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PO-039 - (23792) - THE ROLE OF AROMATIC L-AMINO ACID DECARBOXYLASE IN VASCULAR SMOOTH MUSCLE CELLS

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Aromatic L-amino acid decarboxylase (AADC) is a key enzyme in tryptophan metabolism, producing essential neurotransmitters like serotonin, dopamine, norepinephrine, and epinephrine. While AADC has been studied primarily in the neuronal system, its role in blood vessels remains unclear. This study investigates AADC function in vascular smooth muscle cells (VSMCs) and its involvement in phenotypic switching during vascular disease progression.
Primary VSMCs were isolated and stimulated with IL-1β to evaluate AADC levels by Western blot. VSMCs were transfected with pCMV6-AADC-GFP for overexpression or treated with siRNA-mediated knockdown (siAADC) for knockdown experiments. Mice underwent femoral artery guide wire injury, and arteries were harvested after four weeks. AADC expression was assessed by immunohistochemistry.

Western blot showed AADC was present in VSMCs under basal conditions and upregulated by IL-1β. AADC overexpression reduced contractile markers (SM22α, SM α-actin, SM-MHC), while AADC knockdown attenuated IL-1β-induced proliferation. Arterial injury increased AADC expression levels in medial and neointimal VSMCs, indicating a role of AADC in phenotypic switching and vascular remodeling.

Our study delineates a novel role of AADC in arterial injury response. Through gain- and loss-of-function experiments, we demonstrate that AADC regulates VSMC phenotypic switching and proliferation. These findings unveil an unrecognized function of AADC in vascular biology and its potential as a therapeutic target for vascular disease.

Palavras-chave : aromatic L-amino acid decarboxylase, vascular smooth muscle cells, tryptophan metabolism