

Improved insulin signaling reduces atherosclerosis in mouse models

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Patients with diabetes and metabolic syndrome are at increased risk of atherosclerosis and subsequent heart disease. It is not fully understood why atherosclerosis is increased with diabetes, but it has been proposed that insulin resistance in endothelial cells, which line blood vessels, promotes inflammation.

In this issue of *JCI Insight*, a team led by George King of Harvard Medical School shows that increased insulin signaling in the endothelium of atherosclerosis-prone mice reduces development of disease and improves aorta function.

The anti-atherogenic effects of insulin were linked to induction of nitric oxide and activation of downstream pathways, including endothelin receptor B (EDNRB). EDNRB was decreased in arteries from patients with diabetes.

In a murine model, loss of EDNRB accelerated atherosclerosis, even in animals with enhanced insulin signaling.

Together, the results of this study suggest that strategies to improve insulin signaling in [endothelial cells](#) have the potential to reduce diabetes-associated atherosclerosis.

More information: Kyoungmin Park et al, Insulin decreases atherosclerosis by inducing endothelin receptor B expression, *JCI Insight* (2016). [DOI: 10.1172/jci.insight.86574](https://doi.org/10.1172/jci.insight.86574)

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