Heart disease plaques: 'theory-changing' discovery

Featured Article
Academic Journal
Main Category: Heart Disease
Also Included In: Statins; Biology / Biochemistry
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Scientists have made a new discovery on how plaques grows inside the walls of arteries, opening doors for new clinical treatments for heart disease, according to a study published in the journal *Nature Medicine*.

Researchers from the University of Toronto and Massachusetts General Hospital say the discovery opposes how scientists previously believed plaques developed within cardiovascular disease.

The scientists, who conducted their experiment on mice, found that macrophages replicate inside plaques. Macrophages are white blood cells involved in atherosclerosis, which is the leading cause of heart disease, brought on by a build up of fatty acids inside the arteries.

The researchers say that previously, it was assumed that the growth of macrophages was more to do with cells outside the plaques called monocytes.

Clint Robbins, assistant professor in the Department of Laboratory Medicine and Pathobiology, and Immunology at the University of Toronto and lead study author, explains:

> *"Until now, the thinking was that inflammatory macrophages arise mainly from the recruitment of their precursors - monocytes - from the bloodstream.*
>
> *Our study shows that the accumulation of macrophages also depends on their proliferation locally within the developing plaque."*

Finding could change research into heart drugs

The study authors say that this discovery could be important to treatment of cardiovascular disease, as many pharmaceutical companies are focusing on developing treatments to block the recruitment of white blood cells into plaques.

However, they warn that if macrophages "self-sustain" through local cell division, as this research suggests, blocking the recruitment of white blood cells into plaques may not be the best way of developing treatments.

Filip Swirski, a scientist in the Center for Systems Biology at Massachusetts General Hospital and the study's principal investigator, says:

> *"I think this work will force some major re-evaluations. 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."*

Swirski adds that this approach may be better than focusing on circulating monocytes, as interrupting processes within plaques could leave the monocytes to "other beneficial immune responses that monocytes control."

Additionally, the researchers say that this approach may help improve the widespread use of statin therapies against atherosclerosis.

The scientists are now investigating whether statins could be used to reduce the spread of microphages within plaques.

The study authors warn that there is much more research needed to see how their finding in mice can relate to humans, but note that they did find evidence of macrophage growth of plaques in human carotid arteries.

They add that the next step will be to test their findings at different stages of atherosclerosis. They also aim to see whether all macrophages replicate, or only subsets.

Written by Honor Whiteman

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Visitor Opinions (latest shown first)

Why Test Statins?

*posted by **John** on 13 Aug 2013 at 5:08 am*

Oh, the money. Big Pharma must be funding this research.

I'm curious about other unwanted cell proliferation diseases -- blood cell growth as found in proliferative retinopathy and cancer are two that come to mind. A growing suspect in these cases is sugar. Could this be the link to sugar as the cause of modern heart disease as many of us uneducated lay people suspect?

Yes, test those statins. Cholesterol suppression is obviously linked to cell proliferation.

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