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Study examines the rise of plaque in arteries

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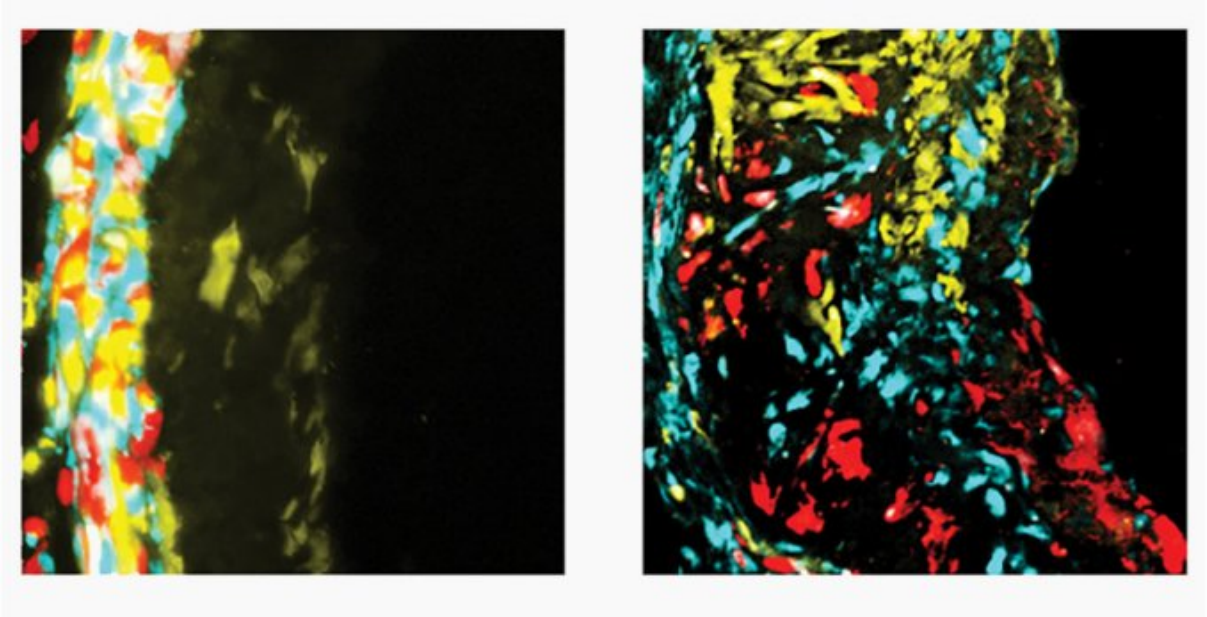

Induction of atherosclerosis in mice models results in plaques (on the right side of each panel) with marked smooth muscle-derived cells of a single color (yellow in left panel) or multiple colors (right panel). These plaques are from mice transplanted with bone marrow that is normal (left panel) or lacking integrin beta3 (right panel). Credit: Yale

The accumulation of cholesterol plaques in artery walls can lead to atherosclerosis, or the hardening of arteries that contributes to heart attacks and strokes. In a new study, Yale researchers investigate how plaque cells develop at the molecular level, and their findings could help produce targeted treatments for the disease.

Smooth muscle cells, the dominant type of cell found in artery walls, are known to be involved in plaque build-up, but it has not been clear how this occurs. The research team, led by senior study author Daniel Greif, used mice models and primary human cells to study smooth muscle cells and their contribution to atherosclerotic plaques.

They discovered that a single smooth muscle cell gives rise, through a process of clonal expansion, to the majority of cells found in the plaque. Additionally, they learned that a gene known as integrin beta3 regulates the migration of a single smooth muscle cell progenitor from the artery wall into plaque. Once inside the plaque, the progenitor cell reproduces and changes into other cell types. Further, the researchers determined that the gene's role in plaque development occurs in smooth muscle cells as well as in bone marrow-derived cells.

Together, the findings shed light on the complex mechanisms behind the progression of atherosclerosis, and suggests potential targets for future therapies, the researchers note.

The study is published in *Nature Communications*.

More information: Ashish Misra et al. Integrin beta3 regulates clonality and fate of smooth muscle-derived atherosclerotic plaque cells, *Nature Communications* (2018). DOI: 10.1038/s41467-018-04447-7

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