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Exogenous hydrogen sulfide ameliorates high glucose-induced myocardial injury & inflammation via the C1RP-MAPK signaling pathway in H9c2 cardiac cells

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Aims: Hydrogen sulfide (H₂S) is a novel signaling molecule with potent cytoprotective actions. In this study, we hypothesize that exogenous H₂S may protect cardiac cells against high glucose (HG)-induced myocardial injury and inflammation with the involvement of the C1RP-MAPK signaling pathway.


Main Methods: H9c2 cardiac cells cultured under HG conditions were transfected with siRNA and different inhibitors for detecting the effects of sodium hydrogen sulfide (NaHS) (a H₂S donor) on cell biological processes. The cardiac cell viability and LDH activity were determined by CCK-8 and LDH kit. ELISA was employed to measure the levels of inflammatory factors, while 2',7'-dichlorofluorescein diacetate (DCFH-DA) to evaluate reactive oxygen species (ROS). Mitochondrial membrane potential (MMP) was identified by rhodamine 123 staining. TUNEL staining and Hoechst 33258 staining were employed to observe cardiac cell apoptosis. Besides, we determined the

expression of C1RP-MAPK signaling pathway- and apoptosis-related factors by protein immunoblot analysis.

Key Findings: HG culturing induced toxicity, LDH, higher level of inflammatory factors, ROS, MMP, and apoptosis in cardiac cells, attenuated the viability of cardiac cells, and activated the C1RP-MAPK signaling pathway. Notably, C1RP silencing aggravated the above condition. H₂S or blockade of the MAPK signaling pathway reversed the above conditions induced by HG.

Significance: The present study provides evidence for the protective effect of exogenous H₂S on HG-induced myocardial injury and inflammation in H9c2 cardiac cells and suggests that the activation of C1RP-MAPK signaling pathway might be one of the mechanisms underlying the protective effect of H₂S.

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