Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Stress in Metabolic Disorders and Drug-Induced Injury: The Potential Role of Nrf2 and PPARs Activators

CALL FOR PAPERS

Oxidative stress plays a major role in metabolic disorders and a wide range of chronic diseases such as diabetes mellitus, obesity, metabolic syndrome, aging, cancer, osteoporosis, rheumatoid arthritis, cardiovascular diseases, and neurodegenerative disorders. In addition, drug-induced organ injury is well known to be associated with oxidative stress and inflammation. Considerable evidence indicates that oxidative stress and inflammation are the key pathophysiological processes underpinning these disorders. Therefore, modulation of oxidative stress represents an important strategy for the treatment of multiple human diseases.

The transcription factor nuclear factor erythroid 2 related factor 2 (Nrf2) is the master regulator of the basal and inducible expression of a large network of cytoprotective and antioxidant genes. Under basal conditions, Nrf2 is bound to Kelch-like ECH-associated protein 1 (Keap1) which functions as a sensor protein against electrophiles and reactive oxygen species (ROS). Upon cell stimulation, Nrf2 dissociates from Keap1 and activated Nrf2 is translocated into the nucleus where it binds to the antioxidant response element (ARE) and leads to expression of target genes including heme oxygenase-1, NAD(P)H:quinone oxidoreductase 1, superoxide dismutase, catalase, and glutathione peroxidase glutathione-s-transferase. Thus, Nrf2 plays a role as multiorgan protector against oxidative stress via inducing target genes. In recent years, Nrf2 has shown promise as a novel therapeutic target in diseases with underlying oxidative and inflammatory stress components.

Peroxisome proliferator-activated receptors (PPARs) are proteins that belong to the nuclear receptor family of ligand-activated transcription factors. The three main forms of peroxisome proliferator-activated receptors (PPARα, PPARβ/δ, and PPARγ) belong to a superfamily of nuclear receptors that function as transcription factors regulating the expression of multiple genes. Upon ligand binding, they form heterodimers with retinoid X receptor (RXR) and result in modulation of gene transcription. PPARs regulate a variety of biological processes in various tissues. Among their effects, PPARα controls lipid metabolism and inflammatory processes, PPARβ/δ regulates glucose utilization, cell differentiation, and inflammation, and PPARγ is involved in adipocyte differentiation, glucose metabolism, and inflammatory pathways. Upon activation, PPARs are known to exert anti-inflammatory and antioxidant properties via suppressing nuclear factor-κB, decreasing ROS production, and upregulating the expression of antioxidant enzymes.

Recent reports point to coactivation and possible interaction between PPARs and Nrf2 through multiple mechanisms. Ongoing and future research will probably provide efficient PPARs and Nrf2 modulating agents for preventing and treating metabolic and other common disorders. Our aim is to bring together novel research and insight views on the role of Nrf2 and PPARs in modulating oxidative stress and inflammation. We invite investigators to contribute original research as well as review articles that illustrate the usefulness of PPARs and Nrf2 as novel therapeutic targets for both metabolic and drug-induced disorders.

Potential topics include but are not limited to the following:

- Pharmacological targeting of PPARs and Nrf2 in the context of disease
- PPARs and Nrf2 ligands as therapeutic agents in metabolic and drug-induced disorders
- Role of PPARs and Nrf2 in different diseases
- Crosstalk between PPARs and Nrf2 signaling pathways
- Molecular mechanisms of synthetic and natural agents targeting Nrf2 and/or PPARs isoforms
- Possible interaction mechanisms between Nrf2 and PPARs
- Role of oxidative stress and inflammation in metabolic and drug-induced disorders
- Mechanisms of drug-induced organ damage
- Regulatory mechanisms within the Nrf2 and PPARs pathways
- Possible toxicity associated with the exposure to PPAR or Nrf2 ligands

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