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Molecular aspects of hydrogen sulfide (H₂S) in cardiovascular pathology

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Abstract

Cardiovascular diseases are the most leading reasons for mortality worldwide. In the past few years, hydrogen sulfide (H₂S) and its donors have been strongly emerged and suggested as promising therapeutic agents in cardiovascular disease, due to their ability to reverse a wide spectrum of pathophysiological processes. Since, H₂S and its donors have established their role as vasodilatory, neuromodulatory, anti-inflammatory and antioxidant agents. However, the interference of H₂S with the other biological molecules during the pathophysiological conditions, particularly in cardiac failure represents an enigma and still unclear. For instance, it is beyond debate that the interaction of H₂S and NO is controversial. We discuss the cardiovascular protective properties of H₂S, the interference and cross-talk between H₂S and NO, along with its interference with the other mediators under normal and pathological conditions. In this review, we demonstrate the rational and underlying mechanisms for its cardiovascular protective role against the development of cardiovascular diseases, including systemic and pulmonary hypertension; hypoxia in carotid bodies; periadventitial vasorelaxation; cardiac injury induced by ischemia, oxidative stress, and CaMKII. Furthermore, the molecular and possible mechanisms for the progression of atherosclerosis, and the anti-atherosclerotic role of H₂S were discussed.

Key words: Hydrogen sulfide; Oxidative stress; Hypertension; Periadventitial vasorelaxation; Myocardial dysfunction; Atherosclerosis.

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A pioneer study on the anti-ulcer activities of copper nicotinate complex [CuCl (HNA) 2] in experimental gastric ulcer induced by aspirin-pylorus ligation model (Shay model), Comparative evaluation of the anti-ulcer activity of curcumin and omeprazole during the acute phase of gastric ulcer, Strategies for diabetes and pathways of vitamin D, Gastric ulcer's diseases pathogenesis, complications and strategies for prevention, Molecular pathogenesis of gastric ulcers and strategies for prevention

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