KSN 2020
FULLY VIRTUAL MEETING

Abstract Submission No. : IL-9043

Role for immunity in salt-sensitive hypertension

Inkyeom KIM
School of Medicine, Kyungpook National University, Korea, Republic of

High-salt or high-fructose intakes are risk factors for hypertension via oxidative stress and inflammation. T helper (Th)17 lymphocytes play an important role in the development of salt-sensitive hypertension. We tested the hypothesis that activation of pathogenic Th17 lymphocytes induces hypertension after high-salt or high-fructose intake in Dahl salt-sensitive (SS) but not Dahl salt-resistant (SR) rats.
Eight-week-old male SS and SR rats were offered 20% fructose solution or tap water only for 4 weeks. Systolic blood pressure was measured by the tail-cuff method. T lymphocytes (Th17 and T regulatory (Treg)) profiling was determined via flow cytometry. The expression of Th17 -related (interleukin (IL)-17A, IL-17RA,IL-23R and retinoic acid receptor-related orphan receptor (ROR) γt) and Treg-related (IL-10, CD25, forkhead box (Fox) P3, and TGF-ß) factors were measured via ELISA or qRT-PCR. Th17 lymphocytes isolated from high fructose-fed SS rats were intraperitoneally injected into recipient SS and SR rats. Moreover, recombinant IL-23 protein was subcutaneously injected into SS and SR rats to induce hypertension. High-fructose intake induced hypertension via the activation of pathogenic Th17 lymphocytes in SS but not SR rats. Injection of activated Th17 lymphocytes isolated from fructose-fed SS rats induced hypertension via increase of serum IL-17A in only recipient SS rat. In addition, injection of IL-23 induced hypertension via activation of pathogenic Th17 lymphocytes in only SS rats.
Activation of pathogenic Th17 lymphocytes induces hypertension after high-fructose intake in SS but not SR rats. These results implicate that immunologic tolerance plays an important role in the protection against hypertension in SR.