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Novel Therapeutic Target for Abdominal Aortic Aneurysm (AAA)

Abdominal aortic aneurysm (AAA) is a life-threatening disease with no effective pharmacological therapy today. The mechanisms underpinning AAA pathogenesis remain poorly understood, and current therapy is limited to open or endovascular surgical repair. Biomechanical factors play a significant role in the development and progression of AAA, where the aortic wall stress fails to withstand the intraluminal hemodynamic forces. The aortic wall stress is closely regulated by the perivascular sympathetic nervous system (SNS), where the sympathetic innervation of vascular smooth muscle cells (VSMCs) results in the contractile forces within the aortic wall. Dysfunction in the perivascular innervation to the abdominal aorta could be a critical contributor to AAA pathogenesis that leads to the progressive dilation and destruction of the aortic wall, a potential mechanism that has not been previously explored for AAA. We hypothesize that the formation of AAA is initiated by the loss of functional sympathetic innervation to the abdominal aortic smooth muscles, resulting in the dysregulation of aortic biomechanics, VSMC phenotype switching and ECM degradation. Interventions that provide the abdominal aorta with functional perivascular SNS signals through pharmacological treatment or re-innervation could potentially attenuate this process by maintaining aortic VSMC contractile phenotype and elasticity. In this talk, I will introduce our animal surgical models of AAA and the development of novel vascular devices to combat AAA. The expected contribution is the establishment of a new mechanism for AAA pathogenesis with the development of novel disease models, which will ultimately lead to new therapeutic targets and effective treatment strategies.