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New signal stabilizes atherosclerotic plaques

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Atherosclerosis is an inflammatory disease with accumulation of cholesterol in the vessel walls. The atherosclerotic plaque is built up throughout life and when it ruptures it leads to heart attack or stroke. T cells are important immune cells able to direct the immune response; they are present in the plaques at all stages and signal to other cells through contact or secretion of cytokines, a type of hormone-like signal molecules. In the present study the researchers have identified a cytokine produced by T cells that can stabilize atherosclerotic plaques and protect them from rupture.

The researchers made the finding when investigating a new mouse model together with scientists at Yale University and Howard Hughes Medical Institute in the United States.

"When we analyzed the mouse model the result puzzled us. The outcome was opposite to our initial hypothesis", says Anton Gisterå, one of the researchers who conducted the study. "We had to conduct a series of new experiments to understand what was going on, and ended up identifying the cytokine interleukin-17 as a signal that can stabilize plaques".

The possibility to pharmaceutically use this in patients to stabilize their plaques was not assessed in this study, but the findings provide important information on the role of the immune system in atherosclerosis.

"Traditionally, scientists and physicians have viewed atherosclerosis as merely a buildup of cholesterol in the arteries, and the influence of

inflammation has not been fully attributed", says Göran K. Hansson, team leader of the Experimental Cardiovascular Research group at the Center for Molecular Medicine, and principal investigator of the study. "We need to explore the inflammatory pathways to find new therapies aside from lowering lipids. We have effective statin therapy, but a substantial risk of heart attacks still remains for treated individuals."

The researchers conclude that their results points toward a possible therapeutic approach to stabilize atherosclerotic plaques. Also, in certain patient groups treated with interleukin-17 blocking therapies for other diseases, this stabilizing pathway might be hampered, therefore these patients should be monitored carefully regarding their cardiovascular health.

More information: 'Transforming growth factor-B signaling in T cells promotes stabilization of atherosclerotic plaques through an interleukin-17 dependent pathway', Anton Gisterå, Anna-Karin Robertson, John Andersson, Daniel FJ Ketelhuth, Olga Ovchinnikova, Stefan K Nilsson, Anna M Lundberg, Ming O Li, Richard A Flavell, Göran K Hansson, *Science Translational Medicine*, online 31 July 2013.

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