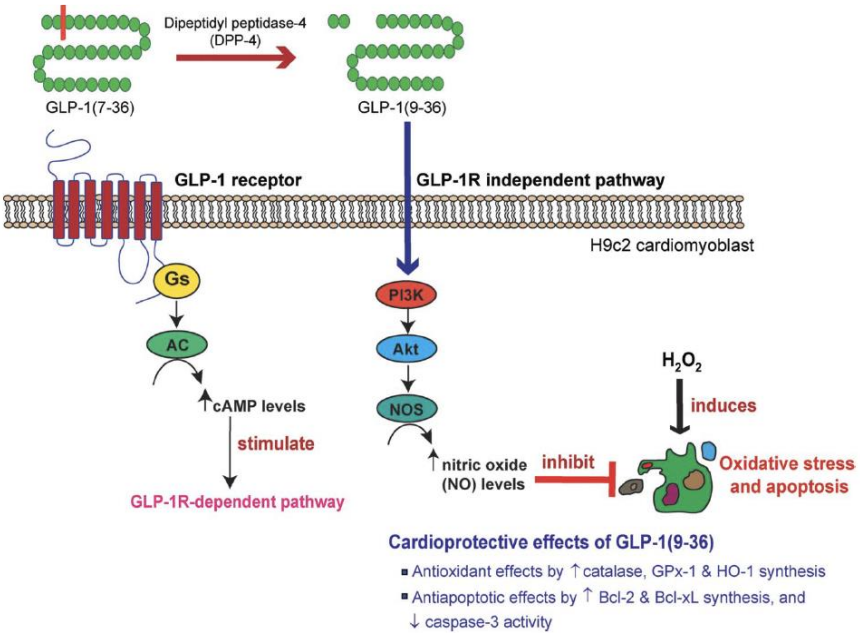
Cardioprotective effects of glucagon-like peptide-1 (9-36) against oxidative injury in H9c2 cardiomyoblasts: Potential role of the PI3K/Akt/NOS pathway

Glucagon-like peptide (GLP)-1(7-36), a major active form of GLP-1 hormone, is rapidly cleaved by dipeptidyl peptidase-4 to generate a truncated metabolite, GLP-1(9-36) which has a low affinity for GLP-1 receptor (GLP-1R). GLP-1(7-36) has been shown to have protective effects on cardiovascular system through GLP-1R-dependent pathway. Nevertheless, the cardioprotective effects of GLP-1(9-36) have not fully understood. The present study investigated the effects of GLP-1(9-36), including its underlying mechanisms against oxidative stress and apoptosis in H9c2 cells. Here, we reported that GLP-1(9-36) protects H9c2 cardiomyoblasts from hydrogen peroxide (H_2O_2)-induced oxidative stress by promoting the synthesis of antioxidant enzymes, glutathione peroxidase-1, catalase, and heme oxygenase-1. In addition, treatment with GLP-1(9-36) suppressed H_2O_2-induced apoptosis by attenuating caspase-3 activity and upregulating antiapoptotic proteins, Bcl-2 and Bcl-xL. These protective effects of GLP-1(9-36) are attenuated by blockade of PI3K-mediated Akt phosphorylation and prevention of nitric oxide synthase-induced nitric oxide production. Thus, GLP-1(9-36) represents the potential therapeutic target for prevention of oxidative stress and apoptosis in the heart via PI3K/Akt/nitric oxide synthase signaling pathway.



กลไกการออกฤทธิ์ของ GLP-1(9-36) ในการปกป้องหัวใจจากภาวะ oxidative injury

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