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Resumo	Heart failure (HF) is the final common pathway of many cardiovascular diseases. Metalloproteinases and their inhibitors, such as MMP9 and TIMP-1, assist in maintaining the extracellular matrix, leading to tissue remodeling observed after HF. Previous studies have shown that L-Arginine (LA) appears to have beneficial effects for the treatment of HF, contributing to vasodilation, the reestablishment of the endothelial function and an increase in muscle contractile force. This study analyzed heart tissue remodeling in an animal model of HF induced by aortocaval fistula (ACF) and submitted to LA treatment. After 4 weeks of ACF, animals were treated with LA for 4 weeks (SHAM-LA, HF-LA) or for 8-12 weeks with saline (SHAM, HF8, HF12). Rats were euthanized and the hearts removed for histological processing. The samples were stained with Hematoxylin-Eosin (HE), Masson's Thichome (MT), or submitted to immunohistochemistry (IHC) for MMP9 and TIMP-1. Light microscopy analysis showed cardiac striated muscle without fibrosis in all experimental groups. Immunostaining of MMP9 and TIMP-1 were positive for all experimental groups. LA administration significatively reduced MMP9 content after HF. These data indicate molecular changes in metalloproteinases expression prior to tissue remodeling and point out LA as an adjuvant therapy to pharmacological treatment of patients with HF.
Fomento	