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Deficiency of exocyst component Sec10 in myeloid cells accelerates hypertension

Gaeun Yoon, JeeIn Kim
Department of Biochemistry & Molecular Biology, Keimyung University School of Medicine, Korea, Republic of

Objectives: Hypertension is a complex condition contributing to world-wide premature death. Approximately 95% of hypertension cases are classified as essential hypertension, of which cause is unknown. Hypertension is closely associated with inflammation; however, whether the inflammation is a cause or effect of hypertension is not well understood. Extracellular vesicle secretion correlates with inflammasome activity. The purpose of this study is to investigate the effect of exocyst Sec10-defficiency in bone marrow-derived myeloid cells in development of hypertension through renal inflammation and angiotensin II expression. Sec10 is known to be a central component of the exocyst complex essential for exocytosis, which plays a critical role in biological processes such as cell growth, cell-cell communication, and cell migration.

Methods: Twenty-one to twenty-nine week-old female myeloid-specific sec10-deficient (Lyz-Sec10 KO) mice and their wild type littermates were used for animal experiments. Body weights and blood pressures (BP) were measured. Expression of pro-inflammatory cytokines, angiotensinogen adhesion molecules, and sodium transporters in the kidney were determined by QPCR or western blot analysis. Ruffle formation and migration of bone marrow-derived macrophages (BMM) were determined.

Results: Lyz-Sec10 KO mice showed higher BP, greater renal inflammation than wild type mice. Lyz-Sec10 KO mice accelerated BMM ruffle formation and migration. Lyz-Sec10 KO mice increased serum Ang II concentration and renal sodium transporter expression.

Conclusions: Conclusively, deficiency of exocyst Sec10 in myeloid cells induces hypertension through increased renal inflammation and serum Ang II by enhanced migration of BMM.