

## Sulforaphane prevents oxidative stress and cell death in rat cardiomyocytes

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Cardiovascular diseases (CVDs) are the major cause of death in developed countries. Oxidative stress plays a major role in the pathophysiology of cardiac disorders. Several studies have highlighted the cardinal role played by the overproduction of reactive oxygen species in the pathogenesis of ischemic myocardial damage and consequent cardiac dysfunction. Sulforaphane (SF) is a molecule within the isothiocyanate (ITC) group of organosulfur compound commonly found in cruciferous vegetables. It is reported to be capable of stimulating cellular antioxidant defenses and inducing phase 2 detoxifying enzymes, which can protect cells against oxidative damage. Although SF is known for its anticancer benefits, its role in cardioprotection is emerging. The aim of this study was to investigate the effects of SF in preventing cell damage induced by oxidative stress. Primary rat cardiomyocytes were exposed to different concentrations of SF for 24 h and subsequently treated with H<sub>2</sub>O<sub>2</sub> to induce oxidative stress. Cell viability, and the expression of oxidative stress markers were studied. A transmission electron microscopy (TEM) analysis was carried out to evaluate the effect of SF on cell morphology. Results showed a higher cell viability and a lower level of oxidative stress in cells pre-treated with SF before peroxide exposure in respect to H<sub>2</sub>O<sub>2</sub> treated cardiomyocytes. TEM analysis showed a well preserved morphology in cells pre-treated with SF before H<sub>2</sub>O<sub>2</sub>. These findings demonstrate that sulforaphane prevents H<sub>2</sub>O<sub>2</sub> - induced oxidative stress and cell death in rat cardiomyocytes, suggesting a potential protective role of SF in CVDs

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### Keywords

Cardiomyocytes; sulforaphane; oxidative stress; cardiovascular diseases.