ACTIVITIES OF *ADANSONIA DIGITATA* ON THE LEVEL OF MALONDIALDEHYDE IN THE LUNG TISSUE OF OVALBUMIN INDUCED ASTHMA IN GUINEA PIGS.

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FACULTY OF LIFE SCIENCE

UNIVERSITY OF BENIN

BENIN CITY.

APRIL, 2024

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A PROJECT WORK SUBMITTED TO THE DEPARTMENT OF BIOCHEMISTRY, FACULTY OF LIFE SCIENCES, UNIVERSITY OF BENIN, BENIN CITY, IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE AWARD OF BANCHELOR OF SCIENCES (BLSC) IN BIOCHEMISTRY.

APRIL, 2024

CERTIFICATION

This is to certify that this project research was carried out by **IGBEKHA OMOYEMEH REJOICE** with matriculation number **LSC1906518,** in partial fulfillment of the requirement for the award of a bachelor degree, Bsc (Hons) in biochemistry.

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DEDICATION

This seminar is dedicated to God almighty for his strength, wisdom and understanding, to my family for their love care and support throughout my undergraduate study.

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ABSTRACT

Asthma is an inflammatory disease of the lungs that causes oxidative stress (Almohawes and Alruhaimi, 2019). Asthma is characterized by airway inflammation, oxidative stress and increased Malondialdehyde level. It doesn't have a cure cannot be treated but it was discovered that a plamt known as *adansonia digitata* commonly referred to as African Baobab. The leaves of this tree serve as a primary source of food and traditional medicine for numerous African populations, and they are consumed either fresh or in a dried form (Besco *et al*., 2007). The plant have effects on asthma due to its renowned for its pharmacological properties including anti-inflammatory and anti-oxidant properties. This study aims at providing valuable insights into the therapeutic potential of *adansonia digitata* in managing asthma by its modulation of Malondialdehyde levels and contribute to supporting the use of natural products for management of asthma.

CHAPTER ONE

1.0 INTRODUCTION AND LITERATURE REVIEW

Adansonia digitata is a native African multi-purpose tree species that belongs to the Malvaceae family and it is commonly called African Baobab because it is indigenous to the African continent and the southern Arabian Peninsula. It is revered in Africa for its medicinal and nutritional value. They are mainly found in sub-Saharan Africa's dry, scorching savannas, where they dominate the landscape and make a visible watercourse nearby. It is also widely distributed across arid savannah regions in Madagascar, mainland Africa, the Arabian Peninsula, and Australia, in Nigeria it is mostly found in the northern part of the country. The leaves of this tree serve as a primary source of food, traditional medicine for numerous African populations, and they can be consumed either in a fresh or in a dried form (Besco *et al.,* 2007). Various parts of the baobab, including the fruit pulp, seeds, leaves, flowers, roots, and bark are edible offers protection, used as raw materials for many useful items and have been the subject of scientific research due to their beneficial properties (Rahula *et al.,* 2015). The baobab is also known as the monkey-bread tree.

Fig 1.1: A Baobab tree in Nigeria (Honours from the Kanem-Borno Empire, 2019).

The application of the tree is can both be medicinal and non-medicinal serving purposes such as the treatment of bronchial asthma, dermatitis, sickle cell anemia, diuretic and antidiabetic effects, relief from diarrhea and dysentery, as a laxative, and in treating hiccough in children.

Adansonia digitata for centuries have been used in traditional medicine to treat a wide range of ailments, including respiratory issues. Recently, scientific investigations have sought to validate the efficacy of *adansonia digitata* in addressing asthma, a chronic and debilitating respiratory disease characterized by inflammation, airway constriction and oxidative stress.

Additionally, it exhibits antioxidant properties, acts as an anti-inflammatory agent, serves as an antidote for poison, and demonstrates anti-trypanosome activity (Sundarambal *et al.,* 2015).

The term asthma" is derived from the Greek word "asthmaino," which means panting or gasping (Diamant *et al.,* 2007). Hippocrates, the ancient Greek physician first described asthma, which is an inflammatory lung disease characterized by bronchial hyperactivity, bronchospasm, mucus secretion, edema, and cellular infiltration. Asthma presents as recurrent symptoms that vary in frequency and intensity such as coughing, chest tightness, and shortness of breath. derived from the Greek word "asthmaino," means panting or gasping It is an inflammatory lung disease characterized It manifests as recurrent symptoms, including wheezing, coughing, , with varying frequency and severity. Symptoms could get worse during physical activity or at night.

Pathologically, asthma is characterized by hyperplasia of structural components, collagen deposition underneath the epithelium, and eosinophilic and lymphocytic inflammation in the bronchial mucosa. Immunoglobulin E bound to allergens interacts with mast cells in the mucosa of the airways to cause the early asthmatic response. This interaction results in the release of mediators that include histamine, TNF-α, chymase, prostaglandins, leukotrienes C4 and D4,

interleukins, prostaglandin D2, tryptase, and amphiregulin after degranulation (Brunton *et al*., 2008). As a result of smooth muscle contraction, vasodilation, increased vascular permeability, constriction of the smooth muscle in the bronchi, and increased mucus secretion, these mediators permeate into the mucosa of the airways, ultimately leading to lower airway obstruction (Galli *et al*., 2008). The recruitment, activation, and migration of eosinophils, lymphocytes, neutrophils, and macrophages into the lamina propria, epithelium, and airway lumen are the hallmarks of the late asthmatic response, which happens 6–9 hours after allergen exposure. Growth factors, chemokines, and cytokines—particularly interleukins 5, 9, and 13—produced during the early asthmatic response are responsible for this process, which further releases mediators and constricts the airways (Galli *et al.,* 2008). the cytokines are believed to draw in and activate eosinophils, trigger B lymphocytes to produce IgE, and directly assist bronchial epithelial cells in producing mucus (Brightling *et al.,*2002) and (Miller, 2001),. When an asthmatic responds late to allergens, eosinophils predominate and reach their peak 24 hours after the allergen challenge (Smith and Broadley, 2007; Toward and Broadley, 2004). Many biological products are released by eosinophils, such as growth factors, oxygen-free radicals, Th2 cytokines, eicosanoids, and toxic granule products. These compounds lead to enhanced vascular permeability, hyperresponsiveness of the airways, and smooth muscle contraction (Bousquet, *et al.,* 2000). Granule products such major basic protein, eosinophil cationic protein, and eosinophil-derived neurotoxic directly contribute to the development of airways hyperresponsiveness and epithelial shredding (Gleich *et al.,* (1993). Elastase, metalloproteases, and growth factors are among the other products that are involved in tissue remodeling and fibrosis (Lagente *et al*., 2005). It is assumed that a mix of environmental and genetic variables produce asthma. Among the environmental impacts are air pollution and allergies. Pharmaceuticals like beta blockers and

aspirin may also be contributing causes. A diagnosis is often made using the pattern of symptoms, the patient's response to therapy over time, and lung function tests using spirometry. Asthma is classified using peak expiratory flow rate, forced expiratory volume in one second (FEV1), and frequency of symptoms. In addition, it can be categorized as atopic or non-atopic, where atopy indicates a tendency to trigger a type 1 hypersensitivity reaction. Asthma is a significant global health concern, affecting over 340million people worldwide. Its prevalence is increasing, particularly in low-and middle-income countries, where access to effective treatment is limited. The current management of asthma, a relies heavily on conventional which can have adverse side effects and limited efficacy. Therefore, there is an urgent need to explore alternative or complementary therapeutic approaches, such as traditional herbal remedies like *adansonia digitata.*

LITERATURE REVIEW

1.1 Asthma

Asthma is one of the most prevalent chronic illnesses that irritates and narrows the airways is asthma, which affects both adults and children and is a serious non-communicable disease. Constant wheezing, chest tightness, shortness of breath, and coughing are symptoms of asthma; the coughing usually happens at night or in the early morning. People of all ages can suffer from asthma, however it typically first manifests in childhood. Asthma is classified as an allergic disease that is characterized by a series of allergic reactions that arise when an individual's immune system comes into touch with a specific trigger (Drazen *et al.,* 2020).

1.1.0 Types of Asthma

Atopic asthma

The onset of atopic asthma usually occurs in childhood or adolescence and is linked to certain triggers that cause wheezing. Atopic characteristics such rhinitis and eczema, as well as a family history of allergic illnesses, are frequently linked to atopic asthma (Diamant *et al.,* 2007 and Townshend *et al.,* 2007). the disease is typically brought on by an allergic reaction to particular allergens such house dust mites, grass and tree pollen, and pet dander (Ward *et al.,*2010). When an atopic person is exposed to an allergen, their B cells generate an excessive amount of Immunoglobulin E (IgE). As a result of IgE's binding to inflammatory cells, inflammatory mediators that promote bronchoconstriction and airway inflammation are released (Holgate and Douglass 2010). People are predisposed to elevated IgE levels, the development of asthma and airway hyper responsiveness due to atmospheric pollution and maternal smoking during pregnancy (Ward *et al.,* 2010).

Non-atopic asthma

Since not all cases of asthma are caused by atopic, variables unrelated to the atopic illness also matter (Anderson, 2005). Non-atopic asthma is the term used to describe the condition that some adults develop in response to viral respiratory infections. With few evident triggers other than illness, this kind of asthma can be more persistent. IgE is not a factor in non-atopic asthma (Ward *et al.,* 2010).

1.1.1 Pathophysiology of Asthma

Asthmas pathophysiology involves multiple factors and very complex. Asthma is a chronic inflammatory disease of the conducting zone of the airways, mainly the bronchi and bronchioles, which makes the smooth muscles around it contract more easily. This contributes to wheezing symptoms and episodes of airway constriction along with other variables. Research has highlighted the role of chronic airway inflammation, triggered by various allergens and irritants (Holgate *et al.,* 2015). With or without therapy, the narrowing may usually be reversed. Every now and then, the airways themselves change. The clinical symptoms of asthma are most likely explained by the combination of airway remodeling and inflammation. We are starting to understand the mechanisms by which complicated genetic processes and external environmental signals combine to promote the inflammatory process that is characteristic of asthma (Desmond et al 2010). Common changes in the airways include an increase in eosinophil levels and a thickening of the lamina reticularis. Over time, the number of mucous glands may increase and the smooth muscle of the airways may enlarge. T lymphocytes, macrophages, and neutrophils are additional cell types implicated. There may be additional immune system components involved as well, like histamine, chemokines, and cytokines. Eosinophil and mast cells are examples of inflammatory cells that release cytokines and mediators that aid in airway remodeling and bronchoconstriction (Lambrecht and Hammad, 2015).

1.1.2 Pathology

Mucus hyper secretion

The expansion of mucous glands and the multiplication of mucus-secreting cells in the airways are the results of asthma. Viscid mucous plugs that might obstruct the airways arise as a result of increased mucus secretion (Ward *et al.,* 2010).

Epithelial damage

The layer of cells lining the airways, known as the epithelium, can become damaged and peel away in asthma. The loss of enzymes that break down inflammatory mediators, the exposure of sensory nerves, which may result in reflex neural effects on the airway, and the loss of barrier function, which may allow allergens to penetrate, are some of the ways that epithelial shedding can contribute to airway hyper-responsiveness (Barnes, 1996). The sub epithelial layer may also undergo changes, such as collagen laying down (Rees, 2010).

Oedema

The walls of the airway can have dilated and leaky capillaries. Increased airway secretions, decreased mucociliary clearance, and oedema are the effects of microvascular leakage, and these conditions may exacerbate hyperresponsiveness and airway constriction (Barnes, 1996). Despite the clinical complexity of asthma, the pathologist currently recognizes only one disease mechanism. In deadly asthma, tenacious plugs of mucus, cells, and exudate obstruct the airways. The epithelium lining the airways is fragile, and the reticular basement membrane is becoming thicker. The thickening of the airway wall is caused by bronchial artery dilatation, congestion and oedema, a strong inflammatory cell infiltration, an increase in the bulk of bronchial smooth muscle, and mucus-secreting glands. These adjustments can significantly alter the impact of airway stiffness on airflow and contribute to the classic indicators of inflammation, including as redness, heat, discomfort, swelling, and mucus secretion. (P.K, 1992).

Bronchospasm

Another symptom of asthma is bronchospasm. It explains how the bronchial smooth muscle contracts sharply to restrict the airways. Remodeling of airways Changes in structural cells and tissues in the lower respiratory tract can result in remodelling of the airway and irreversible fibrotic damage when asthma is poorly managed or undertreated (Rees, 2010).I n the past, asthma was believed to be a completely treatable condition. Reversible airflow blockage has also been called into question recently. Chronic airway modifications have been shown to occur, and these changes may be a factor in the gradual blockage of airflow (Lee *et al.,*2002).

The intricate process of remodeling an airway encompasses all of its constituent tissues, ranging from the adventitia to the epithelium. These models are being extensively characterised and are providing valuable insights into mechanisms that are likely to be quite relevant to human asthma. (ED *et al.,*2007).

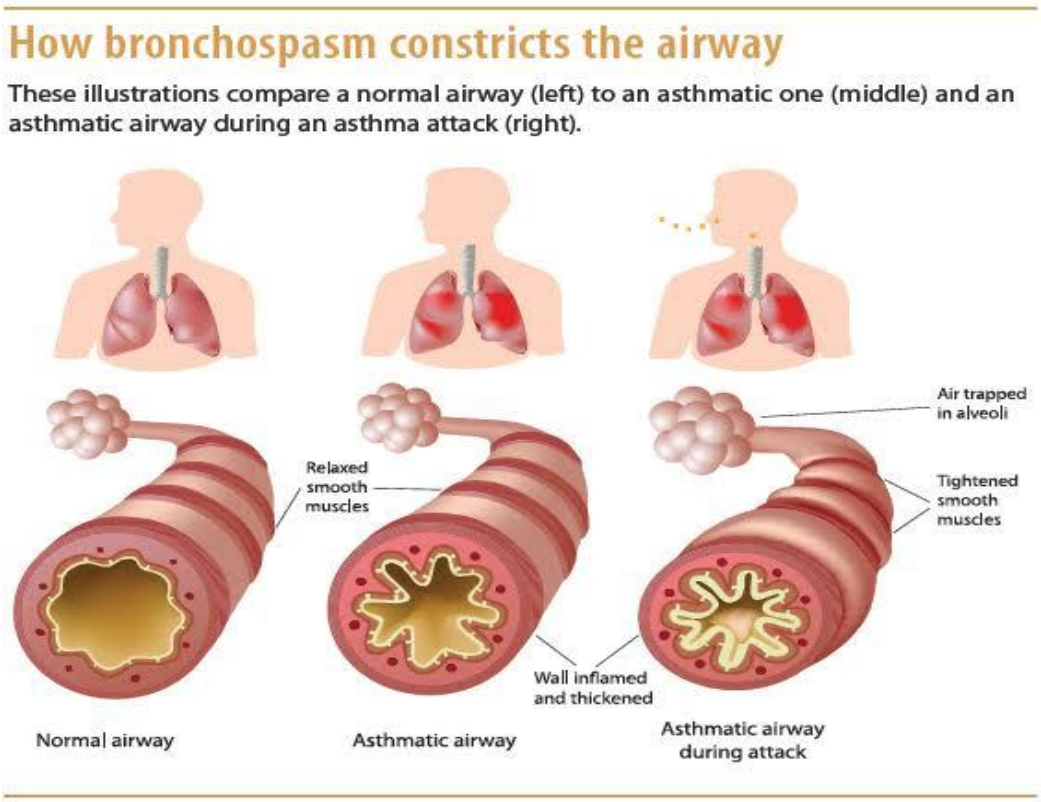

Fig 1.2: An Illustration of how bronchospasm constricts the airway (Shari, 2015).

All of the alterations have the potential to modify the physiology of the airways, leading to an increase in inflammation, hyper responsiveness, and airway constriction. Animal models have shown effective in simulating structural alterations such as epithelial metaplasia, airway fibrosis, and airway smooth muscle hyperplasia. Our treatment strategy for asthma has therefore concentrated on symptomatic management by lowering airway inflammation and bronchospasm. (Jack *et al.,* 1999) It is presently.

1.1.3 Classification of asthma

Since asthma is a complex ailment that arises from a variety of factors, there are different variations of the illness that are categorized based on severity and age. The same allergens that produce symptoms in the respiratory system in adults and children include molds, mildew, airborne pollution, and cigarette smoke.

Occupational asthma

Asthma can be caused by a variety of occupational situations which have been documented. In developed nations, occupational asthma has emerged as the most prevalent lung condition associated with the workplace. The precise number of adult cases of asthma that have been newly diagnosed as a result of occupational exposure is unknown, though. In the US, occupational factors may account for as much as 15% of asthma cases. When "on the job," breathing in dust, fumes, gasses, or other potentially hazardous materials might result in occupational asthma. (Baur *et al.,*2012). Symptoms frequently worsen on the days or nights you work, get better on your days off, and then get worse again when you go back to work.You might have been well before and these are your first symptoms of asthma, or you might have had

asthma as a youngster and it has come back. If you already have asthma, some things you may be exposed to at work may make it worse. Within each industry, occupational asthma rates differ. High concentrations of irritants from the petroleum or chemical industries, such as ammonia, sulfur dioxide, or hydrochloric acid, can cause occupational asthma. You may experience wheezing and other asthma symptoms right away if you are exposed to any of these compounds in high amounts. When exposed to these irritants, workers who already have asthma or another respiratory condition may also notice an aggravation in their symptoms. A lot of occupational asthma cases are influenced by allergies. This kind of asthma usually doesn't appear until months or years after a worker is exposed to a chemical at work. It takes time for your body's immune system to produce allergy antibodies or other protective reactions in response to a specific chemical. Workers in the washing powder sector, for instance, could become allergic to the Bacillus subtilize enzymes, and bakers might experience symptoms of occupational asthma and allergy reactions after handling different types of flour or baking enzymes. Allergies to animal proteins can occur in fisherman, veterinarians, and lab animal workers. When combining powdered drugs or breathing in particles of protein from latex gloves, healthcare workers run the risk of developing asthma. Similarly, occupational asthma has been reported in roughly 5% of workers who use powdered natural rubber latex gloves or who work with laboratory animals. Workers who are repeatedly exposed to small chemical molecules in the air, as in the case of paint hardeners or those employed in the plastic and resin industries, may also develop occupational asthma. For instance, isocyanates are chemicals that are utilized extensively in a variety of sectors, such as foam, rubber, and plastic manufacture, spray painting, and insulation installation. Up to 10% of workers who are exposed to these substances may develop

occupational asthma. There is variation in the amount of time you are exposed to a drug before it aggravates your asthma. It may take months or even years for symptoms to appear.

However, asthma can be brought on by exposure to a high dose. Lastly, breathing in some aerosolized compounds can directly cause your body's natural molecules, such acetylcholine or histamine, to accumulate in your lungs, which can result in asthma. For instance, the use of pesticides in agriculture might result in an accumulation of acetylcholine, which tightens and contracts the muscles in your airways. (Buar *et al.,* 2012).

Adult-onset asthma

Adults with asthma may have had the condition since childhood, it may have been a recurrence of childhood asthma (remembered or not), or it may be a genuine adult-onset asthma without symptoms in the past (Butland *et al*, 2007). environmental factors, particularly those related to one's job, may contribute to adult-onset asthma with or without allergen sensitivity (Kogevinas *et al.,* 2007),. Adult asthma is more common in women, even though it can also develop in response to certain medication treatments (such as β-blockers, non-steroidal anti-inflammatory medicines), the use of hormone replacement therapy, and occupational exposure to sensitizing chemicals or irritants.

Childhood Asthma

Children are more likely to experience severe episodes of an intermittent form of asthma. While some kids may experience daily symptoms, reactivity to allergenic substances is a typical trait among kids with asthma. Children with asthma suffer significant consequences from secondhand cigarette smoke.

1.1.4 Causes of asthma

The intricate and little-understood relationships between environmental and genetic factors that cause asthma. These have an impact on the condition's severity as well as its responsiveness to therapy (Choundhry *et al.,* 2007). It is believed that both altering environmental factors and shifting epigenetics—heritable traits unrelated to DNA sequence—are contributing to an increase in the prevalence of asthma (Tan, 2015). It is more likely for people who develop asthma before the age of 12 to have genetic predispositions, and for those who develop asthma beyond that age, it is more likely to be environmental.

Environmental Factors

Allergens, air pollution, and other environmental contaminants have all been connected to the onset and exacerbation of asthma. Inhaled allergens are important environmental factors in the pathogenesis of asthma and most likely its persistence. Moreover, the interaction between host factors (genetics) and environmental stimuli can result in the development of airway inflammation, altered pulmonary physiology, and asthma symptoms in the susceptible host. Certain substances are known to cause asthma attacks in people who come into contact with them. The following are some common asthma triggers: formaldehyde, glutaraldehyde, anhydrides, glues, dyes, metal working fluids, oil mists, latex, pesticides, welding fumes, metal or wood dusts, spraying isocyanate paint during auto repairs, and molds. Smoking raises the risk of experiencing symptoms similar to asthma during and after childbirth. Both the onset and severity of asthma have been related to low air quality caused by outside factors like ozone or traffic pollution. More than half of child cases in the US occur in areas where the air quality is.

Poor air quality is more common in minority and low-income communities (Brooks N and Sethi, 1997). Exposure to indoor volatile organic compounds, such as formaldehyde, has been linked to good outcomes for asthma symptoms (Mcgwin *et al.,* 2010). Certain PVC items include phthalates, which have been connected to asthma in both adults and children. While there is evidence linking pesticide exposure to the onset of asthma, a cause-and-effect relationship has not yet been shown (Mamane *et al.,* 2015). A meta-analysis indicates that gas stoves contribute significantly to asthma cases. Asthma and indoor allergen exposure are related. Typical indoor allergens include dust mites, cockroaches, mildew, and animal dander (fur or feathers) (Arshad SH, 2010).

Studies have revealed that attempts to lower the number of dust mites had little effect on allergy sufferers' symptoms (Calderon et al., 2015). There is little data to show that reducing mold growth through structural renovations can alleviate symptoms of adult asthma. When contracted in early childhood, certain viral respiratory infections, such as rhinovirus and respiratory syncytial virus, may raise the chance of developing asthma. However, several other infections may reduce the risk (Murray JF, 2010).

Hypothesis of hygiene

According to the hygiene hypothesis, less childhood exposures to non-pathogenic bacteria and viruses are unintentionally and directly fueling the global increase in asthma cases. It has been proposed that modern society's smaller families and improved hygiene play a role in the decreased exposure to bacteria and viruses. While late-life exposure to bacterial endotoxins may cause bronchoconstriction, early childhood exposure may prevent the development of asthma (Rao, 2011).

The lower incidence of asthma in households with pets and on farms lends credence to the hygiene idea. Early antibiotic use has been linked to the development of asthma. Additionally, due to the absence of a beneficial bacterial colonization the baby would have acquired from going through the birth canal, cesarean deliveries are associated with an increased risk of asthma (estimated at 20–80%). Wealth and asthma are interrelated, which could be connected to the hygiene hypothesis because those with lower incomes are often more likely to be exposed to germs and viruses (Von, 2004).

Genetic Factors

Family history has an impact on asthma risk, and multiple genes may be involved (Edward G and Douglas KS 2010). There is a 25% probability that the other identical twin will have the illness if the first has it. By the end of 2005, asthma had been associated with 25 different genes in at least six different populations. These genes included GSTM1, IL10, CTLA-4, SPINK5, LTC4S, IL4R, and ADAM33. Many of these genes are involved in immunological function or in regulating inflammation. Not even within this set of genes supported by several, well-repeated research, have the results been consistent across all groups studied. In one study of genetic associations, over 100 genes were connected to asthma in 2006. Asthma can only appear when specific genetic variants coexist with specific environmental exposures. Exposure to endotoxin, a bacterial byproduct, and a specific single nucleotide polymorphism in the CD14 area are two instances. Numerous environmental variables, such as cigarette smoke, dogs, and farms, might expose people to endotoxins. Thus, a person's genetic composition and endotoxin exposure both affect their chance of acquiring asthma (Martinez, 2007).

Medical issues

Atopic is a combination of allergic rhinitis, asthma, and atopic eczema (Rapini *et al.,* 2007) . The greatest risk factor for developing asthma is a history of atopic disease, with eczema and hay fever sufferers having a significantly greater incidence of the illness. Asthma has been associated with eosinophilic granulomatosis with polyangiitis (formerly known as Churg-Strauss syndrome), vasculitis, and an autoimmune disease (Jennette *et al.,*2013). In certain individuals, symptoms of asthma may coexist with certain types of urticaria. Asthma risk is correlated with obesity, and both have increased recently (Holguin *et al.,* 2010).

Several factors could be involved, such as a condition that promotes inflammation caused by adipose tissue and a build-up of fat that impairs respiratory function. For those who are vulnerable, beta blocker drugs like propranolol can aggravate asthma. However, in patients with mild or moderate illness, cardioselective beta-blockers seem safe (Salpeter *et al.,* 2002). Aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), and angiotensin-converting enzyme inhibitors are additional medications that may pose issues for asthmatics. Pregnant women who use acid suppressing medications (H2 blockers and proton pump inhibitors) run the risk of their unborn child developing asthma (Lia *et al.,* 2018).

Exacerbation

Some people experience weeks or months of stable asthma followed abruptly by an acute asthma attack. Individuals differ in how they respond to different stimuli. Many triggering stimuli can cause significant exacerbations in most people. Dust, mold, cockroach allergens, and animal dander—particularly hair from cats and dogs—can all cause asthma attacks at home. Women and children frequently get acute assaults as a result of perfumes. Upper respiratory tract infections caused by bacteria or viruses can exacerbate the illness (Baxi, 2010).

Psychological stress may exacerbate symptoms because it is believed to modify the immune system, which in turn heightens the inflammatory response of the airways to allergens and irritants. When school-age children return to school in the fall, their asthma flare-ups reach their height. This could be the result of several things, such as inadequate treatment compliance, elevated exposure to allergens and viruses, and changed immunological tolerance. Although seasonal omalizumab medication administered four to six weeks before to the start of the school year may be expensive, it may help minimize fall asthma exacerbations despite the paucity of data supporting potential strategies (Pike *et al.,* 2018).

1.1.5 Prevention

The effectiveness of therapies aimed at preventing the onset of asthma is supported by weak data. The World Health Organization recommends reducing exposure to risk factors like air pollution, tobacco smoke, chemical irritants like perfume, and the incidence of lower respiratory disorders (Henneberger, 2007). Other promising strategies include limiting fetal smoking exposure, breastfeeding, and increasing exposure to childcare or large families; nevertheless, there is not enough evidence to recommend any of them for this indication. Restrictions on nutrition during pregnancy or nursing do not appear to alleviate asthma in children; hence, this advice is not recommended (Prescott *et al.,* 2005).

Although the evidence is still in its early stages, some study has indicated that antioxidants, a Mediterranean diet, and omega-3 supplementation may help avoid crises. Reducing or eliminating chemicals from the workplace that are known to aggravate sensitive people may be beneficial. It is unknown how yearly influenza shots affect the likelihood of exacerbations. The World Health Organization does, however, recommend vaccination. Prohibitions against smoking help to lessen asthma attacks.

1.1.6 Management of asthma

While there is no treatment for asthma, symptoms can typically be managed (Ripoll, 2011). The best ways to treat asthma are to identify triggers, such as cigarette smoke, dogs, or other allergens, and stay away from them. If avoiding triggers is not enough, using medication is recommended. Substantial evidence indicates that the costs of uncontrolled asthma (hospitalization, emergency admission, days off work and school) can be significantly reduced by interventions which improve disease management. Among other factors, the severity of the illness and the frequency of symptoms influence the choice of pharmaceuticals. Certain asthma therapies are categorized using two categories: fast-acting and long-acting. Particularly for moderate-to-severe cases, asthma represents a substantial financial burden. Fifty percent of asthma costs are related to indirect costs. It is reasonable to believe that medications (37%) and general practitioner care (16%) included in the direct costs are associated with managing controlled asthma; the remaining costs, primarily hospital expenses, are associated with treating failed control. Consequently, it would seem that improperly managed illness accounts for almost three quarters of the whole expense based on these estimates. Preventive care and expense savings According to international guidelines, preventative therapy need to be started earlier in the course of treating asthma.

The medications on the list below have demonstrated potential in easing the symptoms of asthma; nevertheless, their "real world" effectiveness is limited because, even with treatment, only around half of asthmatics worldwide have their symptoms under optimal control. "Refractory" asthma refers to people whose asthma is not well controlled by the recommended dosages of their medications; "difficult to treat" asthma refers to people whose asthma is not well controlled by the recommended dosages of their medications

because they are unable to take them because of financial constraints, ineffective inhaler technique, or a desire to avoid the side effects of corticosteroids. The difference between "difficult to treat" and "refractory" asthma patients is actually unachievable for those who have never been prescribed the necessary dosages of asthma medications.

Another issue is that most people with asthma were intentionally excluded from asthma efficacy trials, which provide the guidelines for medication treatment. For example, smoking decreases the efficiency of inhaled corticosteroids, which are the mainstay of asthma therapies, therefore smokers who are otherwise qualified are never included in asthma efficacy therapy trials. It is recommended to utilize bronchodilators to temporarily relieve symptoms. For people who have intermittent attacks, no additional medicine is needed. If a minor chronic disease is present (more than two attacks per week), low-dose inhaled corticosteroids are recommended; other treatments include oral leukotriene antagonists or mast cell stabilizers.

Inhaled corticosteroids are administered at higher dosages to individuals who have attacks on a daily basis. Oral corticosteroids are added to these therapy in cases of moderate or severe exacerbations. Individuals with asthma are more prone to worry, stress, and depression (Kew *et al.,* 2016). This is associated with poorer asthma control. Cognitive behavioral therapy may help asthmatics with their anxiety levels, asthma control, and quality of life (Kew *et al.,* 2016). Using a documented action plan and increasing public awareness of asthma have been found to be crucial elements of asthma management. It is probably helpful to give instructional sessions that contain knowledge unique to an individual's culture.

Further investigation is required to ascertain whether enhancing asthma awareness and readiness among school personnel and families through home-based and school-based interventions leads to sustained enhancements in the safety of children suffering from asthma. School-based asthma

self-management programs may lower hospital admissions and ER visits by enhancing student understanding of asthma, its triggers, and the value of routine practitioner review. Additionally, these therapies can lessen the number of days that kids encounter asthma symptoms and might result in minor gains in the quality of life that comes with having asthma

To find out if a customized asthma action plan is essential and useful, or if shared-decision-making is beneficial for managing adults with asthma, more research is required (Gatheral *et al.,* 2017). Pulse oximeters are used by some asthmatics to track their own blood oxygen levels during an asthma attack. There is, however, no proof that it is used in these situations.

1.2. *Adansonia digitata*

1.2.0. Scientific classification

Kingdom: *Plantae*

Phylum: Tracheophyta

Class: Magnoliopsida

Order: Malvales

Family: Malvaceae

Genus: Adansonia

Species: digitata

Botanical name: *Adansonia digitata*

The baobab tree, or *Adansonia digitata,* is a unique and well-known plant that is native to Africa and a member of the malvaceae family. Its unique appearance, extraordinary lifespan, and varied benefits to human cultures and ecosystems have earned it the title "the tree of life." The presence

of flavonoids, phytosterols, amino acids, fatty acids, vitamins, and minerals was discovered through phytochemical analysis.

Baobab has been widely utilized in traditional medicine since ancient times, and some plant sections exhibit intriguing anti-oxidant and anti-inflammatory qualities (Emmy *et al*., 2010).

Fig 3: Leaves of Baobab tree (Alamy, 2021).

Fig 1.4 :**Fruit of the Baobab tree monkey bread (Aurore V., 2022**)

Fig 1.5: Seed of the Baobab tree (ShutterStock).

1.2.1 Botanical characteristic

Originally from Africa, namely Sub-Saharan Africa, the baobab tree, *Adansonia digitata*, is an amazing plant. Its enormous, bloated trunk, which may reach diameters of up to ten meters, is what makes it so remarkable. Baobabs are distinguished by their smooth bark, which frequently has a light grayish tint. One of the baobab's most significant traits is its unusual development pattern. When many other trees lose their leaves in the dry season, the baobab retains its greenery, giving it the moniker "the upside-down tree." The intricate leaves on its branches typically feature five to seven leaflets each.

The massive, white flowers of baobabs typically open at night. Sometimes referred to as "monkey bread" or "baobab fruit," the fruit of the tree is a large, rectangular capsule with seeds embedded in a pulpy substance.

1.2.2 Ecological Significance

In the environments where it is found, the baobab tree is essential. Its capacity to retain water in its trunk throughout the dry season makes it an invaluable resource for the surrounding community and wildlife.

Throughout many dry and semi-arid habitats, the baobab tree is considered a keystone plant. Because of its enormous trunk's ability to hold water, it can withstand extreme weather and provide vital water during dry seasons for wildlife (Silva *et al.,* 2019).

Baobab trees offer a range of species vital habitat. Baobab tree hollows serve as both breeding grounds and refuge for birds, bats, and insects. A wide range of herbivorous animals feed on the fruits and leaves.

In environments that would otherwise be nutrient-deficient, the organic components provided by the falling baobab leaves aid to improve soil fertility.

1.2.3 Cultural and societal importance

Cultural Symbolism: The baobab tree has a deep cultural significance in Africa. In many African societies, it is a symbol of resilience, longevity, and continuity. It is usually observed close to places of worship and public meeting places.

Conservation and Tourism:. Numerous African nations have taken steps to preserve and advance these well-known trees as a result of realizing the baobab's significance to their cultures and ecosystems. Baobab trees are becoming popular tourist attractions that support local economies.

1.2.4 Nutritional Properties

Renowned for its exceptionally nutrient-dense fruit pulp, leaves, and seeds is *Adansonia digitata*. Its potential as a source of polysaccharides, important nutrients like calcium and vitamin C, and antioxidants has been the subject of recent studies (Chadare *et al.,* 2019). Furthermore, because of its high fiber content, baobab fruit extract has drawn interest for its potential as a functional food ingredient (Ndiaye *et al.,* 2018).

1.2.5 Traditional Uses

For innumerable centuries, the baobab tree has provided food. Native American tribes use the tree's components for materials, food, and medicine. Vitamin C and antioxidant-rich baobab fruit pulp is utilized as a nutritional supplement and in traditional medicine (Chadare *et al.,* 2019). It has been traditionally used as an analgesic, immunostimulant, pesticidal, and anti-inflammatory due to the presence of saponins, sterols, and triterpenes. Several parts of the baobab tree, such as the fruit, leaves, and bark, have been used in traditional African medicine to treat a range of illnesses. The fruit pulp is well-known for its potential health advantages, which include immune system and digestive support Furthermore, the fruit pulp was subjected to phytochemical

screening and the antioxidant assay. The fruit pulp's characteristics revealed that it included significant amounts of ascorbic acid, minerals, and other health advantages, in addition to a strong antioxidant capacity. Furthermore, fruit pulp can be used to generate commercial products and as a dietary supplement, especially in rural regions. For these reasons, consumption is recommended (Olakunle and Fausat, 2012).

1.2.6 Phytochemistry and Pharmacological Uses

Astringent, demulcent, diaphoretic, rheumatic pain reliever, inflammatory ulcer healer, haemoptysis, diarrhoea-dysentery, anti-cancer, anti-diabetic, and diuretic are some of its uses. The plant parts are used to cure a variety of illnesses, including microbial infections, malaria, and diarrhea. Because of its vitamin C level, which is seven to ten times higher than that of oranges, it is said to be an excellent antioxidant. Baobab possesses a wide range of biological qualities, including antibacterial, antiviral, antioxidant, and anti-inflammatory effects (Kamatou *et al.* ,2011).

The health benefits of baobab have been attributed to its bioactive compounds, namely phenols, flavonoids, proanthocyanins, tannins, catechins, and carotenoids. Baobab fruit is also an important source of vitamin C and micronutrients, including zinc, potassium, magnesium, iron, calcium, and protein, which may reduce nutritional deficiencies (Maria *et al.,*2023). The nutritional and therapeutic qualities of baobab fruit make it valuable. While the health benefits of baobab pulp are widely recognized, there is a dearth of research connecting the pulp's nutraceutical qualities to its capacity to remove reactive species (both RNS and ROS).

Methods: Angolan baobab pulp preparations were analyzed for their nutritional composition and antioxidant capacity. Therefore, the assessment of in vitro scavenging ability against the most

physiologically significant reactive oxygen species (ROS) and reactive nitrogen species (RNS) was the main focus of the study for the first time.

Results: The fruit pulp from Angola had significant levels of total dietary fiber (52%), and ash (8.0%).

Fruit pulp's vitamin E concentration was first documented. Antioxidant activity and bioactive substances were measured using green solvents. The largest concentrations of flavonoids (768.7 mg/100 g) and phenolics (1573.0 mg/100 g) were found in hydroalcoholic extracts. As a result, hydroalcoholic extracts demonstrated increased antioxidant activity as well as a greater ability to scavenge ROS ($O_2^{\bullet-}$, H_2O_2, HOCl, ROO•) and RNS (NO, $ONOO^-$), with the highest levels of activity observed for NO and $ONOO^-$. Conclusion: The nutritional value and beneficial antioxidant effects of Angolan baobab fruit were reported for the first time, both as a functional food and as a nutraceutical additive (Ana et al 2024).

1.2.7 Chemical Constituents

The fruit and leaves include carbohydrates, lipids, proteins, and vitamins in addition to arabinose, galactose, glucose, rhamnose, galacturonic, and glucuronic acids. Seed oil contains oleic and myristic acids. Palmitic and stearic acids, along with stem bark, contain quercitain, 7-o-xylopyranoside, and ß-sitosterol. . In this study, extracts from baobab fruit pulp collected from Offa (north central Nigeria) were investigated using a number of parameters including proximate composition:(Moisture–10.2%, Ash–7.67%, Fat–0.4%, Crude fibre–5.7%, Crude protein–2.16%, Carbohydrate–73.87%), metabolizable energy–307.6 kcal/g, ascorbic acid content–264.3 mg/100g, pH-3.33, titratable acidity-28.17%, alcohol insoluble solid-47.5%, total soluble solid-

62%, filtratable solid-55.65%. Elemental analysis of the fruit pulp was also carried out to determine the mineral contents.

1.3 Malondialdehyde

The primary form of malondialdehyde (MDA) is the enol form (Nair *et al.,* 2008). Whereas the trans isomer is more common in water, the cis isomer is preferred in organic solvents. The hydrolysis of 1,1,3,3_ tetramethoxypropane, a chemical that is readily obtained from the market, produces malondialdehyde, a highly reactive substance that is rarely seen in pure form in lab settings (Nair *et al.,* 2018).

The chemical substance having the nominal formula CH2(CHO)2 is malondialdehyde (MDA). Malondialdehyde is a highly reactive liquid that is colorless and exists as the enol (Nair *et al.,* 2008). It is a naturally occurring substance that is readily depronated to yield enolate sodium. Since MDA is produced by reactive oxygen species (ROS), it is tested in vitro as an indicator of oxidative stress.

Biochemistry

When polyunsaturated lipids are broken down by reactive oxygen species, malondialdehyde is created (Pryor *et al.,* 1975). This material is a reactive aldehyde that causes toxic stress in cells and forms covalent protein adducts called advance lipoxidation end products (ALE), which are comparable to advanced glycation end products (AGE) (Farmer *et al.,* 2007). The synthesis of this aldehyde functions as a biomarker to gauge the level of oxidative stress experienced by various organs (Del Rio *et al.,* 2005).

DNA adducts are created when deoxyadenosine and MDA interact; the most notable of these is the mutagenic M,G adduct (Marnett, 1999). 2-amino pyrimidines are created when malondialdehyde and the guanidine group in the arginine residue interact. Human Aldiah

Aldehyde dehydrogenase is capable of oxidizing malondialdehyde. Malondialdehyde and other thiobarbituric reactive compounds (Tbars) condense with two equivalents of thiobarbituric acid to produce a brilliant red derivative that may be detected spectrophotometrically. One more focused and alternate reagent is 1-methyl-2-phenylindole. It has been found that heated edible oils, such as sunflower and palm oils, contain malondialdehyde. It might be mutagenic and is reactive. One study found that the corneas of patients with bullous keratopathy and keratoconus had higher concentrations of malonediadehyde.

Patients with osteoarthritis also have MDA in the tissues that make up their joints. One of the end products of polyunsaturated fatty acid peroxidation in the cells is malondialdehyde (MDA). Overproduction of MDA is caused by a rise in free radicals. According to Stefan *et al* (2004), malondialdehyde level is widely used as a measure of oxidative stress and the antioxidant status in cancer patients.

CHAPTER TWO

2.0 MATERIALS AND METHODOLOGY

2.1 Materials

Beakers

Conical flask

Glass stirrer

Volumetric flask

Measuring cylinder

Spatula

Hand gloves

Syringe and needles

Sensitive weighing balance

Ceramic mortar and pestle

Dissecting sets

EDTA sample containers

2.1.0 Chemicals and Reagents

Methanol, Saline water, Ovalbumin (obtained from the department of Biochemistry, University of Benin), Aluminum hydroxide (obtained from the department of Biochemistry, University of Benin), Tween-80 solution (obtained from the department of Biochemistry), Distilled water (obtained from the department of Biochemistry, university of Benin), Chloroform.

2.2 Source of Plant

The plant was obtained from the northern part of Nigeria, Kaduna. It was verified by Dr. Akinobosun of Plant Biology and Biotechnology.

2.3 Method

2.3.0 Preparation of the plant extract

Plant leaves from *Adansonia digitata* were dried and ground. It was then put in a soxhlet extractor to get crude extract. The crude extract was then oven dried.

Fig 2.1: Prepared *Adansonia digitata* extract (Department of Biochemistry, university of Benin).

Materials

Dried and ground *Adansonia digitata* plant material, ethanol (95-99% pure), distilled water.

Equipment: Soxhlet apparatus consists of the Soxhlet extractor, condenser and round-bottom flask], round-bottom flask (250mL), heating mantle or hot plate with magnetic stirrer, filter paper, cotton wool, rotary evaporator (optional), weighing balance, glassware (beakers, graduated cylinders, etc), rubber stoppers and tubing.

2.3.1 Procedure

An exact weight was determined for 500 mg of dried and crushed *Adansonia digitata* plant material. The weight was recorded. Plant material was placed in the thimble, and the round-bottom flask was placed under the extractor to assemble the Soxhlet apparatus. After that, the device was connected to a condenser that was connected to a cooling water source. Enough ethanol (which served as the solvent) was added to the round-bottom flask. This solvent was continuously pumped through the plant material during the extraction process. Either the heating mantle or a hot plate with a magnetic stirrer was used to heat the ethanol solution within the round-bottom flask. To guarantee that the ethanol was boiled but not dried out, safety measures were taken. The ethanol flowed up the Soxhlet extractor as it vaporized, drawing the compounds out of the plant material and then condensing back into the round-bottom flask. The extracted compounds were progressively collected in the round-bottom flask as a result of numerous extraction cycles lasting many hours. Once there were enough extraction cycles (usually 6–8 hours), the heat source was turned off and the system was allowed to cool. After unplugging the Soxhlet apparatus, the round-bottomed container holding the crude extract was carefully taken out. After the crude extract was concentrated using a rotary evaporator to remove the majority of

the ethanol, the concentrated extract was obtained. After that, the extract underwent additional purification using techniques including filtration and column chromatography. In order to remove any remaining solvent, the filtered extract was lastly dried using a desiccator or other suitable methods. After that, the extract was refrigerated to preserve its freshness.

2.4 Animals

20 Guinea pigs were bought from the Southern part of Nigeria, Ambrose Ali University, Ekpoma, Edo state.

The 20 Guinea pigs weighing between 200g and 420g of both sexes, were obtained. Three weeks were spent housing the animals in the animal house for their acclimatization before the experiment began. The animals were kept in a room with a relative humidity of 44-56% and on a photo schedule of 12 hours of light and 12 hours of darkness. Water was available at all times, along with a regular diet of pellets and newly cut guinea grass. The guinea pigs were kept in experimental cages at the Animal House of the Department of Biochemistry, University of Benin.

Four groups were formed from the 200g- 420g Guinea pigs of both sexes. There were five animals in each group.

Fig 2.2: Guinea Pig (department of Biochemistry, University of Benin).

2.5 Experiment

Measurement of body weight

Each Guinea pig was weighed before acclimatization to obtain their body weight. The average weight of the animals was 360g. The measurement of the body weight of the Guinea pigs was done to determine the dosage of ovalbumin and the treatment to be provided.

Sensitization was carried out in days:

Day 1- The four groups of guinea pigs were sensitized to ovalbumin on the first day of the experiment by intraperitoneally administering 2mg of ovalbumin and 200mg of aluminium hydroxide diluted in 10mL of normal saline solution. A 0.5mL sample of the resulting solution was given to each animal in these groups. After that, they were left but continually fed.

Day 7- They were re-sensitized to ovalbumin six days after the initial sensitization, by intraperitoneally administering 1mg of ovalbumin and 100mg of aluminium hydroxide diluted in 5mL of normal saline solution. 0.25 mL of the solution was given to each animal.

Day 14- The animals in the groups were weighed on the fourteenth day of the experiment. The animals in each of the four groups were exposed to a 1% ovalbumin aerosol for 20 seconds while housed in an airtight exposure chamber (a closed polyacrylic box) coupled to an ultrasonic nebulizer. The time that the Guinea pigs remained in nebulization was defined as Inhalation time. Weighing and dissolving 20g of ovalbumin (OVA) in 200mL of regular saline solution produced the ovalbumin aerosol. The ovalbumin was first weighed and combined with 20mL of regular saline solution. 180mL of regular saline was added once the ovalbumin had completely dissolved. The animals in the first two groups received a dosage of 200 mg/kg body weight of the extract after the animals in the four groups were challenged.

However, the dried *Adansonia digitata* extract was weighed and diluted in 19mL of distilled water and 1mL of Tween-80 solution prior to being given to the animals. The extract's solubility was improved by using the Tween-80 solution. The animals in the first two groups received the extract for seven (7) days following the initial exposure at the normal dosage of 200 mg/kg body weight. Additionally, the fifth group of animals received an ovalbumin sensitization injection consisting of 2 mg of ovalbumin and 200 mg of dissolved aluminium hydroxide in 10 mL of regular saline solution.

Subsequently, the animals were divided into four groups based on body weights between 200 and 420g.

Day 21- On day 21, aerosol exposure of the animals was performed using aerosol containing 1% OVA (w/v) for 20sec.

Day 28- Exposure of the 4 groups to ovalbumin aerosol in an airtight chamber until hypoxia occurred. The guinea pigs were sacrificed after 3 hours of exposure.

2.6 Principle

This study aimed to simulate the mechanisms underlying bronchial asthma. Ovalbumin sensitization caused the animals to produce IgE antibodies, which are necessary for allergic or inflammatory responses. However, the guinea pigs' airways had an allergic reaction as a result of the IgE antibodies cross-linking to the allergen following their exposure to the ovalbumin aerosol. The inflammatory cells, such as mast cells and eosinophils, produced inflammatory mediators, including histamine, leukotrienes, prostaglandins, cytokines, and others, as a result of this allergic reaction. These mediators of inflammation wind up obstructing the airways, which causes bronchospasm in the guinea pigs. For every guinea pig, the pre-convulsion time (PCT)

was recorded. This is the interval between the aerosol exposure and the start of dyspnea that precedes the emergence of convulsion. The guinea pigs were given three hours to recuperate following the second exposure. The following formula was used to determine how much protection the treatment would provide:

Percentage protection = $(1-T_1/T_2)\times 100\%$

where: T_1= PCT before the administration of the extract

T_2= PCT after administration of the extract.

2.7 Sacrification of animals

Materials used: Hand gloves, Syringes and needles, Buffer, EDTA bottles, Lithium Heparin bottles, Dissecting set.

Apparatus for bench work

The apparatus used for bench in this study includes beakers, digital weighing balance, conical flask, nose mask, hand gloves, stop watch, pipette.

Experimental design

On the final week of treatment, the animals were sacrificed. Ovalbumin aerosol saturated chamber was used on both exposed and control animals. The lungs were dissected, connective tissues removed, and preserved at 20°C in a container of normal saline solution until needed for biochemical analysis. Cervical dislocation and carotid hemorrhage were used to sacrifice the animals.

The lungs were dissected out and placed in a dish with a buffer.

Staining

To identify one animal from every other in each group, various body parts of each animal were stained with picric acid.

2.8 Determination of Malondialdehyde (MDA)

Malondialdehyde was determined using the thiobarbituric acid assay (Buege and Aust, 1978)

Principle

Malondialdehyde which is a product of lipid peroxidation react with thiobabituric acid (TBA) to give a red species.

2.9 Procedure

A volume of plasma (1.0ml) was added to 2.0ml of TCA-TBA-HCL and mixed thoroughly. The solution was heated for 15mins in a boiling water bath. After cooling, the flocculent precipitate was removed by centrifuged at 1000g for 10min. The absorbance was determined using the formula;

$$\text{MDA (mol/mg protein)} = \frac{A \times V \times 100}{M \times V \times Y}$$

A= Absorbance

V= Total volume of reaction mixture

M= Molar extinction coefficient

V= volume of the sample

Y= mg protein.

CHAPTER THREE

3.0 RESULTS AND ANALYSIS

3.1 Pre-convulsive time of Guinea pigs treated with *adansonia digitata*

Group 1	Before treatment (seconds)	After treatment (seconds)	% protection
200mg/kg wt	20	52	61.53
200mg/kg wt	20	49	59.18
200mg/kg wt	20	55	63.63
200mg/kg wt	20	57	64.19
200mg/kg wt	20	56	64.28

Group 2	Before treatment (seconds)	After treatment (seconds)	% protection
200mg/kg wt	20	60	66.66
200mg/kg wt	20	47	57.44
200mg/kg wt	20	70	71.42
200mg/kg wt	20	65	69.23
200mg/kg wt	20	78	74.35

3.2 Effects of *Adansonia digitata* extracts on level of malondialdehyde in lung tissues.

3.2 Malondialdehyde concentration in lung tissues.

Group 1 (Sensitized, exposed and treated).

S/N	MALONDIALDEHYDE CONC. (mol/g)
1	0.4426821
2	0.4259897
3	0.3338101
4	0.5891935
5	0.451599

Group 2 (Sensitized, exposed and treated)

S/N	MALONDIALDEHYDE CONC. (mol/g)
1	0.3940974
2	0.4040255
3	0.3960648
4	0.4285291
5	0.4576749

Group 3 (Sensitized and exposed)

S/N	MALONDIALDEHYDE CONC. (mol/g)
1	0.3157038
2	0.4670396
3	0.5716007
4	0.3833907
5	0.4344337

Group 4 (Control)

S/N	MALONDIALDEHYDE CONC. (mol/g)
1	0.5747803
2	0.3707477
3	0.4596647
4	0.4683975667
5	0.4683975667

Table 3.3: Effects of *adansonia digitata* extract level of malondialdehyde in lung tissues

GROUPS	MALONDIALDEHYDE CONC. (mol/g)
1	**0.448±0.041**
2	**0.416±0.012**
3	**0.434±0.043**
4	**0.468±0.032**

The result above is expressed as Mean ± S.E.M. Generally, various alphabets when used in the table indicates significant difference while similar alphabets indicates no significant difference ($p>0.05$) between the values.

For the result expressed in the table above, the use of similar alphabets indicates no significant difference. So there is no significant difference ($p> 0.05$) in the level of malondialdehyde between Group 4 which is the control group and Group 1that is treated treated group. There is also no significant difference in the level of malondialdehyde in Group 2 and Group 1. Significant difference was also not observed in Group 1 and Group 3 when compared with the control (Group 4).

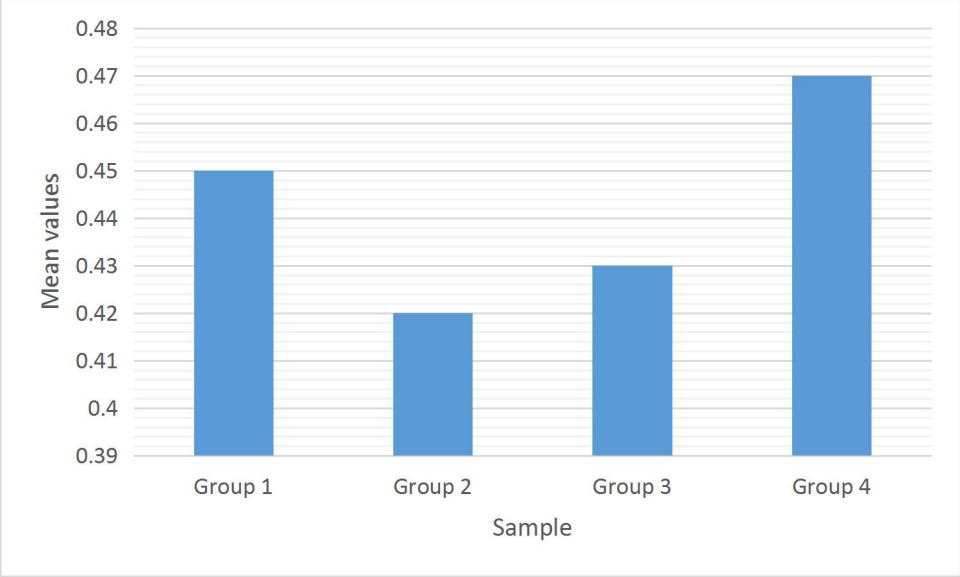

Fig 2.3 : Graphical representation showing the effects of *Adansonia digitata* extract on level of malondialdehyde in lung tissues.

CHAPTER FOUR

4.0 DISCUSSION

The results presented in Table 3.2 show the Malondialdehyde concentration in the lung tissues of guinea pigs from different experimental groups. Malondialdehyde (MDA) is a compound that is derived from the peroxidation of polyunsaturated fatty acids. It has been used a biomarker to measure oxidative stress (Raffaele *etal*., 2023) in various biological samples in patients who are affected by a wide range of diseases. It plays the role as a marker of oxidative stress in allergy-related diseases such as asthma.

Asthma is an allergic disease and as such, involves series of allergic reaction that occurs when a particular trigger comes in contact with the person's immune system. It is also associated with decreased antioxidant defenses such as superoxide dismutase, catalase and glutathione. In the developed world, attention has recently focused on asthma because of its rapidly increasing prevalence, affecting up to one or four urban children (Lily, 2005). The prevalence ranges about 7 million cases, in over 25 million affected people in the U.SA.

Asthma is often under-diagnosed and under-treated, particularly in low- and middle-income countries. People with under-treated asthma can suffer sleep disturbance, tiredness during the day, and poor concentration. Asthma sufferers and their families may miss school and work, with financial impact on the family and wider community. If symptoms are severe, people with

asthma may need to receive emergency health care and they may be admitted to hospital for treatment and monitoring. In the most severe cases, asthma can lead to death (Mannino et al., 2002).

The study involved four groups of guinea pigs: Group 1 (sensitized, exposed and treated), Group 2 (sensitized, exposed and treated), Group 3 (sensitized only) and Group 4 (control). These groups were meant to investigate the effects of Ovalbumin sensitization, induction and exposure on the level of malondialdehyde in the lung tissue, as well as the potential effect of the plant *adansonia digitata.*crude extract.

The effect of a plant of the Malvaceae family known as *Adansonia digitata* extracts on asthma was tested using animal model (guinea pigs) and sensitized with asthma by accessing the levels of malondialdehyde in the lung tissue of these animals.

Group 2 (sensitized, exposed and treated) showed lower malondialdehde activity compared to the control which is Group 4. The mean malondialdehyde concentration for Group 2 (0.416±0.012) was lower compared to that of the control group (0.468±0.032).

Group 2 (sensitized, exposed and treated) compared to Group 3 (only sensitized) showed lower Malondialdehyde activity in the lung tissue. The mean malondialdehyde concentration for Group 2(0.416±0.012) and that of Group 3 (0.434±0.043).

For the malondialdehyde, the treatment with *Adansonia digitata* showed positive effect. The level of significance observable in those sensitized with ovalbumin, and administered extract (group 1), and also with those that was sensitized only (group 3) the treatment was negative.

It is important to note that the results do not provide information about the specific treatments or interventions involving *adansonia digitata* crude extract in the different groups. However, the observed differences in the malondialdehyde activity between the control group and the ovalbumin-sensitized and exposed groups (Group 1 and Group 2) highlight the potential impact of ovalbumin induced asthma on the antioxidant defense system in the lung tissue.

Further analysisand interpretation of these results may require additional information about the the experimental design, the specific treatments or interventions involving *adansonia digitata* crude extract and other relevant parameters such as inflammatory markers, airway hyperresponsiveness or histological changes in the lung tissue.

Overall these findings contribute to the u nderstanding of the role of oxidative stress and antioxidant defense mechanism in the pathogenesis of asthma and provide a basis for further investigations into the potential therapeutic effects of natural products like *adansonia digitata* crude extract in modulating antioxidant enzyme activities and mitigating oxidative stress in asthma.

4.1 CONCLUSION

The results obtained in this project showed no significant effect in the level of malondialdehyde in the lung tissue of the guinea pigs that was sensitized, exposed and treated with *Adansonia digitata* extract ($P>0.05$), as well as that of those that was sensitized only. With the study of the research made by other Scientists previously, there can be significant effect and also no significant effect on the malondialdehyde levels in ovalbumin sensitized guinea pigs but treated with other plant extracts or drugs other than the ones used in this project work. However,

environmental conditions, experimental procedures and the type of treatment used may affect the results obtained in any experiment carried out. It is also possible that the percentage protection showed by the Guinea pigs was not due to the anti-oxidant properties of the plant *adansonia digitata*, because there was no significant difference ($P>0.05$).

Overall these findings contribute to the understanding of the role of oxidative stress and antioxidant defense mechanism in the pathogenesis of asthma and provide a basis for further investigations into the potential therapeutic effects of natural products like *adansonia digitata* crude extract in modulating antioxidant enzyme activities and mitigating oxidative stress in asthma.

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APPENDIX

Weight of guinea pigs

Group 1 (sensitized, exposed and treated).

1	381.8
2	375.9
3	409.5
4	363.7
5	395.0

Group 2 (sensitized, exposed and treated).

1	381.6
2	352.0
3	316.5
4	384.4
5	300.0

Group 3 (sensitized only).

1	345.0
2	303.7
3	289.7
4	321.8
5	315.0

Group 4 (control)

1	418.9
2	361.5

3	414.8
4	398.1
5	398.1

Results of malondialdehyde (MDA) concentration.

	MDA Abs	V mL	Mv	**MDA mol/g Prot**
TISSUE 1	0.221	3	1.56	
T2	0.198	3	1.56	**0.42599**
T3	0.204	3	1.56	**0.33381**
T4	0.284	3	1.56	**0.589194**
T5	0.241	3	1.56	**0.451599**
T6	0.173	3	1.56	**0.394097**
T7	0.233	3	1.56	**0.404025**
T8	0.15	3	1.56	**0.396065**
T9	0.225	3	1.56	**0.428529**
T10	0.26	3	1.56	**0.457675**
T11	0.125	3	1.56	**0.315704**
T12	0.201	3	1.56	**0.46704**
T13	0.246	3	1.56	**0.571601**
T14	0.132	3	1.56	**0.383391**
T15	0.282	3	1.56	**0.57478**
T16	0.217	3	1.56	**0.370748**
T17	0.182	3	1.56	**0.459665**

Notes

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Descriptives

VAR00002

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum
					Lower Bound	Upper Bound	
1.00	5	.4480	.09176	.04104	.3341	.5619	.33
2.00	5	.4158	.02671	.01194	.3826	.4490	.39
3.00	5	.4340	.09568	.04279	.3152	.5528	.32
4.00	5	.4678	.07232	.03234	.3780	.5576	.37
Total	20	.4414	.07303	.01633	.4072	.4756	.32

Descriptives

VAR00002

	Maximum
1.00	.59
2.00	.46
3.00	.57
4.00	.57
Total	.59

ANOVA

VAR00002

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.007	3	.002	.411	.747
Within Groups	.094	16	.006		
Total	.101	19			

Post Hoc Tests

Multiple Comparisons

Dependent Variable: VAR00002

Tukey HSD

(I) VAR00001	(J) VAR00001	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
1.00	2.00	.03220	.04850	.909	-.1065	.1709
	3.00	.01400	.04850	.991	-.1247	.1527
	4.00	-.01980	.04850	.976	-.1585	.1189
2.00	1.00	-.03220	.04850	.909	-.1709	.1065

	3.00	-.01820	.04850	.981	-.1569	.1205
	4.00	-.05200	.04850	.711	-.1907	.0867
3.00	1.00	-.01400	.04850	.991	-.1527	.1247
	2.00	.01820	.04850	.981	-.1205	.1569
	4.00	-.03380	.04850	.897	-.1725	.1049
4.00	1.00	.01980	.04850	.976	-.1189	.1585
	2.00	.05200	.04850	.711	-.0867	.1907
	3.00	.03380	.04850	.897	-.1049	.1725

Homogeneous Subsets

VAR00002

Tukey HSD[a]

VAR00001	N	Subset for alpha = 0.05 1
2.00	5	.4158
3.00	5	.4340
1.00	5	.4480
4.00	5	.4678

Sig.		.711

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 5.000.