Myeloid CD11c(+) Antigen-Presenting Cells Ablation Prevents Hypertension in Response to Angiotensin II Plus High-Salt Diet

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Resumen

Increasing evidence shows that antigen-presenting cells (APCs) are involved in the development of inflammation associated to hypertension. However, the potential role of APCs in the modulation of renal sodium transport has not been addressed. We hypothesized that APCs participate in renal sodium transport and, thus, development of high blood pressure in response to angiotensin II plus a high-salt diet. Using transgenic mice that allow the ablation of CD11c(high) APCs, we studied renal sodium transport, the intrarenal renin-angiotensin system components, blood pressure, and cardiac/renal tissue damage in response to angiotensin II plus a high-salt diet. Strikingly, we found that APCs are required for the development of hypertension and that the ablation/restitution of APCs produces rapid changes in the blood pressure in mice with angiotensin II plus a high-salt diet. Moreover, APCs were necessary for the induction of intrarenal renin-angiotensin system components and affected the modulation of natriuresis and tubular sodium transporters. Consistent with the prevention of hypertension, the ablation of APCs also prevented cardiac hypertrophy and the induction of several indicators of renal and cardiac damage. Thus, our findings indicate a prominent role of APCs as modulators of blood pressure by mechanisms including renal sodium handling, with kinetics that suggest the involvement of tubular cell functions in addition to the modulation of inflammation and adaptive immune response.

Palabras clave

Palabras clave de autor:angiotensin II; antigen-presenting cells; epithelial sodium channel; hypertension; inflammation

KeyWords Plus:RENAL SODIUM TRANSPORTERS; PROXIMAL TUBULAR CELLS; VASCULAR DYSFUNCTION; DENDRITIC CELLS; T-LYMPHOCYTES; PRESSURE CONTROL; ORGAN DAMAGE; IFN-GAMMA; MICE; KIDNEY

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