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Resumo	<p>Kynurenic acid (KYNA) is derived from tryptophan, formed by the kynurenic pathway. KYNA is being widely studied as a biomarker for neurological and cardiovascular diseases, as it is found in ischemic conditions as a protective agent; however, little is known about its effect after ischemia-reperfusion in the vascular system. We induced ischemia for 30 min followed by 5 min reperfusion (I/R) in the rat aorta for KYNA evaluation using functional assays combined with proteomics. KYNA recovered the exacerbated contraction induced by phenylephrine and relaxation induced by acetylcholine or sodium nitroprussiate in the I/R aorta, with vessel responses returning to values observed without I/R. The functional recovery can be related to the antioxidant activity of KYNA, which may be acting on the endothelium-injury prevention, especially during reperfusion, and to proteins that regulate neurotransmission and cell repair/growth, expressed after the KYNA treatment. These proteins interacted in a network, confirming a protein profile expression for endothelium and neuron repair after I/R. Thus, the KYNA treatment had the ability to recover the functionality of injured ischemic-reperfusion aorta, by tissue repairing and control of neurotransmitter release, which reinforces its role in the post-ischemic condition, and can be useful in the treatment of such disease.</p>
Fomento	-