

Aspirin and atherosclerosis

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Aspirin has become one of the most widely used medications in the world, owing to its ability to reduce pain, fevers, inflammation, and blood clotting. In animal studies, aspirin has also been shown to prevent atherosclerosis, though none of its known mechanisms of action would seem to account for this. In a new study, though, researchers have uncovered the mechanism that may explain aspirin's ability to prevent arterial plaque buildup.

Using cell culture and mouse models, Sampath Parthasarathy and colleagues observed that aspirin –specifically its active byproduct salicylate– can greatly increase the expression of two proteins: paraoxonase 1 (PON1) and apolipoprotein A1 (ApoA1); in the mouse studies, low dose aspirin supplements could increase PON1 and ApoA1 levels by 7- and 12- fold, respectively.

Both of these proteins are beneficial components of the HDL complex, the "good cholesterol" that helps prevent atherosclerosis; ApoA1 removes bad cholesterol from the bloodstream while PON1 is an antioxidant that breaks down toxic lipid peroxides.

The researchers also noted that the heightened expression of PON1 was accompanied by an increase in a receptor called AHR (aryl hydrocarbon receptor); this was intriguing as a chemical known to attach to AHR is resveratrol, the "heart healthy" component of red wine.

This study is online in the October issue of *Journal of Lipid Research*. Article link: <u>www.jlr.org/cgi/content/full/49/10/2188</u>



Source: American Society for Biochemistry and Molecular Biology

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