



Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care

KSN SEOUL, KOREA
2026

The 46th Annual Meeting of the Korean Society of Nephrology

THE KOREAN SOCIETY
OF NEPHROLOGY

Jun 11 (Thu) – 14 (Sun), 2026

COEX, Seoul, Korea

Abstract Type : Poster exhibition

Abstract Submission No.: A-0971

Abstract Topic : Dialysis

In Vivo Evidence of p-Cresyl Sulfate-Induced Neointimal Hyperplasia in a Murine Model

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Objectives : Vascular access failure remains a major cause of morbidity in hemodialysis patients, primarily driven by smooth muscle cell (SMC) proliferation-related neointimal hyperplasia. The uremic toxin p-cresyl sulfate (p-CS) has been implicated in poor vascular access outcomes. Extending our previous ex vivo findings, this study aimed to demonstrate the direct role of p-CS in inducing neointimal hyperplasia in vivo using a murine model.

Methods : C57BL/6 mice underwent either a subtotal nephrectomy (1/2 Nx) or a sham operation at 8 weeks of age. Subsequently, the mice received intraperitoneal injections of p-CS for 4 weeks, after which they were sacrificed. The extent of neointimal hyperplasia in the thoracoabdominal aorta was then assessed. The mice were assigned to the following groups: sham (n = 7), 1/2 Nx + p-CS (n = 10), 1/2 Nx + p-CS + N-acetylcysteine (NAC) (n = 8), 1/2 Nx + p-CS + probenecid (n = 8), and 1/2 Nx + probenecid (n = 8).

Results : The cross-sectional area of neointimal hyperplasia in the thoracoabdominal aorta was significantly larger in the 1/2 Nx + p-CS group compared to the sham control group. Notably, concurrent treatment with either NAC or probenecid effectively prevented this p-CS-induced neointimal expansion.

Conclusions : Our in vivo findings confirm that p-CS directly induces neointimal hyperplasia. Further studies are warranted to explore the clinical implications of targeting p-CS to prevent vascular access dysfunction.

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