HYDROGEN SULPHIDE INVOLVEMENT IN VASCULAR TONE IN DIABETIC RATS

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Recently, in the scenery of mediators involved in the regulation of vascular tone, prominently a novel gastrasmitter is coming up, the hydrogen sulphide (H_2S). H_2S is produced endogenously from L-cysteine by cysthationine β-synthase (CBS) and cysthationine-γ-lyase (CSE). Intravenous *bolus* of H_2S caused a transient but significant decrease in arterial blood pressure in the rat, effect antagonized by glibenclamide, an ATP-dependent potassium channels (K_{ATP}) inhibitor [1]. In the same manner, in a *in vitro* study, H_2S induced a dose-dependent relaxation by acting on K_{ATP} channels in the rat aorta [1]. The same results were observed in the mesenteric artery bed but in this case the H_2S-mediated vasodilation was also partially mediated by endothelium-derived hyperpolarazing factor (EDHF) [2]. H_2S seems to be implicated in diabetes, in fact it has been demonstrated that H_2S formation is increased in liver and pancreas in diabetic rats [3]. We investigated the biosynthesis of H_2S in. mesenteric bed and aorta, comparing the physiological *status* to a streptozotocin (STZ)-induced diabetes in rats. Hence, we evaluated the vascular response to H_2S by using sodium-hydrogen sulphide (NaHS, stable donor of H_2S) in both tissue and its implication with EDHF in diabetic rats. Here, we reported that in diabetic rats H_2S production was reduced in diabetic rats in both vascular district. In mesenteric bed NaHS-induced relaxation was similar in both groups. In contrast this effect, in Krebs medicated with indometacin *plus* L-NAME, condition that revealed the effect of EDHF, was higher in STZ than in CTR group. This data could indicate that H_2S may stimulate the release of EDHF in diabetic condition as protective effect. In aorta, NaHS effect was significantly higher in CTR rat suggesting a more susceptible K_{ATP} channels activation. In conclusion, H_2S may contrast the diabetic vascular hyperactivity either by EDHF release or by a major K_{ATP} channels activation; further understanding of the underlying mechanism for H_2S could open future prospective for clinical trials in diabetes.

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