ABSTRACT

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	<i>vivo</i> mechanism of action

It is known from clinical studies that the intake of polyphenols from food acts as a prevention of cardiovascular diseases. However, the parent compounds themselves often have low bioavailability. The emphasis is therefore on their bioactive metabolites. 3-hydroxy phenylacetic acid (3-HPAA) is among such metabolites. The aim of this study was to verify the mechanism of the vasodilatory effect of 3-HPAA using standardized *ex vivo* method on an isolated pig coronary artery. The administration of 3-HPAA resulted in relaxation of maximally contracted segments of porcine artery by mechanism partially dependent on the integrity of endothelium. By inhibiting endothelial nitric oxide synthase, the relaxation was significantly impaired. The blockage of SK_{Ca} and IK_{Ca} channels, muscarinic receptors, cyclooxygenase or L-type calcium channels did not affect relaxation. Thus, 3-HPPA causes dose-dependent vasodilatation of nitric oxide.

Key words: 3-hydroxy phenylacetic acid, ex vivo, vessel, vasodilatation, mechanism