Anti-inflammatory effect of hydrogen sulfide is mediated via the activation of Prdx6 in TNBS-induced rat colitis

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Inflammatory bowel diseases (IBD) are chronic, immune-mediated disorders, which affect the gastrointestinal (GI) tract with periodic inflammation and ulceration. It has been showed that oxidative stress contributes to epithelial and vascular injuries. It is increasingly clear that peroxiredoxins (Prdxs) seem to engage an essential role in inflammation and redox balance with antioxidant cellular components (GSH, SOD). Several investigations demonstrated that hydrogen sulfide (H2S), a gasotransmitter, has a wide range of regulatory functions and plays vital roles in many physiological and pathological processes. In pathological conditions H2S reduces inflammation by upregulating antioxidant enzymes. Therefore, the aim of our study was to investigate the effects of H2S treatment on antioxidant enzymes, primarily on the members of Prdxs (Prdx1,2,4,6) in an experimental model of IBD. To model colitis 2,4,6-trinitrobenzenesulfonic acid (TNBS) was administered intracolonically (i.c.) to Wistar–Hannover male rats. Then animals were treated (2 times/day) with a H2S donor, Lawesson's reagent per os. Our results showed that H2S treatment significantly decreased the extent of the colonic lesions. Furthermore, among Prdx isoforms only the Prdx6 showed a significant upregulation after H2S treatment compared to TNBS. Additionally, the level of glutathione (GSH) and the activity of superoxide dismutase (SOD) were significantly elevated in H2S treated group compared to TNBS, while H2S administration significantly attenuated the level of 3-nitrotyrosine (3-NT), an oxidative stress marker. Taken together, our results suggest that H2S may exert its anti-inflammatory effects through the activation of Prdx6, GSH and SOD antioxidant enzymes.