Short Communication

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The Role of Dysfunctional HDL in Clinical Practice and Research

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Introduction

Cardiovascular disease (CVD) remains one of the leading causes of mortality globally [1]. An established risk factor for CVD is dyslipidemia, characterized by high levels of low-density lipoprotein cholesterol (LDL), elevated triglycerides and low levels of high-density lipoprotein cholesterol (HDL) [2]. HDL has long been considered to offer protection against CVD due to its role in reverse cholesterol transport and anti-inflammatory properties [3]. In clinical practice, HDL measurements are utilized to assess cardiovascular risk, inform treatment decisions, and guide preventive strategies. Increasing HDL levels is a therapeutic goal to improve the overall lipid profile, reduce cardiovascular risk, and guide medication decisions. It should be noted that recent research has suggested the functional properties of HDL, such as its ability to promote reverse cholesterol transport and reduce inflammation, are equally important in assessing the significance of a protective role in CVD risk [4]. Recent research findings suggest HDL may lose cardio-protective functions (known as dysfunctional HDL) and may even promote atherosclerosis and heart disease [5-8]. Dysfunctional HDL has been linked to various cardiovascular diseases, including atherosclerosis and heart disease. One of the key mechanisms leading to HDL dysfunction is oxidative modification [9]. Under conditions of inflammation and oxidative stress, HDL particles can become oxidized, rendering them less effective in promoting reverse cholesterol transport and anti-inflammatory actions with downstream effects of persistent inflammation with increased levels of LDL and triglycerides in peripheral tissues. This process may turn HDL into a pro-inflammatory agent, contributing to endothelial dysfunction, CVD and atherosclerosis. Additionally, glycation, the non-enzymatic binding of sugars to proteins, can also alter HDL function[10]. Glycated HDL has reduced capacity to promote cholesterol efflux and may contribute to the development of atherosclerotic plaques. The glycation of HDL may be a consequence of prolonged hyperglycemia in individuals with diabetes [11]. Study authors have reported that individuals with a higher proportion of dysfunctional HDL are at an increased risk of developing CVD [12].

This underscores the importance of identifying and understanding the role of dysfunctional HDL in cardiovascular health. Accurate identification and measurement of dysfunctional HDL is essential for assessing CVD risk accurately. Continued research is necessary to develop specific assays and diagnostic markers to distinguish functional from dysfunctional HDL. Consequently, several therapeutic approaches should continue to be explored to mitigate the impact of dysfunctional HDL. These include lifestyle modifications, such as diet and exercise, as well as pharmacological interventions that target oxidative stress and inflammation. The use of HDL in clinical practice holds significant clinical implications. It can serve as a valuable tool for assessing cardiovascular risk, guide treatment decisions, and monitor the effectiveness of interventions. However, HDL's clinical utility is not without challenges, such as variations in measurement methods and the emerging complexity of assessing HDL function. Identifying the mechanisms that lead to HDL dysfunction and developing effective therapeutic strategies to address this issue are critical

steps in a deeper understanding of CVD progression. Further research and clinical studies are needed to better characterize dysfunctional HDL, improve diagnostic tools, and evaluate the efficacy of emerging therapies. Understanding and addressing the role of dysfunctional HDL in heart disease may ultimately lead to more effective prevention and treatment strategies in the future.

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