iJRASET
International Journal For Research in Applied Science and Engineering Technology



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 13 **Issue:** XI **Month of publication:** November 2025

DOI: https://doi.org/10.22214/ijraset.2025.75687

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538
Volume 13 Issue XI Nov 2025- Available at www.ijraset.com

Smoking and Cardiovascular Health with Herbal Treatment

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Abstract: ***This article provides a comprehensive review of the impact of smoking on cardiovascular health, highlighting the epidemiology, pathophysiological mechanisms, and associated risks, including atherosclerosis, endothelial dysfunction, and a prothrombotic state. The review synthesizes extensive research demonstrating that both active and passive exposure to cigarette smoke significantly elevate the risk of various cardiovascular diseases, principally through inflammatory, oxidative, and thrombotic pathways, and by unfavorably altering lipid profiles. Additionally, the article explores the potential benefits of herbal interventions—such as Moringa oleifera, Ginseng, and Ginkgo biloba—in reducing cardiovascular risk factors and improving patient outcomes, attributing their efficacy to multi-target anti-inflammatory, antioxidant, and lipid lowering properties. The article concludes that while traditional cessation strategies have reduced global smoking rates, integrating herbal therapies with established treatments could enhance cardiovascular prevention, though further rigorous clinical trials are warranted to confirm their long-term safety and efficacy.***

Keywords: Cardiovascular Health, Smoking, Cardiovascular Diseases (CVD), Atherosclerosis, Endothelial Dysfunction, Oxidative Stress, Prothrombotic State, Tobacco Smoke, Anti-hypertensive, Cardiovascular Risk Factor

I. INTRODUCTION

Worldwide, cardiovascular diseases (CVDs) constitute a leading cause of morbidity and mortality. Numerous racial and ethnic groups in the US are impacted by CVDs, which have a very high yearly cost of almost $200 billion in medical treatment, medications, and lost productivity.[1].Along with the other risk factors for atherosclerotic cardiovascular disease (ASCVD), smoking increases the likelihood of a number of negative consequences, including arrhythmias, heart failure, death, and a lower quality of life [2].

Among the many illnesses brought on by smoking are peripheral arterial diseases, coronary artery disease, cerebrovascular disease, and aneurysms. Smoking exposure, whether active or passive, unavoidably increases the risk of heart and vascular problems.[3]. These substances can have extra harmful effects on health in addition to being toxic, mutagenic, and carcinogenic.

Among the specific substances included in CS are nicotine, tar, carbon monoxide, ammonia, formaldehyde, acrolein, acetone, polyaromatic aromatic hydrocarbons (PAHs), hydroxyquinone, nitrogen oxides, and cadmium. The smoking rate, which was 32.7% of adults worldwide in 2000, dropped to 22.3% in 2020 as a result of smoking cessation counseling, nicotine replacement treatment, medication, and advancements in cultural and societal awareness. [4]. In a similar vein, South Korea's smoking rate dropped from 25.8% in 2012 to 20.6% in 2020 [5].

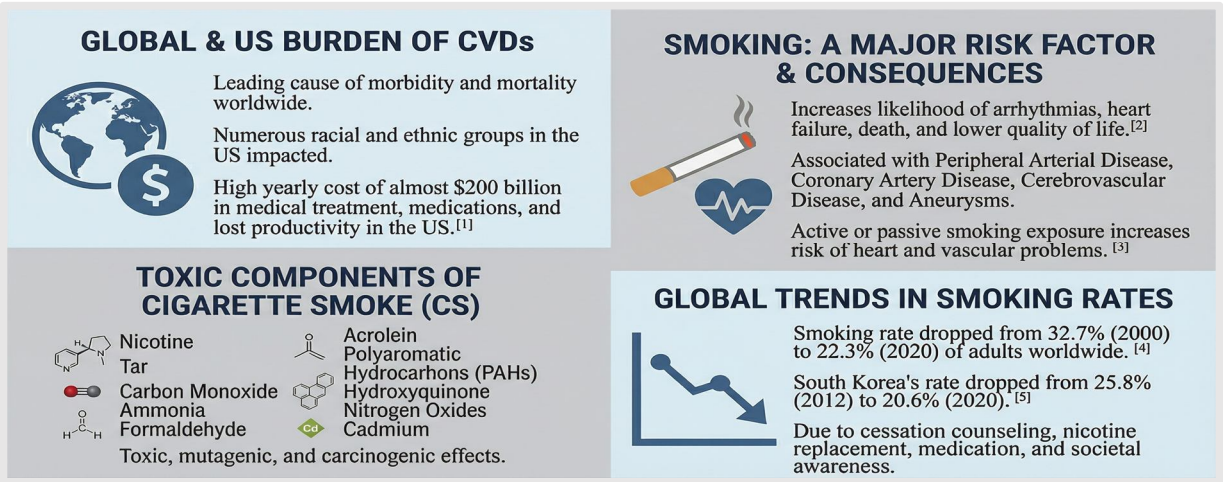


Figure 1: Worldwide impact of cardiovascular disease and smoking

II. EPIDEMIOLOGY

However, unchangeable elements like gender, age, and family history have distinct effects. An independent risk factor is family history, especially premature atherosclerotic disease, which is defined as CVD or death from CVD in a first-degree relative before the age of 55 (for men) or 65 (for women). Additionally, there is data that suggests the existence of CVD risk factors may affect gender differently. For example, women were more likely than males to have CVD if they had diabetes or smoked more than 20 cigarettes a day. With every decade of life, the prevalence of CVD rises dramatically [6]. With estimated indirect costs of $237 billion annually and a projected increase to $368 billion by 2035, cardiovascular disease (CVD) is the most expensive disease, even more so than diabetes and Alzheimer's.[7]. The prevalence rises dramatically with age and varies slightly between genders, with men experiencing a larger incidence at earlier ages. The incidence gap gets smaller [8-9].

III. PATHOPHYSIOLOGY

Tobacco smoke's pathophysiologic effects on cardiovascular disease the link between smoking and CAD has been unequivocally demonstrated by epidemiological research. However, the pathobiological mechanisms behind this connection remains unclear. Atherothrombosis is caused by a number of established pathophysiological mechanisms that are impacted by tobacco use.

A. Endothelial Dysfunction

Epidemiological research has definitively established the connection between smoking and coronary artery disease (CAD).

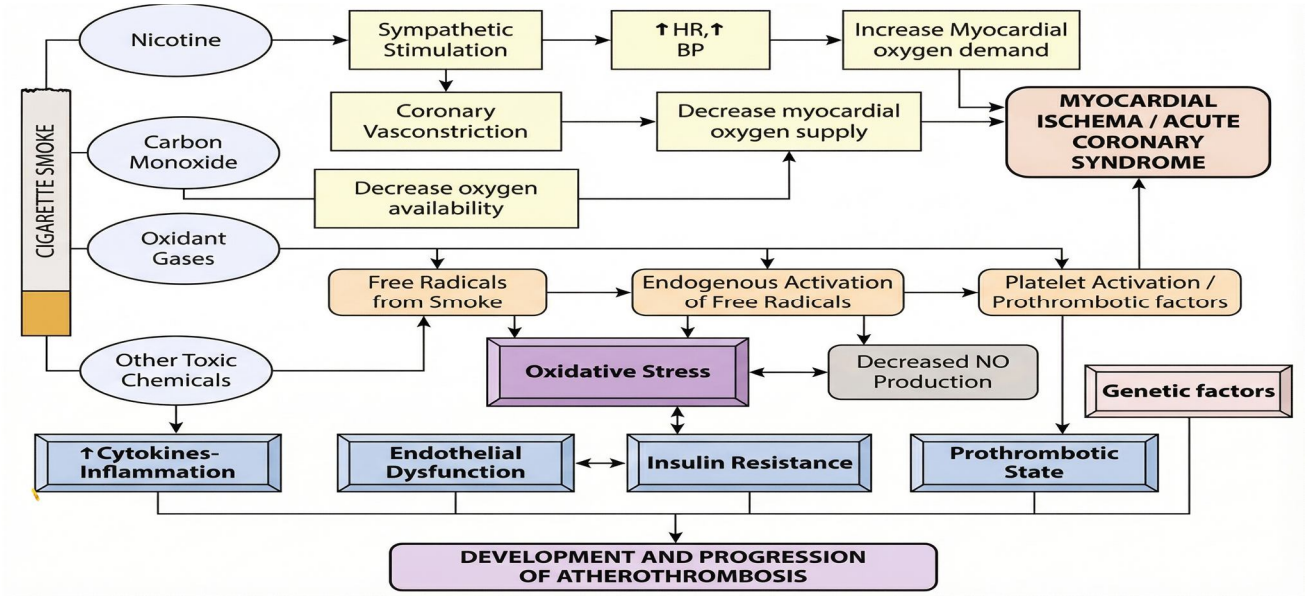


Figure 2: Development & Progression of Atherothrobosis

However, the underlying pathobiological mechanisms of this association remain poorly understood. Tobacco influences several recognized pathophysiological pathways that contribute to the onset of atherothrombosis. Vascular and endothelial dysfunction. Smoking can harm the vascular wall, which leads to a decrease in prostacyclin production and an increase in platelet-vessel wall interactions. This damage can diminish the elastic properties of the aorta, causing stiffening and injury to the wall. Both smoking and exposure to secondhand smoke hinder endothelium-dependent vasodilation in healthy coronary arteries and lower coronary flow reserve. Additionally, smoking is a known risk factor for coronary vasospasm. The impact on endothelial function is primarily due to oxidative substances that promote the oxidation of low-density lipoprotein (LDL) while decreasing the production of nitric oxide.[11]

B. Impact on Lipid Profile

Research indicates that smokers exhibit elevated levels of serum cholesterol, triglycerides, very low-density lipoprotein cholesterol, and low-density lipoprotein cholesterol (LDL-C) compared to nonsmokers, along with reduced serum levels of high-density lipoprotein cholesterol (HDL-C) and apolipoprotein A-1[11]. In a recent randomized study, it was found that quitting smoking led to an increase in total HDL and larger HDL particles; however, there was no change in LDL-C levels or LDL particle size. Cigarette smoking increases the oxidative modification of plasma LDL-C, which could contribute to atherogenesis and has been shown to disrupt endothelial function [23]

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C. Actions that Promote Inflammation

Numerous inflammatory indicators, such as peripheral leukocytosis, Creatives protein, homocysteine, interleukin-6, and tumor necrosis factor, have been linked to elevated levels of cigarette smoke. It is well recognized that inflammation plays a crucial role in atherogenesis. White blood cell counts were found to be considerably higher among smokers, and this was closely linked to the development of carotid atherosclerotic plaques. In patients with chest discomfort and early coronary artery disease. found that smokers had higher numbers of neutrophils, lymphocytes, and monocytes than nonsmokers. Local processes inside the atherosclerotic plaque, which are marked by inflammation and elevated expression of matrix metalloproteinases, were found to be correlated with these systemic immunologic changes.[22]

D. Prothrombotic Condition

Prothrombotic condition It is generally recognized that smoking cigarettes causes a prothrombotic state, as evidenced by smokers' greater incidence of sudden mortality and acute myocardial infarction compared to angina pectoris. By altering thrombotic factors, fibrinolytic factors, and platelet mediated pathways, among other pathogenic mechanisms, smoking causes a prothrombotic state. Smoking cigarettes raises the levels of fibrinogen and increases the expression of tissue factor. By preventing tissue plasminogen activator release from the endothelium and raising plasminogen activator inhibitor-1 levels, it modifies fibrinolysis.

Furthermore, platelets isolated from long-term smokers have a greater tendency to aggregate both spontaneously and in response to stimulation, which activates platelet mediated pathways of thrombosis . Increased production of platelet dependent thrombin is linked to smoking.

IV. SIGN AND SYMPTOMS

People with cardiovascular disease (CVD) may have certain physical symptoms. These symptoms include the following:

1) One of the most common signs of cardiovascular illness is chest pain or discomfort.
2) Having trouble breathing, particularly when exercising.
3) Weakness or exhaustion without apparent cause.
4) Lightheadedness or dizziness.
5) Abdominal, ankle, or leg swelling may indicate heart failure or other cardiovascular disorders.
6) Typical signs of heart attacks, strokes, and cardiovascular illnesses.
7) The underlying blood vessel disease frequently shows no symptoms. The first indication of an underlying illness could be a heart attack or stroke.
8) The following are signs of a heart attack

Discomfort or pain in the middle of the chest, as well as discomfort or pain in the arms, left shoulder, elbows, jaw, or back Symptom identification and reporting may be impacted by variables including depression and cognitive
function, and symptoms are frequently experienced differently by men and women. By promptly identifying individuals who may be at higher risk, monitoring and assessing symptoms with instruments that adequately
account for depression and cognitive function may assist to enhance patient care.[13]



Figure 3: causes of development of CVD

V. HERBAL TREATMENT

Multitudinous herbal interventions have demonstrated promising cardiovascular benefits. A number of medicinal sauces well linked to treat CVD are Moringaoleifera, Ginseng, Ginkgo biloba, Celosia argentea, Gentianalutea , Allium sativum, Curcuma longa, and Zingiber officinale. Mechanistic perceptivity reveal that herbal interventions frequently target multiple pathways involved in CVD pathogenesis. These mechanisms encompass anti-inflammatory, antioxidant, anti-thrombotic antihypertensive, and lipid- lowering goods. also, some sauces enhance endothelial function, promote nitric oxide product, and ply vasodilatory goods, contributing to bettered cardiovascular health. Clinical studies have handed substantiation of the efficacy of certain herbal interventions in reducing CVD threat factors and perfecting patient issues. still, further rigorous, large-scale clinical trials are demanded to establish their long-term safety and effectiveness. It's pivotal to consider implicit condiment-medicine relations and standardize tablets for dependable remedial issues. [21]

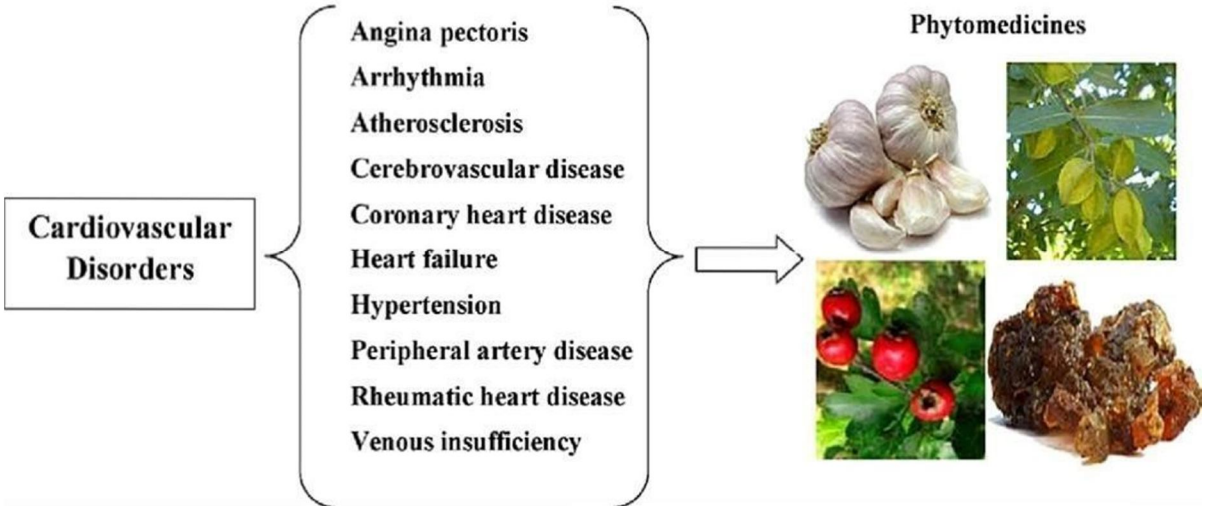


Figure 4: CVD reason and drug approch

A. Moringa oleifera

Reducing the onset and progression of CVD is crucial given its high fatality rate. In order to prevent future harm to the heart and blood vessels, pharmacological treatments have been developed.[24] Additionally, natural products containing different phytochemical substances are being studied. Moringa oleifera Lam. (MO) is one example of a natural product. MO has been proposed as an anti-apoptotic, enhancing cardiac contractility and preventing harm to the structural integrity of the heart. Additionally, by reducing oxidative stress and inflammation, MO may have a cardioprotective effect. The purpose of this review is to examine the effectiveness of MO's phytochemical substances as a protective agent against vascular dysfunction and cardiac damage in the CVD model. [25].

B. Turmeric

Turmeric is a naturally occurring polyphenol that comes from the rhizome of Curcuma longa. It interacts with gut microbiota and has anti-inflammatory, cardiovascular, mental health, and anti-aging properties.[17].

C. Ginseng

Ginseng is a natural substance frequently utilized in culinary applications and traditional herbal medicine within China. The clinical safety profile of ginseng is well documented. It is linked to cardioprotective, antioxidant, anti-inflammatory, and anticoagulant effects Consequently, there exists significant potential for the clinical application of ginseng in managing cardiovascular diseases. In a rat study, ginseng enhanced cardiac contractility without affecting the heart rate by activating PPARδ and increasing intracellular calcium levels along with cardiac troponin I phosphorylation.[18] This indicates that ginseng may enhance cardiac functions without inducing negative effects such as arrhythmias.

Ginsenosides are the primary bioactive compounds found in Panax ginseng extracts. They enhance myocardial blood supply and the heart's pumping efficiency by markedly increasing myocardial contractility, vasodilation, and blood flow. The antiapoptotic and anti-inflammatory properties of ginsenoside Rg3 significantly mitigated cardiac dysfunction induced by myocardial ischemia/reperfusion (I/R) [19]. Ginsenoside Rh2, the pharmacologically active component of red ginseng, significantly enhances cardiac function by reducing cardiac fibrosis. The antiinflammatory effects of ginsenoside Rb3 notably alleviate inflammation induced ventricular systolic dysfunction .

Ginsenoside Rb2 significantly improved cardiac function by reducing infarct size in an in vivo model of myocardial ischemia/reperfusion (MI/R) injury; it also diminished in vitro H_2O_2-induced stress in H9c2 cardiomyocytes . Presently, there is a wealth of literature and experimental evidence demonstrating that ginsenosides are the principal pharmacologically active constituents in ginseng that enhance cardiac function through various mechanisms .Therefore, ginseng represents a promising candidate for the prevention and treatment of cardiovascular diseases [20].

D. Ginkgo biloba

Ginkgo biloba's constituent terpenoids (ginkgolides and bilobalide) and flavonoids (ginkgo-flavone glycosides) are primarily responsible for its pharmacological activity and therapeutic effects . [14]The anti-inflammatory and antioxidant properties of these Ginkgo biloba components are widely recognized. .[15] Cardiovascular, pulmonary, and central nervous system disorders are among the many conditions that benefit from ginkgo biloba's antioxidant and anti-inflammatory properties.[16].

E. Zingiber officinale

6-shogaol and 9-gingerol are responsible for the antihypertensive effects of ginger. These compounds reduce LDL and cholesterol, prevent atheroma plaque formation, and increase vascular elasticity. Additionally, by lowering intercellular adhesion molecule levels, they prevent the synthesis of inflammatory mediators that lead to endothelial dysfunction [21]

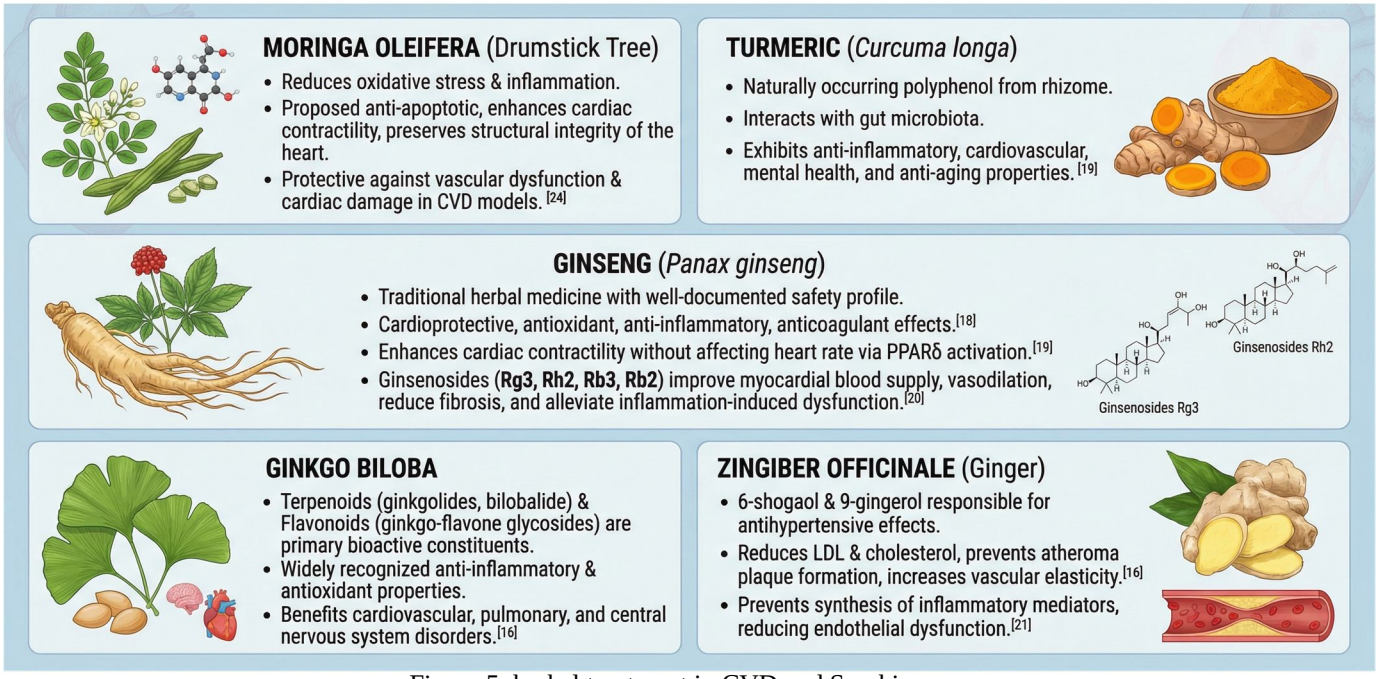


Figure 5: herbal treatment in CVD and Smoking

VI. CONCLUSION

Smoking is a major risk factor for cardiovascular diseases, causing damage primarily through inflammation, oxidative stress, and blood clotting pathways. While smoking rates have declined, the cardiovascular burden remains significant. Herbal treatments like Moringa, Ginseng, and Ginkgo show promise in reducing cardiovascular risks through multiple mechanisms, but more clinical trials are needed to confirm their long-term safety and effectiveness. Combining herbal therapies with conventional treatments could improve cardiovascular outcomes for smokers.

VII. ACKNOWLEDGMENT

The authors would like to express their sincere gratitude to Dr. Sayyad Sajauddin Gulab (Principal) and Ms. Rubina Imran Sheikh (Assistant Professor & our guide) for their expert guidance, constant encouragement, and supervision throughout this study. We also extend our heartfelt thanks to the management of Central India College of Pharmacy, Lonara, Nagpur, Maharashtra, for providing the necessary library facilities, infrastructure, and digital resources required to conduct this comprehensive review. Furthermore, we thank the faculty members for their valuable insights and our families and friends for their continuous moral support.

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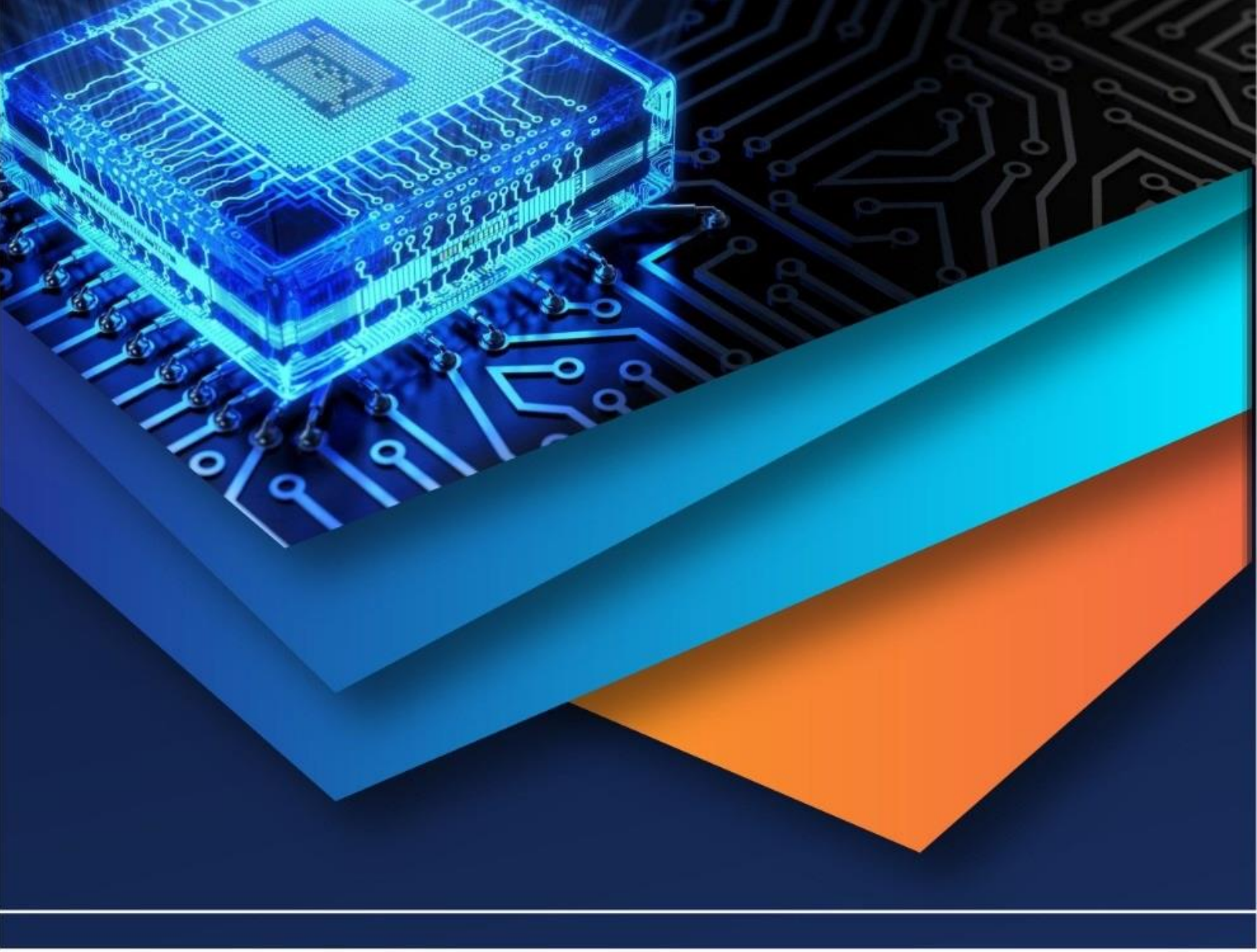
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