NON-NEURONAL CHOLINERGIC SYSTEM IN RAT AORTA

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We have showed before that both cholinesterases are present in rat aorta, while inhibition of butyrylcholinesterase impairs physiology of the isolated organ. The endothelium-dependent vasodilatory effect of acetylcholine (ACh) on vessels is well known, but physiological or pathological importance of this effect in live animals is questionable, and origin of possibly acting ACh unclear. Hypothesizing that aorta is a non-neuronal cholinergic tissue, the main aim of this project was to examine the presence of the proteins involved in the synthesis, storage, release, and degradation of ACh. Target-specific primers were used in RT-qPCR for determination of relative expressions and proteins were visualized by immunohistochemistry using commercially available antibodies. We confirmed the presence of high-affinity transporter and vesicular acetylcholine transporter in aorta, but no choline acetyltransferase was detected. Instead, relatively high levels of carnitine acetyltransferase were observed thus we assume this enzyme to be responsible for ACh synthesis in aorta. Additionally, present organic cation transporters OCT2 and OCT3 (but not OCT1) suggest possible involvement in ACh transmembrane transport. We confirmed the presence of both cholinesterases in rat aorta, more precisely in the smooth muscle, while no protein or activity was detected in the endothelium. Our results confirm aorta to be a non-neuronal cholinergic tissue carrying a full machinery for synthesis, storage and release and degradation of ACh.

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