The inhibitory effect of trilinolein on norepinephrine-induced beta-myosin heavy chain promoter activity, reactive exygen species generation, and extracellular signal-regulated kinase phosphorylation in neonatal rat cardiomyocytes

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摘要

Abstract

The myocardial protective effects of trilinolein, isolated from the traditional Chinese herb Sanchi (Panax notoginseng), are thought to be related to its antioxidant activity. However, the intracellular mechanism underlying the protective effect of trilinolein in the heart remains unclear. In the present study, we investigated the effect of trilinolein on norepinephrine (NE)-induced protein synthesis in cardiomyocytes. Cultured neonatal rat cardiomyocytes were stimulated with NE, then protein content, [3H]-leucine incorporation, and -myosin heavy chain (-MyHC) promoter activity were examined. The effect of trilinolein on NE-induced intracellular reactive oxygen species (ROS) generation was measured with a redox-sensitive fluorescent dye (2',7'-dichlorofluorescin diacetate) and extracellular signal-regulated kinase (ERK) phosphorylation by Western blotting. Trilinolein inhibited NE-increased protein synthesis, -MyHC promoter activity, and intracellular ROS. Both trilinolein and the antioxidant, N-acetyl-cysteine, decreased NE- and H2O2-induced protein synthesis, -MyHC promoter activity, and ERK phosphorylation. These data indicate that trilinolein inhibits NE-induced protein synthesis via attenuation of ROS generation in cardiomyocytes.