Hydrogen sulphide and cardiovascular homeostasis

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In the mammalian cardiovascular system, H_2S joins carbon monoxide (CO) and endothelial derived relaxing factors, (EDRFs)-nitric oxide (NO), as the third gasotransmitter. In the vasculature, cystathionine-γ-lyase (CSE) is the main enzyme responsible for H_2S biosynthesis starting from the substrate e.g. L-cysteine. There is a growing body of evidence that supports a role for H_2S in regulating the vascular homeostasis. H_2S (NaHS) is known to induce a concentration-dependent relaxation of large conduit arteries. Interestingly, H_2S also relaxes peripheral resistance vessels such as mesenteric arteries suggesting a role for H_2S also in the regulation of vascular resistance and systemic blood pressure. This vasodilatory effect is dependent on the activation of K_{ATP} channels. However, a cross-talk exists between the L-Argine/NO and L-cysteine/H_2S pathways. Furthermore, it has been shown that H_2S acts as an endogenous non-selective inhibitor of phosphodiesterase activity. Compelling evidence involves the H_2S pathway also in heart failure. Despite the rapid growth of the field, it should be noted that several aspects of H_2S physiology in the cardiovascular system remain unsolved and the lack of reliable inhibitors and donors remains a major limitation.