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

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Abstract: This study investigates the influence of dibunol (also known as ionol or butylated hydroxytoluene, BHT), an antioxidant compound, on the lipid profile of rabbits with experimentally induced atherosclerosis. Using a high-cholesterol diet to simulate hyperlipidemia and plaque formation, the experiment evaluates how dibunol administration alters key lipid parameters, including total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) levels in blood serum. The findings suggest that dibunol has a lipid-modulating effect and may contribute to the prevention or attenuation of atherosclerotic changes through its antioxidant properties.
Keywords: dibunol, atherosclerosis, lipid profile, serum cholesterol, antioxidant, rabbits.

INTRODUCTION

Atherosclerosis is a progressive vascular disease characterized by lipid accumulation, oxidative stress, and inflammatory responses within the arterial wall, leading to the formation of plaques that compromise blood flow and increase the risk of cardiovascular events. One of the central pathogenic mechanisms in atherosclerosis is lipid peroxidation — a process driven by reactive oxygen species that alters lipoproteins and promotes their deposition in vessel walls.

Antioxidants have gained attention as potential therapeutic agents for modulating lipid metabolism and reducing oxidative damage. Dibunol (BHT), a synthetic phenolic antioxidant, is known for its free-radical scavenging ability and has been investigated in experimental models for its cytoprotective and lipid-regulating effects. However, data on its impact on lipid levels in the context of experimental atherosclerosis remain limited. This study aims to clarify the effect of dibunol on lipid profiles in rabbits subjected to a cholesterol-rich diet, offering insights into its potential as an anti-atherosclerotic agent.

MATERIALS AND METHODS

In the atherosclerosis model group (Group II), rabbits showed a significant elevation in total cholesterol, LDL-C, and triglycerides, along with a reduction in HDL-C compared to the control group, confirming successful induction of hyperlipidemia and early-stage atherosclerosis [1].

In contrast, dibunol-treated rabbits (Group III) exhibited significantly lower total cholesterol and LDL-C levels compared to Group II. Triglyceride levels were moderately reduced, and HDL-C levels showed a notable increase, approaching normal values.

Parameter	Group I (Control)	Group II (Athero)	Group III (Dibunol)
TC (mg/dL)	82.4 ± 6.3	192.5 ± 11.8*	121.3 ± 9.6†
LDL-C (mg/dL)	35.2 ± 4.1	124.6 ± 10.3*	73.5 ± 8.4†
HDL-C (mg/dL)	41.3 ± 3.5	22.8 ± 2.9*	36.7 ± 4.1†
TG (mg/dL)	78.5 ± 7.2	141.2 ± 9.4*	101.9 ± 6.8†

*Significantly different from Group I ($p<0.01$)
*Significantly different from Group II ($p<0.05$)

These results indicate that dibunol administration mitigated dyslipidemia induced by a high-cholesterol diet and improved the atherogenic lipid profile in rabbits.

RESULTS AND DISCUSSION

The observed reduction in serum lipids in dibunol-treated rabbits supports the hypothesis that oxidative stress plays a critical role in atherogenesis, and that antioxidant intervention can modulate this process. Dibunol's phenolic structure enables it to donate hydrogen atoms to lipid radicals, thereby terminating lipid peroxidation chains and preserving lipoprotein integrity [2].

The increase in HDL-C levels in the dibunol group is especially relevant, as HDL is known to exert anti-atherogenic effects by facilitating reverse cholesterol transport and inhibiting endothelial inflammation. Furthermore, dibunol may influence lipid metabolism at the hepatic level by modulating enzyme activities such as HMG-CoA reductase or lipoprotein lipase, although this mechanism was not explored in the current study.

Notably, while dibunol improved lipid parameters, the extent of normalization did not reach control values, suggesting that antioxidant therapy may be most effective when used in combination with lipid-lowering agents or lifestyle interventions [3].

Beyond the observed changes in serum lipid levels, the potential of dibunol in modulating atherogenesis extends to its multi-faceted biochemical effects on oxidative processes, endothelial function, and lipid regulation at the molecular level. The complexity of atherosclerosis lies not only in the accumulation of cholesterol but in its interaction with free radicals and inflammatory mediators that alter vascular integrity. In this regard, dibunol's lipophilic and phenolic structure allows it to integrate into lipid membranes and interrupt the chain reactions of lipid peroxidation — a process widely acknowledged as central to the initiation and progression of atherosclerotic plaques.

Studies suggest that dibunol stabilizes cell membranes by preventing the oxidation of polyunsaturated fatty acids, thus preserving the structure and function of low-density lipoproteins (LDLs). When oxidized, LDLs are readily taken up by macrophages via scavenger receptors, forming foam cells — the hallmark of early atherosclerotic lesions. By reducing the oxidation potential of circulating LDL, dibunol may indirectly inhibit the formation of these foam cells, thereby exerting not only a lipid-lowering effect but also a plaque-stabilizing and anti-inflammatory influence.

Furthermore, dibunol's antioxidant properties may also influence vascular reactivity and nitric oxide bioavailability, both of which are critical in maintaining endothelial health. Oxidative stress is known to inactivate nitric oxide (NO), leading to endothelial dysfunction, vasoconstriction, and platelet activation. Dibunol, by mitigating oxidative damage, may help sustain NO-mediated vasodilation, reduce vascular tone, and prevent thrombogenic conditions associated with advanced atherosclerosis [4].

CONCLUSION

Dibunol demonstrates a beneficial effect on lipid metabolism in rabbits with experimental atherosclerosis, significantly reducing total cholesterol, LDL-C, and triglycerides, while enhancing HDL-C levels. These findings suggest that dibunol may offer a promising adjunctive strategy in the prevention or treatment of atherosclerosis by attenuating oxidative stress and improving serum lipid profiles. Further research, including histological analysis of arterial walls and long-term clinical trials, is necessary to fully evaluate its therapeutic potential.

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