



Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care

KSN SEOUL, KOREA
2026

The 46th Annual Meeting of the Korean Society of Nephrology



Jun 11 (Thu) – 14 (Sun), 2026

COEX, Seoul, Korea

Abstract Type : Poster exhibition

Abstract Submission No.: A-0973

Abstract Topic : Basic Research

Nicotine And High-Salt Diet Synergistically Dysregulate Kidney Immune Microenvironment And Impair Vascular Endothelial Function

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Objectives : Smoking and high-salt diet are independent risk factors for kidney disease and may synergistically amplify kidney injury. However, their combined effects on renal immune microenvironment and vascular endothelial cells remain poorly understood. We investigated immunological and endothelial consequences of concurrent nicotine and high-salt exposure.

Methods : Mice were divided into four groups based on nicotine exposure and high-salt diet feeding and sacrificed at 2 and 4 weeks. Additional groups underwent 4-week nicotine exposure followed by 2-week cessation or continuous exposure. Nicotine was administered via intraperitoneal injection. Kidney mononuclear cells were isolated and analyzed by flow cytometry. Induced pluripotent stem cell-derived endothelial cells (iPSC-ECs) were treated with nicotine (50, 100µM) or high-salt conditions (165mM NaCl) under normoxia or hypoxia to assess proliferation, tube formation, and reactive oxygen species (ROS) generation.

Results : High-salt diet increased macrophage infiltration at 2 weeks compared to control group. At 4 weeks, nicotine plus high-salt diet group showed increased mature B cells and decreased IgM⁺IgD⁻ B cells. Regulatory CD4⁺ T cells were significantly reduced by nicotine plus high-salt diet exposure at 2 weeks, while CD4⁺ effector memory T cells expanded significantly at 4 weeks. Prolonged nicotine exposure (6 weeks) increased B1-a cells and reduced naïve CD4⁺ T cells. Regarding endothelial tube formation, nicotine treatment at 100 µM significantly reduced the number of tube junctions in iPSC-ECs with the greatest mesh size, indicating disrupted angiogenic maturation, compared to vehicle and 50µM groups. High-salt conditions (165mM NaCl) suppressed proliferation and elevated ROS under both normoxic and hypoxic conditions via ionic rather than osmotic mechanisms.

Conclusions : Combined nicotine and high-salt diet exposure promotes renal immune dysregulation through accelerated B cell maturation, regulatory T cell depletion, and effector memory T cell expansion, alongside impaired endothelial tube formation and increased oxidative stress, supporting simultaneous smoking cessation and dietary salt restriction for kidney disease prevention.