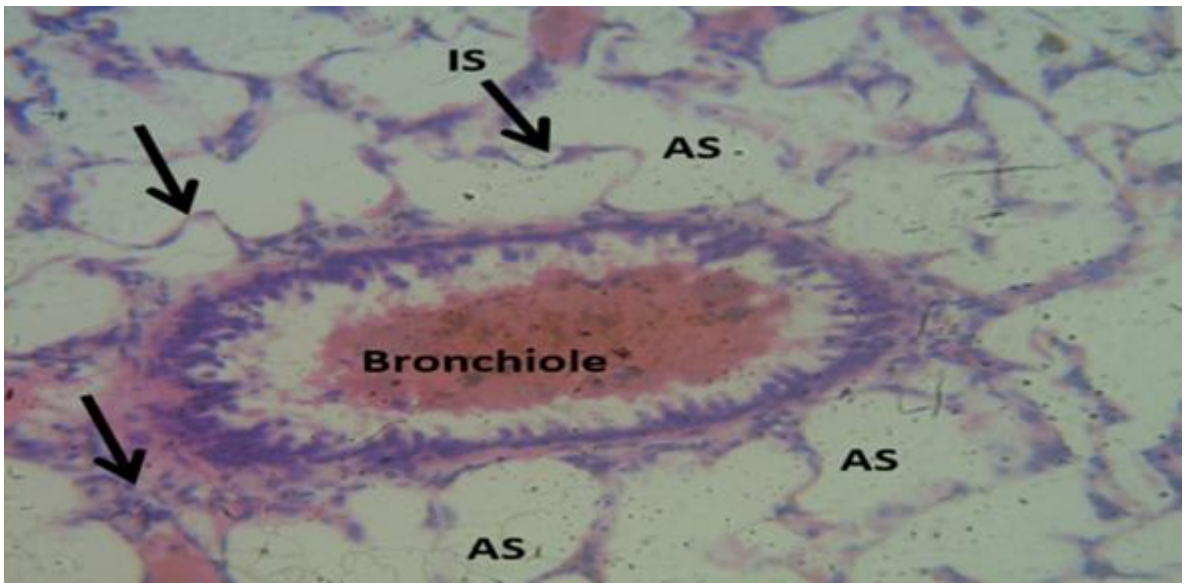

Figure 6: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with Dexamethasone for one week, showing thin inter-alveolar septa and alveolar sac wall, with regressive mononuclear cell activities (arrows).
Diagnosis: Reduced inflammation of the lung tissue.

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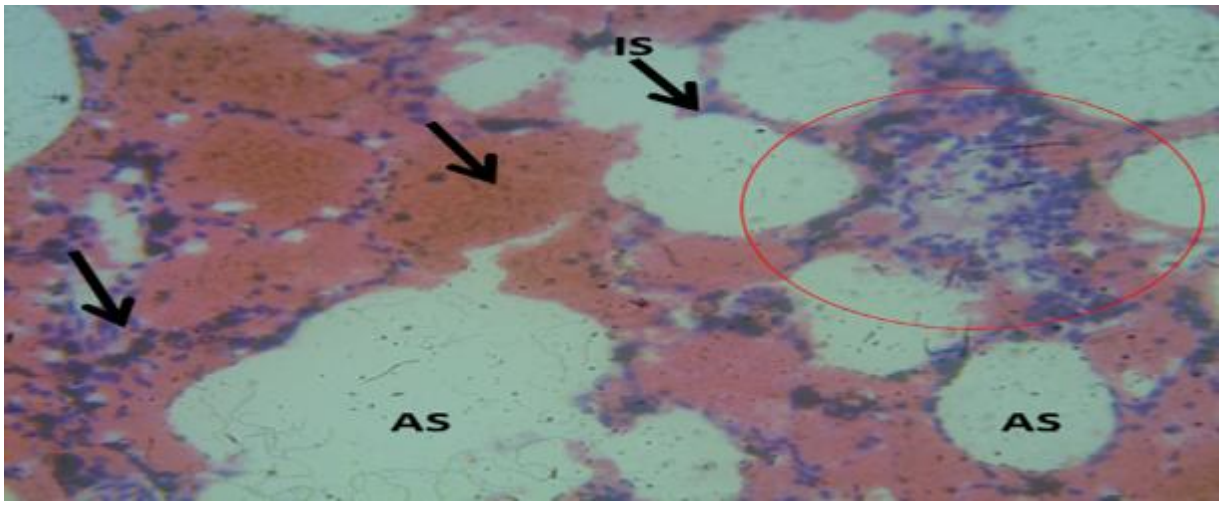

Figure 7: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and not treated with any drug, showing focal mononuclear activities associated with hyalinization and thickening of the alveolar sac wall and interalveolar septa, with hemosiderin deposits (arrows).
Diagnosis: Severe inflammation and hyperaemia of lung tissue.

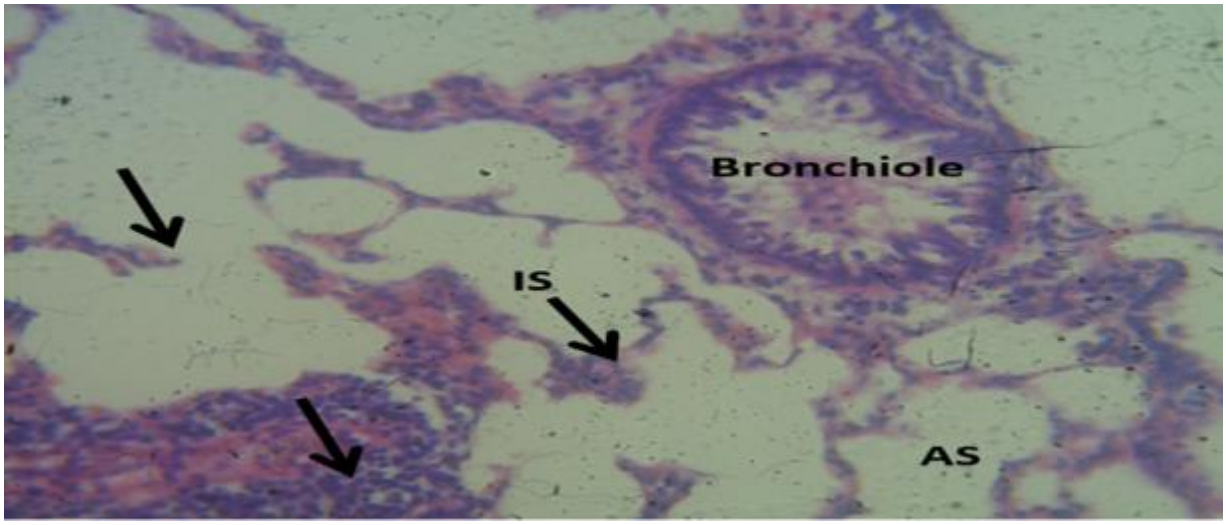

Figure 8: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with 1000mg/kg of ginger for one week showing distorted inter-alveolar septa (IS) and alveolar sac wall with regressive lymphocytic activities (arrows).
Diagnosis: Moderate inflammation and degeneration of the lung tissues.

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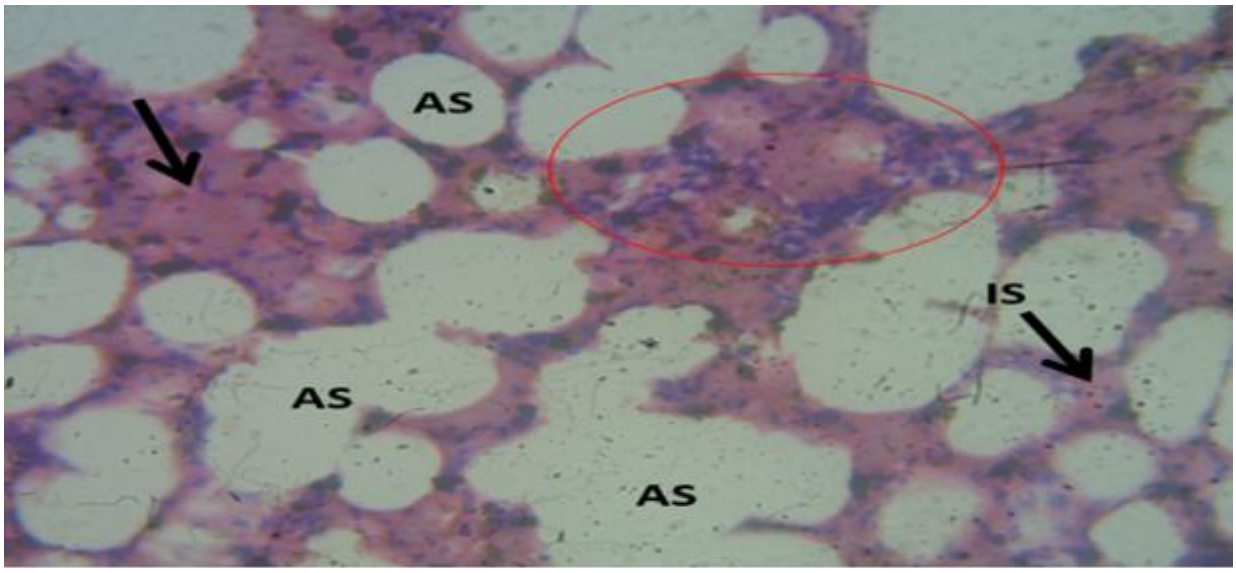

Figure 9: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with 250mg/kg of ginger for one week, showing inter-alveolar septa (IS); mononuclear cells activities (circled) associated with hyalinization of the alveolar sac (AS)wall causing congestion of the alveolar sac wall (arrows).
Diagnosis: Moderate inflammation of the lung tissue.

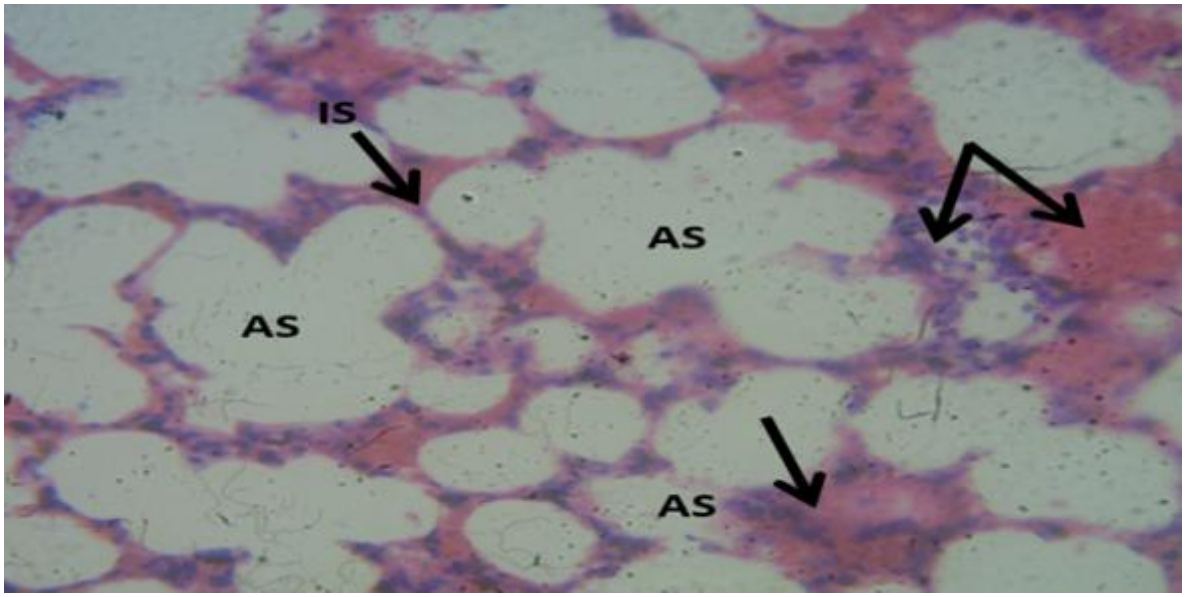

Figure 10: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol-extracted peanut for one week and treated with 500mg/kg of ginger for one week showing inter-alveolar septa (IS); reduced mononuclear activities and hemosiderin deposit clogging the alveolar sac (AS), causing congestion of the alveolar sac wall (arrows).
Diagnosis: Moderate inflammation of the lung tissue.

In the **second week**, the group treated with the standard drug, Dexamethasone (Figure 11), revealed thin alveolar sac walls, regressive activities of the mononuclear cells and moderate hyperaemia. All these features depict a decrease in inflammation, efficient gas exchange, an increased amount of blood flow to the lung tissues and most importantly, confirming the effectiveness of dexamethasone.

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Lung sections of rats dosed with 250mg/kg of extract (Figure 12) displayed inflammation and degeneration of the lung tissue. The presence of small vacuoles within the cells reveals degeneration and metabolic stress. Thick and damaged alveolar sac walls, congested blood vessels and regressed immune cells. These characteristics suggest that, at this dose, the extract is insufficient to reverse the existing asthmatic injury, hence, the continuous inflammation and deterioration of the lungs.

For Wistar rats dosed with 500mg/kg of ginger (Figure 13), moderate inflammation and partial damage of the lung tissues were observed, suggesting that although some improvement was apparent, the damage persisted as "moderate". This indicates an incomplete recovery or significant initial injury as a result of a short treatment period.

The group that received 1000mg/kg of the extract (Figure 14) exhibited a remarkable and near-complete restoration of normal pulmonary architecture. Inflammatory infiltrates were absent or minimal, alveolar septa were thin, and bronchioles maintained normal epithelial lining and lumen diameter. These findings were comparable to those observed in the standard drug–treated group, suggesting a strong dose- and time-dependent protective effect of ginger extract.

Overall, the histopathological results demonstrated a continuous, dose- and time-dependent amelioration of asthma-induced lung damage following ginger extract treatment. The high-dose ginger extract exhibited comparable protective effects to the standard anti-asthmatic drug, particularly after two weeks of treatment.

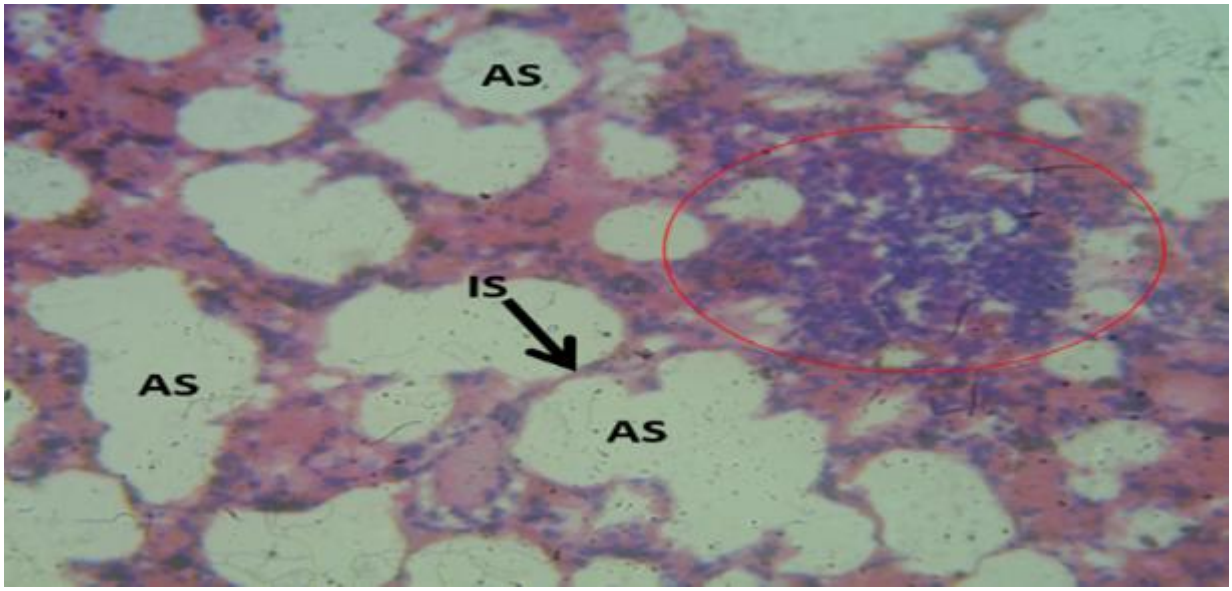

Figure 11: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol-extracted peanut for one week and treated with dexamethasone for two weeks, showing thin alveolar sac wall, with regressive mononuclear cell activities (arrows).
Diagnosis: Reduced Inflammation with moderate hyperaemia of the lung tissue

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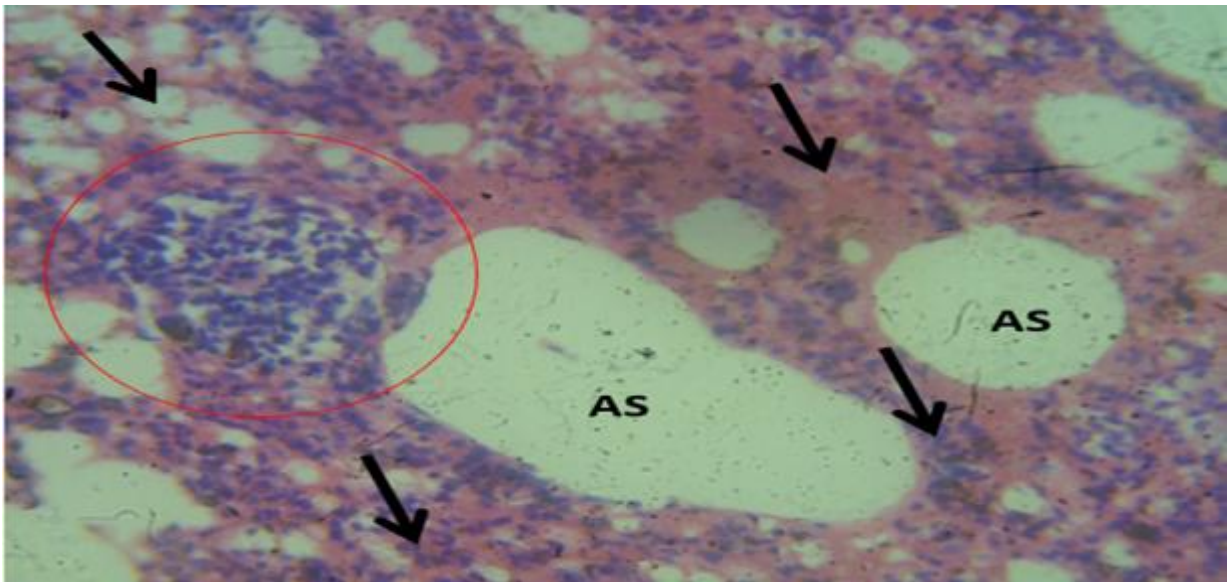

Figure 12: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with 250mg/kg of Ginger for two weeks, showing vacuolation and hyalinized alveolar sac wall (arrows), causing congestion with focal infiltration of regressive lymphocytic activities (circled).
Diagnosis: Inflammation and degeneration of the lung tissue.

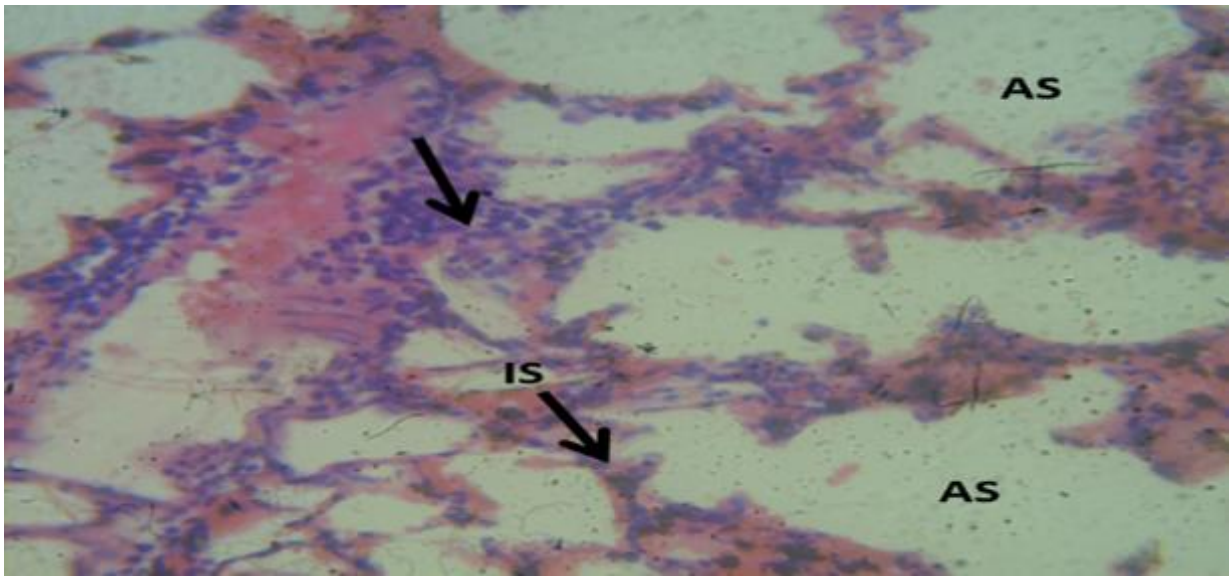

Figure 13: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with 500mg/kg of Ginger for two weeks, showing disrupted alveolar sac (AS) wall with reduced lymphocytic activities (arrows).
Diagnosis: Moderate inflammation and distortion of the lung tissues.

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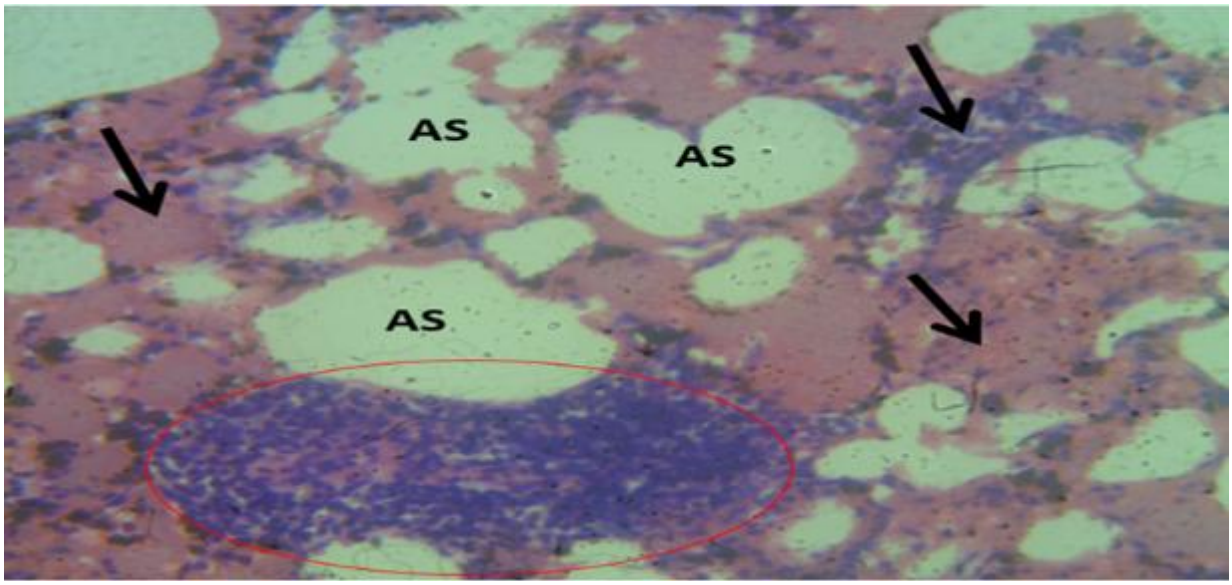

Figure 14: Photomicrograph (H&E X400) of the lungs of animals exposed to ethanol extract of peanut for one week and treated with 1000mg/kg of ginger for two weeks, showing focal mononuclear cell activities within the alveolar sac wall associated with hyalinization and hemosiderin deposits (arrows).
Diagnosis: moderate inflammation with hyperaemia of the lung tissue.

3.3. Discussion

The present study evaluated changes in the immunoglobulin levels as well as the numerous alterations that occurred in the lung histology of albino Wistar rats following the induction of asthma and treatment with *Zingiber Officinale* in comparison with the standard drug, Dexamethasone. This study aimed to understand how ginger affects immune response and lung inflammation during asthma treatment.

Immunoglobulins are essential in inflammatory and allergic responses, especially in asthma. IgE is produced from B-cells in response to inhaled allergens, which are also stimulated by T-helper 2 cells and interacting cytokines like stem cell factor (SCF). The released IgE leads to the activation of mast cells, triggering degranulation and the release of inflammatory mediators. These mediators contribute to airway inflammation, mucus secretion, and bronchoconstriction. The production of IgE is regulated through interactions between CD4+T helper cells and B lymphocytes, further linking adaptive immunity to allergic responses.

In this study, the data shown on tables 5 & 6, as well as figures 2 & 4, demonstrate changes in IgE following treatment with ginger extract and standard drug, Dexamethasone, indicating that both treatments reduced allergic response related to asthma. The decrease in IgE shows a reduced mast cell activity and a lesser release of inflammatory mediators and β-hexosaminidase. These findings correspond with earlier studies, including those conducted by Itharat et al [21], which reported that the inhibition of the release of β-hexosaminidase indicates anti-allergic activities.

Histological examination of lung tissues further supported these results. The lung photomicrographs (Figures 4.5 - 4.14) showed reduced inflammation, less airway wall thickening, and improved lung structure in the treated groups compared with the negative control group. These improvements were observed when ginger extract and dexamethasone were administered during both pre- and post-treatment periods following asthma induction.

The results from this study suggest that *Zingiber Officinale* has anti-inflammatory and immune-regulating effects that help reduce airway inflammation and improve lung structure in asthma. This supports the potential use of ginger as a supportive treatment in asthma management.

3.4. Conclusion

This study provides compelling experimental evidence supporting the anti-inflammatory and immunomodulatory potential of *Zingiber Officinale* in an experimental asthma model. Histopathological evaluation of lung tissues revealed that treatment with ginger extract markedly attenuated airway inflammation, as demonstrated by reduced inflammatory cell infiltration and improved lung structure, with effects comparable to those

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observed following dexamethasone administration. These findings indicate that ginger is effective in limiting the pathological changes associated with asthma.

The anti-inflammatory efficacy of *Zingiber Officinale* was clearly dose-dependent, with medium (500mg/kg) and high (1000mg/kg) doses producing greater therapeutic effects than the low dose (250mg/kg). This dose-response relationship highlights that appropriate dosing is critical for achieving optimal therapeutic effects when using plant-based treatments.

Beyond its effects on the lung histology, ginger extract demonstrated a positive influence on immune function, characterised by increased levels of IgG, IgA, IgM, and IgD relative to the negative control. This enhanced influence suggests an immune-supportive role for ginger consistent with previous reports and indicative of broader immunomodulatory benefits. At the same time, ginger treatment resulted in a significant reduction in IgE levels across all administered doses, indicating the suppression of all allergic responses linked to asthma and comparable to that achieved with dexamethasone.

Overall, these findings support the potential of *Zingiber Officinale* as a useful and alternative therapeutic agent in asthma management. The observed dose-dependent anti-inflammatory and immune functions suggest that ginger, could be used as a combination therapy with standard anti-inflammatory drugs like dexamethasone. This combination may enhance therapeutic efficacy while reducing the long-term usage of the corticosteroid. This study provides a foundation for future advancements and clinical investigations aimed at developing safe and effective treatments for asthma.

3.5. Recommendation

Based on the findings of this study, extensive and comprehensive research is recommended to explore the possible therapeutic effects of *Zingiber Officinale* on other body systems. Further studies should also focus on identifying the mechanisms through which ginger extract produces its anti-inflammatory and immunomodulating effects, particularly on diseases affecting the respiratory system. These studies will play a major role in determining the safe and effective use of ginger as an adjunct treatment option.

3.6. Contribution to Knowledge

This study has contributed to the scientific body of knowledge as it has further proven that ginger exhibits an anti-inflammatory response, hence, the reduction of IgE and reduced inflammation of the lungs. Therefore, ginger should be used as a combination therapy with other standard drugs like Dexamethasone for the treatment of asthma.

3.7. Acknowledgement

The authors would like to thank all research assistants for their contributions to the success and outcome of this study.

3.8. Ethical Statement

This study was conducted in accordance with the ethical guidelines established by the National Institute of Health (NIH) for the ethical treatment of animals in research. The study procedure was approved by the Research Ethics Committee of the University of Port Harcourt, Rivers State, Nigeria.

3.9. Data Availability

The supporting data of this study is available upon request from the corresponding author.

3.10. Conflict of Interest

The authors have no conflict of interest in this study.

3.11. Funding/Support

No form of funding or support was received for this study.

3.12. Authors Contribution

Author *JCI* developed the study and carried out the statistical analysis. Author *CGE* performed the experiments, maintained the data, conducted literature searches and authored the first draft of the manuscript. Both authors reviewed and approved the final paper.

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