Vasodilatory effect of pentoxifylline in isolated equine digital veins

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Résumé en anglais	The direct vasodilatory action of pentoxifylline (1-(5-oxohexyl)-3,7-dimethylxanthine) and its signalling pathway was evaluated in equine digital veins. Cumulative concentration-response curves to pentoxifylline (1 nM to 300 μM) were recorded in phenylephrine-precontracted equine digital vein rings under different experimental conditions. Relaxation to pentoxifylline was partially inhibited by endothelium removal, but was unaltered by CGS-15943 (a non-xanthine adenosine receptor antagonist; 3 μM). Nitric oxide synthase (NOS), soluble guanylate cyclase and cyclooxygenase (COX) inhibitors (Nω-nitro-L-arginine methyl ester (100 μM), ODQ (30 μM) and indomethacin (10 μM), respectively) significantly reduced the maximum relaxation induced by pentoxifylline. Moreover, pentoxifylline-induced relaxation was strongly reduced by Rp-8-Br-PET-cyclic guanosine monophosphate-S (a protein kinase G inhibitor; 3 μM), but remained unaffected by H-89 (a protein kinase A inhibitor; 2 μM). Pentoxifylline-induced relaxation was associated with a 3.4-fold increase in tissue cGMP content. To investigate whether pentoxifylline can affect cAMP- and cGMP-mediated relaxations, curves to forskolin, to sodium nitroprusside (SNP) and 8-bromo-cGMP were also recorded in endothelium-denuded equine digital vein rings pretreated with pentoxifylline (10 and 100 μM). Pentoxifylline only potentiated the SNP-mediated relaxation at the highest concentration (100 μM). Thus, pentoxifylline relaxed equine digital veins via endothelium-dependent and endothelium-independent components. The effect was mediated through both the NOS and COX pathways and could also result from inhibition of cGMP specific-phosphodiesterase activity at the highest concentrations used.

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