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Resumo	<p>Background: Patients with heart failure (HF) display erectile dysfunction (ED). However, the pathophysiology of ED during HF remains poorly investigated.</p> <p>Objective: This study aimed to characterize the aortocaval fistula (ACF) rat model associated with HF as a novel experimental model of ED. We have undertaken molecular and functional studies to evaluate the alterations of the nitric oxide (NO) pathway, autonomic nervous system and oxidative stress in the penis.</p> <p>Methods: Male rats were submitted to ACF for HF induction. Intracavernosal pressure in anesthetized rats was evaluated. Concentration-response curves to contractile (phenylephrine) and relaxant agents (sodium nitroprusside; SNP), as well as to electrical field stimulation (EFS), were obtained in the cavernosal smooth muscle (CSM) strips from sham and HF rats. Protein expression of endothelial NO synthase (eNOS) and neuronal NO synthase (nNOS) and phosphodiesterase-5 in CSM were evaluated, as well as NOX2 (gp91phox) and superoxide dismutase (SOD) mRNA expression. SOD activity and thiobarbituric acid reactive substances (TBARs) were also performed in plasma.</p> <p>Results: HF rats display erectile dysfunction represented by decreased ICP responses compared to sham rats. The neurogenic contractile responses elicited by EFS were greater in CSM from the HF group. Likewise, phenylephrine-induced contractions were greater in</p>



CSM from HF rats. Nitregic response induced by EFS were decreased in the cavernosal tissue, along with lower eNOS, nNOS and phosphodiesterase-5 protein expressions. An increase of NOX2 and SOD mRNA expression in CSM and plasma TBARs of HF group were detected. Plasma SOD activity was decreased in HF rats.

Conclusion: ED in HF rats is associated with decreased NO bioavailability in erectile tissue due to eNOS/nNOS downregulation and NOX2 upregulation, as well as hypercontractility of the penis. This rat model of ACF could be a useful tool to evaluate the molecular alterations of ED associated with HF.

Fomento