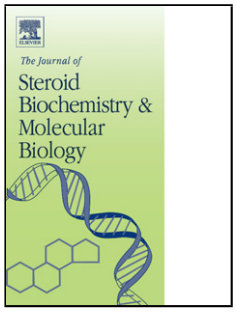

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Deletion of JNK2 prevents vitamin-D-deficiency-induced hypertension and atherosclerosis in mice

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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