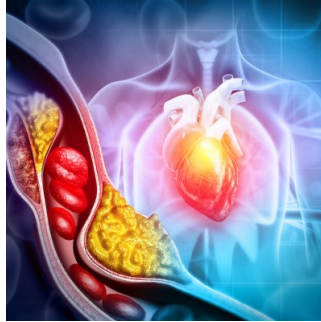


New Approach to Design Cholesterol-Lowering Therapies



A collaborative effort between the Centro Nacional de Investigaciones Cardiovasculares (CNIC) in Madrid and Aarhus University in Denmark has uncovered a crucial mechanism capable of triggering the regression of atherosclerotic plaques. Published in *Nature Cardiovascular Research*, their research pinpoints a novel target for potential future treatments: cells originating from smooth muscle cells within the arterial wall.

The identified mechanism hinges on a specific type of inflammatory signalling within a subset of smooth muscle cells that typically fuels the growth of atherosclerotic plaques. Jacob F. Bentzon, leading the teams at CNIC and Aarhus University, highlights the significance of this discovery, envisioning tailored therapies to amplify the positive impacts of cholesterol-lowering medications, potentially fostering more effective lesion regression among individuals with advanced atherosclerosis.

Smooth muscle cells constitute a vital component of the arterial wall, and their proliferation and transformation into various cell types during atherosclerosis significantly contribute to cardiovascular and cerebrovascular ailments such as heart attacks and strokes.

High blood cholesterol is the primary culprit behind atherosclerosis, with lifestyle modifications or targeted medications like statins offering effective means to stave off high-risk atherosclerosis.

For those already grappling with advanced atherosclerosis, cholesterol reduction not only mitigates the risk of irreversible lesions triggering heart or cerebral infarctions but also unveils mechanisms behind these protective effects that were previously elusive.

Laura Carramolino, the study's lead author, underscores that initiating cholesterol-lowering measures in mice with advanced atherosclerosis reduces the population of smooth muscle-derived cells responsible for plaque growth while preserving those pivotal for plaque stabilisation. This nuanced understanding was made possible through advanced techniques enabling a granular analysis of smooth muscle-derived cell behaviour in atherosclerosis, coupled with seamless collaboration with CNIC Technical Units.

Source: [CNIC](#)
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