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Role of p-Cresyl Sulfate in Neointimal Hyperplasia and Vascular Dysfunction: Evidence from a Murine Model

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Objectives : Vascular access failure is a leading cause of hospitalization and morbidity in patients undergoing hemodialysis, primarily due to neointimal hyperplasia resulting from vascular smooth muscle cell (SMC) proliferation. Our preliminary research demonstrated that p-Cresyl sulfate (p-CS) directly induces SMC proliferation and promotes oxidative stress and endothelial dysfunction, leading to neointimal hyperplasia. This study aimed to further investigate whether p-CS promotes SMC proliferation and endothelial dysfunction in a mouse model.
Methods : C57 mice underwent either a 1/2 nephrectomy (1/2 NR) or a sham operation at 8 weeks of age, followed by p-CS injections for 4 weeks. The extent of neointimal hyperplasia was assessed at 20 weeks in 7 groups: sham (n = 7), 1/2 NR + p-CS (n = 10), 1/2 NR + p-CS + N-acetylcysteine (NAC) (n = 8), 1/2 NR + p-CS + probenecid (n = 8), and 1/2 NR + probenecid (n = 8).
Results : The neointimal hyperplasia area of the thoracoabdominal aorta was significantly larger in the 1/2 NR + p-CS group compared to the sham group, while NAC and probenecid treatment effectively prevented this increase.
Conclusions : Our findings confirm that p-CS directly induces neointimal hyperplasia in a mouse model. Further studies are warranted to explore the clinical implications of p-CS in vascular access dysfunction.