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Emerging Roles of CYP450, Nrf2, and NF-κB in Multisystem Chronic Diseases

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ABSTRACT

Cytochrome P450 enzymes (CYP450), nuclear factor erythroid 2-related factor 2 (Nrf2), and nuclear factor-κB (NF-κB) have long been studied individually in toxicology, oxidative stress, and immune activation. Emerging evidence now positions these molecules as central, interacting regulators of multisystem chronic diseases that span metabolic disorders, cardiovascular dysfunction, autoimmune conditions, neurodegeneration, and cancer. CYP450 enzymes govern the metabolism of endogenous and exogenous compounds, generating metabolites that influence redox balance and immune signaling. Nrf2 orchestrates antioxidant and cytoprotective responses, mitigating damage from reactive oxygen species (ROS) and environmental stressors. NF-κB integrates inflammatory and stress signals to regulate immune responses and cell survival. Cross-talk among these pathways shapes cellular homeostasis: CYP450-derived reactive metabolites and ROS can activate or suppress Nrf2 and NF-κB; Nrf2 influences expression of metabolic enzymes and dampens NF-κB-mediated inflammation; NF-κB modifies expression of CYP450 genes and interacts with redox sensors. Dysregulation across this network contributes to chronic inflammation, impaired detoxification, oxidative damage, and maladaptive remodeling in multiple organ systems. This review synthesizes current knowledge on the interplay of CYP450, Nrf2, and NF-κB, highlights mechanistic insights into disease pathogenesis, and discusses therapeutic opportunities aimed at modulating these interconnected pathways.

Keywords: CYP450; Nrf2; NF-κB; oxidative stress; chronic inflammation

INTRODUCTION

Chronic diseases affecting multiple organ systems are leading contributors to global morbidity and mortality[1]. Conditions such as type 2 diabetes, cardiovascular disease, chronic liver and kidney disorders, neurodegenerative diseases, and autoimmune syndromes are increasingly understood as products of dysregulated metabolism, persistent inflammation, and oxidative stress[2]. At the molecular level, three regulatory systems such as CYP450 enzymes, Nrf2 signaling, and NF-κB activation—play central and interconnected roles in these processes. Cytochrome P450 enzymes (CYP450s) comprise a large family of heme-containing monooxygenases that mediate Phase I metabolism of a wide array of endogenous substrates (steroids, fatty acids) and xenobiotics (drugs, pollutants)[3]. While vital for detoxification, CYP450 activity often generates reactive intermediates and ROS that impact redox balance and can initiate stress responses[4]. Nrf2 is a master regulator of antioxidant defenses and cellular survival pathways. In response to redox perturbations, Nrf2 translocates to the nucleus where it induces expression of genes encoding antioxidant enzymes, detoxification proteins, and stress response factors[5]. By limiting ROS accumulation and damage, Nrf2 supports tissue resilience in chronic stress states. NF-κB is a

transcription factor central to immune and inflammatory responses. Activated by cytokines, pattern recognition receptors, ROS, and stress signals, NF-κB drives expression of pro-inflammatory cytokines, adhesion molecules, and survival factors[6]. While essential for host defense, chronic NF-κB activation underlies persistent inflammation in many diseases. Importantly, CYP450, Nrf2, and NF-κB do not operate in isolation: metabolic products from CYP450 influence redox sensors, Nrf2 modulates inflammatory signaling and detoxification capacity, and NF-κB alters metabolic and stress-response gene expression. Understanding how these pathways converge offers insight into mechanisms of disease and points to novel therapeutic strategies.

2. Cytochrome P450: Metabolism Beyond Detoxification

2.1 Overview of CYP450 Function

Cytochrome P450 enzymes (CYP450s) are a large family of heme-containing monooxygenases that catalyze oxidative reactions, inserting an oxygen atom into lipophilic substrates to facilitate their metabolism and elimination[7]. While highly expressed in the liver, CYP450s are also present in extra-hepatic tissues, including the kidney, lung, intestine, and brain, reflecting their widespread role in systemic metabolism[8]. Major isoforms such as CYP1A2, CYP2E1, CYP3A4, and CYP2D6 metabolize both endogenous compounds—including steroids, fatty acids, and hormones—and exogenous xenobiotics, such as medications and environmental chemicals[9]. By regulating the turnover of hormones, lipid mediators, and signaling molecules, CYP450 enzymes influence key physiological processes including energy homeostasis, vascular tone, and immune signaling[10]. Their broad substrate specificity and inducible expression allow the body to respond dynamically to environmental, dietary, and pharmacologic stimuli[11].

2.2 CYP450-Driven Reactive Intermediates and Redox Stress

CYP450-mediated metabolism, while critical for detoxification, frequently generates reactive intermediates and reactive oxygen species (ROS) as by-products[12]. For instance, CYP2E1, responsible for ethanol and small molecule metabolism, produces substantial ROS, leading to oxidative stress in hepatocytes[13]. Chronic induction or dysregulation of CYP450 activity, whether due to environmental toxins, drug exposure, or metabolic disorders can overwhelm cellular antioxidant defenses[14]. Accumulated ROS initiate lipid peroxidation, protein oxidation, and DNA damage, triggering inflammatory and apoptotic pathways. Consequently, CYP450 activity becomes a double-edged sword: essential for metabolic clearance but capable of contributing to cellular injury when homeostasis is disrupted.

2.3 CYP450 in Chronic Disease Pathogenesis

Aberrant CYP450 function is implicated in diverse chronic diseases. In liver disease, CYP2E1-mediated metabolism of acetaminophen and other hepatotoxins produces reactive species that drive hepatocellular injury[15]. In cardiovascular disease, CYP450 metabolites of arachidonic acid, including epoxyeicosatrienoic acids, modulate vascular tone, endothelial function, and inflammatory responses. Metabolic syndrome involves altered CYP450 expression that contributes to dyslipidemia and insulin resistance, exacerbating systemic inflammation[16]. In the central nervous system, brain CYP450s metabolize neuroactive steroids, and their dysregulation can potentiate neuroinflammation and neuronal vulnerability. Collectively, these roles highlight the dual nature of CYP450 enzymes: essential for physiological metabolism yet capable of promoting oxidative stress and chronic pathology when dysregulated.

3. Nrf2: Master Regulator of Antioxidant and Cytoprotective Responses

3.1 Nrf2 Regulation and Mechanism

Nuclear factor erythroid 2-related factor 2 (Nrf2) is a transcription factor that serves as a primary sensor and regulator of cellular redox homeostasis. Under basal conditions, Nrf2 is bound to Kelch-like ECH-associated protein 1 (Keap1) in the cytoplasm[17]. Keap1 functions as an adaptor for ubiquitin ligase complexes, targeting Nrf2 for proteasomal degradation and maintaining low basal levels. Upon oxidative or electrophilic stress, critical cysteine residues on Keap1 are modified, resulting in conformational changes that prevent Nrf2 ubiquitination. Stabilized Nrf2 translocates to the nucleus, where it binds to antioxidant response elements (AREs) in the promoters of target genes[18]. These genes encode Phase II detoxification enzymes such as glutathione S-transferases, antioxidant proteins including NAD(P)H quinone dehydrogenase 1 (NQO1) and heme oxygenase-1 (HO-1), as well as transporters involved in efflux of toxic metabolites. This coordinated transcriptional program enhances cellular resilience against oxidative damage, electrophilic stress, and chemical insults.

3.2 Nrf2 in Metabolic and Inflammatory Diseases

Nrf2 activation plays a protective role in multiple chronic disease contexts. In metabolic disorders, Nrf2 improves insulin sensitivity, reduces inflammatory signaling in adipose tissue, and mitigates hepatic lipid accumulation,

limiting progression to non-alcoholic fatty liver disease[19]. In the cardiovascular system, Nrf2 reduces endothelial oxidative stress, inhibits inflammatory cascades, and attenuates atherogenesis. Within the central nervous system, Nrf2 modulates glial activation, decreases ROS accumulation, and protects neurons from oxidative injury, contributing to neuroprotection[20]. However, persistent or aberrant Nrf2 activation in certain cancers can promote tumor survival, chemoresistance, and proliferation, demonstrating that context-dependent effects must be considered in therapeutic strategies.

3.3 Nrf2 and Detoxification Capacity

By upregulating Phase II detoxification enzymes and transporter proteins, Nrf2 enhances the ability of cells to conjugate and remove reactive metabolites generated by CYP450 activity[21]. This cross-talk between Nrf2 and metabolic enzymes illustrates how antioxidant responses are integrated with xenobiotic metabolism to prevent cellular damage, limit inflammation, and maintain systemic homeostasis[22].

4. NF-κB: Central Integrator of Inflammation and Stress

4.1 NF-κB Activation and Signaling

Nuclear factor-κB (NF-κB) is a family of transcription factors that serve as master regulators of immune responses, inflammation, and cellular stress signaling. c[23]. Upon exposure to pro-inflammatory stimuli such as cytokines (tumor necrosis factor-α [TNF-α], interleukin-1β [IL-1β]), pathogen-associated molecular patterns (PAMPs), damage-associated molecular patterns (DAMPs), or reactive oxygen species (ROS), intracellular signaling cascades converge on IκB kinase (IKK) complexes. IKKs phosphorylate IκB, targeting it for ubiquitination and proteasomal degradation[24]. Freed NF-κB subunits then translocate to the nucleus, where they bind specific κB motifs in target gene promoters. This initiates transcription of pro-inflammatory cytokines, chemokines, adhesion molecules, and anti-apoptotic proteins, coordinating innate and adaptive immune responses[25]. NF-κB signaling is tightly regulated by feedback loops, post-translational modifications, and cross-talk with other pathways, including MAPKs, Nrf2, and metabolic sensors, ensuring context-specific responses.

4.2 NF-κB in Chronic Inflammation and Disease

Dysregulated or persistent NF-κB activation is central to the pathogenesis of numerous chronic diseases[26]. In autoimmune disorders, prolonged NF-κB signaling promotes the survival and activation of autoreactive lymphocytes, contributing to tissue damage. In metabolic disease, NF-κB links obesity-associated inflammation to insulin resistance by upregulating pro-inflammatory cytokines in adipose tissue and the liver[27]. In cancer, chronic NF-κB activity fosters tumor progression through enhanced cell proliferation, angiogenesis, immune evasion, and resistance to apoptosis. Neuroinflammatory conditions, including Alzheimer's and Parkinson's disease, involve NF-κB-mediated microglial activation, which amplifies oxidative stress and neuronal injury[28]. The integration of NF-κB signaling with redox balance, metabolic cues, and immune pathways underscores its central position as a convergent hub in multisystem chronic diseases, making it an attractive target for therapeutic intervention.

5. Roles in Specific Multisystem Diseases

5.1 Metabolic Syndrome and Diabetes

In metabolic syndrome and type 2 diabetes, dysregulated lipid metabolism and nutrient overload increase CYP450-mediated oxidation of fatty acids, generating reactive oxygen species (ROS)[29]. These ROS act as signaling molecules that activate NF-κB, promoting pro-inflammatory cytokine production in adipose tissue, liver, and vascular endothelium. The resulting chronic low-grade inflammation exacerbates insulin resistance and contributes to hyperglycemia[30]. Nrf2 activation provides a counter-regulatory antioxidant response, enhancing expression of detoxification enzymes and antioxidant proteins. However, persistent nutrient excess and oxidative stress can overwhelm Nrf2 defenses, tipping the balance toward cellular damage and metabolic dysfunction.

5.2 Cardiovascular Disease

CYP450-derived metabolites of arachidonic acid, such as epoxyeicosatrienoic acids, modulate vascular tone, platelet aggregation, and endothelial homeostasis[31]. Dysregulation of these pathways can lead to endothelial dysfunction and heightened vascular reactivity. Concurrent NF-κB activation in endothelial and smooth muscle cells amplifies inflammation, promoting atherosclerotic plaque formation and vascular remodeling[32]. Nrf2 exerts protective effects by limiting oxidative injury, enhancing antioxidant defenses, and preserving endothelial function, highlighting the interplay between redox balance and inflammatory regulation in cardiovascular health.

5.3 Liver Disease

Drug-induced and metabolite-mediated hepatotoxicity relies heavily on CYP450 activity, which generates ROS and reactive intermediates that injure hepatocytes[33]. NF-κB activation amplifies local inflammation, recruiting immune cells and perpetuating tissue damage. Nrf2 induction partially mitigates oxidative stress, but chronic or repeated injury can overwhelm this protective response, leading to fibrosis and progressive liver disease[34].

5.4 Neurodegeneration

Brain-expressed CYP450s metabolize neuroactive steroids and xenobiotics, influencing neuronal signaling and redox balance[35]. Disrupted CYP450 activity generates ROS that activate microglia through NF-κB signaling, driving neuroinflammation and neuronal injury. Nrf2 activation in neurons and glia counteracts oxidative stress, but age-related decline or chronic pathology can reduce its efficacy, contributing to neurodegenerative progression.

5.5 Cancer

Persistent oxidative stress from CYP450 activity and chronic NF-κB-driven inflammation fosters DNA damage, genomic instability, and oncogenic transformation[36]. Nrf2's cytoprotective role, while limiting oxidative injury in normal cells, may paradoxically enhance survival of preneoplastic or malignant cells, contributing to chemoresistance and tumor progression[37].

CONCLUSIONS

CYP450 enzymes, Nrf2 signaling, and NF-κB activation form an interconnected regulatory network that influences metabolism, redox homeostasis, and immune responses. Disruption of this network contributes to the pathogenesis of diverse chronic diseases affecting multiple systems. Understanding how these pathways intersect offers avenues for targeted therapy and improved management of complex chronic conditions.

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