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Anti- Inflammatory Effect of Zingiber officinale Extract on Male Wistar Albino Rat Induced with Albumin

1Cyril Onyekachi Edoga, 1Stephanie Chetam Uduak and 2Cyril Ifeanyichukwu Ezugwu

1Department of Applied Biology and Biotechnology, Faculty of Biological Sciences, Enugu State University of Science and Technology, ESUT.

2Deputy Director Emergency Department, Ahad Al-Masaraha General Hospital Jizan Region, Kingdom of Saudi Arabia.

Abstract-

The research employed a completely randomized experimental design and the study investigated the anti-inflammatory and pro-apoptotic effects of Zingiber officinale ethanol extract in male Wistar albino rats induced with albumin. Thirty rats were assigned into five groups: a blank control, a negative (inflammation-induced) control, a standard treatment group, and two treatment groups receiving low and high doses of Zingiber officinale extract. The serum levels of key inflammatory and apoptotic biomarkers (TNF-α, Bcl-2, Bax, and Caspase-3) were measured using ELISA techniques. Results showed a marked increase in TNF-α in the negative control (322.66 ± 0.0395 pg/ml) compared to the blank control (139.5 ± 0.0364 pg/ml), confirming inflammation-induced immune disruption. Treatment with Zingiber officinale extract significantly reduced TNF-α, particularly in the high-dose group (140.65 ± 0.0538 pg/ml), restoring levels close to baseline. Similarly, Bcl-2 decreased in the negative control (0.423 ± 0.0271 ng/ml) relative to the blank (1.072 ± 0.0017 ng/ml) but was improved in the high-dose treated group (0.757 ± 0.0447 ng/ml). Caspase-3 activity, suppressed in the negative control (0.479 ± 0.0528 ng/ml), was restored in both low- and high-dose treatment groups (0.730 ± 0.0078 ng/ml; 0.733 ± 0.0826 ng/ml). Conversely, Bax expression showed a minimal response, remaining suppressed in both treated groups (0.065–0.066 pg/ml) compared to the blank (0.206 ± 0.0021 pg/ml). Overall, the findings reaffirm the anti-inflammatory and pro-apoptotic potential of Zingiber officinale, supporting its traditional use in managing inflammation and highlighting its promise as a cost-effective natural therapeutic agent for immune modulation.

Keywords: *Zingiber officinale, TNF-α, Bcl-2, Bax, and Caspase-3*

***Corresponding Author: 1Cyril Onyekachi Edoga**

Introduction

Background of the study

An intricate and essential physiological reaction to tissue damage, infection, or toxic stimuli is inflammation. According to Chen et al. (2018), it is a typical component of the body's defensive mechanism that can occasionally be therapeutic and protective and other times contribute to the onset of different disorders. Chronic inflammation can cause tissue damage and has been linked to diseases like arthritis, cardiovascular disease, cancer, and neurological disorders, despite the fact that acute inflammation helps to maintain tissue homeostasis (Chen et al., 2018). The search for safer, natural anti-inflammatory drugs has increased as a result of this.

Microbial infections, physical damage, chemical irritants, immune system malfunction, and oxidative stress are the main causes of inflammation. Inflammatory mediators like cytokines, which are tiny signaling proteins generated by immune cells that aid in regulating inflammation and immunological response, are released when certain stimuli activate immune responses. Prostaglandins, reactive oxygen species (ROS), and TNF-α and IL-6 are some of the substances that contribute to inflammation (Pázmándi et al., 2024). Although they are necessary for the body's defense, an excess of them might have negative consequences and cause chronic inflammation (Zhou et al., 2006). Various chemicals are utilized to replicate various features of human inflammatory reactions in animals. These include carrageenan-induced paw edema, lipopolysaccharide (LPS)-induced systemic inflammation, and formalin-induced arthritis (Ojewole, 2006). Ovalbumin, the primary protein in egg white, is injected into the body of a male wistar albino rat in this study, which focuses on albumin-induced inflammation (Ismail & Al-Nahari, 2009). This study will assess important biomarkers involved in immune signaling and cellular apoptosis in order to look into Zingiber officinale's possible anti-inflammatory and pro-apoptotic qualities. Markers such as during inflammation, the body's immune cells produce a chemical signal called tumor necrosis factor-alpha (TNF-α). Although too much of it can harm healthy tissues and exacerbate inflammatory illnesses, it aids the body in fighting infection (Pázmándi et al., 2024). An enzyme called caspase-3 aids in apoptosis, or programmed cell death, the body's method of safely eliminating unhealthy or undesirable cells. When a cell is intended to die, it degrades vital cell components (Ali et al., 2022). A protein called Bcl-2 keeps cells from dying. By preventing other proteins from causing cell death, it aids in cell survival (Zick et al., 2011).Bax: this does the opposite of Bcl-2. It helps trigger cell death when a cell is damaged or unhealthy. It plays a key role in starting the apoptosis process (Elnahas et al., 2021).

Zingiber officinale, is a medicinal plant that has long been used in traditional medicine to treat infections, pain, inflammation, and other conditions. Its bioactive ingredients, such as gingerol, shogaol, and paradol, which all have strong anti-inflammatory and antioxidant qualities, are primarily responsible for its efficacy. By lowering oxidative stress, these substances combat free radicals, inhibit the synthesis of pro-inflammatory mediators, and alter or affect proteins linked to apoptosis, including Bax, Bcl-2, P53, and Caspase-3 (Ismail and Al-Nahari, 2009). Research has shown that giving Zingiber officinale to male wistar albino rats significantly improves a number of biological indicators that show the plant protects against inflammation and stress. While Zingiber officinale anti-inflammatory properties are well documented, very few studies has explored its effect in albumin induced inflammation models, especially its impact on apoptotic markers and cytokines like TNF-α, Bax, Bcl-2, and Caspase-3. This study seeks to fully elucidate the anti-inflammatory and apoptotic effects of Zingiber Officinale extract on male wistar albino rats.

***Corresponding Author: 1Cyril Onyekachi Edoga**

Justification of the Study

Researchers are looking for natural alternatives due to the growing number of disorders associated with inflammation and the adverse effects of many conventional drugs. One of these natural treatments, Zingiber officinale, is well-known for its anti-inflammatory properties (Pázmándi etal., 2024). Although Zingiber officinale has demonstrated promise in a number of trials, it is unclear how exactly it will affect models in which albumin causes inflammation. The purpose of this study is to investigate how Zingiber officinale might be useful in certain situations, possibly providing information about its function as a natural anti-inflammatory agent.

Aim of the Study

The primary aim of this study is to determine the anti-inflammatory effect of Zingiber officinale extract on male Wistar albino rats induced with albumin.

Specific objectives of the Study

The specific objective of the study were to:

● determine the effect of Zingiber officinale extract on Caspase-3 on male Wistar albino rats induced with albumin,

● determine the effects of Zingiber officinale extract on Bcl-2 on male Wistar albino rats induced with albumin,

● determine effects of Zingiber officinale extract on Bax on male Wistar albino rats induced with albumin, and

● determine the effects of Zingiber officinale extract on TNF-α on male Wistar albino rats induced with albumin.

Materials and Methods

Materials

Reagent

Distilled water, normal Saline, Tween 80, Ethanol, Ibuprofen (NSAIDs), Albumin.

Equipments

Rat cage, Capillary tubes, Centrifuge,Blood collection bottle, Beaker, Stirrer, Dissection kit, Dropper or pipette, Soxhlet apparatus , Whatnam filter paper, Weighing balance, Water bath, Analytical drying oven, Mechanical Grinder, Measuring cylinder, Gloves ,Cotton wool, Wash bottle .

Methods

Collection of plants extracts

Fresh rhizomes of Zingiber officinale were obtained from Nkpokiti market, IMT Road, Enugu State Nigeria. The rhizomes were identified and authenticated by Prof.C.S. Eze of the Department of Applied Biology and Biotechnology. ESUT

Preparation of the plant extract

The preparation of the plant extract follows a known protocol described by Ajayi et al. (2011)the Zingiber officinale rhizomes were washed to avoid contamination and placed in the tray of the analytical drying oven for 48 hrs at 105 degree centigrade. The dried Zingiber officinale rhizomes were then pulverized in a mechanical grinder. 76.4g of the powder was measured and poured into a sterilized soxhlet apparatus as the method of extraction is by soxhlet apparatus. 300ml of (98%) absolute ethanol (the extraction solvent) was poured into the boiling flask of the soxhlet apparatus. The extraction took place for 5 hrs after which both the ethanol and the pulverized powder were seated in the thimble of the soxhlet apparatus and appeared yellowish in color. Afterwards it was filtered using the Whatman no 1 filter paper and the citrate was then in a water bath at 50 degree centigrade to remove the ethanol and obtain a semi solid lipid like solvent or crude sap.

Animal model, Formulation and Administration of plant extract dose and full experimental design

Animal collection

Thirty male Wistar albinorats were obtained from theUniversity of Nigeria Enugu Campus

(UNEC) along with their feed. They were grouped into a labeled group of A-E, six in each group and were placed in a wired cage house. They were acclimatized for three weeks at the Animal House of Power Tech Analytical and Scientific Research Laboratory before the commencement of the experiment.

Formulation and Administration of plant extract and standard drug

The plant extract after being obtained as crude sap was dissolved in 3% of tween 80 (3 drops of tween 80 and 90% water) and the soluble plant extract was obtained.The now soluble plant extract was administered through intubation via oral. Administration was calculated and given according to the body weight of the rat. Rat weight was between 72-120g. Dosage volume was given in, low dose (100 mg/g) and high dose (200 mg/g).

A standard drug ibuprofen was also procured and 0.8 mg was crushed and dissolved in water and (100 mg/g) was given to a particular group.

Experimental design

The animals were conveniently sampled and grouped into five groups (six rats per group)

Group A (Blank control): were neither induced nor treated but received feed and water regularly.

Group B (Negative control): were induced with concentrated egg albumin but received no treatment.

Group C (Standard control): induced with concentrated egg albumin and treated with standard inflammation drug (ibuprofen)

Group D (low dose group) was induced with concentrated egg albumin and treated with 100 mg/g of the plant extract.

Group E (The high dose group) induced with egg albumin and treated with 200 mg/g of the plant extract.

The treatment was administered orally via intubation once every day for 3 consecutive weeks the egg albumin induction.

Inflammation Induction

0.5ml of Albumin (ovalbumin) was injected intraperitoneally into groups B-E of the male Wister albino rats, and inflammation was left to peak under under 6 hrs.

Sample Collection

The method of collection of blood sample was via ocular puncture.

Biochemical Analysis

Zingiber officinale extract was used, and rat serum samples were added in serial dilutions. Detection markers including Caspase-3, Bcl-2, Bax, and TNF-α were measured using commercially available ELISA (enzyme-linked immunosorbent assay) kits. The absorbance was read at 450 nm using a microplate reader, and the concentration of each marker was determined using standard calibration curves.

In addition, antibody responses, specifically IgG and IgM titers against Zingiber officinale, were assessed using an indirect ELISA method. For this, microplates were coated with Zingiber officinale extract, followed by the addition of rat serum samples. Enzyme-linked secondary antibodies and TMB (tetramethylbenzidine) substrate were then applied, and the optical density was measured to determine antibody levels. For immune cell profiling, flow cytometry was employed. Peripheral blood mononuclear cells (PBMCs) were isolated from whole blood using Ficoll-Paque density gradient centrifugation. The PBMCs were stained with fluorescently labeled monoclonal antibodies targeting specific surface markers such as CD3 (T cells), CD4 and CD8 (T-cell subsets), and CD45RA (B cells). The samples were analyzed using a flow cytometer, and the data were interpreted with appropriate flow cytometry software (McKinnon, 2018).

Statistical Analysis

The data were carried out using the Statistical Package for Social Science (SPSS, version 16). The values of the measured parameters were expressed as mean ± SEM. Two-way analysis of variance (2-way ANOVA) was used to determine the anti-inflammatory and pro-apoptotic effects of ethanol extract of Zingiber officinale in male Wistar albino rats induced with albumin. Differences between means were separated using Duncan's multiple range tests, and tests for significance were set at the 0.05 probability level.

RESULTS

Tumor Necrosis Factor-Alpha (TNF-α)

The serum TNF-α levels in male Wistar albino rats are presented in Table 1. The results shows a significant increase ($p < 0.05$) in TNF-α levels in the group B (322.66 ± 0.0395 pg/ml) when compared to the group A (139.5 ± 0.0364 pg/ml), indicating that inflammation was induced using albumin.Treatment with Zingiber officinale extractled to a marked reduction in TNF-α levels. The group E (140.65 ± 0.0538 pg/ml) shows TNF-α levels similarly statiscal to the group A ($p > 0.05$), which suggests a high dose of Zingiber officinale reversed the inflammatory effect effectively. Very similar to that is the the group D (151.491 ± 0.0835 pg/ml) and the group C (146.962 ± 0.0173 pg/ml) also shows significantly reduced TNF-α levels compared to the group B ($p < 0.05$), although it is a higher than the group E.These results suggest that Zingiber officinale displays strong anti-inflammatory effects, which are capable of lowering elevated TNF-α levels in a dose-dependent manner. The near balance of the group E to the group A shows a restoration of physiological TNF-α balance (Table 1).

Table1: Effect of Zingiber officinale ethanol extract on TNF-A (pg/ml) of male wistar albino rats induced with inflammation.

GROUPS	TNF-A (pg/ml)
A (Blank Control)	139.5 ± 0.0364[a]
B (Negative Control)	322.66 ± 0.0395[b]
C (Standard Control)	146.962 ± 0.0173[a]
D (Low-Dose Treated Group)	151.491± 0.0835[a]
E (High-Dose Treated Group)	140.65 ± 0.0538[a]

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different ($p<0.05$)

BAX

In this study, the expression of BAX was measured to assess the potential of Zingiber officinale to modify apoptotic signaling during inflammation. The results revealed notable variations in BAX levels across the experimental groups.The blank control group (Group A), which consisted

of healthy rats not exposed to inflammatory induction or treatment, exhibited the highest base-

line level of BAX expression (0.206 ± 0.0021 pg/ml). This indicates normal pro-apoptotic activity in the absence of inflammatory stress or pharmacological intervention. On the other hand, the negative control group (Group B) which was induced with inflammation but received no treatment had a decrease in BAX levels (0.074 ± 0.0004 pg/ml) compared to the blank control. This reduction suggests that the inflammatory process may suppress natural pro-apoptotic mechanisms, and contribute into the survival and accumulation of damaged or inflamed cells. The standard control group (Group C), which received a known anti-inflammatory drug(ibuprofen) after inflammation induction, showed a marked increase in BAX expression (0.296 ± 0.0761 pg/ml). Interestingly, this value was even higher than that observed in the blank control group, indicating a strong restoration and possible enhancement of pro-apoptotic signaling in response to treatment. However, the low-dose (Group D: 0.065 ± 0.0421 pg/ml) and high-dose (Group E: 0.066 ± 0.0251 pg/ml) Zingiberofficinale treatment groups exhibited BAX levels similar to the negative control group and was lower than both the blank and standard control groups (Table 2).

Table 2: Effect of Zingiber officinale ethanol extract on BAX (pg/ml) of male wistar albino rats induced with inflammation

GROUPS	BAX (pg/ml)
A (Blank Control)	0.206 ± 0.0021^{a}
B (Negative Control)	0.074 ± 0.0004^{b}
C (Standard Control)	0.296 ± 0.0761^{a}
D (Low-Dose Treated Group)	0.065 ± 0.0421^{b}
E (High-Dose Treated Group)	0.066 ± 0.0251^{b}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different ($p<0.05$)

BCL-2

The blank control group (Group A), which was not subjected to inflammation or treatment, exhibited the highest BCL-2 levels (1.072 ± 0.0017 ng/ml). This serves as the baseline for normal anti-apoptotic activity in healthy, unstressed tissues, where cells maintain a natural balance of survival signals. The negative control group (Group B), which was induced with inflammation but did not receive any treatment, and showed a marked reduction in BCL-2 expression (0.423 ± 0.0271 ng/ml)and possibly made tissues more vulnerable to damage and cell loss due to reduced protection against apoptosis. In other cases, the low-dose treated group (Group D) recorded BCL-2 levels (0.442 ± 0.0693 ng/ml) that were comparable to the negative control. This indicates that the lower dose of Zingiber officinale had minimal restorative effect on BCL-2 expression, and did not really counteract the inflammation-induced decline in anti-apoptotic signaling. , the standard control group (Group C), which received a known anti-inflammatory drug, and the high-dose treated group (Group E), which was administered a higher dose of Zingiber officinale, both exhibited moderately elevated BCL-2 levels (0.794 ± 0.0055 ng/ml and 0.757 ± 0.0447 ng/ml, respectively) compared to the negative control. Although these levels did not fully return to those seen in the blank control group, the increase indicates partial restoration of

BCL-2 expression in response to treatment (Table 3).

Table 3: Effect of Zingiber officinale ethanol extract on BCL2 (ng/ml) of male wistar albino rats induced with inflammation

GROUPS	BCL_2 (ng/ml)
A (Blank Control)	1.072 ± 0.0017^{a}
B (Negative Control)	0.423 ± 0.0271^{b}
C (Standard Control)	0.794 ± 0.0055^{c}
D (Low-Dose Treated Group)	0.442 ± 0.0693^{b}
E (High-Dose Treated Group)	0.757 ± 0.0447^{c}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different ($p<0.05$)

CASPASE-3

The blank control group (Group A), representing healthy animals with no inflammation or treatment, displayed relatively high Caspase-3 levels (0.751 ± 0.0117 ng/ml). This level serves as the groundwork for normal apoptotic activity under non-stressed conditions. The negative control group (Group B), which was induced with inflammation but did not receive any treatment, recorded a notably lower Caspase-3 concentration (0.479 ± 0.0528 ng/ml). The low-dose (Group D) and high-dose (Group E) Zingiberofficinale treated groups showcased elevated Caspase-3 levels (0.730 ± 0.0078 ng/ml and 0.733 ± 0.0826 ng/ml, respectively). These values were very similar to that of the blank control group, indicating a complete restoration of Caspase-3 expression. This showed or suggested that treatment with Zingiber officinale, regardless of dose, effectively reestablished apoptotic signaling that had been disrupted by inflammation. The standard control group (Group C), which received anti-inflammatory drug, showed an intermediate Caspase-3 level (0.617 ± 0.0361 ng/ml). While this value was higher than that of the negative control, it did not reach the levels observed in the blank control or the ginger-treated groups, suggesting partial recovery of apoptotic activity (Table 4).

Table 4: Effect of Zingiber officinale ethanol extract on CASPASE-3 (ng/ml)of male wistar albino rats induced with inflammation

GROUPS	CASPASE-3 (ng/ml)
A (Blank Control)	0.751 ± 0.0117^{a}
B (Negative Control)	0.479 ± 0.0528^{b}
C (Standard Control)	0.617 ± 0.0361^{c}
D (Low-Dose Treated Group)	0.730 ± 0.0078^{a}
E (High-Dose Treated Group)	0.733 ± 0.0826^{a}

The values are expressed as (mean ± SEM)

Mean values with different letters as superscript are significantly different ($p<0.05$

***Corresponding Author:** *1Cyril Onyekachi Edoga*

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DISCUSSION, CONCLUSION AND RECOMMENDATIONS

Discussion

This study investigated the anti-inflammatory effects of Zingiber officinale extract in male Wistar albino rats induced albumin. The study focused on the impact of Zingiber officinale on specific pro-inflammatory and apoptotic markers namely TNF-α, BAX, BCL-2, and Caspase-3. The results are interpreted in light of existing literature to identify points of agreement, reinforcement, contradiction, or novelty. The current study demonstrated that the negative control group had much higher TNF-α levels, indicating that inflammation had been successfully induced. Zingiber officinale treatment, particularly at high doses, decreased TNF-α concentrations, returning them to levels comparable to those of the blank and standard control groups. This result supports earlier research showing Zingiber officinale's ability to inhibit pro-inflammatory cytokines. For example, it is consistent with the findings of Pázmándi et al. (2024), who found that in an ovalbumin-induced inflammation model, Zingiber officinale extract inhibited TNF-α and other Th2 cytokines. The results of Zammel et al. (2022), who demonstrated that Zingiber officinale decreased TNF-α levels in asthmatic rats, are also supported by this.Furthermore, the result agrees with Mashhadi et al. (2013) and Morvaridzadeh et al. (2020), who confirmed that Zingiber officinale supplementation reduced systemic TNF-α in human trials. Thus, the anti-inflammatory role of ginger observed in this study is in strong alignment with existing literature. According to the study, the negative control group's BAX levels were significantly suppressed by inflammation alone. Interestingly, both the low-dose and high-dose treated groups displayed BAX levels that were comparable to the negative control and much lower than the standard and blank controls, despite the fact that Zingiber officinale is known to activate pro-apoptotic markers. This outcome does not entirely corroborate past research showing elevated BAX expression after Zingiber officinale therapy. For instance, following the treatment of Zingiber officinale, Elnahas et al. (2021) and Zick et al. (2011) observed elevated BAX expression in inflammatory or malignant tissues. On the other hand, our results imply that the BAX reaction was dose-sensitive or that the Zingiber officinale extract might have altered apoptosis by other routes in the circumstances of this investigation.Therefore, while the outcome partially supports Zingiber officinale apoptotic influence, the expected BAX elevation was not prominent, indicating the need for further exploration.

The findings indicated that BCL-2, an anti-apoptotic protein, was reduced significantly in the negative control group due to inflammation. While the high-dose and standard control groups had a moderate restoration of BCL-2 expression, but not to the level of the blank control, the low-dose ginger group displayed similarly low BCL-2 levels. This is in line with research by Zick et al. (2011) and Elnahas et al. (2021), which discovered that Zingiber officinale extract raised pro-apoptotic markers including p53 and Bax while decreasing BCL-2 expression. It also reaffirms the report from the 2006 Journal of the Korean Society of Food Science and Nutrition, where [6]-gingerol reduced BCL-2 in breast cancer cells. Therefore, the hypothesis that ginger regulates BCL-2 levels based on dose and condition to balance apoptosis is supported by the partial restoration of BCL-2 in the high-dose group.

In the negative control group, caspase-3 levels were significantly reduced, suggesting that inflammation inhibits apoptosis. However, Caspase-3 levels were considerably recovered to meet or surpass those of the healthy control in both the low-dose and high-dose Zingiber officinale treated groups, indicating activation of the apoptotic pathway. Partial recovery was also observed in the standard control group. [6]-gingerol

***Corresponding Author:** *1Cyril Onyekachi Edoga*

stimulated Caspase-3 through ROS and MAPK signaling in cancer cells, according to previous research by Choi et al. (2016) and Chakraborty etal. (2021), which is strongly supported by this data. Additionally, it confirms Zingiber officinale's capacity to trigger apoptosis in inflammatory settings, as noted in the Morvaridzadeh et al. (2020) systematic review.

Thus, the result agrees with literature showing that Zingiber officinale restores Caspase-3 activity in conditions where inflammation suppresses apoptotic signals.

Conclusion

This study shows that Zingiber officinale ethanol extract has both anti-inflammatory and pro-apoptotic effects in rats with albumin-induced inflammation. The extract significantly lowered TNF-α levels, confirming its strong anti-inflammatory action. It also helped restore normal cell death signals by improving BCL-2 and Caspase-3 activity. However, BAX levels remained low, suggesting that this particular pathway may not respond as strongly in the inflammation model used.Overall, the findings support the traditional and scientific use of Zingiber officinale as a natural remedy for reducing inflammation and promoting healthy cell turnover.

Recommendations

Although Zingiber officinale has been widely studied for its anti-inflammatory and antioxidant properties, there is still a need for further exploration into its pharmaceutical applications, especially in relation to apoptotic regulation and immune response. Based on the results of this study, it is recommended that Zingiber officinale be further investigated as a potential therapeutic agent in the management of inflammation-related conditions and apoptosis dysregulation. Its natural origin, affordability, and safety profile make it a valuable candidate for the development of cost-effective drugs or supplements aimed at treating chronic inflammatory diseases and restoring immune balance.

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