HISTOLOGICAL CHANGES IN THE LARYNX AND SOME HAEMATOLOGICAL AND BIOCHEMICAL CHANGES IN WISTAR RATS FOLLOWING EXPOSURE TO CERAMICS DUST

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BEING A PROJECT WORK SUBMITTED TO THE

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BENIN CITY, EDO STATE,

NIGERIA.

NOVEMBER, 2025

DECLARATION

I declare that:

a. This project is based on the experiment work undertaken by me in the department of anatomy, School of Basic Medical Sciences, University of Benin, under the supervision of DR. (MRS) M.B EHI-OMOSUN.

b. This work has not been previously submitted for the award of a degree elsewhere.

C. All ideas and views are essentially based on this research and where the views of others has been expressed, such were duly acknowledged.

ETSENAME OGHELLE GABRIELLA

CERTIFICATION

This is to certify that this research project titled "**HISTOLOGICAL CHANGES IN THE LARYNX AND SOME HAEMATOLOGICAL AND BIOCHEMICAL CHANGES IN WISTAR RATS FOLLOWING EXPOSURE TO CERAMICS DUST**" is a project work carried out by **ETSENAME OGHELLE GABRIELLA (BMS2101325)** of the Department of Anatomy, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria.

DR. (MRS) M. B. EHI-OMOSUN — DATE

(Project Supervisor)

DR A.B. ENOGIERU — DATE

HEAD OF DEPARTMENT

EXTERNAL EXAMINER — DATE

DEDICATION

This project work is dedicated to Almighty God and myself.

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ABSTRACT

Exposure to industrial dust has been linked to respiratory and systemic toxicity. Ceramics dust, a common occupational contaminant, contains particulate matter that may induce structural and biochemical alterations. This study aimed to investigate the histological changes in the larynx and some haematological and biochemical parameters in adult Wistar rats following exposure to ceramics dust. Twenty-four (24) adult Wistar rats with weight ranging from 120–200g were grouped into four groups (A, B, C and D). The rats in Group A served as the control, the rats in Groups B were exposed to 5g of ceramics dust (low dose), the rats in Group C were exposed to 10g of ceramics dust (medium dose) and the rats in Group D were exposed to 20g of ceramics dust (high dose) for a duration of 1 hour daily for 30 days. At the end of the exposure period, rats were sacrificed, laryngeal tissues harvested for histological assessment, and blood samples collected for haematological and biochemical analyses, including RBC, WBC, hemoglobin, urea, creatinine, and bicarbonate levels. Histology revealed dose-dependent inflammatory infiltration in exposed groups. Haematological analysis showed dose-dependent decrease in hemoglobin and hematocrit, with a more significant decrease in the WBC count of the high dust exposure (20g) when compared with the control group. Biochemical assessment indicated elevated urea levels and decreased bicarbonate levels, suggesting renal and systemic effects of ceramics dust. These findings indicate that ceramics dust exposure causes structural damage to the larynx and alters some haematological and biochemical parameters in Wistar rats. The study underscores the potential health risks of occupational ceramics dust exposure and highlights the need for protective interventions in industrial settings.

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND OF THE STUDY

Ceramics, derived from the Greek word *keramos* meaning "potter's clay," have been integral to human civilization for millennial, serving as essential materials in construction, art, industry, and household applications. From ancient pottery to modern advanced ceramics used in electronics, aerospace, and medical implants, ceramics have shaped human culture and technological advancement (Carter & Norton, 2013). The production and use of ceramics involve processing raw materials such as clay, silica, feldspar, and other mineral oxides, which are often ground into fine powders during manufacturing. These processes generate ceramic dust, a particulate matter that poses potential health risks when inhaled by workers or individuals in proximity to ceramic production environments (Fubini & Areán, 1999).

The relationship between humans and ceramics is multifaceted, encompassing economic, cultural, and industrial dimensions. In industrial settings, ceramics are valued for their durability, thermal resistance, and aesthetic qualities, making them indispensable in tiles, sanitary ware, and refractory materials (Kingery et al., 1976). However, the generation of ceramic dust during mining, grinding, cutting, or polishing of ceramic materials has raised concerns about occupational and environmental health. Ceramic dust often contains crystalline silica, a known respiratory hazard that can lead to conditions such as silicosis, chronic obstructive pulmonary disease (COPD), and even lung cancer (IARC, 1997). Prolonged exposure to such dust may also affect other parts of the respiratory tract, including the larynx, which serves as a critical anatomical structure in phonation and airway protection (Gray, 1918).

The larynx, commonly known as the voice box, is particularly vulnerable to inhaled irritants due to its position in the upper respiratory tract. Histological changes in the larynx, such as squamous metaplasia, epithelial hyperplasia, or inflammatory cell infiltration, have

been observed in animal models exposed to various airborne pollutants, including formaldehyde and wood dust (Swenberg et al, 1980). These changes may reflect adaptive or toxic responses to chronic irritation, potentially compromising laryngeal function. Additionally, systemic effects of dust exposure, including haematological and biochemical alterations, have been documented in animal studies, indicating broader physiological impacts (Ziemann et al., 2017). For instance, exposure to particulate matter has been associated with increased white blood cell counts, oxidative stress, and changes in serum biomarkers such as alkaline phosphatase and total protein levels.

Given the widespread use of ceramics and the occupational exposure risks in ceramic industries, understanding the biological effects of ceramic dust is crucial. Animal models, particularly Wistar rats, are widely used to study the toxicological effects of inhaled particles due to their physiological similarities to humans and their well-characterized responses to respiratory irritants (Oberdörster, 1995). Investigating histological changes in the larynx and systemic haematological and biochemical alterations in Wistar rats exposed to ceramic dust can provide insights into the mechanisms of dust-induced toxicity and inform occupational health strategies.

1.2 STATEMENT OF RESEARCH PROBLEM

The ceramic industry is a significant contributor to global economies, employing millions of workers in mining, manufacturing, and processing activities. However, these workers are often exposed to ceramic dust, which contains respirable particles such as crystalline silica, aluminium silicates, and other mineral compounds (Fubini & Areán, 1999). Chronic inhalation of ceramic dust has been linked to respiratory diseases, including silicosis, bronchitis, and lung cancer, as classified by the International Agency for Research on Cancer (IARC, 1997). The larynx, a critical component of the respiratory tract, is particularly susceptible to damage from inhaled particles due to its role as a conduit for air and its exposure to high concentrations of airborne irritants (Gray, 1918). Histological changes in the larynx, such as epithelial metaplasia or inflammatory responses, may impair phonation, airway protection, and respiratory function, yet these effects remain under-explored in the context of ceramic dust exposure.

Moreover, systemic effects of ceramic dust inhalation, including haematological and biochemical changes, have been reported in animal studies but are not well-characterized for ceramic dust specifically (Ziemann et al., 2017). Alterations in blood parameters (e.g., white blood cell counts) and biochemical markers (e.g., liver enzymes, oxidative stress markers) may indicate broader physiological impacts, including immune activation, inflammation, or organ dysfunction. The lack of comprehensive data on the laryngeal and systemic effects of ceramic dust exposure in animal models limits our understanding of its toxicity and hinders the development of effective preventive measures for workers in the ceramic industry.

This study addresses the gap in knowledge regarding the histological changes in the larynx and associated haematological and biochemical alterations in Wistar rats exposed to ceramic dust. By using an animal model, the research aims to elucidate the potential health risks posed by ceramic dust, particularly to the respiratory tract and systemic physiology, and to provide a foundation for further toxicological and occupational health studies.

1.3 AIM

The aim of this study is to investigate the histological changes in the larynx and the haematological and biochemical alterations in Wistar rats following controlled exposure to ceramic dust.

1.4 OBJECTIVES

The specific objectives of the study are:

1. To evaluate the histological changes in the larynx of Wistar rats exposed to ceramic dust, including epithelial alterations, inflammatory responses, and structural changes.

2. To assess haematological changes, such as white blood cell counts, red blood cell parameters, and platelet levels, in Wistar rats exposed to ceramic dust.

3. To determine biochemical changes, including serum levels of creatinine, urea and bicarbonate, in Wistar rats exposed to ceramic dust.

4. To correlate the observed histological, haematological, and biochemical changes with the duration and intensity of ceramic dust exposure.

1.5 SIGNIFICANCE OF THE STUDY

This study is significant for several reasons:

1. It provides critical insights into the toxicological effects of ceramic dust on the larynx, an understudied organ in the context of occupational dust exposure. Understanding these effects can inform strategies to protect workers in the ceramic industry from respiratory and systemic health risks.

2. The study contributes to the body of knowledge on the systemic impacts of inhaled particulate matter, as evidenced by haematological and biochemical changes, which may serve as early biomarkers of exposure-related toxicity (Ziemann et al., 2017).

3. By using Wistar rats as a model, the study offers a controlled experimental framework to simulate human exposure scenarios, providing data that can guide occupational health policies and interventions.

4. The findings may also have implications for environmental health, as ceramic dust can contaminate air in communities near ceramic production facilities, posing risks to the general population (Fubini & Areán, 1999).

1.6 JUSTIFICATION OF THE STUDY

The justification for this study lies in the growing concern over occupational and environmental exposure to ceramic dust, particularly in regions with significant ceramic industries. Despite the well-documented risks of crystalline silica and other dust-related respiratory diseases, there is limited research specifically addressing the effects of ceramic dust on the larynx and systemic physiology (IARC, 1997). The larynx is a critical organ for

phonation and airway protection, and any damage could have significant functional consequences for affected individuals. Furthermore, systemic effects, such as immune activation or oxidative stress, may exacerbate the health impacts of dust exposure, necessitating a comprehensive investigation of both local and systemic responses.

Wistar rats were chosen as the experimental model due to their established use in toxicological studies and their physiological similarities to humans in terms of respiratory and immune responses (Oberdörster, 1995). The study's focus on histological, haematological, and biochemical parameters provides a holistic approach to understanding ceramic dust toxicity, addressing gaps in the literature and offering a foundation for future human studies. By elucidating the mechanisms of ceramic dust-induced damage, this research can support the development of safer workplace practices, improved dust control measures, and targeted medical interventions for exposed populations.

1.7 LIMITATIONS OF THE STUDY

This study has several limitations that should be acknowledged:

1. The use of Wistar rats, while valuable for controlled experimentation, may not fully replicate the physiological responses of humans to ceramic dust exposure due to species-specific differences in anatomy and immune responses (Oberdörster, 1995).
2. The study is limited to a controlled laboratory setting with specific exposure duration and dust concentrations, which may not fully reflect the variable exposure conditions in real-world occupational or environmental settings.
3. The study focuses on short- to medium-term exposure effects, and long-term chronic exposure outcomes may differ.
4. The availability of resources, such as advanced imaging or molecular analysis techniques, may limit the depth of histological and biochemical analyses, potentially restricting the scope of mechanistic insights.

CHAPTER TWO

LITERATURE REVIEW

2.1 TOXICANT OF STUDY

Ceramic dust is a fine particulate matter generated during the production, processing, or handling of ceramic materials. It consists of a complex mixture of inorganic compounds, primarily silicates, and can pose significant health and environmental risks when inhaled or dispersed. This chapter explores the chemical composition, origins, health impacts, and research gaps related to ceramic dust, with a focus on its effects on the larynx and hematological and biochemical changes in Wistar rats, a commonly used animal model in toxicological studies.

Figure 1: A Porcelain-based ceramic dust. Source: https://share.google/ZNKZdxZwm2xTLiAdS

2.1.1 CHEMICAL CONSTITUENTS OF CERAMIC DUST

Ceramic dust is primarily composed of silica (SiO_2), aluminum oxide (Al_2O_3), and various metal oxides such as calcium oxide (CaO), magnesium oxide (MgO), and iron oxide (Fe_2O_3). Depending on the type of ceramic material, trace amounts of heavy metals like chromium, lead, or cadmium may also be present, especially in glazes or specialty ceramics

(Smith et al., 2018). The crystalline form of silica, such as quartz, is particularly hazardous due to its ability to cause oxidative stress and tissue damage when inhaled. Particle size is a critical factor, with fine particles (PM2.5 and PM10) being more likely to penetrate deep into the respiratory tract, including the larynx (Oberdörster et al., 2005).

2.1.2 ORIGIN AND DISTRIBUTION OF CERAMIC DUST

Ceramic dust originates from both natural and anthropogenic sources. Naturally, it may arise from the erosion of silicate-containing rocks, but the primary source is industrial activities, including ceramic manufacturing, tile production, and pottery. These processes involve grinding, cutting, and firing of raw materials like clay, feldspar, and quartz, which release fine dust particles into the air (Monfort et al., 2014). Ceramic dust is widely distributed in occupational settings, such as factories and workshops, and can also contaminate surrounding environments through improper waste disposal or airborne dispersion (see Figure 1).

2.1.3 PRODUCTION AND CHARACTERISTICS

The production of ceramics involves several stages—mixing, shaping, drying, and firing—that generate dust at various points. The dust is characterized by its fine particle size (often <10 μm), angular morphology, and chemical stability, which make it resistant to degradation in biological systems (Guthrie & Heaney, 1995). The physicochemical properties, such as surface area and reactivity, influence its toxicity, with smaller particles exhibiting greater potential for cellular damage due to their ability to evade clearance mechanisms in the respiratory tract.

2.1.4 HEALTH IMPACTS OF CERAMIC DUST

Inhalation of ceramic dust is associated with respiratory, hematological, and systemic health effects. The larynx, a critical part of the upper respiratory tract, is particularly vulnerable to irritation and histological changes due to its direct exposure to inhaled particles. Studies have shown that ceramic dust can induce squamous metaplasia, inflammation, and epithelial hyperplasia in the larynx of rodents, which may serve as an adaptive response to

chronic irritation (Osimitz et al., 2007). Hematological changes, such as increased white blood cell counts and altered red blood cell indices, have been observed in Wistar rats exposed to dusts with similar compositions, like cement dust (Mojiminiyi et al., 2008). Biochemical alterations, including elevated liver enzymes (ALT, AST) and markers of oxidative stress, suggest systemic toxicity (Hasan et al., 2018).

2.1.5 ENVIRONMENTAL IMPACTS OF CERAMIC DUST

Ceramic dust contributes to air and soil pollution, particularly in areas near manufacturing facilities. Its deposition can alter soil chemistry, affecting plant growth and microbial activity (Monfort et al., 2014). Airborne ceramic dust also reduces air quality, posing risks to both human and ecological health. The persistence of silica particles in the environment exacerbates these impacts, as they are not readily biodegradable.

2.1.6 APPLICATIONS AND SOURCES OF CERAMIC DUST

Ceramic materials are used in construction (tiles, bricks), electronics (insulators), and medical applications (dental implants). Dust is generated during mining, grinding, and firing of raw materials like kaolin, quartz, and feldspar. Occupational exposure is common in ceramic factories, while environmental exposure occurs in communities near these facilities or through improper disposal of ceramic waste (Smith et al., 2018).

2.1.7 TOXICOLOGICAL STUDIES ON CERAMIC DUST

Toxicological studies on ceramic dust are limited compared to other dusts like silica or asbestos. However, research on related particulates, such as cement dust, shows that chronic exposure in Wistar rats leads to hematological changes, including increased packed cell volume (PCV) and hemoglobin levels, as well as histopathological changes in the lungs and other organs (Mojiminiyi et al., 2008). These studies suggest that ceramic dust, with its similar silica content, may induce comparable effects, including inflammation and oxidative stress in the respiratory tract.

2.1.8 EXPERIMENTAL CONTEXT OF CERAMIC DUST STUDIES

Most studies on ceramic dust use animal models like Wistar rats to simulate human exposure. These studies typically involve inhalation or intratracheal instillation of dust to assess respiratory and systemic effects. Wistar rats are chosen for their genetic consistency and well-characterized physiology, making them ideal for studying histological, hematological, and biochemical changes (Mojiminiyi et al., 2008). Experimental designs often include control and exposed groups, with exposure duration ranging from acute (days) to subchronic (weeks).

2.1.9 BIOMEDICAL STUDIES ON CERAMIC DUST EXPOSURE

Biomedical studies have focused on the respiratory effects of ceramic dust, with some evidence suggesting laryngeal and pulmonary damage. For instance, exposure to silica-containing dusts in rats has been shown to cause squamous metaplasia and inflammation in the larynx, similar to findings with formaldehyde exposure (Osimitz et al., 2007). Hematological studies indicate that dust exposure can increase white blood cell counts, reflecting an immune response, while biochemical markers like ALT and AST rise due to liver stress (Hasan et al., 2018). These findings highlight the need for studies specific to ceramic dust.

2.1.10 REGULATIONS AND SAFETY STANDARDS

Regulatory bodies like the Occupational Safety and Health Administration (OSHA) and the World Health Organization (WHO) have set exposure limits for crystalline silica, a key component of ceramic dust, due to its association with silicosis and lung cancer. The permissible exposure limit (PEL) for respirable crystalline silica is 0.05 mg/m^3 in many countries (OSHA, 2016). However, specific regulations for ceramic dust as a whole are lacking, underscoring the need for targeted safety standards.

2.1.11 METHODS FOR CERAMIC DUST CHARACTERIZATION AND ANALYSIS

Ceramic dust is characterized using techniques like X-ray diffraction (XRD) to identify crystalline phases, scanning electron microscopy (SEM) for particle morphology, and inductively coupled plasma mass spectrometry (ICP-MS) for elemental composition (Guthrie & Heaney, 1995). These methods help determine the dust's toxicity potential by analyzing its size, shape, and chemical makeup. Particle size analysis is particularly important, as smaller particles are more likely to cause respiratory damage.

2.1.12 MITIGATION AND CONTROL OF CERAMIC DUST

Mitigation strategies include engineering controls like ventilation systems, wet suppression techniques, and personal protective equipment (PPE) such as respirators. In occupational settings, regular monitoring of air quality and adherence to exposure limits are critical (Monfort et al., 2014). Community-level interventions involve proper waste management to prevent environmental contamination.

2.1.13 GAPS IN EXISTING LITERATURE

Despite the known risks of silica, research on ceramic dust specifically is sparse. Few studies have examined its effects on the larynx, and even fewer have explored hematological and biochemical changes in animal models. The lack of standardized exposure protocols and long-term studies limits our understanding of chronic effects. Additionally, the interaction between ceramic dust and other environmental pollutants remains under-explored.

2.1.14 THEORETICAL FRAMEWORKS AND MODELS

The toxicity of ceramic dust can be understood through the oxidative stress paradigm, where reactive oxygen species (ROS) generated by dust particles cause cellular damage (Oberdörster et al., 2005). The dose-response model is also relevant, as the severity of effects depends on particle concentration, size, and exposure duration. These frameworks guide experimental design and interpretation of results in toxicological studies.

2.1.15 FUTURE DIRECTIONS IN CERAMIC DUST RESEARCH

Future research should focus on long-term exposure studies in animal models to better understand chronic effects on the larynx and systemic physiology. Human epidemiological studies in ceramic workers could provide insights into real-world impacts. Additionally, developing specific regulations for ceramic dust and improving mitigation technologies will be crucial for reducing health risks.

2.2 ORGAN OF STUDY: THE LARYNX

The larynx, a vital organ in the upper respiratory tract, serves as the primary site of interaction with inhaled particles like ceramic dust. Its susceptibility to irritation and histological changes makes it a key focus in toxicological studies.

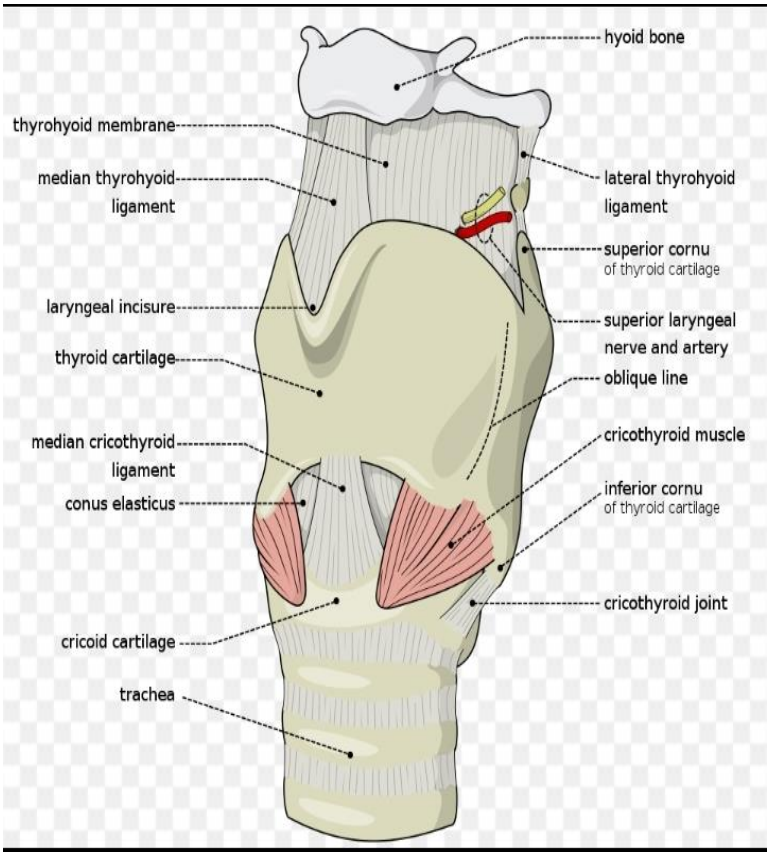

Figure 2: Structure of the human larynx. Source: https://share.google/fGmFb2gWo20FFVCFo

2.2.1 GROSS ANATOMY OF THE HUMAN LARYNX

The larynx, located between the pharynx and trachea, is a cartilaginous structure composed of the thyroid, cricoid, and arytenoid cartilages, among others. It houses the vocal cords and is responsible for phonation, airway protection, and respiration (Standring, 2020). The epiglottis, a flexible cartilage, prevents aspiration during swallowing. The larynx is lined with respiratory epithelium, except for the vocal cords, which are covered by stratified squamous epithelium.

2.2.1.1 MUSCLES OF THE HUMAN LARYNX

The larynx is a cartilaginous structure in the anterior neck involved in phonation, respiration, and airway protection. Its movements are controlled by intrinsic and extrinsic muscles, which alter the position, tension, and shape of the vocal folds (Standring, 2020; Sadler, 2019).

1. Intrinsic Muscles

Intrinsic muscles originate and insert within the larynx. They primarily control vocal cord tension, length, and opening/closing of the glottis. Most are innervated by the recurrent laryngeal nerve (branch of the vagus nerve, CN X), except the cricothyroid muscle, which is innervated by the external branch of the superior laryngeal nerve (Standring, 2020).

Key intrinsic muscles:

i. Cricothyroid: Tenses and elongates vocal cords; adjusts pitch (external branch of superior laryngeal nerve) (Sadler, 2019).
ii. Posterior cricoarytenoid: Only abductor of vocal cords; opens the glottis during inspiration (Standring, 2020).
iii. Lateral cricoarytenoid: Adducts vocal cords; closes the glottis during phonation (Netter, 2019).
iv. Transverse and oblique arytenoids: Adduct arytenoid cartilages; close the posterior glottis (Standring, 2020).

v. Thyroarytenoid: Shortens and relaxes vocal cords; aids in lowering pitch (Sadler, 2019).
vi. Vocalis: Part of thyroarytenoid; fine-tunes vocal cord tension (Kandel et al., 2021).

2. Extrinsic Muscles

Extrinsic muscles connect the larynx to surrounding structures. They stabilize or move the larynx during swallowing and speech (Standring, 2020). They include:

i. Suprahyoid muscles (digastric, stylohyoid, mylohyoid, geniohyoid): Elevate the larynx (Sadler, 2019).
ii. Infrahyoid muscles (sternohyoid, sternothyroid, omohyoid, thyrohyoid): Depress or stabilize the larynx (Netter, 2019).

2.2.1.2 BLOOD SUPPLY

The larynx is supplied by the superior and inferior laryngeal arteries, branches of the superior thyroid artery (from the external carotid) and the inferior thyroid artery (from the thyrocervical trunk), respectively. Venous drainage occurs via the laryngeal veins, which empty into the internal jugular vein (Standring, 2020).

2.2.1.3 LYMPHATIC DRAINAGE

Lymphatic drainage of the larynx is divided into supra- and subglottic regions. The supraglottic larynx drains into the deep cervical lymph nodes, while the subglottic region drains into the pretracheal and paratracheal nodes (Standring, 2020).

2.2.1.4 NERVE SUPPLY

The larynx is innervated by the vagus nerve via its superior and recurrent laryngeal branches. The superior laryngeal nerve supplies sensory innervation to the mucosa above the vocal cords and motor innervation to the cricothyroid muscle. The recurrent laryngeal nerve innervates all other intrinsic laryngeal muscles and provides sensory innervation below the vocal cords (Standring, 2020).

2.2.2 EMBRYOLOGY OF THE HUMAN LARYNX

The larynx develops from the fourth and sixth pharyngeal arches during the fourth week of embryogenesis. The endodermal lining of the laryngotracheal tube forms the laryngeal epithelium, while the surrounding mesenchyme differentiates into cartilage and muscle. By the eighth week, the vocal cords begin to form, and the larynx assumes its functional structure by birth (Sadler, 2018).

2.2.3 HISTOLOGY OF THE HUMAN LARYNX

The laryngeal mucosa consists of pseudostratified ciliated columnar epithelium (respiratory epithelium) in most areas, transitioning to stratified squamous epithelium over the vocal cords and epiglottis. Submucosal glands and lymphoid tissue are present, particularly at the base of the epiglottis and ventral pouch, which are prone to irritation-induced changes like squamous metaplasia (Osimitz et al., 2007). Cartilaginous structures are covered by perichondrium, and intrinsic muscles are composed of striated muscle fibers.

2.2.4 HEMATOLOGICAL AND BIOCHEMICAL EFFECTS OF CERAMIC DUST EXPOSURE

Exposure to ceramic dust in Wistar rats has been shown to cause hematological changes, including increased white blood cell counts, reflecting an inflammatory response, and altered red blood cell indices, such as packed cell volume (PCV) and hemoglobin levels (Mojiminiyi et al., 2008). Biochemical markers, such as elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT), indicate liver stress, while increased creatinine and urea levels suggest kidney involvement (Hasan et al., 2018). These changes are likely mediated by oxidative stress and inflammation induced by silica and metal oxides in the dust.

CHAPTER THREE

RESEARCH METHODOLOGY

3.1 MATERIALS

3.1.1 COLLECTION AND IDENTIFICATION OF CERAMICS DUST

For the aim of this study, Ceramics Dust was obtained from the Department of Industrial Design (Ceramics Subdivision), Faculty of Environmental Sciences, Federal University of Technology, Akure, Ondo State, Nigeria.

3.1.2 EQUIPMENT USED

The following equipment were used in this experimental study: cotton wool, plastic cages, DDGC (Dust Distributor Glass Chamber) for dust administration, a small hand fan for dust circulation, beakers and a digital weight balance. Wood shavings as bedding, grower mash, plates and bowls for feeding the animals, slides and covers, EDTA (tubes), plain bottles, universal syringes, tools for processing tissue and a light microscope.

3.1.3 REAGENTS

Reagents used during the study are: Purified water, alcohol levels in ascending and descending order, xylene, Haematoxylin, Eosin, Paraffin wax, 10% formal saline and commercial Formalin (40%).

3.2 METHODS

3.2.1 PREPARATION OF CERAMICS DUST

The components of the ceramics dust were obtained and processed at the Department of Industrial Design (Ceramics Subdivision), Faculty of Environmental Sciences, Federal University of Technology, Akure, Ondo State, Nigeria. To prepare ceramics dust for the study on the effects of ceramic dust exposure on the larynx of Wistar rats, the following steps were undertaken based on the provided Ceramics Dust (porcelain) composition and preparation method:

Composition of Ceramics (Porcelain) Dust:

- **Ball Clay**: 25% of the whole composition
- **Kaolin**: 25% of the whole composition
- **Feldspar**: 25% of the whole composition
- **Silica**: 25% of the whole composition

Preparation Process:

- **Weighing:** Each component (ball clay, kaolin, feldspar, and silica) was measured using a precision weight balance to ensure accurate proportions of 300 grams per component.
- **Mixing and Sieving:** The components were thoroughly mixed and sieved 10 times to achieve a homogeneous blend, ensuring uniform particle size and complete incorporation of the materials.
- **Drying:** The mixed powder was placed in a kiln (oven) and dried at a temperature of 600°C to remove moisture and prepare the dust for experimental use. Then it is stored in an airtight container.

3.2.2 ANIMAL CARE AND MANAGEMENT

To conduct this experimental study, twenty-six (24) Wistar rats weighing between 120 and 200 grams,were obtained from the Animal House at the University of Benin. These rats were given Top Feed Grower Mesh and clean water for two weeks to adapt (acclimatize) them to their new environment. Before the administration began, each rat was weighed. All procedures involving the rats were carried out in accordance with the accepted ethical guidelines for animal research (Oberdörster et al, 2005).

3.2.3 METHOD OF ADMINISTRATION

The rats that were to be administered to was exposed to the ceramics dust for 1 hour a day for 30 days with the use of a DDGC (Dust Distributor Glass Chamber) and an electric fan to ensure proper circulation of the dust. The dimensions of the DDGC are as follows: Length- 48cm, width- 38cm, height- 28cm and the volume- 51072cm^3. The DDGC was

properly cleaned with water and air dried after each administration to allow for effective and accurate dust administration. The experimental animals were weighed every week to note the changes in the rats' weight over the course of the experiment, and they were given unlimited access to standardized animal feed and clean water.

3.2.4 EXPERIMENTAL DESIGN

Wistar experimental rats totaling twenty-four (24) were randomly selected into four different groups; group A to group D, each including six (6) rats each. Each group's description is listed respectively below:

Table 3.1 Grouping of Experimental Animals.

Table 1 - Grouping of Experimental Animals.

GROUP	ADMINISTRATION PROTOCOL
A	**Control group:** standard feed and water
B	**Toxicant (5g):** standard feed and water+ ceramics dust exposure (1 hour/day for 30 days)
C	**Toxicant (10g):** standard feed and water+ ceramics dust exposure (1 hour/day for 30 days)
D	**Toxicant (20g):** standard feed and water+ ceramics dust exposure (1 hour/day for 30 days)

***Source**: Researcher's Fieldwork (2025).*

The weight of the experimental animals was taken after 6 weeks and the difference between the new weights and previous weights were recorded.

3.2.5 METHOD OF SACRIFICE AND SAMPLE COLLECTION

Before sacrifice, the final weights of the rats were taken using digital weight balance calibrated in grams. Cotton wool was then soaked with chloroform of about 50ml in an enclosed transparent container. The rats were put into the container for about 3-10secs for the anesthesing after which the rats were placed in a supine position on the dissection trolley. An abdomino-thoracic incision was made to expose the thoracic viscera of the rats, thereafter,

blood samples were collected through the heart and inferior vena cava by the procedure of cardiac and venous puncture respectively using 5mks syringes. The blood samples were turned into EDTA bottles for full blood count analysis and Red-top tubes for biochemical test analysis (kidney function test).

Each rat's larynx was harvested and fixed with 10% Formalin to preserve it before being processed in the Department of Anatomy Histopathology for histology analysis. The blood samples were submitted at the University of Benin Teaching Hospital (UBTH) for haematological and biochemical analysis.

3.3 HISTOLOGICAL PROCEDURES

3.3.1 TISSUE PROCESSING

The tissue sections were fixed in 10% Formalin for Haematoxylin and Eosin staining for 48 hours. The sections were then passed through grades of alcohol 70% to 90% alcohol and absolute alcohol to dehydrate. Next the sections were cleared in two changes of xylene to remove all the alcohol. In an oven at 65-75ºc, the sections were impregnated with two changes of molten paraffin wax for two hours. The tissue sections were then embedded in the paraffin wax, before being mounted on a wooden block and trimmed. Sections were made using a rotary microtome. The sizes of tissue sections were between 3-5 microns. The cut sections were floated on hot water bath and were picked on clean albuminized glass slides, and left to dry for 3 hours before staining.

3.3.2 HAEMATOXYLIN AND EOSIN STAINING METHOD

Haematoxylin and Eosin (H&E) staining: Tissue sections were processed at the histology laboratory of the University of Benin, Anatomy Department. The sections were first dewaxed in two changes of xylene, each for three minutes. This was followed by rehydration through a series of alcohol concentrations: absolutely alcohol, 90%, 70%, each for three minutes. The sections were then stained in Haematoxylin for ten minutes, rinsed with running water; a process called Blueing. Afterward, the sections were counterstained with Eosin for five minutes and rinsed in distilled water. Coverslips were mounted using

D.P.X, and the sections were examined under a light microscope at magnification of ×100 and ×400.

3.3.3 HISTOPATHOLOGY AND PHOTOMICROGRAPHY

The Larynx sections were viewed and analyzed then with a Leica Dm750 research microscope with a digital camera (LeicaCC50). At objective magnification of ×100 and ×400, tissue sections were digitally captured.

3.3.4 STATISTICAL ANALYSIS

Data were statistically analyzed using Graph Pad Prism and the relevant analytical valves were obtained. Statistical significance was determined by means of one-way analysis of variance, followed by Fishers multiple comparison LSD, and data were presented as Mean ± Standard Error of Mean (SEM). The post-hoc test was carried out for all groups compared with control and toxicant (20g of ceramics dust) group.

CHAPTER FOUR

4.1 RESULTS FROM STATISTICAL ANALYSIS

Data were statistically analyzed using Graph pad Prism and the relevant analytical values were obtained. Statistical significance was determined by means of one-way analysis of variance, followed by Turkey's multiple comparison post-hoc, and data were presented as Mean ± Standard Error of Mean (SEM). The post-hoc test was carried out for all groups compared with control.

TABLE 4.1 WEIGHT RESULTS

Weight indices across the experimental groups

Table 2 - WEIGHT RESULTS

Test/Groups	Group A (Mean ± SEM)	Group B (Mean ± SEM)	Group C (Mean ± SEM)	Group D (Mean ± SEM)	p-Value
INITIAL WEIGHT (g)	**154.00 ± 5.03**	**147.33 ± 5.17**	**156.33 ± 4.91**	**146.33 ± 6.94**	**0.5379**
FINAL WEIGHT (g)	**179.33 ± 8.17**	**168.33 ± 9.53**	**183.67 ± 5.67**	**190.67 ± 8.74**	**0.3347**
WEIGHT CHANGE (g)	**25.33 ± 6.39**	**21.00 ± 6.00**	**27.33 ± 1.20**	**44.33 ± 3.84**	**0.0394**

***Source**: Researcher's Fieldwork (2025).*

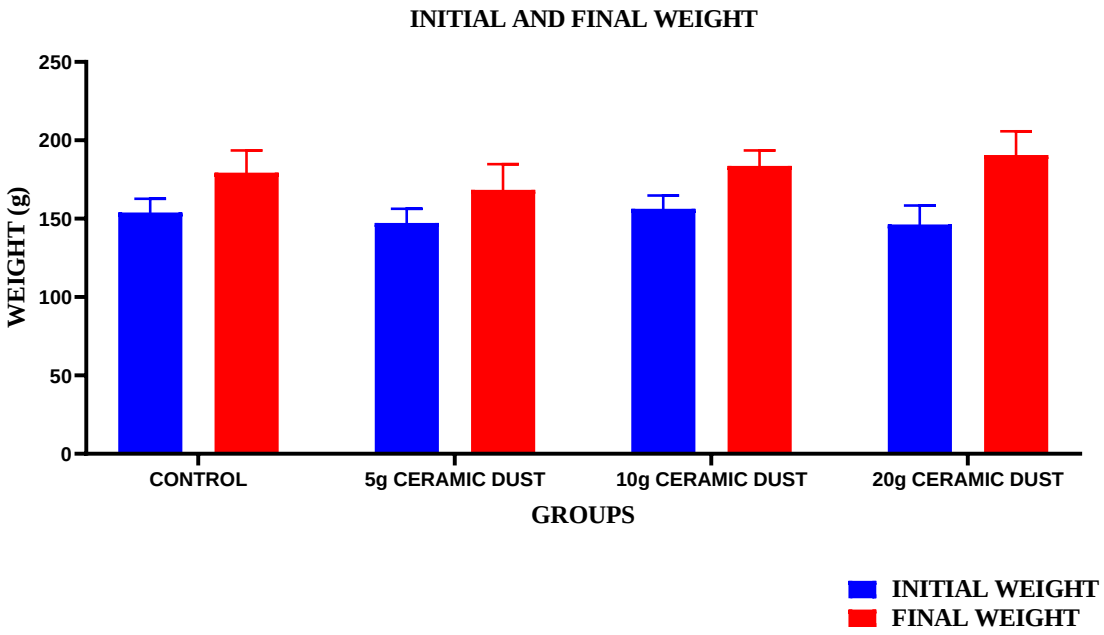

Figure 3 (Chart 1a): Body weight across the experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM.

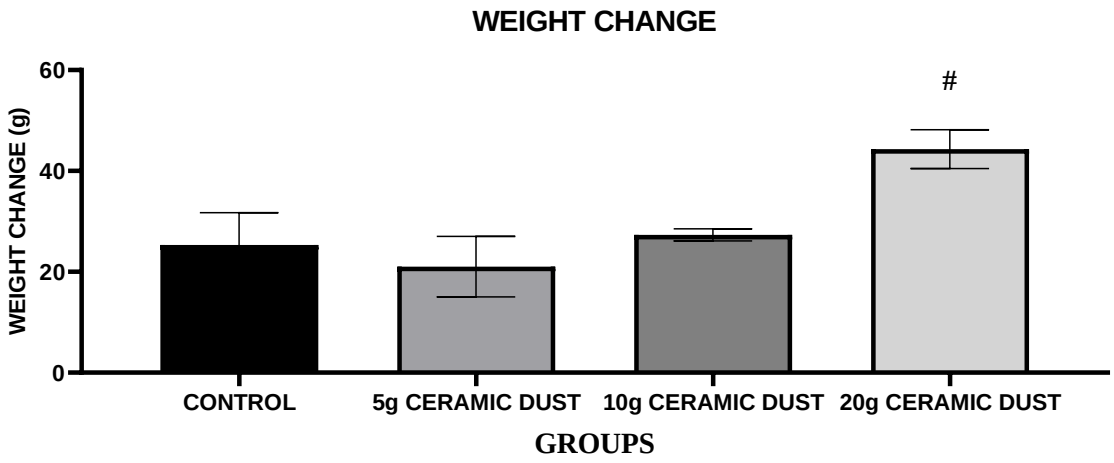

Figure 4 (Chart 1b): Comparing body weight change across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control group, # P<0.05 compared with 5g ceramic dust group.

There was a significant increase ($p<0.05$) in weight change of the group that were exposed to 20g ceramic dust as compared to 5g ceramic dust group.

Table 3 - HAEMATOLOGICAL PARAMETERS

Heamatological indices across the experimental groups

Test/Groups	**Group A**	**Group B**	**Group C**	**Group D**	**p-Value**
	(Mean ± SEM)	**(Mean ± SEM)**	**(Mean ± SEM)**	**(Mean ± SEM)**	
White `Blood Cells (×10³/µL)	**11.93 ± 1.02**	**11.85 ± 0.54**	**11.33 ± 2.86**	**7.63 ± 2.33**	**0.0331**
Lymphocytes Count (×10³/µL)	**9.17 ± 1.09**	**9.80 ± 1.09**	**7.47 ± 1.11**	**5.07 ± 1.26**	**0.0639**
Hemoglobin (g/dL)	**15.17 ± 0.18**	**14.33 ± 0.43**	**14.37 ± 0.27**	**12.20 ± 0.79**	**0.0461**
Hematocrit (%)	**45.63 ± 0.56**	**42.87 ± 1.39**	**43.90 ± 0.62**	**36.60 ± 5.85**	**0.0430**
Mean Corpuscular Volume (fL)	**64.50 ± 1.35**	**65.77 ± 0.73**	**63.43 ± 0.95**	**66.97 ± 1.32**	**0.5797**
Mean Corpuscular Hemoglobin (pg)	**17.37 ± 0.64**	**17.50 ± 0.28**	**17.23 ± 0.28**	**16.57 ± 0.38**	**0.5341**
Platelets (×10³/µL)	**1263.33 ± 377.77**	**1522.33 ± 326.75**	**1134.00 ± 334.53**	**1316.33 ± 325.04**	**0.9248**
Red Blood Cells (×10⁶/µL)	**7.09 ± 0.16**	**6.57 ± 0.21**	**6.93 ± 0.20**	**5.54 ± 0.37**	**0.1120**

***Source**: Researcher's Fieldwork (2025).*

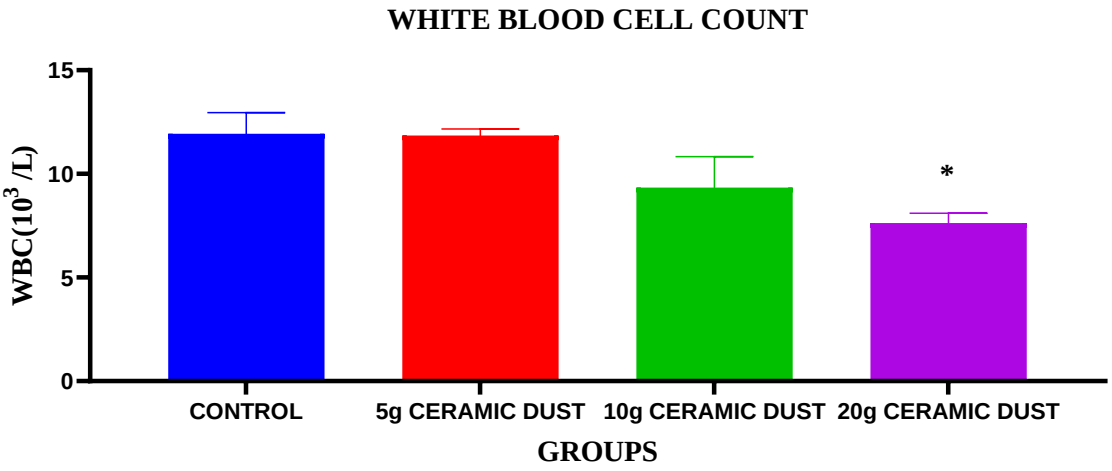

Figure 5 (Chart 2): Comparing White Blood Cell count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was a significant difference (p< 0.05) of White blood cell count in the group that was exposed to 20g Ceramic dust as compared to control group.

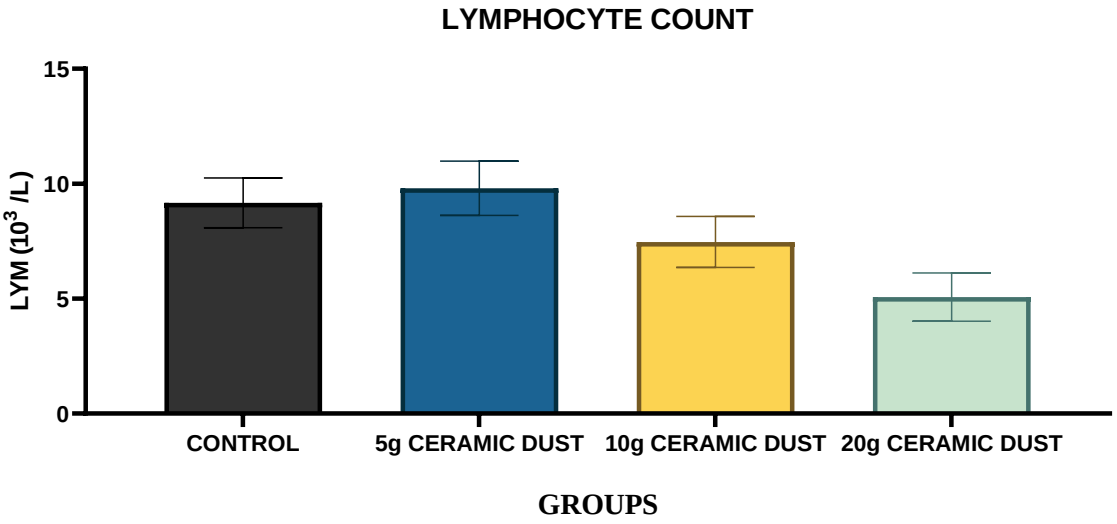

Figure 6 (Chart 3): comparing lymphocyte count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) in the lymphocyte Count across the groups.

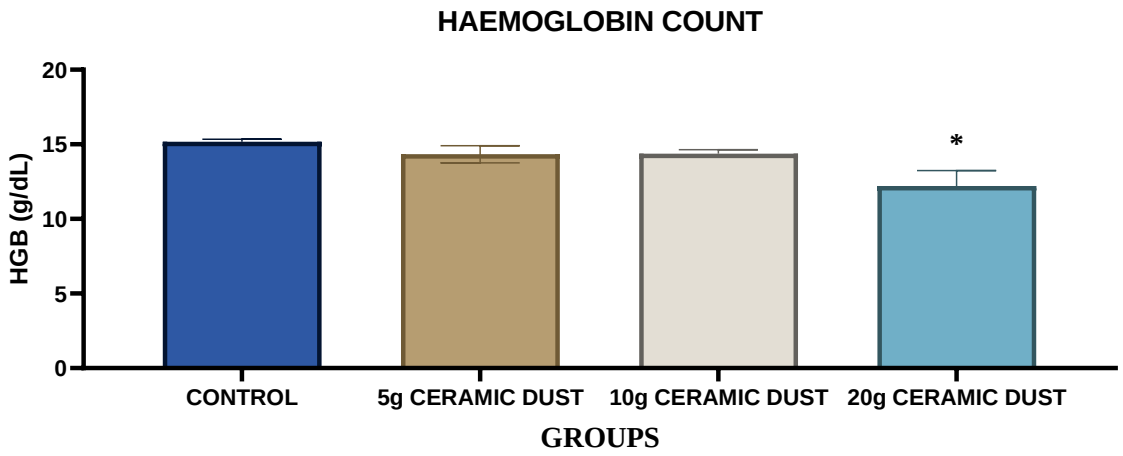

Figure 7 (Chart 4): Comparing Haemoglobin count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was a significant difference (p< 0.05) of haemoglobin count in the group that was exposed to 20g Ceramic dust as compared to control group.

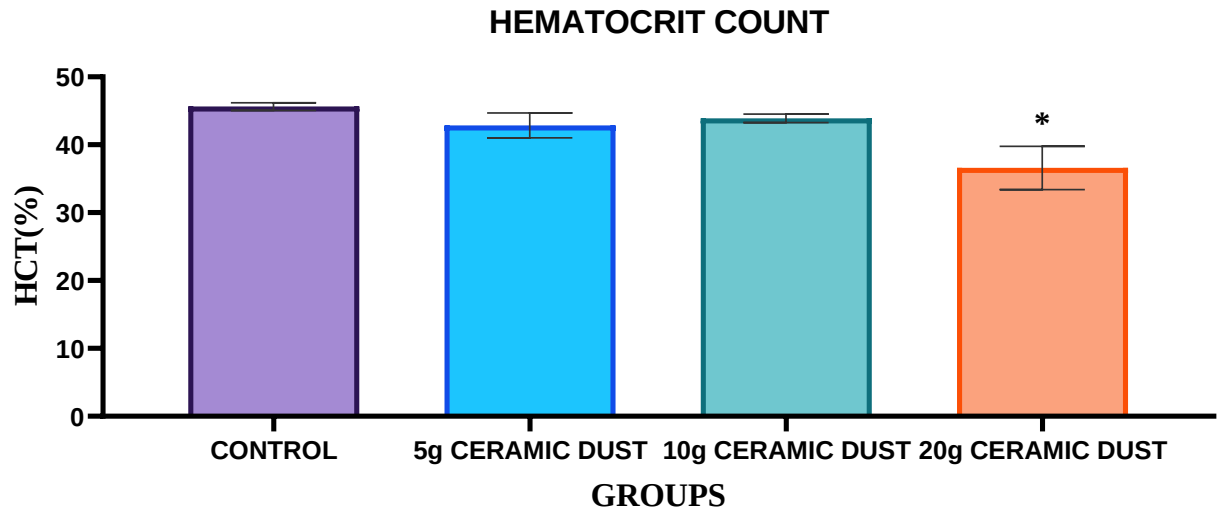

Figure 8 (Chart 5): Comparing Hematocrit count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was a significant difference (p< 0.05) of hematocrit count in the group that was exposed to 20g Ceramic dust as compared to control group.

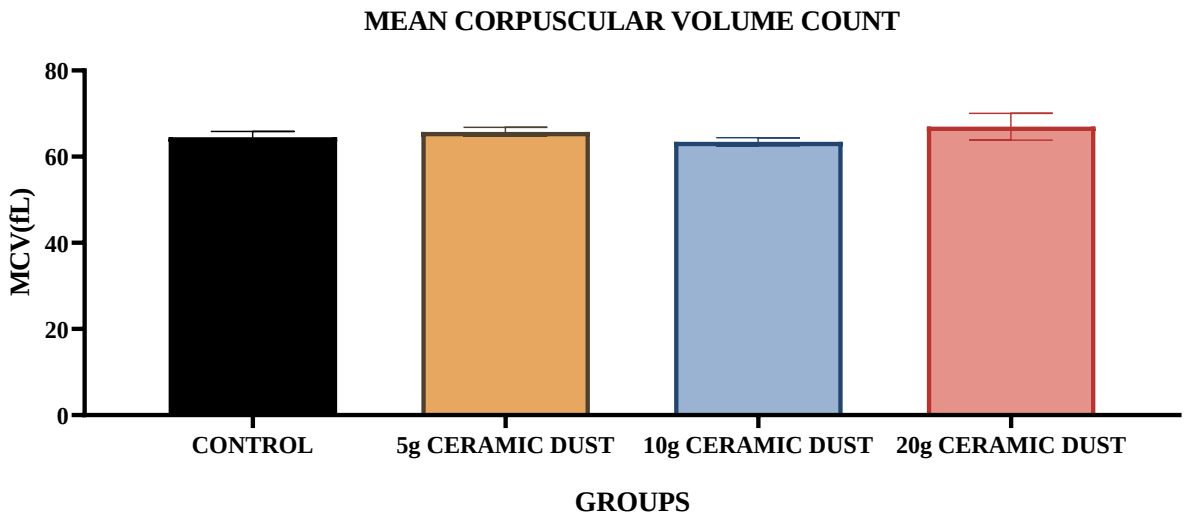

Figure 9 (Chart 6): Comparing MCV count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) in MCV Count across the groups.

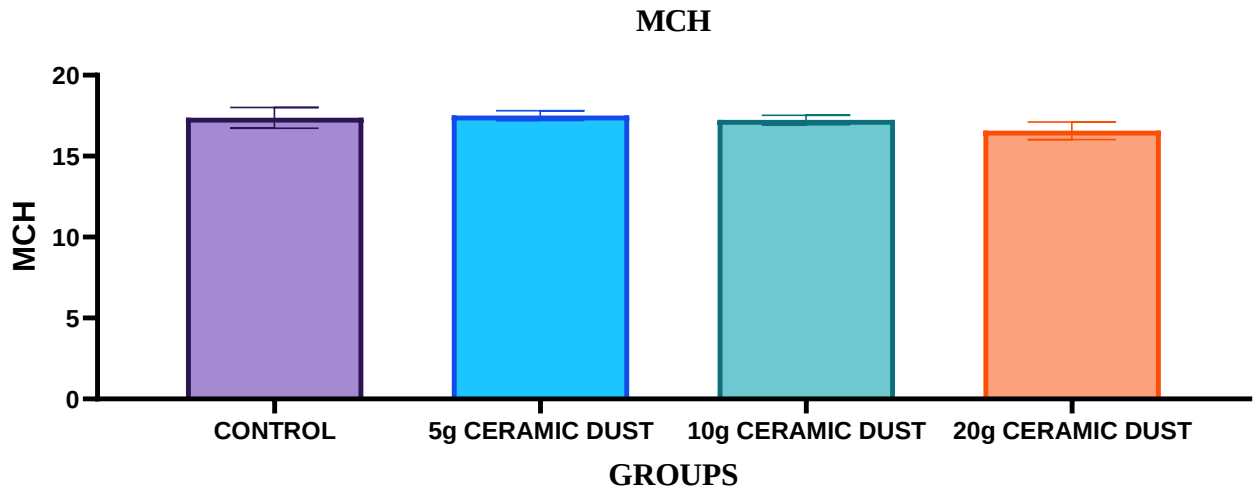

Figure 10 (Chart 7): Comparing MCH COUNT across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) in the MCH Count across the groups.

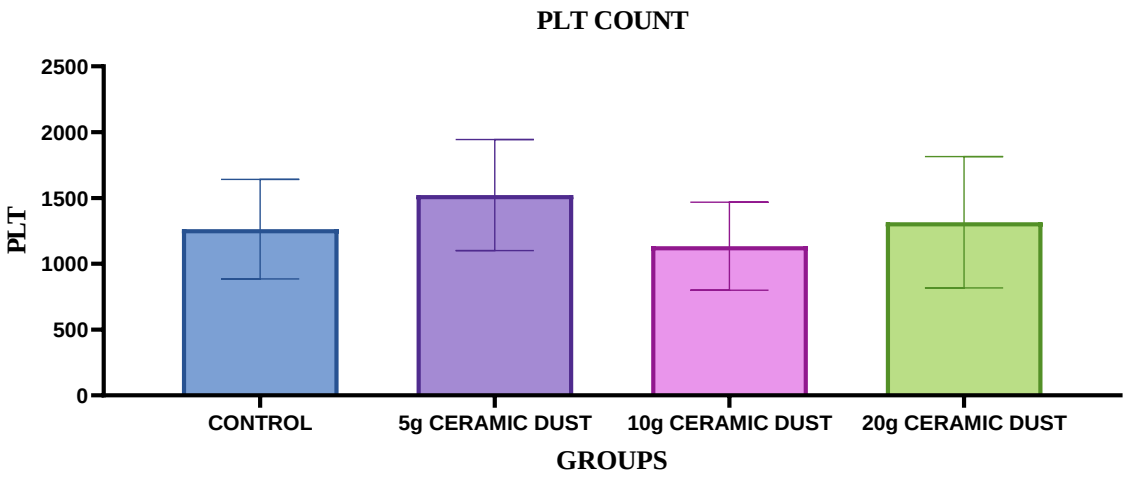

Figure 11 (Chart 8): Comparing Platelet count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) of platelet Count across the groups.

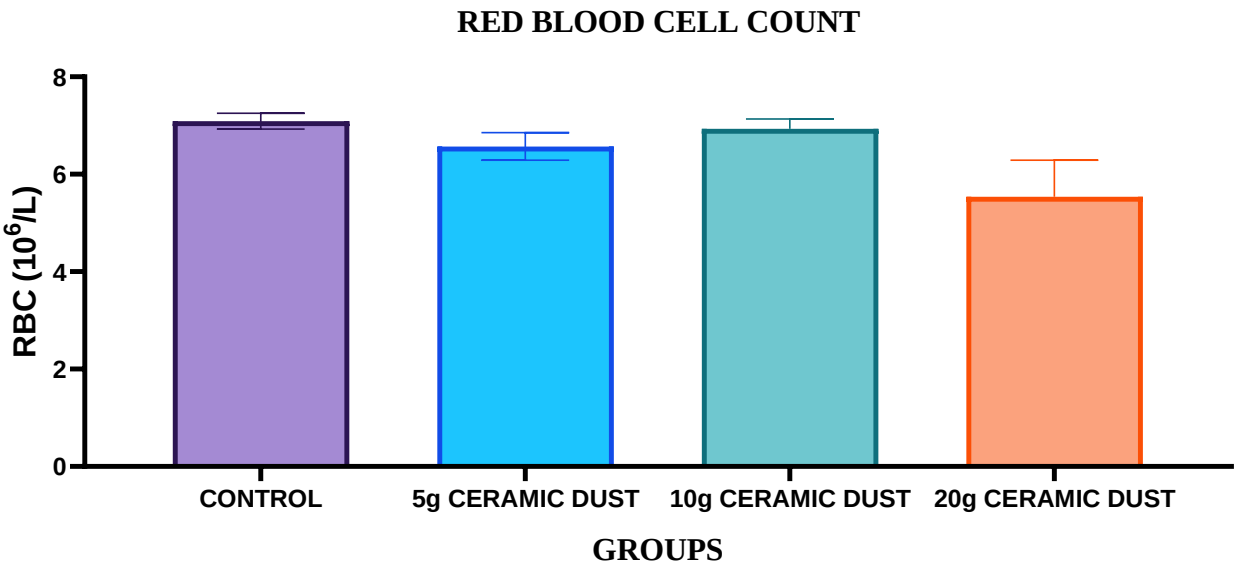

Figure 12 (Chart 9): Comparing Red blood cell count across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) in the Red blood cell count across the groups.

Table 4 - BIOCHEMICAL TEST

Test/Groups	Group A (Mean ± SEM)	Group B (Mean ± SEM)	Group C (Mean ± SEM)	Group D (Mean ± SEM)	P VALUE
UREA (mg/dl)	**30.33 ± 1.20**	**39.00 ± 2.08**	**39.00 ± 4.16**	**43.00 ± 3.21**	**0.0191**
HCO3- (m.mol/l)	**21.67 ± 0.88**	**4.67 ± 0.76**	**22.33 ± 0.33**	**100.67 ± 1.86**	**0.0046**
CR (mg/dl)	**0.73 ± 0.03**	**0.77 ± 0.03**	**0.77 ± 0.03**	**0.73 ± 0.03**	**0.0951**

***Source**: Researcher's Fieldwork (2025).*

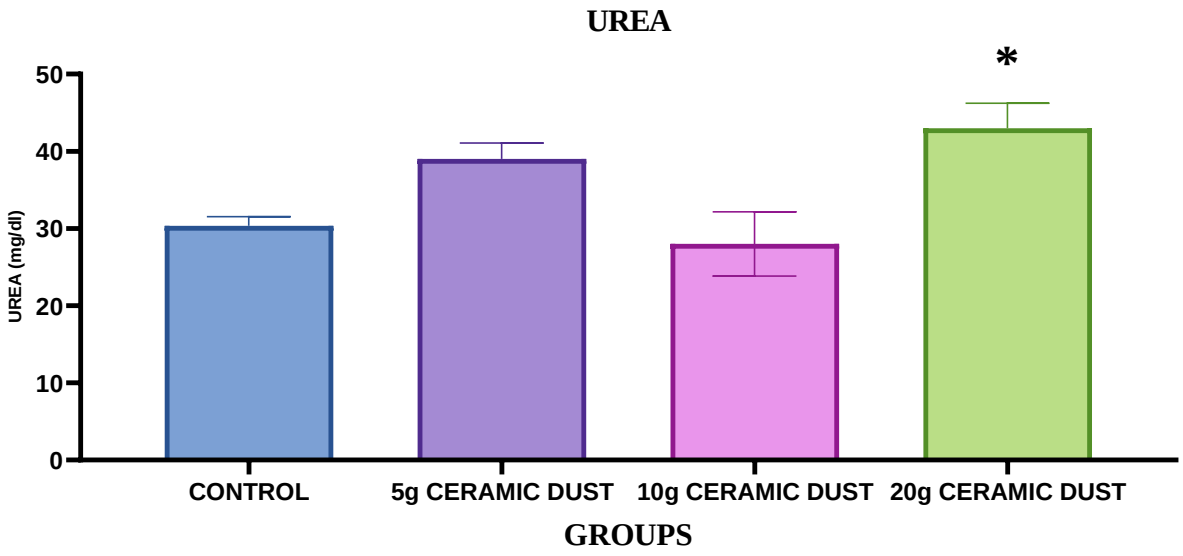

Figure 13 (Chart 10): Comparing Urea concentration across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was a significant difference (p< 0.05) of urea concentration in the group that was exposed to 20g Ceramic dust as compared to control group.

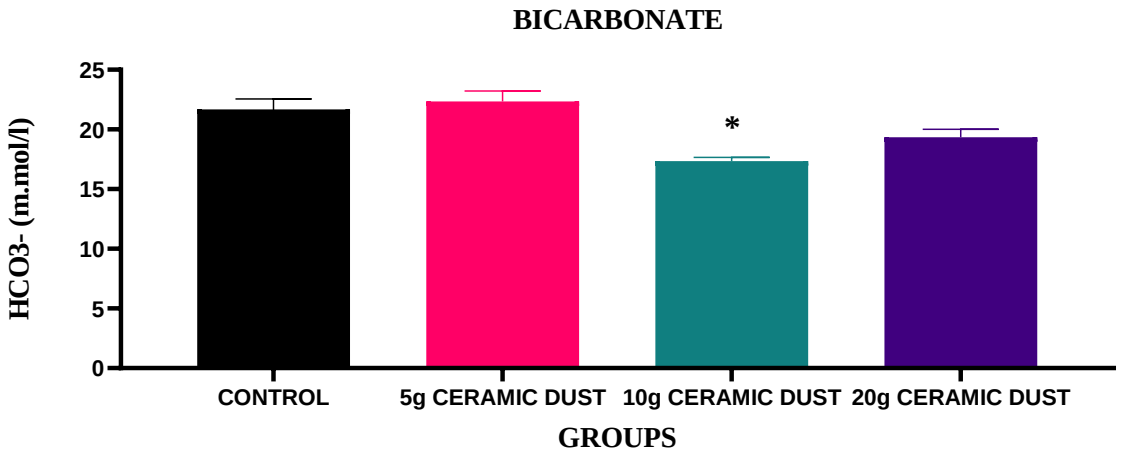

Figure 14 (Chart 11): Comparing Bicarbonate concentration across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was a significant difference (p< 0.05) of bicarbonate concentration in the group that was exposed to 20g Ceramic dust as compared to control group.

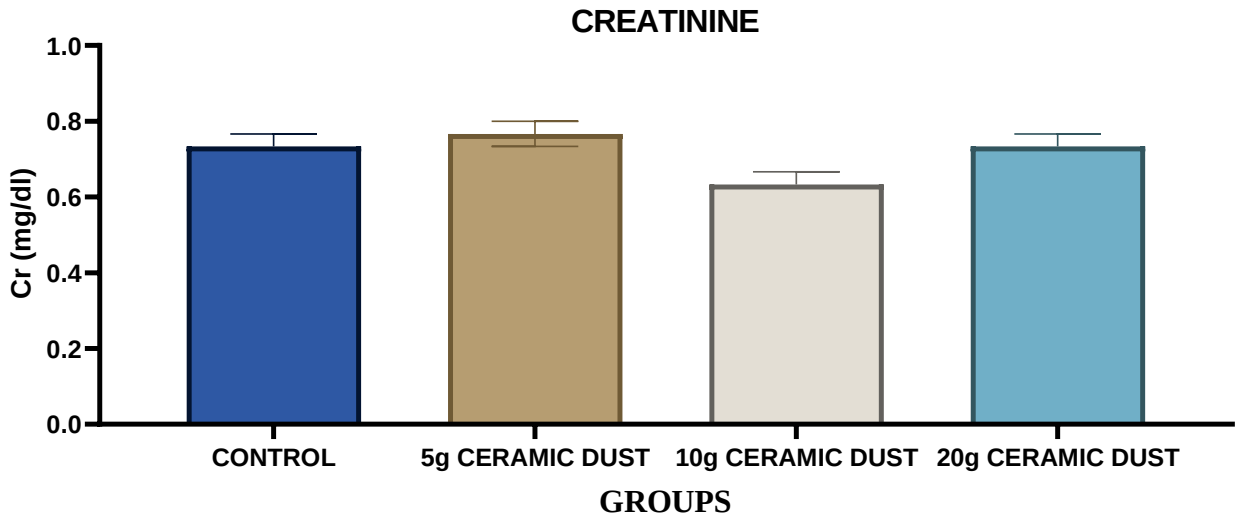

Figure 15 (Chart 12): Comparing Creatinine concentration across experimental groups. Source: Researcher's Fieldwork (2025).

Values are given as mean ± SEM. *P<0.05 compared with Control

There was no significant difference (p> 0.05) of creatinine concentration across the groups

4.2 RESULTS FROM HISTOPATHOLOGICAL ANALYSIS

The control group of the laryngeal sections in **Plates 1 & 2** below demonstrated with H&E staining at x100 and x400 magnifications respectively shows a normal composition of tissue.

Plates 3 & 4 below shows rats given 5g of ceramic dust via inhalation and demonstrated with H&E staining at x100 and x400 magnifications respectively shows a mild infiltrate of inflammatory cells.

Plates 5 & 6 below shows rats given 10g of ceramic dust via inhalation and demonstrated with H&E staining at x100 and x400 magnifications respectively shows a moderate infiltrate of inflammatory cells.

Plates 7 & 8 below shows rats given 20g of ceramic dust via inhalation and demonstrated with H&E staining at x100 and x400 magnifications respectively shows a severe infiltrate of inflammatory cells.

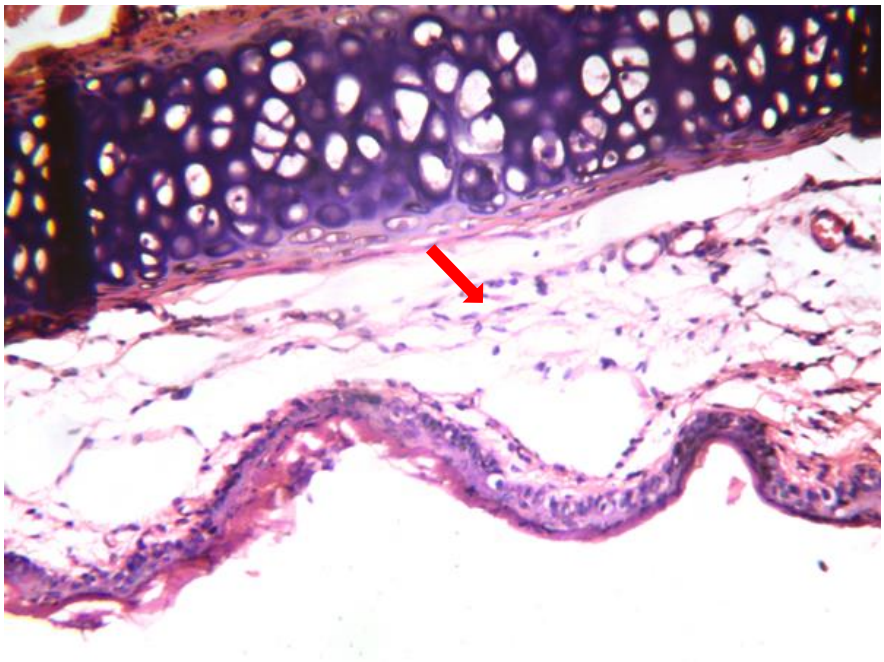

Figure 16 – (Plate 1): Rat larynx; Control; Composed of normal tissue: (H&E; x100). Source: Researcher's Fieldwork (2025).

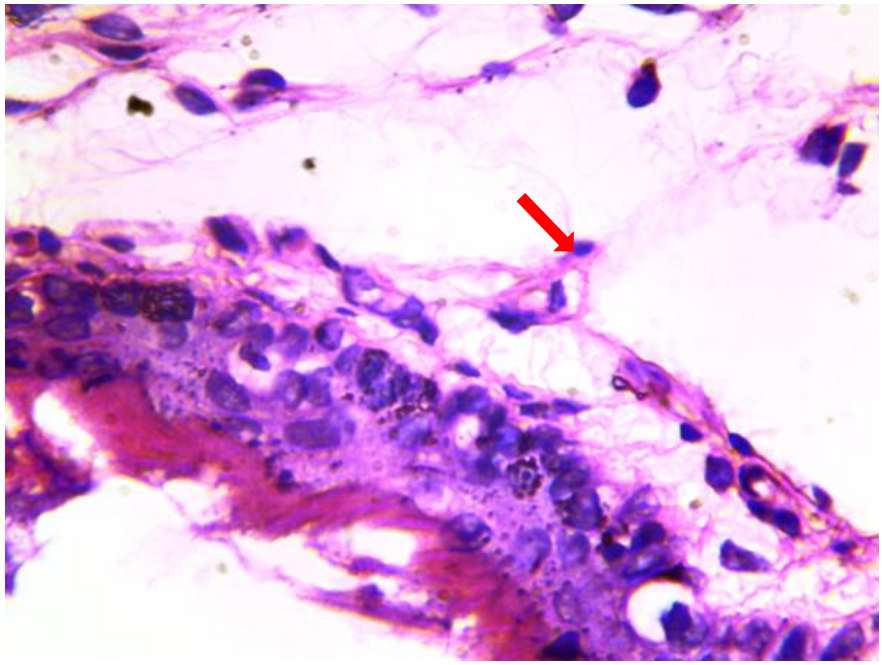

Figure 17 – (Plate 2): Rat larynx; Control; Composed of normal tissue: (H&E; x400). Source: Researcher's Fieldwork (2025).

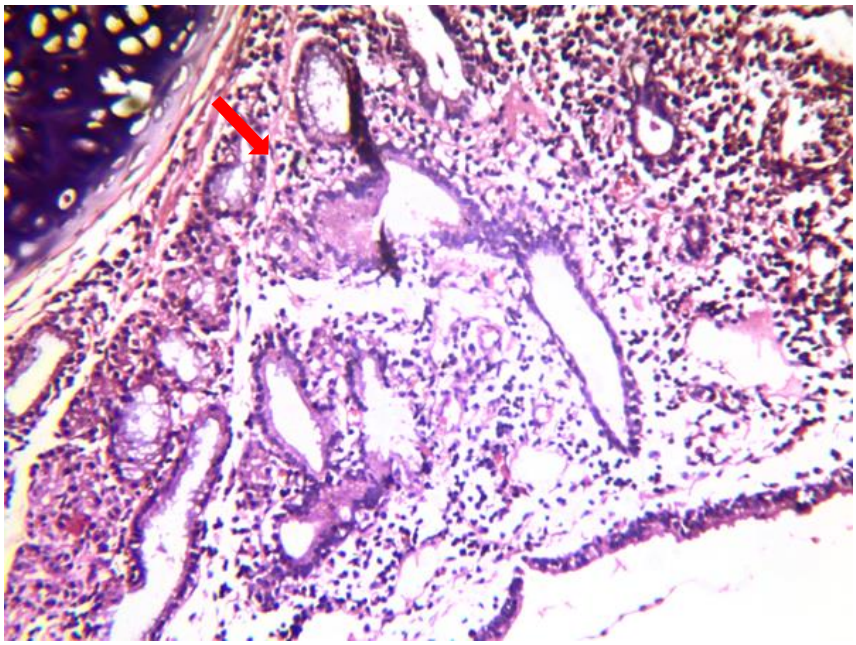

Figure 18 – (Plate 3): Photomicrographs of the Rat larynx given 5g of ceramic dust showing a mild infiltrate of inflammatory cells. (H&E; x100). Source: Researcher's Fieldwork (2025).

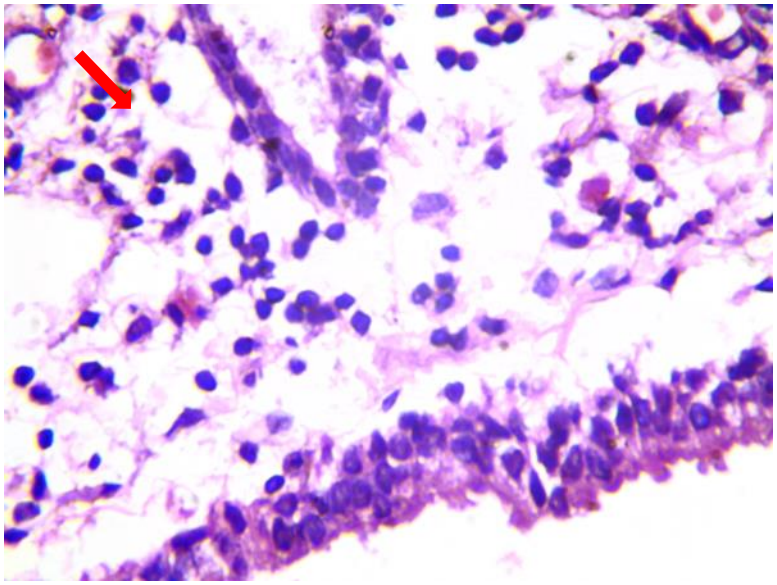

Figure 19 – (Plate 4): Photomicrographs of the Rat larynx given 5g of ceramic dust showing a mild infiltrate of inflammatory cells. (H&E; x400). Source: Researcher's Fieldwork (2025).

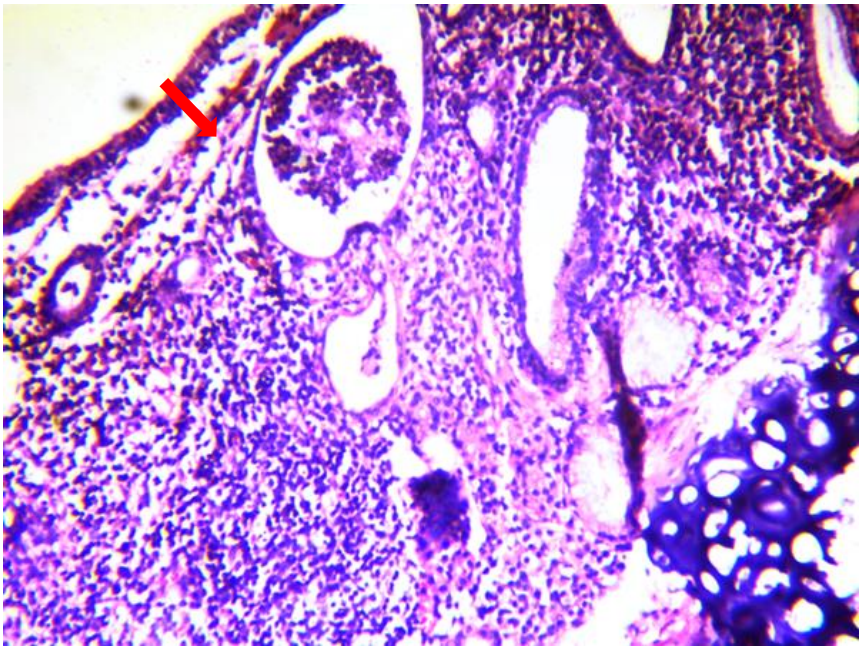

Figure 20 – (Plate 5): Photomicrographs of the Rat larynx given 10g of ceramic dust showing a moderate infiltrate of inflammatory cells. (H&E; x100). Source: Researcher's Fieldwork (2025).

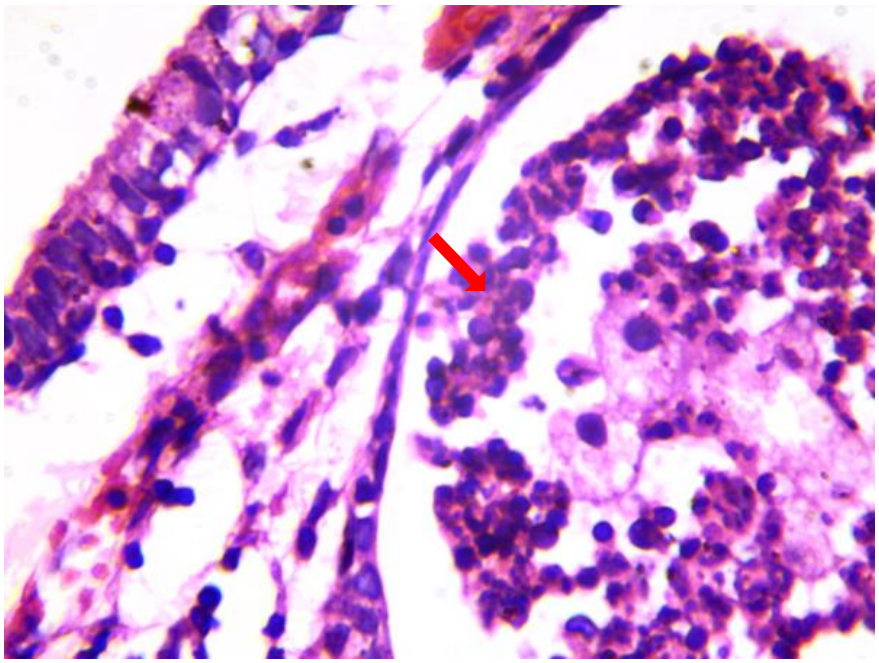

Figure 21 – (Plate 6): Photomicrographs of the Rat larynx given 10g of ceramic dust showing a moderate infiltrate of inflammatory cells. (H&E; x400). Source: Researcher's Fieldwork (2025).

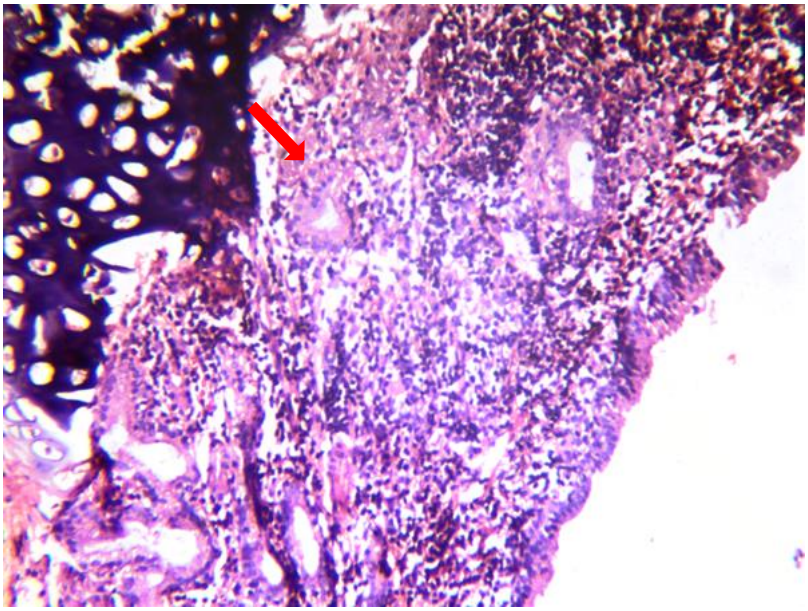

Figure 22 – (Plate 7): Photomicrographs of the Rat larynx given 20g of ceramic dust showing a severe infiltrate of inflammatory cells. (H&E; x100). Source: Researcher's Fieldwork (2025).

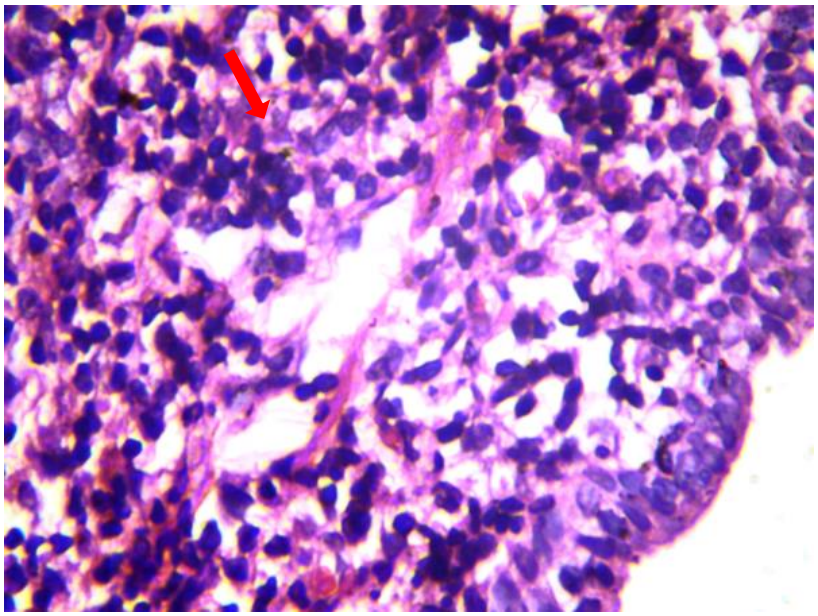

Figure 23 – (Plate 8): Photomicrographs of the Rat larynx given 20g of ceramic dust showing a severe infiltrate of inflammatory cells. (H&E; x400). Source: Researcher's Fieldwork (2025).

CHAPTER FIVE

5.1 DISCUSSION

5.1.1 BODY WEIGHT ANALYSIS

Contrary to expectations based on classic dust toxicity models, all exposure groups exhibited an increase in final body weight after 30 days, with the high-dose group (20g of ceramics dust) showing the most pronounced gain. This paradoxical weight gain challenges the conventional assumption that inhaled particulates induce metabolic stress and anorexia. Similar unexpected weight increases have been reported in rats exposed to low-to-moderate levels of particulate matter, possibly due to hormonal dysregulation or altered energy metabolism (Sun et al., 2005). One plausible mechanism involves dust-induced stimulation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels that promote fat deposition and weight gain despite inflammatory stress (Dhabhar, 2009). Additionally, the silica and metal oxides in ceramic dust may trigger compensatory hyperphagia or reduced physical activity due to respiratory discomfort, indirectly contributing to weight accrual. However, this finding contrasts with studies on cement dust, where weight loss was observed due to severe systemic inflammation (Mojiminiyi et al., 2008). The difference may stem from the lower inflammatory potency of the ceramic dust used in this study or its specific particle size distribution, which allowed deposition primarily in the upper airways rather than deep lung penetration. Further metabolic profiling (e.g., leptin, insulin) is needed to clarify this anomaly.

5.1.2 HAEMATOLOGICAL ANALYSIS

Hematological evaluation revealed a dose-dependent decrease in hemoglobin (Hb) and hematocrit (Hct) across exposure groups, with the high-dose (20 g of ceramics dust) group showing the most significant reduction. This suggests impaired oxygen-carrying capacity and possible microcytic anemia. The mechanism likely involves oxidative damage to erythrocytes by reactive oxygen species (ROS) generated from silica and metal oxides in the dust (Oberdörster et al., 2005). Chronic low-grade hemolysis or suppressed erythropoiesis due to inflammatory cytokines (e.g., IL-6, TNF-α) may also contribute, as seen in silica-exposed workers (Mojiminiyi et al., 2008).

Additionally, white blood cell (WBC) counts were significantly decreased in the high-dose group, contrasting with typical leukocytosis in particulate exposure. This suppression may reflect bone marrow toxicity or lymphocyte apoptosis induced by heavy metal components (e.g., aluminum, iron) in the dust (Smith et al., 2018). Alternatively, the 30-day duration may have allowed adaptation, with initial leukocytosis giving way to immune exhaustion. This finding aligns with reports of immunosuppression in chronic silica exposure (Hamilton et al., 2008) and underscores the complex, non-linear immune response to ceramic dust.

5.1.3 BIOCHEMICAL ANALYSIS

Biochemical markers indicated renal and acid-base disturbances. Urea levels were significantly elevated in a dose-dependent manner, suggesting impaired glomerular filtration or increased protein catabolism. This is consistent with nephrotoxic effects of silica particles, which can translocate systemically and deposit in renal tubules, causing tubular damage (Hasan et al., 2018). The decrease in serum bicarbonate points to metabolic acidosis, likely secondary to renal bicarbonate wasting or lactic acid accumulation from tissue hypoxia (due to reduced Hb/Hct). These changes highlight the systemic reach of inhaled ceramic dust, extending beyond the respiratory tract to affect kidney function and pH homeostasis.

5.1.4 HISTOLOGICAL ANALYSIS

The larynx, as the first major site of dust deposition, showed dose-dependent inflammatory infiltration dominated by modulated neutrophils. This neutrophilic response indicates acute-on-chronic irritation, with silica particles acting as mechanical and chemical irritants (Guthrie & Heaney, 1995). Unlike squamous metaplasia seen in longer-term silica studies (Osimitz et al., 2007), the 30-day exposure primarily elicited neutrophil-rich inflammation without epithelial remodeling, suggesting that metaplastic changes require prolonged exposure (>30 days). The absence of fibrosis or granuloma formation further supports a sub-chronic, reversible inflammatory phase. The modulated (likely activated) state of neutrophils; evidenced by degranulation and ROS release,may exacerbate local tissue damage and contribute to systemic effects via cytokine spillover. These laryngeal

changes mirror early histopathological findings in ceramic workers with voice hoarseness and throat irritation (Monfort et al., 2014).

5.2 CONCLUSION

The 30-day inhalation exposure to ceramic dust induced a complex, dose-dependent toxicological profile in Wistar rats. Despite an unexpected increase in body weight (possibly driven by stress-induced metabolic shifts) the study confirmed anemia (↓Hb, ↓Hct), immunosuppression (↓WBC), renal stress (↑urea), and acid-base imbalance (↓bicarbonate), alongside neutrophil-dominated laryngeal inflammation. These findings reveal that ceramic dust is not merely a local irritant but a systemic toxicant with hematological, renal, and immunological consequences. The results challenge simplistic models of dust toxicity and highlight the need to consider exposure duration, dose, and particle characteristics when assessing risk. While the laryngeal changes appear reversible at this stage, the systemic alterations raise concerns for chronic occupational exposure in ceramic industries.

5.3 RECOMMENDATIONS

1. Exposure duration for the research should be extended to determine whether weight gain persists, anemia progresses, or laryngeal metaplasia/fibrosis develops (Osimitz et al., 2007).
2. Implementation of regular hematological and renal function screening for ceramic workers, alongside voice and laryngeal exams (Monfort et al., 2014).
3. Further research should be carried out to assess ceramics dust effect on the other organs of the respiratory system and body.

4. Investigations on HPA axis activation (cortisol), cytokine profiles (IL-6, TNF-α), and bone marrow function should be carried out to explain weight gain and leukopenia (Hamilton et al., 2008)
5. Further research should be done with the use of SEM, XRD, and ICP-MS to fully characterize the ceramic dust (size, crystallinity, metal content) and correlate physicochemical properties with biological effects (Guthrie & Heaney, 1995)

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