ORIGINAL ARTICLE

INTERNATIONAL JOURNAL OF ARTIFICIAL INTELLIGENCE

ISSN: 2692-5206, Impact Factor: 12,23

American Academic publishers, volume 05, issue 06,2025

Journal: https://www.academicpublishers.org/journals/index.php/ijai

THE EFFECT OF THE ANTIOXIDANT DIBUNOL ON THE LIPID PROFILE OF BLOOD SERUM IN RABBITS WITH EXPERIMENTAL ATHEROSCLEROSIS

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Abstract: Atherosclerosis is a widespread and severe systemic disease and remains a leading cause of cardiovascular morbidity and mortality. This study aimed to investigate the effect of the antioxidant dibunol on the serum lipid profile in rabbits with experimentally induced atherosclerosis. The results demonstrated that dibunol significantly reduced serum cholesterol, triglycerides, and beta-lipoprotein levels, confirming its potential for atherosclerosis prevention and therapy.

Keywords: Atherosclerosis, ischemic heart disease, antioxidant, dibunol, lipid metabolism, cholesterol, triglycerides, beta-lipoproteins, experimental model, prevention.

Relevance of the Study:

Atherosclerosis and its complications, particularly ischemic heart disease, are among the leading causes of death and disability worldwide [1]. According to WHO, in 2021, cardiovascular diseases accounted for approximately 20.5 million deaths globally, constituting nearly one-third of all deaths. Notably, 85% of these were associated with atherosclerotic cardiovascular conditions. In 2019, cardiovascular diseases caused 107,394 deaths in Uzbekistan, placing the country among those with the highest mortality rates from such diseases. Daily smoking prevalence among men stands at 24.1%, a major risk factor for cardiovascular pathology. Given the disease's significant impact on modern healthcare systems, especially within the context of Uzbekistan, the development of effective prevention and treatment methods is imperative. In-depth understanding of the pathogenesis of atherosclerosis and disorders of lipid metabolism is key to developing effective pharmacological agents. From this perspective, antioxidants play a crucial role in the prevention of atherosclerosis [2].

Atherosclerosis: Pathogenetic Basis and Clinical Manifestations

Atherosclerosis is a chronic and progressive disease resulting from the accumulation of cholesterol, lipoproteins (especially LDL), lipids, and mineral substances in arterial walls [6]. These substances damage the endothelium—the inner layer of arteries—undergo oxidation, and

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trigger inflammatory responses [3]. Consequently, atheromatous plaques form beneath the endothelial layer.

Stages of Pathogenesis:

Endothelial Dysfunction: Triggered by risk factors such as smoking, hypertension, and hyperglycemia, leading to microscopic endothelial injury and infiltration of lipoproteins. Lipid Accumulation and Oxidation: Low-density lipoproteins (LDL) oxidize within the vascular wall. Macrophages engulf these oxidized particles, transforming into foam cells. Inflammation: Monocytes, T-lymphocytes, and inflammatory mediators (e.g., IL-1, TNF-α) amplify the inflammatory process, promoting plaque growth and complexity [4]. Plaque Complication and Calcification: Formation of necrotic zones, fibrous collagen layers, and calcium deposits leads to arterial stiffening and loss of elasticity [5]. Increased Risk of Thrombosis: Plaque rupture may result in clot formation, leading to acute ischemic events such as myocardial infarction or stroke.

Clinical Manifestations:

Atherosclerosis affects various vascular beds:Coronary Arteries: Chest pain (angina), arrhythmias, heart failure [7]. Cerebral Arteries: Headache, visual disturbances, speech impairment, imbalance, stroke. Peripheral Arteries (Lower Limbs): Claudication, cold extremities, risk of necrosis. Clinicians distinguish between two stages of the disease:

Asymptomatic (Subclinical) Stage

Manifest (Clinical) Stage – when arterial stenosis becomes significant (>50%), leading to symptom development.

Materials and Methods:

An experimental model of atherosclerosis was established in laboratory rabbits. Cholesterol emulsion in cottonseed oil was administered daily via an elastic gastric tube for 30–60 days. This led to a significant increase in serum levels of cholesterol, triglycerides, and beta-lipoproteins. Subsequently, rabbits in the experimental group received dibunol at a dose of 30 mg/kg body weight. All biochemical parameters were assessed using standard laboratory diagnostic techniques.

Results and Discussion:

In rabbits with induced experimental atherosclerosis, serum cholesterol levels were significantly elevated. Upon administration of dibunol, the following improvements in lipid parameters were observed: Cholesterol: Decreased from 387.42 mg% to 108.4 mg%. Triglycerides: Decreased from 947 mg% to 73.7 mg%. Beta-lipoproteins: Decreased from 685.77 mg% to 351.32 mg%. These findings indicate that dibunol significantly attenuates lipid metabolism disorders, which play a key role in the pathogenesis of atherosclerosis.

Conclusion:

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The results of this experimental study demonstrated that the antioxidant dibunol is effective in preventing and treating atherosclerosis. It exerts a beneficial effect by lowering serum cholesterol, triglycerides, and beta-lipoprotein levels, thereby targeting critical components of atherosclerotic pathogenesis. These findings highlight the potential use of antioxidants in both the prevention and treatment of atherosclerosis.

Recommendations:

Dibunol may be considered as a supportive agent in the prevention and treatment of atherosclerosis. Further clinical studies are necessary to evaluate its efficacy and safety in humans. Moreover, incorporating antioxidant-rich foods into the daily diet and developing antioxidant-based therapeutic strategies for patients with atherosclerosis are highly recommended.

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