T cells key in atherosclerosis-linked inflammation

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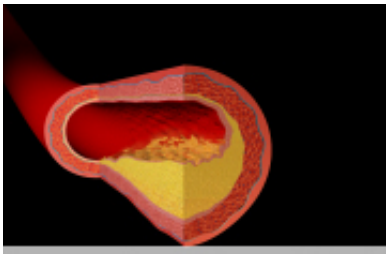
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In a mouse model of atherosclerosis, CD4+ T cells interact with antigen-presenting cells in the presence of cognate antigen, leading to cell activation and proliferation and the secretion of proinflammatory cytokines, according to an experimental study published online Aug. 13 in the *Journal of Clinical Investigation*.

(HealthDay) -- In a mouse model of atherosclerosis, CD4+ T cells interact with antigen-presenting cells (APCs) in the presence of cognate antigen, leading to cell activation and proliferation and the secretion of proinflammatory cytokines, according to an experimental study published online Aug. 13 in the *Journal of Clinical Investigation*.

Ekaterina K. Koltsova, M.D., Ph.D., of the La Jolla Institute for Allergy and Immunology in California, and colleagues compared the behavior and role of APCs in normal and atherosclerotic mice using live-cell imaging of explanted aortas.

The researchers found that, in the presence, but not the absence, of cognate antigen, CD4+ T cells were able to interact with fluorescently labeled APCs in the aortic wall. In atherosclerosis-prone mice, APCs interacted with CD4+ T cells in the aorta, which resulted in cell activation and proliferation and the secretion of cytokines (interferon-γ and tumor necrosis factor-α). Uptake of oxidized and minimally modified low-density lipoproteins was enhanced by these cytokines.

"We conclude that antigen presentation by APCs to CD4+ T cells in the arterial wall causes local T-cell activation and production of proinflammatory cytokines, which promote atherosclerosis by maintaining chronic inflammation and inducing foam cell formation," the authors write.

More information: Abstract
Full Text