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Authors: Jisu Oh, Amy E. Riek, Rong M. Zhang, Samantha  
A.S. Williams, Isra Darwech, Carlos Bernal-Mizrachi



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## Deletion of JNK2 prevents vitamin-D-deficiency-induced hypertension and atherosclerosis in mice

Jisu Oh<sup>a</sup>, Amy E. Riek<sup>a</sup>, Rong M. Zhang<sup>a</sup>, Samantha A.S. Williams<sup>a</sup>, Isra Darwech<sup>a</sup> and Carlos Bernal-Mizrachi<sup>a,b,c</sup>

<sup>a</sup> Division of Endocrinology, Metabolism, and Lipid Research, Washington University, 660 South Euclid Ave., Campus Box 8127, St. Louis, MO 63110, U.S.A.; email: [joh@wustl.edu](mailto:joh@wustl.edu), [aeriek@wustl.edu](mailto:aeriek@wustl.edu), [rongmeizhang@wustl.edu](mailto:rongmeizhang@wustl.edu), [swilliams32@wustl.edu](mailto:swilliams32@wustl.edu), [idarwech@wustl.edu](mailto:idarwech@wustl.edu), [cbernal@wustl.edu](mailto:cbernal@wustl.edu)

<sup>b</sup> Division of Endocrinology, Metabolism, and Lipid Research, Department of Cell Biology and Physiology, Washington University, 660 South Euclid Ave., Campus Box 8127, St. Louis, MO 63110, U.S.A.; email: [cbernal@wustl.edu](mailto:cbernal@wustl.edu)

<sup>c</sup> Division of Endocrinology, Saint Louis VA Medical Center, 915 N Grant Blvd, Saint Louis, MO, 63106.; email: [carlos.bernal\\_mizrachi@va.gov](mailto:carlos.bernal_mizrachi@va.gov)

To whom correspondence should be addressed: Carlos Bernal-Mizrachi, Division of Endocrinology, Metabolism, and Lipid Research, Department of Internal Medicine, Washington University School of Medicine, 660 South Euclid Avenue, Campus Box 8127, St. Louis, MO 63110, U.S.A.; tel.: 1-(314)362-0947; fax: 1-(314)362-7641; email: [cbernal@dom.wustl.edu](mailto:cbernal@dom.wustl.edu)

### Highlights:

- JNK2 signaling links vitamin D deficiency to angiotensin-II-mediated hypertension
- JNK2 activation is necessary for vitamin D deficiency to induce atherosclerosis
- Deletion of JNK2 prevents vitamin-D-induced foam cell formation
- Vitamin-D-deficiency-induced pro-inflammatory monocyte phenotype is JNK2-dependent

### Abstract

The c-Jun N-terminal kinase 2 (JNK2) signaling pathway contributes to inflammation and plays a key role in the development of obesity-induced insulin resistance and cardiovascular disease. Macrophages are key cells implicated in these metabolic abnormalities. Active vitamin D downregulates macrophage JNK activation, suppressing oxidized LDL cholesterol uptake and foam cell formation and promoting an anti-inflammatory phenotype. To determine whether deletion of JNK2 prevents high blood pressure and atherosclerosis known to be induced by vitamin D deficiency in mice, we generated mice with knockout of JNK2 in a background

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