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The contribution of adenosine to free flow exercise hyperaemia

Adenosine (ADO) is a potent endogenous vasodilator, and is known to contribute significantly to metabolic vasodilatation in the coronary circulation. The present investigation was undertaken to determine the role and contribution of ADO to exercise hyperaemia under free-flow condition in gracilis muscles in anaesthetized dogs by using adenosine deaminase (ADA), which converts ADO to vasoinactive inosine.

The dogs (19.5-31.5 kg, n=10) were maintained under deep surgical anaesthesia by sodium pentobarbitone (40 mg kg^{-1} iv), and artificially ventilated. The gracilis muscle was vascularly isolated and perfused under systemic blood pressure through an extracorporeal perfusion circuit and muscular branches with blood from the cardiac end of the femoral artery. Electrical stimulation of the cut end of the obturator nerve (4 Hz, 6V, 0.2 ms) resulted in muscle contractions and an immediate increase in blood flow with a concomitant fall in arterial perfusion pressure. The solvent (control) or ADA (8 U min^{1}) was infused into the arterial limb of the perfusion circuit (n=10; mean $\pm$ SE; Paired t test was used for statistical comparisons).

The vascular conductance (VC) increased from 0.14 $\pm$ 0.01 to 0.39 $\pm$ 0.04 mm Hg mL^{-1} min^{-1} 100 g^{-1} (184.62 $\pm$ 25.1%) at the 7^{th} minute of muscle stimulation in the presence of the solvent, and from 0.17 $\pm$ 0.01 to 0.41$\pm$ 0.04 mm Hg^{-1} mL^{-1} min^{-1}1oo g^{-1} (141.25 $\pm$ 19.4 %; P<0.01) in the presence of ADA. The ADA attenuated 23.06 $\pm$ 3.7 %of the VC at the 7^{th} minute of muscle contractions. However, at the 10^{th} second of muscle stimulation, in the presence of the solvent, the VC increased from 0.14 $\pm$ 0.05 to 0.29$\pm$ 0.04 mm Hg^{-1} mL^{-1} min^{-1}1oo g^{-1} (108.8 $\pm$ 22.1% increase) in the presence of ADA (P,N,S,).

These results have demonstrated that ADO does not contribute to the initiation of exercise vasodilation, but plays a significant role contributes (about 25%) to the mediation of sustained exercise hyperaemia under free flow condition. These observations also confirm that other factor/s are responsible for the initiation of exercise hyperaemia under free flow condition.