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## A protective role of nuclear factor-erythroid 2-related factor-2 (Nrf2) in inflammatory disorders.

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#### Abstract

Nuclear factor-erythroid 2-related factor-2 (Nrf2) is a key transcription factor that plays a central role in cellular defense against oxidative and electrophilic insults by timely induction of antioxidative and phase-2 detoxifying enzymes and related stress-response proteins. The 5'-flanking regions of genes encoding these cytoprotective proteins contain a specific consensus sequence termed antioxidant response element (ARE) to which Nrf2 binds. Recent studies have demonstrated that Nrf2-ARE signaling is also involved in attenuating inflammation-associated pathogenesis, such as autoimmune diseases, rheumatoid arthritis, asthma, emphysema, gastritis, colitis and atherosclerosis. Thus, disruption or loss of Nrf2 signaling causes enhanced susceptibility not only to oxidative and electrophilic stresses but also to inflammatory tissue injuries. During the early-phase of inflammation-mediated tissue damage, activation of Nrf2-ARE might inhibit the production or expression of pro-inflammatory mediators including cytokines, chemokines, cell adhesion molecules, matrix metalloproteinases, cyclooxygenase-2 and inducible nitric oxide synthase. It is likely that the cytoprotective function of genes targeted by Nrf2 may cooperatively regulate the innate immune response and also repress the induction of pro-inflammatory genes. This review highlights the protective role of Nrf2 in inflammation-mediated disorders with special focus on the inflammatory signaling modulated by this redox-regulated transcription factor.

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