

Metformin's potential role in atherosclerosis explored

9 January 2015



Image courtesy of Blausen Medical

causing <u>inhibition</u> of the differentiation of monocytes to macrophages. In ApoE^{?/?} mice, metformin attenuated Ang-II-induced atheromatous plaque formation and aortic aneurysm, partly by decreasing infiltration of monocytes.

"We conclude that AMPK-STAT3 axis plays a pivotal role in regulating monocyte-to-macrophage differentiation and that AMPK activators by decreasing STAT3 phosphorylation through increased AMPK activity inhibit monocyte-tomacrophage <u>differentiation</u>," the authors write.

More information: Abstract

Full Text (subscription or payment may be required)

(HealthDay)—Metformin's role in atherosclerosis may be inhibition of monocyte-to-macrophage differentiation via AMPK-mediated inhibition of STAT3 activation, according to research published online Dec. 31 in *Diabetes*.

Sathish Babu Vasamsetti, from the Indian Institute of Chemical Technology in Hyderabad, and colleagues examined the molecular mechanisms responsible for monocyte-to-macrophage differentiation. They also examined the effect of metformin in regressing Angiotensin-II-mediated atheromatous plaque formation in ApoE^{?/?} mice.

The researchers observed a dose- and timedependent downregulation in AMPK activity during PMA-induced monocyte-to-macrophage differentiation; this was accompanied by upregulation of production of pro-inflammatory cytokines. These were significantly attenuated with AMPK activators metformin and AICAR. In the absence of PMA, compound C-induced inhibition of AMPK activity alone was not effective for promoting monocyte-to-macrophage differentiation. JNK activity inhibited the inflammation induced by PMA, but not differentiation. Both inflammation and monocyte-to-macrophage differentiation were inhibited with inhibition of STAT3 activity. Increased AMPK activation with metformin and AICAR decreased STAT3 phosphorylation,

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APA citation: Metformin's potential role in atherosclerosis explored (2015, January 9) retrieved 30 April 2021 from <u>https://medicalxpress.com/news/2015-01-metformin-potential-role-atherosclerosis-explored.html</u>

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