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Tripeptide DT-109 (Gly-Gly-Leu) attenuates atherosclerosis and vascular calcification in nonhuman primates

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Advanced atherosclerotic lesions and vascular calcification substantially increase the risk of cardiovascular events. However, effective strategies for preventing or treating advanced atherosclerosis and calcification are currently lacking. This study investigated the efficacy of DT-109 (Gly-Gly-Leu) in attenuating atherosclerosis and calcification in nonhuman primates, exploring its broader therapeutic potential. In this study, twenty male cynomolgus monkeys were administered a cholesterol-rich diet of libitum for 10 months. Then, the animals were treated either orally with DT-109 (150 mg/kg/day) or a vehicle (H₂O) for 5 months while continuing on the same diet. Plasma lipid levels were measured monthly and at the end of the experiment, pathological examinations of the aortas and coronary arteries and RNA sequencing of the coronary arteries were performed. To explore possible molecular mechanisms, the effects of DT-109 on smooth muscle cells (SMCs) were examined *in vitro*. We found that DT-109 administration significantly suppressed atherosclerotic lesion formation in both the aorta and coronary arteries. Pathological examinations revealed that DT-109 treatment reduced lesional macrophage content and calcification. RNA sequencing analysis showed that DT-109 treatment significantly downregulated the pro-inflammatory factors *NLRP3*, *AIM2*, and *CASP1*, the oxidative stress factors *NCF2* and *NCF4*, and the osteogenic factors *RUNX2*, *COL1A1*, *MMP2*, and *MMP9*, while simultaneously upregulating the expression of the SMCs contraction markers *ACTA2*, *CNN1*, and *TAGLN*. Furthermore, DT-109 inhibited SMC calcification and *NLRP3* inflammasome activation *in vitro*. These results demonstrate that DT-109 effectively suppresses both atherosclerosis and calcification. These findings, in conjunction with insights from our previous studies, position DT-109 as a novel multifaceted therapeutic agent for cardiovascular diseases.

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INTRODUCTION

Atherosclerosis and its complications, including myocardial infarction and stroke, are leading causes of death worldwide and remain a significant threat to human health. Atherosclerosis is caused by various genetic and environmental risk factors, such as hyperlipidemia, hypertension, diabetes mellitus, and cigarette smoking. In humans, it may take several decades for clinical complications to manifest.¹ Atherosclerosis is widely recognized as a chronic vascular inflammation resulting from the interaction of these risk factors with arterial wall cells and atherogenic lipoproteins. Pathologically, it begins with the deposition of low-density lipoproteins (LDL), particularly oxidized LDL (ox-LDL). This is followed by the adhesion of circulating monocytes to the endothelial cells of the arteries and their migration into the subendothelial space, where they differentiate into macrophages.

These macrophages then engulf the deposited ox-LDL through scavenger receptors, transforming it into foam cells that contribute to the formation of fatty streaks, a hallmark of early-stage atherosclerotic plaque.² Over time, these fatty streaks progress into more advanced lesions characterized by the development of necrotic or lipid cores, rupture, hemorrhage, stenosis, as well as calcification, all of which can lead to severe cardiovascular and cerebrovascular events.³ Vascular calcification is a hallmark of advanced atherosclerosis, characterized by ectopic calcium deposition in arterial walls. This calcification can occur in both the intimal and medial layers of the arteries, contributing to increased plaque vulnerability (intimal calcification) and arterial stiffness (medial calcification) and the risk of cardiovascular events. Although the molecular mechanisms are not yet fully understood, calcification is now recognized as an active rather

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Atherosclerosis In Primates

J Elliott



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Atherosclerosis In Primates Book Review: Unveiling the Magic of Language

In an electronic digital era where connections and knowledge reign supreme, the enchanting power of language has become more apparent than ever. Its power to stir emotions, provoke thought, and instigate transformation is really remarkable. This extraordinary book, aptly titled "**Atherosclerosis In Primates**," published by a highly acclaimed author, immerses readers in a captivating exploration of the significance of language and its profound impact on our existence. Throughout this critique, we will delve into the book's central themes, evaluate its unique writing style, and assess its overall influence on its readership.

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