Pharmacological Evaluation of *Madhuca Longifolia* Against Iso-Induced Myocardial Infarction in Experimental Rats

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***Abstract*—Myocardial infarction (MI) is one of the leading causes of morbidity and mortality worldwide and is primarily associated with oxidative stress, inflammation, and myocardial necrosis. The present study was designed to evaluate the cardioprotective potential of *Madhuca longifolia* against isoproterenol (ISO)-induced myocardial infarction in experimental rats. *Madhuca longifolia*, a medicinal plant belonging to the family *Sapotaceae*, is known for its rich phytochemical composition and diverse pharmacological activities, including antioxidant and anti-inflammatory properties. The cardioprotective activity of *Madhuca longifolia* was assessed using the isoproterenol-induced myocardial infarction model in Wistar rats. Experimental animals were divided into different groups, including normal control, ISO control, standard treatment, and plant extract-treated groups. Myocardial infarction was induced by subcutaneous administration of isoproterenol. Following treatment, various biochemical parameters such as creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), lipid profile, malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) were evaluated. Histopathological examination of cardiac tissues was also performed to assess myocardial damage and protective effects of the plant extract. Treatment with *Madhuca longifolia* significantly reduced the elevated levels of cardiac marker enzymes and lipid peroxidation while restoring endogenous antioxidant enzyme levels when compared with the isoproterenol-treated group. Histopathological studies demonstrated marked protection against myocardial necrosis, inflammatory cell infiltration, and structural damage in the treated animals. The observed cardioprotective effect may be attributed to the presence of flavonoids, phenolic compounds, triterpenoids, and other phytoconstituents possessing potent antioxidant and free radical scavenging activities. The findings of the present study suggest that *Madhuca longifolia* possesses significant cardioprotective activity against isoproterenol-induced myocardial infarction and may serve as a promising natural therapeutic agent for the prevention and management of cardiovascular diseases. Further studies are warranted to isolate the active constituents and elucidate their precise mechanisms of action.**

***Index Terms*—Madhuca longifolia, Myocardial Infarction, Isoproterenol, Cardioprotective Activity, Oxidative Stress, Antioxidant Activity, Cardiac Biomarkers, Histopathology, Experimental Rats, Medicinal Plants.**

I. INTRODUCTION

Cardiovascular diseases (CVDs) represent a major global health burden and are the leading cause of death worldwide. According to global health reports, millions of people die each year due to various cardiovascular disorders, including coronary artery disease, hypertension, stroke, heart failure, and myocardial infarction (Roth *et al.,* 2020). Among these conditions, myocardial infarction (MI), commonly referred to as a heart attack, is one of the most severe and life-threatening cardiovascular events. It occurs when the blood supply to a portion of the heart muscle is significantly reduced or completely blocked, resulting in prolonged ischemia and subsequent necrosis of myocardial tissue. The loss of viable cardiac muscle adversely affects cardiac function and may lead to serious complications such as arrhythmias, heart failure,

cardiogenic shock, and sudden cardiac death (Libby *et al.,* 2013).

The pathogenesis of myocardial infarction involves multiple mechanisms including oxidative stress, inflammation, calcium overload, mitochondrial dysfunction, apoptosis, and endothelial injury. During myocardial ischemia, the oxygen demand of cardiac tissue exceeds the available oxygen supply, resulting in metabolic disturbances and cellular damage. Reperfusion of ischemic tissue, although essential for restoring blood flow, can further aggravate myocardial injury through the excessive generation of reactive oxygen species (ROS). These free radicals initiate lipid peroxidation, protein oxidation, DNA damage, and inflammatory responses, ultimately contributing to the progression of myocardial damage (Zhou *et al.,* 2015).

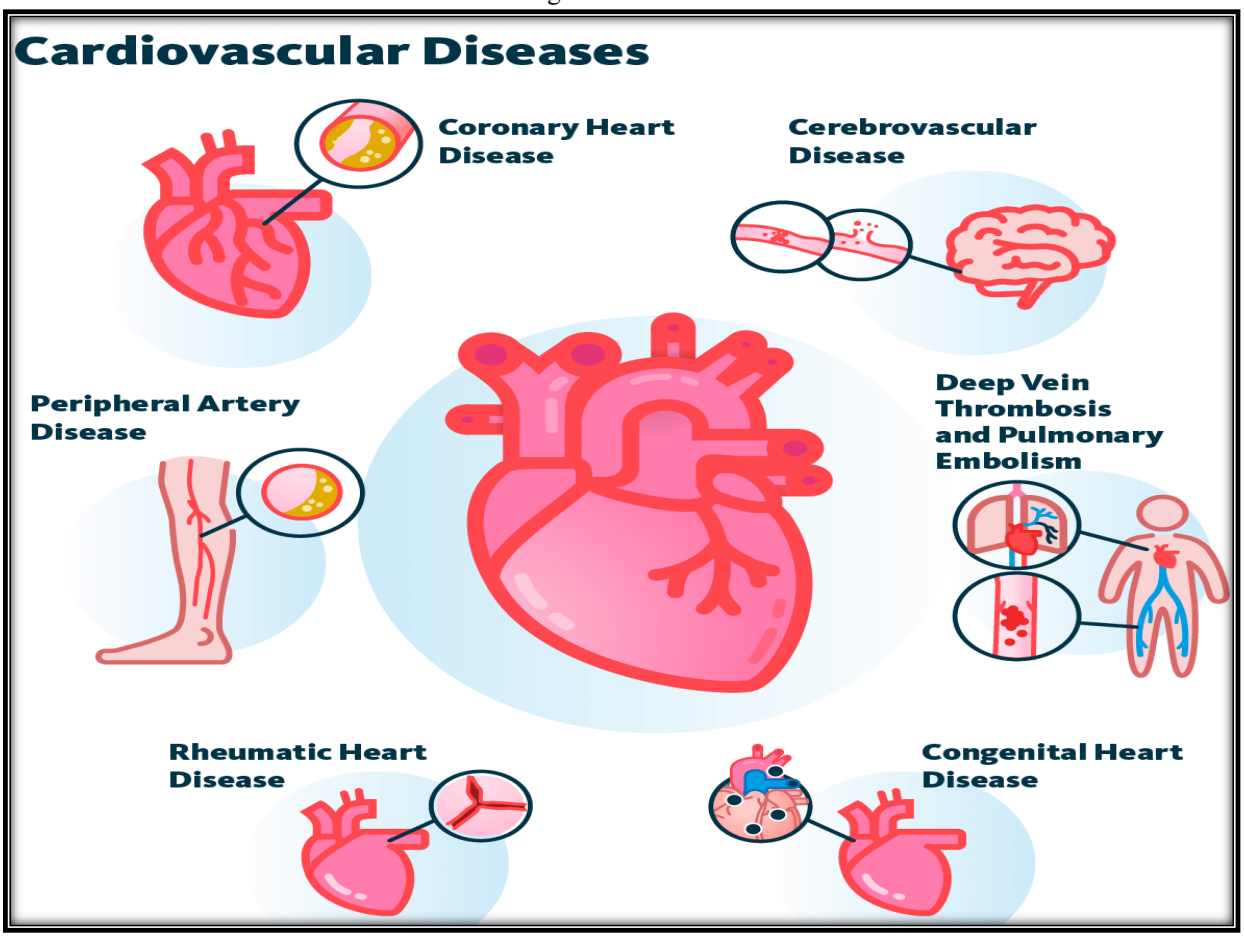

Figure 1: Cardiovascular diseases

Several risk factors are associated with the development of myocardial infarction, including hypertension, diabetes mellitus, hyperlipidemia, obesity, smoking, excessive alcohol consumption, stress, genetic predisposition, and sedentary lifestyle. The increasing prevalence of these risk factors has significantly contributed to the growing incidence of cardiovascular diseases worldwide (Flora *et al.,* 2019). Despite considerable advances in pharmacological and interventional therapies, cardiovascular morbidity and mortality remain high, emphasizing the need for novel preventive and therapeutic strategies. Experimental models of myocardial infarction are widely employed to investigate the pathophysiological mechanisms involved in cardiac injury and to evaluate potential cardioprotective agents. Among these models, isoproterenol-induced myocardial infarction in rats is one of the most commonly used and well-established experimental models. Isoproterenol is a synthetic β-adrenergic receptor agonist that induces severe stress on the myocardium by increasing heart rate,

myocardial contractility, and oxygen consumption. Administration of high doses of isoproterenol leads to myocardial necrosis, oxidative stress, inflammatory infiltration, and biochemical alterations that closely resemble those observed in human myocardial infarction. Therefore, this model is extensively used for screening cardioprotective compounds and evaluating their therapeutic efficacy (Bader Eddin *et al.,* 2025).

II.OVERVIEW OF MYOCARDIAL INFARCTION

Myocardial infarction (MI), commonly known as a heart attack, is a pathological condition characterized by the irreversible necrosis of cardiac muscle resulting from prolonged ischemia. It occurs when coronary blood flow is significantly reduced or completely obstructed, leading to inadequate oxygen and nutrient supply to the myocardium. The most common cause of myocardial infarction is the rupture of an atherosclerotic plaque within a coronary artery, followed by thrombus formation that impedes blood flow. The resulting ischemia causes severe metabolic disturbances and structural damage to cardiac tissue (Fathima, 2021).

The myocardium requires a continuous supply of oxygen to maintain its contractile function and cellular metabolism. When blood flow is interrupted, aerobic metabolism shifts to anaerobic metabolism, resulting in reduced ATP production, accumulation of lactic acid, electrolyte imbalance, and impaired cellular function. If ischemia persists for an extended period, irreversible injury occurs, leading to the death of cardiomyocytes and permanent myocardial damage. The pathogenesis of myocardial infarction involves a complex sequence of biochemical and molecular events. One of the earliest consequences of ischemia is the excessive generation of reactive oxygen species (ROS), which contribute to oxidative stress. These free radicals attack cellular lipids, proteins, and nucleic acids, resulting in membrane damage and cellular dysfunction. Oxidative stress is further accompanied by inflammatory responses characterized by the release of cytokines, chemokines, and inflammatory mediators that exacerbate myocardial injury (Sarkar *et al.,* 2019).

III.MAJOR CONSEQUENCES OF MYOCARDIAL INFARCTION

a) Myocardial Cell Death

Prolonged oxygen deprivation results in irreversible injury and death of cardiac muscle cells. Necrosis and apoptosis of cardiomyocytes reduce the functional capacity of the heart and impair its pumping efficiency (Schwinger, 2021).

b) Cardiac Tissue Necrosis

The infarcted region undergoes coagulative necrosis due to prolonged ischemia. Necrotic myocardial tissue is gradually replaced by fibrous scar tissue, which lacks the contractile properties of normal myocardium (Leancă *et al.,* 2022).

c) Oxidative Damage

Excessive production of reactive oxygen species during ischemia and reperfusion causes lipid peroxidation, protein oxidation, and DNA damage. Oxidative stress contributes significantly to myocardial dysfunction and cell death (Güler *et al.,* 2022).

d) Inflammatory Response

Myocardial injury triggers the recruitment of inflammatory cells such as neutrophils and macrophages. These cells release inflammatory mediators and proteolytic enzymes that can further aggravate tissue damage.

e) Impaired Cardiac Function

Loss of viable myocardial tissue reduces cardiac contractility and ventricular performance. This may lead to decreased cardiac output and compromised systemic circulation.

f) Heart Failure and Arrhythmias

Extensive myocardial damage can result in ventricular remodeling, heart failure, and life-threatening arrhythmias. Electrical instability of damaged cardiac tissue increases the risk of sudden cardiac death (Bacharova *et al.,* 2023).

IV. ROLE OF OXIDATIVE STRESS IN MYOCARDIAL INFARCTION

Oxidative stress plays a pivotal role in the initiation and progression of myocardial infarction and is considered one of the major mechanisms responsible for cardiac injury. It occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense systems of

the body. Under physiological conditions, reactive oxygen species are generated in small amounts and participate in normal cellular signalling processes. However, during myocardial ischemia and reperfusion, excessive ROS production overwhelms the endogenous antioxidant defenses, resulting in oxidative damage to cardiac tissues **(**Afzal *et al.,* 2023).

During myocardial ischemia, reduced oxygen supply disrupts cellular metabolism and impairs mitochondrial function. Upon reperfusion, the sudden restoration of oxygen paradoxically leads to the massive generation of reactive oxygen species, a phenomenon known as ischemia-reperfusion injury. The major ROS generated during this process include superoxide anion radicals ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2), hydroxyl radicals (•OH), and peroxynitrite ($ONOO^-$). These highly reactive molecules attack various cellular components, causing structural and functional alterations in myocardial cells (Kalogeris *et al.,* 2017).

V.ISOPROTERENOL-INDUCED MYOCARDIAL INFARCTION MODEL

Experimental animal models play a crucial role in understanding the pathophysiology of myocardial infarction and evaluating the efficacy of potential cardioprotective agents. Among the various models available, the isoproterenol-induced myocardial infarction model is one of the most widely used and well-established experimental approaches. Isoproterenol (ISO) is a synthetic catecholamine and non-selective β-adrenergic receptor agonist that produces severe stress on the myocardium when administered at high doses. The pathological and biochemical alterations induced by isoproterenol closely resemble those observed in human myocardial infarction, making it a valuable tool for cardiovascular research (Imran *et al.,* 2026).

Isoproterenol stimulates both β1- and β2-adrenergic receptors, resulting in significant changes in cardiac function and metabolism. Administration of supramaximal doses of isoproterenol causes extensive myocardial injury characterized by oxidative stress, inflammation, membrane damage, and necrosis of cardiac muscle fibers. Due to its ability to consistently induce myocardial infarction-like lesions, the ISO-induced model has become a standard experimental method for assessing the cardioprotective effects of synthetic drugs and medicinal plants (Ahmed *et al.,* 2017).

VI. MECHANISM OF ISOPROTERENOL-INDUCED CARDIOTOXICITY

The cardiotoxic effects of isoproterenol are mediated through multiple interconnected mechanisms that ultimately result in myocardial injury and necrosis.

- Excessive Stimulation of β-Adrenergic Receptors

Isoproterenol acts as a potent β-adrenergic receptor agonist and excessively stimulates β1-receptors present in cardiac tissue. This overstimulation leads to increased cardiac contractility and acceleration of heart rate. Persistent activation of these receptors imposes significant stress on myocardial cells, making them more susceptible to injury (Yuan *et al.,* 2024)..

- Increased Heart Rate and Cardiac Workload

Following administration, isoproterenol produces positive chronotropic and inotropic effects, leading to tachycardia and enhanced myocardial contractility. The increased workload elevates the metabolic requirements of the heart and places excessive demand on cardiac tissue, contributing to myocardial stress and dysfunction (Bertero and Maack, 2018).

- Elevated Oxygen Demand

The enhanced cardiac activity induced by isoproterenol substantially increases myocardial oxygen consumption. However, the oxygen supply often becomes insufficient to meet this increased demand, resulting in a state of relative ischemia. This imbalance between oxygen supply and demand is a key factor in the development of myocardial injury.

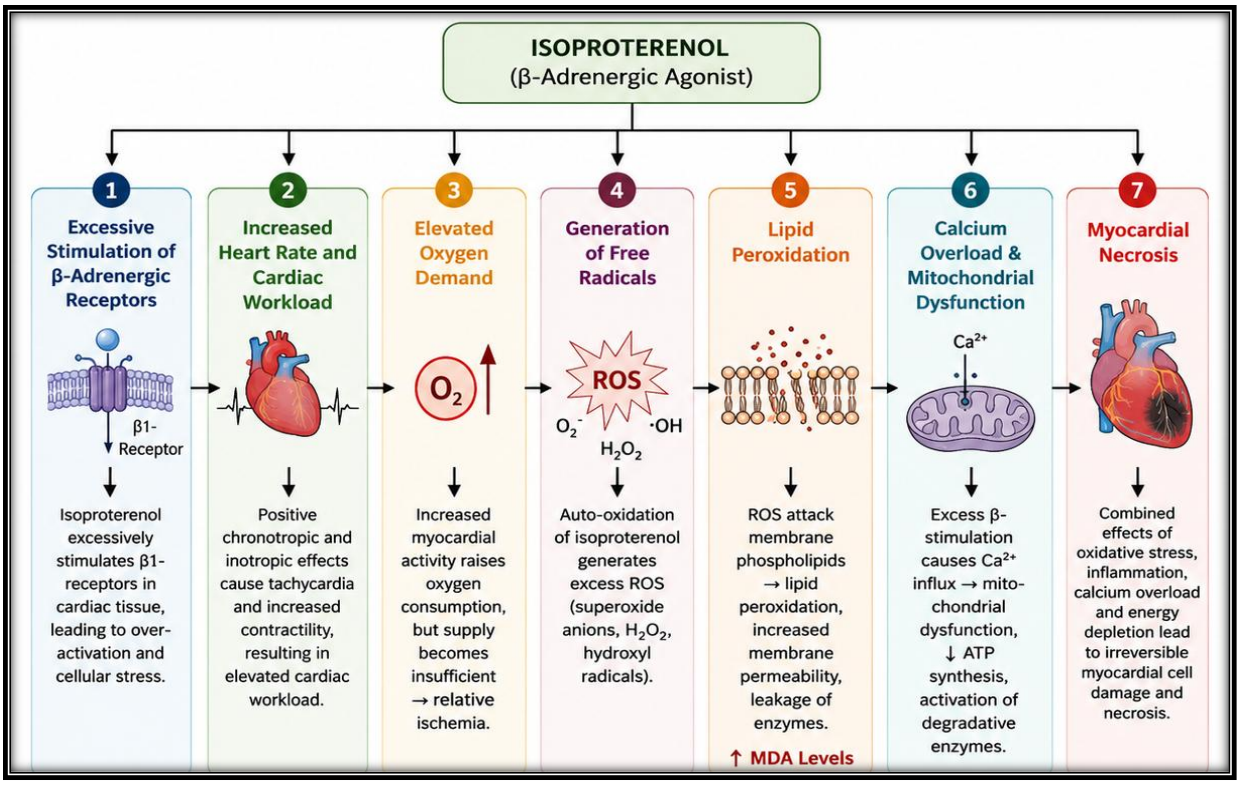

Figure 2: Mechanism of Isoproterenol-Induced Cardiotoxicity

- Generation of Free Radicals

Isoproterenol undergoes auto-oxidation within cardiac tissues, generating large amounts of reactive oxygen species (ROS) such as superoxide anions, hydrogen peroxide, and hydroxyl radicals. Excessive free radical production overwhelms endogenous antioxidant defenses and initiates oxidative stress-mediated damage **(**Imran *et al.,* 2026).

- Lipid Peroxidation

Reactive oxygen species attack membrane phospholipids, leading to lipid peroxidation and disruption of cellular membrane integrity. This process increases membrane permeability, causes leakage of intracellular enzymes, and impairs normal cellular function. Elevated levels of malondialdehyde (MDA), a marker of lipid peroxidation, are commonly observed following isoproterenol administration (Su *et al.,* 2019).

- Calcium Overload and Mitochondrial Dysfunction

Excessive β-adrenergic stimulation leads to abnormal calcium influx into myocardial cells. Increased intracellular calcium levels disrupt mitochondrial function, reduce ATP synthesis, and activate degradative enzymes. These changes further aggravate cellular injury and promote cardiomyocyte death (Balderas *et al.,* 2024).

- Myocardial Necrosis

The combined effects of oxidative stress, inflammation, calcium overload, and energy depletion ultimately result in irreversible myocardial cell damage and necrosis. Histopathological examination typically reveals myocardial fiber degeneration, edema, inflammatory cell infiltration, and extensive necrotic lesions similar to those observed in human myocardial infarction (Ghafoor *et al.,* 2020).

VII. BIOCHEMICAL ALTERATIONS INDUCED BY ISOPROTERENOL

Administration of isoproterenol produces characteristic biochemical changes that serve as markers of myocardial injury:

- Increased serum creatine kinase-MB (CK-MB)
- Elevated lactate dehydrogenase (LDH)
- Increased aspartate aminotransferase (AST)
- Increased alanine aminotransferase (ALT)
- Elevated cardiac troponins

- Increased malondialdehyde (MDA) levels
- Decreased superoxide dismutase (SOD)
- Decreased catalase (CAT)
- Reduced glutathione (GSH) depletion **(**Imran *et al.,* 2026).

VIII. MEDICINAL PLANTS IN CARDIO PROTECTION

Natural products and medicinal plants have gained significant attention due to their therapeutic potential and minimal adverse effects. Plant-derived bioactive compounds such as flavonoids, phenolics, alkaloids, and terpenoids exhibit potent antioxidant and cardioprotective activities (Latif and Nawaz, 2025).

Benefits of Herbal Cardioprotective Agents

- Scavenging free radicals
- Reducing inflammation
- Preventing lipid peroxidation
- Improving lipid profile
- Enhancing endogenous antioxidant defense
- Protecting myocardial cells

IX. ADVANTAGES OF ISO-INDUCED MYOCARDIAL INFARCTION MODEL

The isoproterenol-induced myocardial infarction model offers several advantages that make it highly suitable for cardiovascular research.

- Reproducible and Reliable

The model consistently produces myocardial injury with predictable biochemical and histopathological changes, ensuring reliable experimental outcomes.

- Closely Resembles Human Myocardial Infarction

The pathological features induced by isoproterenol, including oxidative stress, inflammation, enzyme leakage, and myocardial necrosis, closely mimic those observed in human myocardial infarction.

- Easy Induction of Cardiac Injury

Myocardial infarction can be induced simply through subcutaneous or intraperitoneal administration of isoproterenol without requiring complex surgical procedures.

- Cost-Effective and Time-Efficient

The model is economical, easy to perform, and suitable for large-scale pharmacological screening studies.

- Suitable for Screening Cardioprotective Agents

The model is extensively used to evaluate the efficacy of natural products, medicinal plants, antioxidants, and synthetic compounds against myocardial injury (Allawadhi *et al.,* 2018).

X. INTRODUCTION TO MADHUCA LONGIFOLIA

Madhuca longifolia (J. Koenig ex L.) J.F. Macbr., commonly known as Mahua, is an important medicinal and economic tree belonging to the family Sapotaceae. It is a large, evergreen to semi-evergreen tree widely distributed throughout the Indian subcontinent, particularly in India, Nepal, Sri Lanka, Bangladesh, and Myanmar. The plant is highly valued in traditional systems of medicine such as Ayurveda, Siddha, and folk medicine due to its diverse therapeutic applications and nutritional significance. Mahua is considered a multipurpose tree because almost every part of the plant, including its flowers, leaves, bark, fruits, seeds, and roots, possesses medicinal value. The tree plays an important role in rural economies, especially among tribal communities, where it is utilized as a source of food, medicine, fodder, fuel, and traditional beverages. Owing to its remarkable medicinal properties, *Madhuca longifolia* has attracted considerable scientific interest for the investigation of its phytochemical constituents and pharmacological activities (Sinha *et al.,* 2017).

Figure 3: *Madhuca longifolia*

The plant is rich in various bioactive compounds such as flavonoids, triterpenoids, saponins, tannins, glycosides, alkaloids, phenolic compounds, steroids, and carbohydrates. These phytoconstituents contribute to its wide range of biological activities,

including antioxidant, anti-inflammatory, antidiabetic, hepatoprotective, nephroprotective, antimicrobial, antiulcer, analgesic, and cardioprotective effects. Among its pharmacological properties, the antioxidant activity of *Madhuca longifolia* is particularly significant. Oxidative stress is one of the major factors involved in the pathogenesis of myocardial infarction and other cardiovascular disorders. The presence of potent antioxidant phytochemicals in *Madhuca longifolia* enables it to scavenge reactive oxygen species, inhibit lipid peroxidation, enhance endogenous antioxidant defenses, and protect tissues against oxidative damage. These properties suggest its potential role in the prevention and management of cardiovascular diseases (Dubey *et al.,* 2023).

XI. ETHNOMEDICINAL IMPORTANCE OF MADHUCA LONGIFOLIA

Madhuca longifolia (Mahua) has been extensively utilized in traditional systems of medicine such as Ayurveda, Siddha, Unani, and various indigenous folk practices for centuries. The plant is highly valued due to its diverse therapeutic applications and widespread availability in tropical regions of India. Almost every part of the plant, including flowers, leaves, bark, fruits, seeds, and roots, is used for medicinal purposes. The long history of traditional use of *Madhuca longifolia* indicates the presence of numerous bioactive constituents that contribute to its pharmacological activities.

XII. TRADITIONAL USES OF MADHUCA LONGIFOLIA

12.1 Diabetes Mellitus

The leaves, bark, and flowers of *Madhuca longifolia* have traditionally been used for the management of diabetes mellitus. Folk practitioners administer preparations of the plant to help regulate blood glucose levels and improve metabolic functions. Scientific studies have also reported significant antihyperglycemic activity, supporting its traditional use in diabetic conditions (Jha and Mazumder, 2018).

12.2 Inflammatory Disorders

Various parts of the plant are used to alleviate inflammation and pain associated with inflammatory disorders. Leaf and bark extracts are commonly applied in traditional medicine to reduce swelling, redness, and discomfort. The anti-inflammatory activity is attributed to the presence of flavonoids and triterpenoids that inhibit inflammatory mediators (Yatoo *et al.,* 2018).

12.3 Skin Diseases

Madhuca longifolia has long been employed in the treatment of skin ailments such as eczema, dermatitis, itching, wounds, and infections. Paste prepared from the bark or leaves is applied topically to promote wound healing and relieve skin irritation. The antimicrobial and anti-inflammatory properties of the plant contribute to its effectiveness in dermatological conditions.

12.4 Rheumatism

Traditional healers use preparations derived from the bark, leaves, and seed oil for the management of rheumatism and joint pain. The plant is believed to reduce inflammation and improve mobility in individuals suffering from musculoskeletal disorders.

12.5 Ulcers

The flowers and bark of *Madhuca longifolia* are used in traditional medicine for the treatment of gastric and duodenal ulcers. The plant exhibits gastroprotective and antiulcer activities, which may be due to its antioxidant properties and ability to strengthen the gastric mucosal barrier (Amer *et al.,* 2025).

12.6 Bronchitis and Respiratory Disorders

Flower extracts and other plant preparations are traditionally used to relieve respiratory ailments such as bronchitis, cough, asthma, and throat irritation. The soothing and anti-inflammatory properties of the flowers help reduce respiratory discomfort and improve breathing.

12.7 Liver Disorders

The plant is widely used in folk medicine for treating various liver ailments. Traditional preparations are believed to improve liver function and protect hepatic tissues from toxic insults. Several studies have demonstrated hepatoprotective activity, supporting its ethnomedicinal application in liver diseases (Abou Seif, 2016).

12.8 Cardiovascular Ailments

Madhuca longifolia has been used traditionally to promote cardiovascular health and manage disorders associated with the heart and circulatory system. The presence of antioxidant and anti-inflammatory phytoconstituents may help protect cardiac tissues from oxidative damage and improve overall cardiovascular function. These traditional claims have encouraged scientific investigations into its cardioprotective potential, particularly in experimental models of myocardial infarction (Kaur *et al.,* 2025).

XIII. PHARMACOLOGICAL ACTIVITIES OF MADHUCA LONGIFOLIA

13.1 Antioxidant Activity

Oxidative stress is one of the major factors involved in the pathogenesis of several chronic diseases, including myocardial infarction, diabetes, cancer, and neurodegenerative disorders. *Madhuca longifolia* possesses potent antioxidant activity primarily due to the presence of flavonoids, phenolic compounds, tannins, and triterpenoids. These phytoconstituents are capable of scavenging reactive oxygen species (ROS) and protecting biological systems from oxidative damage (Dubey *et al.,* 2023).

The antioxidant potential of *Madhuca longifolia* has been demonstrated in various in vitro and in vivo studies. Plant extracts have shown significant free radical scavenging activity against different reactive oxygen species, thereby reducing oxidative stress and preventing cellular damage. The antioxidant effect of the plant contributes substantially to its therapeutic applications in cardiovascular and metabolic disorders (Devi and Sangeetha, 2016).

13.2 Anti-inflammatory Activity

Inflammation is a protective physiological response; however, excessive or chronic inflammation contributes to the development of various diseases, including cardiovascular disorders. *Madhuca longifolia* possesses significant anti-inflammatory activity and has been traditionally used to treat inflammatory conditions such as arthritis, rheumatism, and tissue swelling.

Experimental studies have demonstrated that extracts of *Madhuca longifolia* reduce inflammation by inhibiting the production and release of inflammatory mediators. The anti-inflammatory effects are mainly attributed to flavonoids, triterpenoids, saponins, and phenolic compounds present in the plant (Prince, 2018).

13.3 Antihyperlipidemic Activity

Hyperlipidemia is one of the major risk factors for cardiovascular diseases, including atherosclerosis, coronary artery disease, and myocardial infarction. Elevated levels of serum cholesterol and triglycerides contribute to plaque formation within blood vessels and increase the risk of cardiovascular complications.

Studies have reported that *Madhuca longifolia* possesses significant antihyperlipidemic activity and helps improve lipid metabolism. The beneficial effects are mainly attributed to flavonoids, saponins, and phenolic compounds that influence lipid synthesis, absorption, and metabolism (Shrirao and Code 2017).

13.4 Cardioprotective Activity

Cardioprotective activity is one of the most important pharmacological properties of *Madhuca longifolia*. Cardiovascular diseases are closely associated with oxidative stress, inflammation, hyperlipidemia, and myocardial injury. Since *Madhuca longifolia* possesses antioxidant, anti-inflammatory, and antihyperlipidemic activities, it exhibits substantial potential in protecting cardiac tissues from damage.

Several experimental studies have demonstrated that extracts of *Madhuca longifolia* protect the myocardium against oxidative stress-induced injury and ischemic damage. The cardioprotective effect is believed to result from multiple mechanisms acting simultaneously to preserve cardiac structure and function (Ullah *et al.,* 2024).

XIV. BIOCHEMICAL PARAMETERS EVALUATED IN THE STUDY

14.1 Cardiac Marker Enzymes

Cardiac marker enzymes are intracellular enzymes released into the bloodstream following damage to the myocardial cell membrane. Elevated serum levels of these enzymes are considered important indicators of myocardial injury and necrosis.

- Creatine Kinase-MB (CK-MB)

Creatine Kinase-MB is one of the most specific biomarkers of myocardial injury. It is predominantly

present in cardiac muscle and is released into circulation following myocardial cell damage. In isoproterenol-induced myocardial infarction, significant elevation of CK-MB levels occurs due to myocardial necrosis. A reduction in CK-MB levels following treatment indicates cardioprotective activity (Imran *e al.,* 2026).

- Lactate Dehydrogenase (LDH)

Lactate Dehydrogenase is an intracellular enzyme involved in carbohydrate metabolism and is present in various tissues, including the heart. Reduction of LDH levels in treated groups suggests protection against myocardial damage.

- Aspartate Transaminase (AST)

Aspartate Transaminase, also known as Serum Glutamate Oxaloacetate Transaminase (SGOT), is present in cardiac muscle, liver, and skeletal muscle. A decrease in AST levels after treatment indicates preservation of myocardial integrity.

- Alanine Transaminase (ALT)

Alanine Transaminase, also known as Serum Glutamate Pyruvate Transaminase (SGPT), is primarily found in the liver but may also increase during severe myocardial injury. Lower ALT levels in treated animals suggest reduced tissue damage.

- Cardiac Troponins

Cardiac troponins (Troponin I and Troponin T) are highly specific regulatory proteins found in cardiac muscle fibers. Elevated cardiac troponin levels indicate severe myocardial damage, while reduced levels following treatment demonstrate cardioprotection (Marston and Zamora, 2020).

14.2 Antioxidant Parameters

Oxidative stress is a major contributor to myocardial injury. Therefore, evaluation of endogenous antioxidant enzymes is essential for assessing the antioxidant and cardioprotective effects of *Madhuca longifolia*.

1. Superoxide Dismutase (SOD)

Superoxide Dismutase is the first line of defense against reactive oxygen species.

2. Catalase (CAT)

Catalase is an important antioxidant enzyme that decomposes hydrogen peroxide into water and oxygen.

3. Reduced Glutathione (GSH)

Reduced Glutathione is a major intracellular non-enzymatic antioxidant (Shrirao and Code, 2017).

14.3 Lipid Peroxidation Marker

Malondialdehyde (MDA)

Malondialdehyde is a major end product of lipid peroxidation and is widely used as a biomarker of oxidative stress (Ayala *et al.,* 2014).

XV. CONCLUSION

The assessment of cardiac marker enzymes (CK-MB, LDH, AST, ALT, and cardiac troponins), antioxidant parameters (SOD, CAT, and GSH), and lipid peroxidation marker (MDA) provides a comprehensive evaluation of myocardial injury and cardioprotective activity. Elevated cardiac enzymes and MDA levels, together with decreased antioxidant enzyme activities, indicate severe myocardial damage induced by isoproterenol. Restoration of these biochemical parameters following treatment with *Madhuca longifolia* would confirm its antioxidant and cardioprotective potential against experimental myocardial infarction.

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