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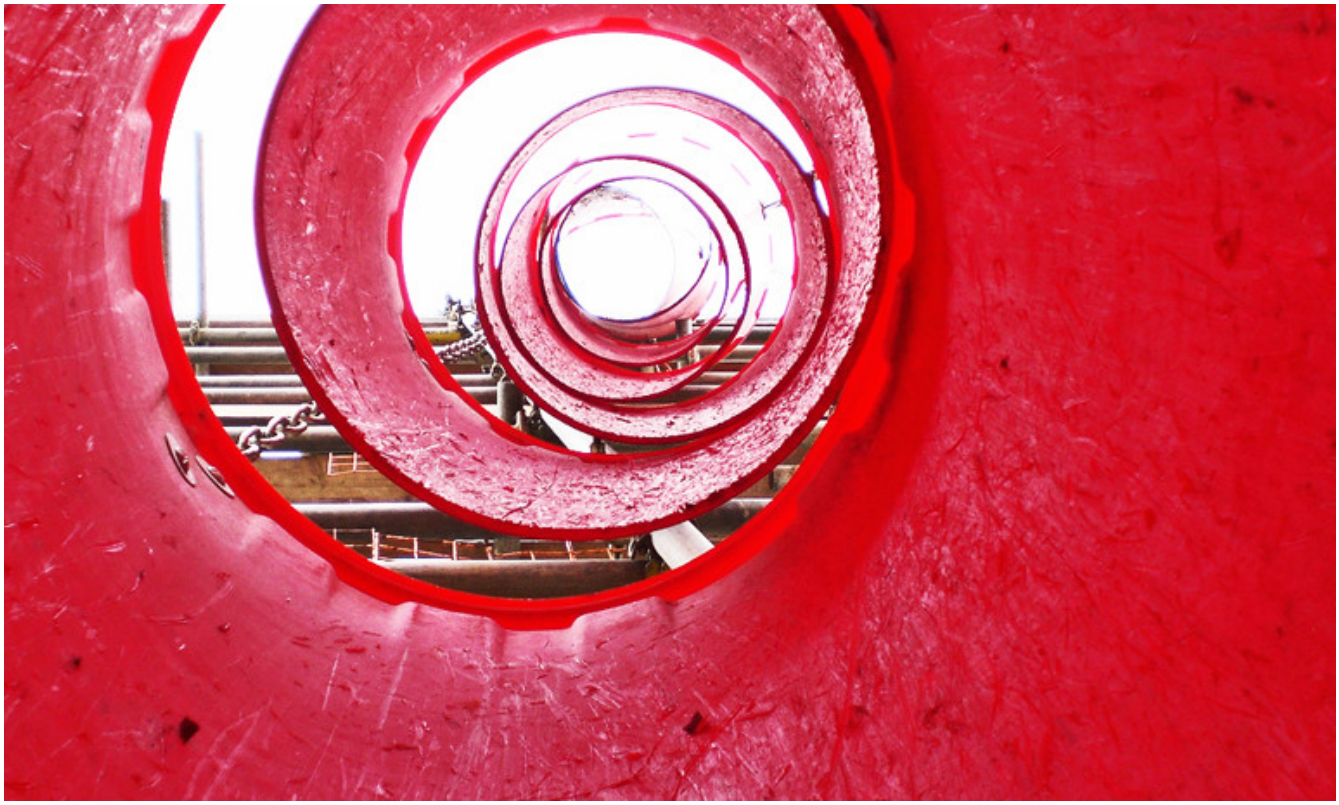

New insights into how plague grows inside arteries could help improve the current standard of care in treating atherosclerosis: statin therapy. (Credit: Ian Gallagher/Flickr)

University of Toronto *right*Original Study

Posted by Michael Kennedy-Toronto on August 28, 2013

Plaque may work from inside to clog arteries

A new understanding of how deadly plaque grows inside artery walls could affect the way doctors treat the condition.

Researchers from the University of Toronto and Massachusetts General Hospital found that macrophages, white blood cells that drive hardening of the arteries—a condition known as atherosclerosis—replicate inside plaques. This growth is not reliant on cells outside the plaques called monocytes, as scientists had assumed.

"Until now, the thinking was that inflammatory macrophages arise mainly from the recruitment of their precursors—monocytes—from the bloodstream," says Clint Robbins, lead author on the study and an Assistant Professor in the Departments of Laboratory

Medicine and Pathobiology, and Immunology. “Our study shows that the accumulation of macrophages also depends on their proliferation locally within the developing plaque.”

The effect of the research on clinical treatments could be large. Many pharmaceutical companies are pouring resources into potential therapies that can block the recruitment of white blood cells into plaques. But if macrophages self-sustain through local cell division, blocking recruitment may not be the best strategy.

“I think this work will force some major re-evaluations,” says Filip Swirski, the study’s principal investigator who is a scientist in the Center for Systems Biology at Massachusetts General Hospital and an Assistant Professor at Harvard Medical School.

“People have been thinking of targeting monocyte influx to treat atherosclerosis, but they need to consider macrophage proliferation as an additional or alternative approach, especially in established disease.”

That approach might be better than targeting circulating monocytes, since interrupting disease-causing processes within plaques could spare other beneficial immune responses that monocytes control, says Swirski.

As well, it could help improve the current standard of care in treating atherosclerosis: statin therapy. Statins, in addition to lowering blood lipids that contribute to plaque, have anti-inflammatory properties. The researchers are now looking at whether statins might limit the spread of macrophages within plaques.

“Additional targeting of macrophage proliferation may further decrease inflammation in atherosclerosis and prove clinically advantageous,” says Robbins, who is also a scientist in the Toronto General Research Institute at University Health Network.

The researchers conducted their study in mice, and they caution that much more research is needed to see how the work will translate to humans. But encouragingly, they found evidence of macrophage growth in plaques from human carotid arteries. The study is published in the journal *Nature Medicine*.

Next, the team will compare macrophage proliferation to monocyte recruitment during different stages of atherosclerosis, and look at whether all macrophages, or only subsets, replicate.

The US National Institutes of Health, the Massachusetts General Hospital, the Heart and Stroke Richard Lewar Centre of Excellence in Cardiovascular Research, and the Department of Laboratory Medicine and Pathobiology at the University of Toronto funded the study.

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