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Redox-Sensitive Immune Pathways: Nrf2, NF-κB, and the Balance Between Inflammation and Cytoprotection

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ABSTRACT

The balance between inflammation and cytoprotection in the immune system is critically regulated by redox- sensitive pathways - notably Nuclear factor erythroid- 2- related factor 2 (Nrf2) and Nuclear Factor kappa- light- chain- enhancer of activated B cells (NF-κB). Nrf2 orchestrates antioxidant and cytoprotective gene expression, safeguarding cells from oxidative and electrophilic stress, while NF-κB governs pro- inflammatory and immune- activating programs. The interplay - or "cross- talk" - between these pathways determines whether a cell mounts an inflammatory response or engages protective, redox- balanced processes. Excessive reactive oxygen species (ROS) can hyperactivate NF-κB, driving inflammation, tissue damage, and chronic disease; whereas Nrf2 activation restores redox homeostasis, suppressing NF-κB–mediated inflammation and promoting cellular resilience. This review discusses the molecular mechanisms regulating Nrf2 and NF-κB activation, their mutual regulation, and how their balance affects immune responses, inflammation, and cytoprotection. We explore how natural and pharmacologic agents modulate this balance, the implications for inflammatory diseases, tissue protection, and potential risks of disrupting redox- sensitive signaling. Recognizing the Nrf2–NF-κB balance as a central node in immunoregulation offers a refined framework for therapeutic strategies aiming to harness cytoprotection without impairing immune competence or triggering unchecked inflammation.

Keywords: Nrf2, NF-κB, Redox signaling, Inflammation, Cytoprotection

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INTRODUCTION

Cells constantly generate reactive oxygen species (ROS) as part of normal metabolism, immune responses, and environmental exposures[1]. ROS serve essential physiological functions, including cellular signaling, microbial killing by phagocytes, and regulation of gene expression. However, when ROS production exceeds the capacity of antioxidant defenses, oxidative stress occurs - threatening lipids, proteins, DNA, and disrupting cellular homeostasis[2]. To counteract this, cells have evolved redox- sensitive regulatory systems that detect changes in oxidative/electrophilic status and mobilize protective or inflammatory responses as needed[3]. Two of the most central players in these systems are Nrf2 and NF-κB. Nrf2 is a transcription factor that under basal conditions is sequestered in the cytoplasm by its repressor Kelch-like ECH-associated protein 1 (Keap1), which directs Nrf2 for proteasomal degradation[4]. Under oxidative or electrophilic stress, critical cysteine residues in Keap1 are modified or upstream kinases phosphorylate Nrf2, leading to Nrf2 stabilization, nuclear translocation, and binding to antioxidant response elements (ARE) in the genome[5]. This induces a battery of cytoprotective genes encoding antioxidant enzymes, detoxifying proteins, and factors that restore redox balance and support cell survival. In contrast, NF-κB is a master regulator of inflammation: upon appropriate stimuli (e.g., cytokines, microbial products, oxidative stress), inhibitory proteins (e.g., IκB) are phosphorylated, degraded, allowing NF-κB to translocate into the nucleus and drive transcription of inflammatory cytokines, chemokines, adhesion molecules, and enzymes such as iNOS and COX-2[6].

The outcome - whether a cell favors cytoprotection or inflammation - depends on the balance and cross- regulation between Nrf2 and NF-κB[7]. Understanding how these pathways interact is critical for harnessing redox signaling in immunological regulation, preventing tissue damage, and designing therapeutic approaches for inflammatory and oxidative-stress–related diseases[8].

Mechanisms of Nrf2 and NF-κB Activation and Their Cross- Talk

Nrf2 Activation and Cytoprotection

Under basal, homeostatic conditions, Nrf2 is bound to Kelch-like ECH-associated protein 1 (Keap1) in the cytoplasm, which facilitates its ubiquitination and proteasomal degradation[9]. This tight regulation maintains low constitutive levels of Nrf2, preventing unnecessary activation of antioxidant and cytoprotective genes[10]. However, when cells encounter oxidative or electrophilic stress - due to elevated ROS, reactive electrophiles, or metabolic disturbances - critical cysteine residues in Keap1 are modified, altering its conformation and disrupting the Keap1–Nrf2 complex. This prevents Nrf2 degradation, allowing it to accumulate and translocate into the nucleus[11]. Within the nucleus, Nrf2 dimerizes with small Maf proteins and binds to antioxidant response elements (AREs) in the promoter regions of target genes[12]. These genes encode a wide array of cytoprotective proteins, including phase II detoxifying enzymes such as glutathione S-transferases, antioxidant enzymes like Heme oxygenase-1 (HO-1) and NAD(P)H quinone oxidoreductase 1 (NQO1), and proteins involved in NADPH generation, heme metabolism, and xenobiotic detoxification[13]. Activation of these targets enhances the cell's antioxidant capacity, neutralizes ROS, supports metabolic detoxification, and restores redox homeostasis. This adaptive response positions Nrf2 as a molecular "guardian," protecting cells from oxidative damage, minimizing apoptosis, and preserving tissue integrity[14]. Its cytoprotective effects extend across multiple tissue types, including immune cells, vascular endothelium, epithelial barriers, and parenchymal organs[15]. In immune cells, Nrf2 ensures survival under inflammatory and oxidative stress conditions, supports energy metabolism, and modulates redox-sensitive signaling pathways critical for proper immune function[16].

NF-κB Activation and Inflammatory Response

NF-κB is a master regulator of inflammation, maintained in an inactive state in the cytoplasm through binding with inhibitory proteins, primarily IκBs[17]. Exposure to stimuli such as oxidative stress, pro-inflammatory cytokines, or pathogen-associated molecular patterns activates upstream kinases, including the IκB kinase (IKK) complex. Phosphorylation of IκB by IKK marks it for ubiquitination and subsequent proteasomal degradation[18]. Once liberated, NF-κB - typically a p50/p65 heterodimer - translocates to the nucleus, where it initiates transcription of numerous pro-inflammatory genes, including cytokines (IL-1, IL-6, TNF-α), chemokines, adhesion molecules, and enzymes like inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2)[19].

In immune cells, NF-κB signaling orchestrates the recruitment and activation of immune effectors, promotes microbial defense, and coordinates tissue repair[20]. However, excessive or dysregulated NF-κB activation - often driven by mitochondrial ROS and other oxidative stress sources - can lead to chronic inflammation, tissue injury, and the development of pathologies such as autoimmune disorders, cardiovascular disease, and neurodegeneration[21]. Mitochondrial ROS function as critical second messengers in this process, amplifying inflammatory signaling and linking metabolic stress to immune activation.

The precise regulation of Nrf2 and NF-κB, and the dynamic interplay between cytoprotective and inflammatory responses, is therefore essential for maintaining cellular homeostasis and preventing pathology[22]. Understanding these mechanisms provides a foundation for therapeutic strategies aimed at modulating redox-sensitive pathways to balance inflammation with cytoprotection[23].

Cross-talk and Mutual Regulation: Achieving Balance

Importantly, Nrf2 and NF-κB pathways are not independent but engage in a dynamic interplay. Activation of Nrf2 can suppress NF-κB–mediated inflammation[24]. Mechanistically, increased antioxidant enzyme expression reduces intracellular ROS, thereby attenuating ROS- mediated NF-κB activation. Additionally, Nrf2 upregulation can prevent degradation of IκB-α (the NF-κB inhibitor), blocking NF-κB nuclear translocation[25]. For example, induction of HO-1 - a canonical Nrf2 target - has been shown to inhibit NF-κB–driven expression of adhesion molecules, inflammatory cytokines and mediators in various tissues[26]. Nrf2 activation also reduces inflammasome activation (e.g., NLRP3 inflammasome), thereby limiting production of

IL-1β and preventing pyroptotic or inflammatory cell death.

Conversely, persistent activation of NF-κB and chronic inflammation can suppress Nrf2 signaling. NF-κB may recruit repressors (e.g., histone deacetylases such as Histone deacetylase 3, HDAC3) to ARE regions, limiting Nrf2-mediated transcription of antioxidant genes[27]. In effect, there is a "tug-of-war" between inflammatory signaling and cytoprotective, antioxidant responses. The balance determines whether cells respond to stress with inflammation (to fight infection or insult) or by initiating protective, antioxidant defense - or sometimes both[28]. The outcome can shape disease progression, tissue damage, repair, and longevity.

Biological and Clinical Implications

Physiologic and Pathologic Scenarios

In acute infections or tissue injury, transient activation of NF-κB is beneficial, mobilizing immune defenses[29]. Simultaneously, activation of Nrf2 may protect host tissues from collateral oxidative damage. In this context, a coordinated Nrf2–NF-κB response ensures effective immunity with minimal tissue injury. However, in chronic diseases (e.g., metabolic disorders, chronic inflammatory diseases, neurodegeneration, cardiovascular disease), persistent oxidative stress and low-level inflammation can shift the balance unfavorably[30]. Chronic NF-κB activation may suppress Nrf2-mediated antioxidant defenses, fueling a vicious cycle of ROS generation, inflammation, tissue damage, fibrosis[31]. Conversely, sustained Nrf2 activation - while cytoprotective - might suppress necessary inflammatory responses, impairing pathogen clearance or normal immune signaling[32]. Indeed, the role of Nrf2 in modulating immune cell polarization (e.g., macrophage phenotype from pro-inflammatory “M1” to anti-inflammatory “M2”) suggests that excessive Nrf2 activity could dampen effective immune defense under certain contexts. Such duality underlies the notion of Nrf2 as a “double-edged sword”: while beneficial for cytoprotection, detoxification, inflammation resolution, its persistent overactivation (e.g., in cancer cells) may contribute to chemoresistance, reduced apoptosis of damaged cells, or impaired immune surveillance.

Therapeutic Opportunities: Modulating the Redox Balance

Given the centrality of Nrf2–NF-κB balance in inflammation and cytoprotection, these pathways present attractive therapeutic targets[33]. Dietary phytochemicals, natural compounds, and small molecules can modulate this balance: many anti- inflammatory and antioxidant molecules suppress NF-κB signaling while activating Nrf2- ARE pathways. For instance, agents that activate Nrf2 (e.g., inducing its nuclear translocation, or inhibiting Keap1) can enhance antioxidant gene expression, reduce oxidative stress, suppress excessive inflammatory signaling, and protect tissues from ROS-mediated damage[34]. This has implications in chronic inflammatory diseases, autoimmune conditions, cardiovascular disease, neuroinflammation, and even in mitigating drug-induced organ toxicities[35]. At the same time, careful modulation is necessary: therapies must preserve the capacity of NF-κB to respond to infections or acute insults, while suppressing pathological, chronic inflammation. Over-suppression of NF-κB may compromise immune defense; over-activation of Nrf2 may aid survival of damaged or malignant cells[36]. Strategies may include context- specific Nrf2 activators, transient or controlled activation, tissue-targeted delivery, or combination therapies that restore redox balance without compromising necessary immune function. Identification of upstream regulators (e.g., kinases, redox sensors, histone modifiers) and modulators (e.g., phytochemicals, small molecules) remains a key area of research.

CONCLUSION

The dynamic interplay between Nrf2 and NF-κB - two redox- sensitive transcription factors - forms a foundational axis that governs whether cells commit to inflammation or engage cytoprotective antioxidant responses. This balance is not binary but highly nuanced, dependent on context, stimulus intensity, cell type, and regulatory feedback. Through their cross- regulation, Nrf2 and NF-κB shape immune responses, inflammation, tissue repair, and long-term cellular health. Therapeutic strategies that modulate this balance - activating Nrf2, inhibiting excessive NF-κB, or both in context-specific manners - hold considerable promise for treating chronic inflammatory, oxidative-stress–related diseases, preventing tissue damage, and improving outcomes. However, given their dual roles, such interventions require careful calibration to avoid impairing necessary immune defenses or promoting survival of pathologic cells. Understanding redox- sensitive immune pathways like Nrf2 and NF-κB as central to immunoregulation and cytoprotection offers a refined framework for future research. Continued exploration into their molecular regulation, cross-talk mechanisms, and modulation by natural or pharmacologic agents may unlock new therapies that harness the body’s own redox biology for improved health and disease resilience.

REFERENCES

1. Ogbodo John Onyebuchi, Chinazom Precious Agbo, Ugoci Olivia Njoku, Martins Obinna Ogugofor, Egba Simeon Ikechukwu, Stella Amarachi Ihim, Adaeze Chidiebere Echezona Kenneth Chibuike Brendan, Aman Babanrao Upaganlawar, and ChandrashekarDevidas Upasani. Alzheimer’s Disease: Pathogenesis and Therapeutic Interventions, Current Aging Science, 2021; 21:1-25.
2. Hong Y, Boiti A, Vallone D, Foulkes NS. Reactive Oxygen Species Signaling and Oxidative Stress: Transcriptional Regulation and Evolution. Antioxidants. 2024; 13(3):312. https://doi.org/10.3390/antiox13030312U

3. Uhuo E N, Egba S I, Nwuke P C, Obike C A and Kelechi G K. Antioxidative properties of Adansonia digitata L. (baobab) leaf extractexert protective effect on doxorubicin induced cardiac toxicity in Wistar rats. Clinical Nutrition Open Science 2022; 45:3-16
4. Ugwu, CE., Sure, SM., Dike, CC., Okpoga, NA and Egba, SI. Phytochemical and *in vitro* antioxidant activities of methanol leave extract of *Alternanthera basiliana*. Journal of Pharmacy Research, 2018; 12(6): 835-839
5. Uroko Robert Ikechukwu., Egba Simeon Ikechukwu., Okwor Josephat Ani., Agbafor Amarachi., Nwuke Chinedu Paulinus., Sangodare Rose Simon Adeyi., Ezembu Tochukwu Ukamaka (2020) Methanol Extract of Acanthus montanus (acanthaceae) leaves ameliorates oxidative stress and improves haematological indices in rats. African Journal of Pharmaceutical Research and Development, 12(1): 026-037
6. Mohite, P., Yadav, V., Pandhare, R., Maitra, S., Saleh, F. M., Saleem, R. M., Al-malky, H. S., Kumarasamy, V., Subramaniyan, V., Abdel-Daim, M. M., & Uti, D. E. (2024). Revolutionizing Cancer Treatment: Unleashing the Power of Viral Vaccines, Monoclonal Antibodies, and Proteolysis-Targeting Chimeras in the New Era of Immunotherapy. ACS Omega, 9(7), 7277–
7. Saha S, Buttari B, Panieri E, Profumo E, Saso L. An Overview of Nrf2 Signaling Pathway and Its Role in Inflammation. Molecules. 2020 Nov 23;25(22):5474. doi: 10.3390/molecules25225474. PMID: 33238435; PMCID: PMC7700122.
8. Emanuele S, Celesia A, D'Anneo A, Lauricella M, Carlisi D, De Blasio A, Giuliano M. The Good and Bad of Nrf2: An Update in Cancer and New Perspectives in COVID-19. International Journal of Molecular Sciences. 2021; 22(15):7963. https://doi.org/10.3390/ijms22157963
9. Tian W, Rojo de la Vega M, Schmidlin CJ, Ooi A, Zhang DD. Kelch-like ECH-associated protein 1 (KEAP1) differentially regulates nuclear factor erythroid-2-related factors 1 and 2 (NRF1 and NRF2). J Biol Chem. 2018 Feb 9;293(6):2029-2040. doi: 10.1074/jbc.RA117.000428. Epub 2017 Dec 18. PMID: 29255090; PMCID: PMC5808764.
10. Chauhan W, Zennadi R. Keap1-Nrf2 Heterodimer: A Therapeutic Target to Ameliorate Sickle Cell Disease. Antioxidants. 2023; 12(3):740. https://doi.org/10.3390/antiox12030740
11. McCord JM, Gao B, Hybertson BM. The Complex Genetic and Epigenetic Regulation of the Nrf2 Pathways: A Review. Antioxidants (Basel). 2023 Feb 3;12(2):366. doi: 10.3390/antiox12020366. PMID: 36829925; PMCID: PMC9952775.
12. Jung KA, Kwak MK. The Nrf2 system as a potential target for the development of indirect antioxidants. Molecules. 2010 Oct 20;15(10):7266-91. doi: 10.3390/molecules15107266. PMID: 20966874; PMCID: PMC6259123.
13. Zhang M, An C, Gao Y, Leak RK, Chen J, Zhang F. Emerging roles of Nrf2 and phase II antioxidant enzymes in neuroprotection. Prog Neurobiol. 2013 Jan;100:30-47. doi: 10.1016/j.pneurobio.2012.09.003. Epub 2012 Sep 29. PMID: 23025925; PMCID: PMC3623606.
14. Stojanovic B, Milivojcevic Bevc I, Dimitrijevic Stojanovic M, Stojanovic BS, Jovanovic M, Lazarevic S, Milosevic B, Radosavljevic I, Tasic-Uros D, Markovic N, et al. Nrf2 as a Molecular Guardian of Redox Balance and Barrier Integrity in IBD. Antioxidants. 2025; 14(12):1407. https://doi.org/10.3390/antiox14121407
15. Verza FA, DA Silva GC, Nishimura FG. The impact of oxidative stress and the NRF2-KEAP1-ARE signaling pathway on anticancer drug resistance. Oncol Res. 2025 Jul 18;33(8):1819-1834. doi: 10.32604/or.2025.065755. PMID: 40746900; PMCID: PMC12308266.
16. Cores Á, Piquero M, Villacampa M, León R, Menéndez JC. NRF2 Regulation Processes as a Source of Potential Drug Targets against Neurodegenerative Diseases. Biomolecules. 2020 Jun 14;10(6):904. doi: 10.3390/biom10060904. PMID: 32545924; PMCID: PMC7356958.
17. Oeckinghaus A, Ghosh S. The NF-kappaB family of transcription factors and its regulation. Cold Spring Harb Perspect Biol. 2009 Oct;1(4):a000034. doi: 10.1101/cshperspect.a000034. PMID: 20066092; PMCID: PMC2773619.
18. Christian F, Smith EL, Carmody RJ. The Regulation of NF-κB Subunits by Phosphorylation. Cells. 2016; 5(1):12. https://doi.org/10.3390/cells5010012
19. Gupta SC, Sundaram C, Reuter S, Aggarwal BB. Inhibiting NF-κB activation by small molecules as a therapeutic strategy. Biochim Biophys Acta. 2010 Oct-Dec;1799(10-12):775-87. doi: 10.1016/j.bbagrm.2010.05.004. Epub 2010 May 21. PMID: 20493977; PMCID: PMC2955987.
20. Mao H, Zhao X, Sun SC. NF-κB in inflammation and cancer. Cell Mol Immunol. 2025 Aug;22(8):811-839. doi: 10.1038/s41423-025-01310-w. Epub 2025 Jun 25. PMID: 40562870; PMCID: PMC12310982.
21. Iacobazzi D, Convertini P, Todisco S, Santarsiero A, Iacobazzi V, Infantino V. New Insights into NF-κB

Signaling in Innate Immunity: Focus on Immunometabolic Crosstalks. Biology. 2023; 12(6):776. https://doi.org/10.3390/biology12060776

22. Pant T, Uche N, Juric M, Zielonka J, Bai X. Regulation of immunomodulatory networks by Nrf2-activation in immune cells: Redox control and therapeutic potential in inflammatory diseases. Redox Biol. 2024 Apr; 70:103077. doi: 10.1016/j.redox.2024.103077. Epub 2024 Feb 10. PMID: 38359749; PMCID: PMC10877431.
23. Stojanovic B, Milivojcevic Bevc I, Dimitrijevic Stojanovic M, Stojanovic BS, Jovanovic M, Lazarevic S, Milosevic B, Radosavljevic I, Tasic-Uros D, Markovic N, et al. Nrf2 as a Molecular Guardian of Redox Balance and Barrier Integrity in IBD. Antioxidants. 2025; 14(12):1407. https://doi.org/10.3390/antiox14121407
24. Sivandzade F, Prasad S, Bhalerao A, Cucullo L. NRF2 and NF-қB interplay in cerebrovascular and neurodegenerative disorders: Molecular mechanisms and possible therapeutic approaches. Redox Biol. 2019 Feb;21:101059. doi: 10.1016/j.redox.2018.11.017. Epub 2018 Nov 28. PMID: 30576920; PMCID: PMC6302038.
25. Alum EU, Uti DE, Offor CE. Redox Signaling Disruption and Antioxidants in Toxicology: From Precision Therapy to Potential Hazards. *Cell Biochem Biophys* (2025). https://doi.org/10.1007/s12013-025-01846-8
26. Navarro E, Esteras N. Multitarget Effects of Nrf2 Signalling in the Brain: Common and Specific Functions in Different Cell Types. Antioxidants. 2024; 13(12):1502. https://doi.org/10.3390/antiox13121502
27. Krishnamoorthy R, Gatasheh MK, Famurewa AC, Subbarayan S, Vijayalakshmi P. Neuroprotective Potential of Jimson Weed in Methotrexate-Induced Neurotoxicity: Insights into Anti-Oxidative, Anti-Inflammatory, and Anti-Apoptotic Mechanisms via Modulation of Caspase-3, Interleukin-6, and Tumor Necrosis Factor-Alpha: In Silico. Endocr Metab Immune Disord Drug Targets. 2025 Aug 15. doi: 10.2174/0118715303350736241220090850.
28. Chovatiya R, Medzhitov R. Stress, inflammation, and defense of homeostasis. Mol Cell. 2014 Apr 24;54(2):281-8. doi: 10.1016/j.molcel.2014.03.030. PMID: 24766892; PMCID: PMC4048989.
29. Bąska P, Norbury LJ. The Role of Nuclear Factor Kappa B (NF-κB) in the Immune Response against Parasites. Pathogens. 2022 Mar 2;11(3):310. doi: 10.3390/pathogens11030310. PMID: 35335634; PMCID: PMC8950322.
30. Sharifi-Rad M, Anil Kumar NV, Zucca P, Varoni EM, Dini L, Panzarini E, Rajkovic J, Tsouh Fokou PV, Azzini E, Peluso I, Prakash Mishra A, Nigam M, El Rayess Y, Beyrouthy ME, Polito L, Iriti M, Martins N, Martorell M, Docea AO, Setzer WN, Calina D, Cho WC, Sharifi-Rad J. Lifestyle, Oxidative Stress, and Antioxidants: Back and Forth in the Pathophysiology of Chronic Diseases. Front Physiol. 2020 Jul 2;11:694. doi: 10.3389/fphys.2020.00694. PMID: 32714204; PMCID: PMC7347016.
31. Ochulor Okechukwu C., Njoku Obioma U., Uroko Robert I and Egba Simeon I**.** Nutritional composition of *Jatropha tanjorensis* leaves and effects of its aqueous extract on carbon tetrachloride induced oxidative stress in male Wistar albino rats. Biomedical Research 2018; 29(19): 3569-3576
32. Dash UC, Bhol NK, Swain SK, Samal RR, Nayak PK, Raina V, Panda SK, Kerry RG, Duttaroy AK, Jena AB. Oxidative stress and inflammation in the pathogenesis of neurological disorders: Mechanisms and implications. Acta Pharm Sin B. 2025 Jan;15(1):15-34. doi: 10.1016/j.apsb.2024.10.004. Epub 2024 Oct 16. PMID: 40041912; PMCID: PMC11873663.
33. Gado F, Ferrario G, Della Vedova L, Zoanni B, Altomare A, Carini M, Aldini G, D'Amato A, Baron G. Targeting Nrf2 and NF-κB Signaling Pathways in Cancer Prevention: The Role of Apple Phytochemicals. Molecules. 2023; 28(3):1356. https://doi.org/10.3390/molecules28031356
34. Nwuruku AO, Edwin N. Targeting Oxidative Stress in Cancer Management: The Role of Antioxidant Phytochemicals. KIU J. Health Sci., 4(2): 1-10. https://doi.org/10.59568/KJHS-2024-4-2-01
35. Alum EU. Role of phytochemicals in cardiovascular disease management: Insights into mechanisms, efficacy, and clinical application. Phytomedicine Plus, 5(1),100695. https://doi.org/10.1016/j.phyplu.2024.100695.
36. Bellezza I, Mierla AL, Minelli A. Nrf2 and NF-κB and Their Concerted Modulation in Cancer Pathogenesis and Progression. Cancers. 2010; 2(2):483-497. https://doi.org/10.3390/cancers2020483

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