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Nrf2-ARE stress response mechanism: a control point in oxidative stress-mediated dysfunctions and chronic inflammatory diseases.

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Abstract

Nrf2, a redox sensitive transcription factor, plays a pivotal role in redox homeostasis during oxidative stress. Nrf2 is sequestered in cytosol by an inhibitory protein Keap1 which causes its proteasomal degradation. In response to electrophilic and oxidative stress, Nrf2 is activated, translocates to nucleus, binds to antioxidant response element (ARE), thus upregulates a battery of antioxidant and detoxifying genes. This function of Nrf2 can be significant in the treatment of diseases, such as cancer, neurodegenerative, cardiovascular and pulmonary complications, where oxidative stress causes Nrf2 derangement. Nrf2 upregulating potential of phytochemicals has been explored, in facilitating cure for various ailments while, in cancer cells, Nrf2 upregulation causes chemoresistance. Therefore, Nrf2 emerges as a key regulator in oxidative stress-mediated diseases and Nrf2 silencing can open avenues in cancer treatment. This review summarizes Nrf2-ARE stress response mechanism and its role as a control point in oxidative stress-induced cellular dysfunctions including chronic inflammatory diseases.

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