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Immune-Mediated Toxic Reactions: The Contribution of ROS, Cytokines, and Autoantibodies

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ABSTRACT

Immune-mediated toxic reactions represent a significant subset of pathological conditions where dysregulated immune responses contribute directly to tissue damage and organ dysfunction. Central to these reactions are three interrelated mediators: reactive oxygen species (ROS), pro-inflammatory cytokines, and autoantibodies. ROS, generated by activated immune cells, cause oxidative damage to lipids, proteins, and nucleic acids, amplifying inflammatory cascades. Cytokines orchestrate immune cell recruitment and activation, but their excessive or persistent release can exacerbate tissue injury. Autoantibodies, hallmark features of autoimmune conditions, target self-antigens, inducing complement activation, cell lysis, and chronic inflammation. The interplay between ROS, cytokines, and autoantibodies creates a self-perpetuating cycle of immune-mediated toxicity, contributing to the pathogenesis of autoimmune diseases, drug hypersensitivities, and chronic inflammatory conditions. Understanding the mechanistic roles of these mediators provides insight into disease progression and highlights therapeutic targets aimed at modulating oxidative stress, cytokine signaling, and autoantibody production. This review synthesizes current knowledge on the cellular and molecular mechanisms of immune-mediated toxic reactions, emphasizing the integration of redox biology, immunology, and autoimmunity in the development of tissue injury and chronic disease.

Keywords: Immune-mediated toxicity, Reactive oxygen species, Cytokines, Autoantibodies, Inflammation

INTRODUCTION

Immune-mediated toxic reactions are pathological processes in which the immune system itself contributes to tissue injury [1]. Unlike infectious or traumatic injury, the damage in these reactions is driven by endogenous immune mechanisms, often involving overactivation or dysregulation of cellular and molecular mediators. Conditions such as autoimmune diseases, drug-induced hypersensitivity reactions, chronic inflammatory disorders, and transplant rejection exemplify immune-mediated toxicity [2]. At the molecular level, reactive oxygen species (ROS), pro-inflammatory cytokines, and autoantibodies form a triad that mediates immune-induced cellular damage [3]. ROS, including superoxide anions, hydrogen peroxide, and hydroxyl radicals, are generated primarily by activated neutrophils, macrophages, and other immune cells [4]. While ROS play physiological roles in microbial defense and cellular signaling, their excessive or uncontrolled production leads to oxidative damage to proteins, lipids, and nucleic acids. Cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), coordinate immune responses by regulating leukocyte recruitment, activation, and differentiation [5]. Overproduction of cytokines, however, contributes to a hyperinflammatory environment, amplifying tissue injury and sustaining immune activation. Autoantibodies, characteristic of autoimmune conditions, recognize self-antigens, initiating complement-mediated cytotoxicity, immune complex deposition, and

chronic inflammation. The combination of ROS, cytokines, and autoantibodies forms a self-reinforcing network that underlies the pathophysiology of immune-mediated toxic reactions[6]. Understanding these interactions is critical for the development of targeted therapies aimed at mitigating immune-mediated tissue damage[7].

2. Reactive Oxygen Species in Immune-Mediated Toxicity

2.1 Sources of ROS in Immune Reactions

Reactive oxygen species are generated by a variety of cellular mechanisms within the immune system, serving both protective and potentially damaging roles [8]. Phagocytes, including neutrophils, macrophages, and dendritic cells, produce ROS during the respiratory burst, an essential process for microbial killing [9]. NADPH oxidase is a primary enzymatic source, converting molecular oxygen into superoxide anions. Additional ROS arise from mitochondrial electron transport chain leakage, where incomplete reduction of oxygen produces superoxide, and from enzymatic reactions catalyzed by xanthine oxidase, myeloperoxidase, and other oxidases [10]. Under physiological conditions, controlled ROS generation aids pathogen clearance, modulates intracellular signaling, and participates in immune regulation. In pathological states, including drug hypersensitivity reactions, autoimmune disorders, and chronic inflammation, ROS production becomes excessive and persistent [11]. For example, in rheumatoid arthritis, activated synovial macrophages and neutrophils generate high concentrations of ROS that damage cartilage, synovial tissue, and joint structures [12]. Similarly, in systemic lupus erythematosus, ROS contribute to oxidative modification of self-antigens, enhancing immune complex formation and causing endothelial injury. The sustained production of ROS in these conditions shifts the balance from protective immune function to tissue-destructive processes [13].

2.2 Molecular Consequences of ROS Overproduction

Excessive ROS damage critical cellular macromolecules [14]. Lipid peroxidation compromises membrane integrity and fluidity, leading to altered ion transport and cellular signaling. Proteins undergo carbonylation and oxidation, disrupting enzyme activity, receptor function, and structural stability [15]. DNA is particularly vulnerable; oxidative lesions induce strand breaks and base modifications that activate DNA repair pathways. If repair is insufficient or overwhelmed, cells may undergo apoptosis, senescence, or accumulate mutations that promote chronic pathology [16]. ROS also influence intracellular signaling by activating redox-sensitive pathways such as nuclear factor kappa B and mitogen-activated protein kinases [17]. These pathways enhance transcription of pro-inflammatory cytokines, creating a feedback loop where oxidative stress promotes inflammation, and inflammation further increases ROS production [18]. This self-reinforcing cycle is central to immune-mediated tissue injury, amplifying damage during autoimmune disease, drug-induced hypersensitivity, and chronic inflammatory conditions.

3. Cytokines as Mediators of Immune Toxicity

3.1 Pro-inflammatory Cytokines

Cytokines are critical immune mediators that regulate leukocyte recruitment, activation, differentiation, and tissue responses [19]. Key pro-inflammatory cytokines, including tumor necrosis factor-alpha, interleukin-1 beta, interleukin-6, and interferon-gamma, coordinate host defense and tissue repair. However, excessive cytokine release generates a hyperinflammatory environment that contributes to tissue injury and cellular dysfunction [20]. Overproduction amplifies ROS generation, induces apoptosis or necrosis, and disrupts normal organ function. In cytokine release syndrome, often observed with certain immunotherapies, massive cytokine elevations result in vascular leakage, hypotension, and multi-organ dysfunction [21]. Chronic elevation of TNF-α and IL-6 in autoimmune diseases drives tissue remodeling, fibrosis, and functional impairment, highlighting the pathological potential of cytokine overproduction [22].

3.2 Cytokine–ROS Interactions

ROS and cytokines interact in a bidirectional manner, forming a self-perpetuating cycle that sustains immune-mediated toxicity [23]. ROS activate redox-sensitive transcription factors, such as nuclear factor kappa B, leading to increased pro-inflammatory cytokine expression. Conversely, cytokines stimulate ROS production in immune and non-immune cells, amplifying oxidative stress and tissue injury [24]. This synergistic interplay underlies the progression of autoimmune disorders, drug-induced toxicity, and chronic inflammatory diseases, making the ROS–cytokine axis a central driver of immune-mediated tissue damage.

4. Autoantibodies and Immune Complex-Mediated Damage

4.1 Mechanisms of Autoantibody-Induced Toxicity

Autoantibodies are immunoglobulins that recognize self-antigens, initiating a cascade of immune responses that can directly damage tissues [25]. Binding of autoantibodies to target antigens activates the classical complement pathway, leading to the formation of membrane attack complexes that lyse cells. In addition, autoantibody-antigen

complexes, or immune complexes, deposit in tissues such as kidneys, skin, and blood vessels, triggering local inflammation [26]. Recruitment of neutrophils, monocytes, and macrophages to these sites amplifies tissue injury by releasing ROS, proteolytic enzymes, and pro-inflammatory cytokines. Clinical manifestations of autoantibody-mediated toxicity are organ-specific. For example, autoimmune hemolytic anemia results from red blood cell destruction, pemphigus vulgaris arises from autoantibodies against epidermal proteins, and glomerulonephritis develops due to immune complex deposition in the kidneys.

4.2 Autoantibodies and Redox Imbalance

Autoantibody activity not only triggers inflammation but also perturbs redox homeostasis [27]. Immune complex deposition induces neutrophil respiratory bursts, producing high levels of ROS that damage surrounding tissues. Chronic autoantibody-mediated inflammation recruits additional phagocytes, further amplifying ROS generation and cytokine release [28]. This creates a self-reinforcing cycle where oxidative stress enhances autoantibody pathogenicity and cytokine production, perpetuating tissue injury over time. Redox imbalance caused by autoantibodies contributes to cellular apoptosis, extracellular matrix remodeling, and organ dysfunction in chronic autoimmune and inflammatory conditions.

5. Immune-Mediated Toxicity in Clinical Contexts

5.1 Autoimmune Diseases

Immune-mediated toxicity is central to autoimmune diseases such as systemic lupus erythematosus, rheumatoid arthritis, and autoimmune thyroiditis [29]. In SLE, autoantibodies against nuclear antigens form circulating immune complexes that deposit in the kidneys, skin, and blood vessels, inducing complement activation, ROS generation, and pro-inflammatory cytokine release [30]. In rheumatoid arthritis, persistent ROS and cytokine activity in the synovium promote cartilage degradation and bone erosion. Chronic oxidative and inflammatory stress drives tissue remodeling, fibrosis, and functional impairment.

5.2 Drug-Induced Hypersensitivity Reactions

Certain medications trigger immune-mediated toxic reactions by inducing ROS accumulation, cytokine release, or autoantibody formation [31]. Acetaminophen overdose, for instance, produces hepatocyte necrosis through ROS-mediated mitochondrial damage. Beta-lactam antibiotics can provoke hypersensitivity reactions via cytokine-driven immune activation. Drug-induced autoimmune syndromes, such as drug-induced lupus, illustrate how autoantibody production amplifies tissue injury and contributes to multi-organ involvement.

5.3 Chronic Inflammatory Disorders

Low-grade, persistent immune activation in conditions such as metabolic syndrome, atherosclerosis, and chronic infections also drives immune-mediated tissue damage [32]. Activated immune cells release ROS and pro-inflammatory cytokines, compromising vascular endothelium, cardiac tissue, and metabolic organs. This ongoing oxidative and inflammatory stress promotes progressive organ dysfunction, highlighting the centrality of immune-mediated toxicity in chronic disease pathophysiology.

6. Mechanistic Interplay: ROS, Cytokines, and Autoantibodies

Reactive oxygen species, cytokines, and autoantibodies function as an integrated network that drives immune-mediated toxicity. Autoantibodies initiate immune complex formation by binding self-antigens, which recruits neutrophils and macrophages to affected tissues [33]. These immune cells generate high levels of ROS and secrete pro-inflammatory cytokines, creating a localized inflammatory environment. ROS, in turn, activate redox-sensitive transcription factors such as nuclear factor kappa B, further amplifying cytokine production. Cytokines stimulate ROS generation in both immune and non-immune cells, perpetuating oxidative stress and tissue damage [34]. This self-reinforcing loop leads to chronic inflammation, cellular apoptosis, extracellular matrix remodeling, fibrosis, and progressive organ dysfunction. The interplay among these mediators explains why immune-mediated toxicity often persists despite removal of the initial trigger and highlights the importance of targeting multiple pathways to achieve effective therapeutic control [35].

7. Therapeutic Implications

7.1 Antioxidant-Based Interventions

Antioxidant therapies aim to neutralize ROS and prevent oxidative tissue damage. Both enzymatic antioxidants, such as superoxide dismutase and catalase, and non-enzymatic compounds, including vitamins C and E or N-acetylcysteine, have shown efficacy in experimental models [36]. Clinical application is challenging due to tissue-specific targeting limitations and context-dependent effects, where antioxidants may interfere with essential ROS signaling.

7.2 Cytokine Modulation

Cytokine-targeted therapies have proven effective in reducing immune-mediated tissue injury. TNF-α inhibitors, IL-6 receptor antagonists, and IL-1 blockers dampen hyperinflammatory responses, improving clinical outcomes in autoimmune and chronic inflammatory diseases [37]. However, broad cytokine suppression can compromise host defense, emphasizing the need for precision approaches that selectively modulate pathological signaling.

7.3 Autoantibody-Targeted Strategies

Autoantibody-focused interventions, including plasmapheresis, B-cell depletion (rituximab), and immunomodulatory therapies, reduce tissue damage by lowering pathogenic antibody levels [38]. Combining these strategies with antioxidant and cytokine-targeted therapies offers synergistic benefits, addressing multiple arms of immune-mediated toxicity simultaneously and improving long-term disease management.

CONCLUSION

Immune-mediated toxic reactions result from complex interactions among ROS, pro-inflammatory cytokines, and autoantibodies. Each mediator contributes to tissue injury, and their interplay establishes self-reinforcing cycles that sustain immune activation and oxidative damage. These processes underlie a broad spectrum of pathological conditions, including autoimmune diseases, drug hypersensitivity, and chronic inflammatory disorders. Understanding the integrated roles of ROS, cytokines, and autoantibodies is essential for developing effective therapeutic interventions. Targeted modulation of oxidative stress, cytokine signaling, and autoantibody activity offers the potential to mitigate immune-mediated toxicity while preserving essential immune functions. Future research should focus on unraveling context-specific mechanisms and designing multi-targeted strategies that restore immune homeostasis, prevent tissue injury, and improve clinical outcomes.

REFERENCES

1. Chen L, Deng H, Cui H, Fang J, Zuo Z, Deng J, Li Y, Wang X, Zhao L. Inflammatory responses and inflammation-associated diseases in organs. Oncotarget. 2017 Dec 14;9(6):7204-7218. doi: 10.18632/oncotarget.23208. PMID: 29467962; PMCID: PMC5805548.
2. Raziyeva K, Kim Y, Zharkinbekov Z, Kassymbek K, Jimi S, Saparov A. Immunology of Acute and Chronic Wound Healing. Biomolecules. 2021; 11(5):700. https://doi.org/10.3390/biom11050700
3. Mittal M, Siddiqui MR, Tran K, Reddy SP, Malik AB. Reactive oxygen species in inflammation and tissue injury. Antioxid Redox Signal. 2014 Mar 1;20(7):1126-67. doi: 10.1089/ars.2012.5149. Epub 2013 Oct 22. PMID: 23991888; PMCID: PMC3929010.
4. Khan S, Huda B, Bhurka F, Patnaik R, Banerjee Y. Molecular and Immunomodulatory Mechanisms of Statins in Inflammation and Cancer Therapeutics with Emphasis on the NF-κB, NLRP3 Inflammasome, and Cytokine Regulatory Axes. International Journal of Molecular Sciences. 2025; 26(17):8429. https://doi.org/10.3390/ijms26178429
5. Padgett LE, Broniowska KA, Hansen PA, Corbett JA, Tse HM. The role of reactive oxygen species and proinflammatory cytokines in type 1 diabetes pathogenesis. Ann N Y Acad Sci. 2013 Apr;1281(1):16-35. doi: 10.1111/j.1749-6632.2012.06826.x. Epub 2013 Jan 16. PMID: 23323860; PMCID: PMC3715103.
6. Liu S, Liu J, Wang Y, Deng F, Deng Z. Oxidative Stress: Signaling Pathways, Biological Functions, and Disease. MedComm (2020). 2025 Jul 1;6(7):e70268. doi: 10.1002/mco2.70268. PMID: 40599237; PMCID: PMC12209598.
7. Manful CF, Fordjour E, Ikumoinein E, Abbey L, Thomas R. Therapeutic Strategies Targeting Oxidative Stress and Inflammation: A Narrative Review. BioChem. 2025; 5(4):35. https://doi.org/10.3390/biochem5040035
8. de Almeida AJPO, de Oliveira JCPL, da Silva Pontes LV, de Souza Júnior JF, Gonçalves TAF, Dantas SH, de Almeida Feitosa MS, Silva AO, de Medeiros IA. ROS: Basic Concepts, Sources, Cellular Signaling, and its Implications in Aging Pathways. Oxid Med Cell Longev. 2022 Oct 19;2022:1225578. doi: 10.1155/2022/1225578. PMID: 36312897; PMCID: PMC9605829.
9. 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/antiox13030312
10. Tavassolifar MJ, Vodjgani M, Salehi Z, Izad M. The Influence of Reactive Oxygen Species in the Immune System and Pathogenesis of Multiple Sclerosis. Autoimmune Dis. 2020 Jun 25;2020:5793817. doi: 10.1155/2020/5793817. PMID: 32789026; PMCID: PMC7334772.
11. Mittal M, Siddiqui MR, Tran K, Reddy SP, Malik AB. Reactive oxygen species in inflammation and tissue injury. Antioxid Redox Signal. 2014 Mar 1;20(7):1126-67. doi: 10.1089/ars.2012.5149. Epub 2013 Oct 22. PMID: 23991888; PMCID: PMC3929010.

12. Bellanti F, Coda ARD, Trecca MI, Lo Buglio A, Serviddio G, Vendemiale G. Redox Imbalance in Inflammation: The Interplay of Oxidative and Reductive Stress. Antioxidants. 2025; 14(6):656. https://doi.org/10.3390/antiox14060656
13. Liu S, Liu J, Wang Y, Deng F, Deng Z. Oxidative Stress: Signaling Pathways, Biological Functions, and Disease. MedComm (2020). 2025 Jul 1;6(7):e70268. doi: 10.1002/mco2.70268. PMID: 40599237; PMCID: PMC12209598.
14. Juan CA, Pérez de la Lastra JM, Plou FJ, Pérez-Lebeña E. The Chemistry of Reactive Oxygen Species (ROS) Revisited: Outlining Their Role in Biological Macromolecules (DNA, Lipids and Proteins) and Induced Pathologies. Int J Mol Sci. 2021 Apr 28;22(9):4642. doi: 10.3390/ijms22094642. PMID: 33924958; PMCID: PMC8125527.
15. Jomova K, Raptova R, Alomar SY, Alwasel SH, Nepovimova E, Kuca K, Valko M. Reactive oxygen species, toxicity, oxidative stress, and antioxidants: chronic diseases and aging. Arch Toxicol. 2023 Oct;97(10):2499-2574. doi: 10.1007/s00204-023-03562-9. Epub 2023 Aug 19. PMID: 37597078; PMCID: PMC10475008.
16. Shreeya T, Ansari MS, Kumar P, Saifi M, Shati AA, Alfaifi MY, Elbehairi SEI. Senescence: A DNA damage response and its role in aging and Neurodegenerative Diseases. Front Aging. 2024 Mar 21;4:1292053. doi: 10.3389/fragi.2023.1292053. PMID: 38596783; PMCID: PMC11002673.
17. Childs BG, Baker DJ, Kirkland JL, Campisi J, van Deursen JM. Senescence and apoptosis: dueling or complementary cell fates? EMBO Rep. 2014 Nov;15(11):1139-53. doi: 10.15252/embr.201439245. Epub 2014 Oct 13. PMID: 25312810; PMCID: PMC4253488.
18. Sarma PA, Abbadie C, de Launoit Y, Cleri F. Cell Senescence and the DNA Single-Strand Break Damage Repair Pathway. DNA. 2024; 4(4):530-552. https://doi.org/10.3390/dna4040036
19. Al-Qahtani AA, Alhamlan FS, Al-Qahtani AA. Pro-Inflammatory and Anti-Inflammatory Interleukins in Infectious Diseases: A Comprehensive Review. Tropical Medicine and Infectious Disease. 2024; 9(1):13. https://doi.org/10.3390/tropicalmed9010013
20. Khaiboullina S. Special Issue: The Role of Cytokines in Disease. Int J Mol Sci. 2026 Jan 15;27(2):876. doi: 10.3390/ijms27020876. PMID: 41596520; PMCID: PMC12841538.
21. Shimabukuro-Vornhagen A, Gödel P, Subklewe M, Stemmler HJ, Schlößer HA, Schlaak M, Kochanek M, Böll B, von Bergwelt-Baildon MS. Cytokine release syndrome. J Immunother Cancer. 2018 Jun 15;6(1):56. doi: 10.1186/s40425-018-0343-9. PMID: 29907163; PMCID: PMC6003181.
22. Arvanitis P, Tziotis A, Papadimatos S, Farmakiotis D. Pathogenesis, Diagnosis, and Management of Cytokine Release Syndrome in Patients with Cancer: Focus on Infectious Disease Considerations. Current Oncology. 2025; 32(4):198. https://doi.org/10.3390/curroncol32040198
23. Selvaraj NR, Nandan D, Nair BG, Nair VA, Venugopal P, Aradhya R. Oxidative Stress and Redox Imbalance: Common Mechanisms in Cancer Stem Cells and Neurodegenerative Diseases. Cells. 2025; 14(7):511. https://doi.org/10.3390/cells14070511
24. Chen YS, Tian HX, Rong DC, Wang L, Chen S, Zeng J, Xu H, Mei J, Wang LY, Liou YL, Zhou HH. ROS homeostasis in cell fate, pathophysiology, and therapeutic interventions. Mol Biomed. 2025 Oct 30;6(1):89. doi: 10.1186/s43556-025-00338-8. PMID: 41162811; PMCID: PMC12572517.
25. Elkon K, Casali P. Nature and functions of autoantibodies. Nat Clin Pract Rheumatol. 2008 Sep;4(9):491-8. doi: 10.1038/ncprheum0895. PMID: 18756274; PMCID: PMC2703183.
26. Böröcz K, Szinger D, Simon D, Berki T, Németh P. Regulators and Conductors of Immunity: Natural Immune System in Health and Autoimmunity. International Journal of Molecular Sciences. 2025; 26(11):5413. https://doi.org/10.3390/ijms26115413
27. Garcia-Llorens G, El Ouardi M, Valls-Belles V. Oxidative Stress Fundamentals: Unraveling the Pathophysiological Role of Redox Imbalance in Non-Communicable Diseases. Applied Sciences. 2025; 15(18):10191. https://doi.org/10.3390/app151810191
28. Leiva-Castro C, Múnera-Rodríguez AM, Torres-Joya G, Palomares-Jerez F, López-Enríquez S. Modulating inflammation and oxidative stress in rheumatoid arthritis: a systematic review of nutraceutical interventions. Inflammopharmacology. 2025 Nov;33(11):6357-6375. doi: 10.1007/s10787-025-01976-8. Epub 2025 Sep 30. PMID: 41026372; PMCID: PMC12618442.
29. Pollard KM, Hultman P, Kono DH. Toxicology of autoimmune diseases. Chem Res Toxicol. 2010 Mar 15;23(3):455-66. doi: 10.1021/tx9003787. PMID: 20078109; PMCID: PMC3076021.
30. Yasmeen F, Pirzada RH, Ahmad B, Choi B, Choi S. Understanding Autoimmunity: Mechanisms,

Predisposing Factors, and Cytokine Therapies. International Journal of Molecular Sciences. 2024; 25(14):7666. https://doi.org/10.3390/ijms25147666

31. Sakamoto E, Katahira Y, Mizoguchi I, Watanabe A, Furusaka Y, Sekine A, Yamagishi M, Sonoda J, Miyakawa S, Inoue S, Hasegawa H, Yo K, Yamaji F, Toyoda A, Yoshimoto T. Chemical- and Drug-Induced Allergic, Inflammatory, and Autoimmune Diseases Via Haptenation. Biology (Basel). 2023 Jan 12;12(1):123. doi: 10.3390/biology12010123. PMID: 36671815; PMCID: PMC9855847.
32. Acierno C, Nevola R, Barletta F, Rinaldi L, Sasso FC, Adinolfi LE, Caturano A. Multidrug-Resistant Infections and Metabolic Syndrome: An Overlooked Bidirectional Relationship. Biomedicines. 2025 May 30;13(6):1343. doi: 10.3390/biomedicines13061343. PMID: 40564061; PMCID: PMC12190144.
33. Mayadas TN, Tsokos GC, Tsuboi N. Mechanisms of immune complex-mediated neutrophil recruitment and tissue injury. Circulation. 2009 Nov 17;120(20):2012-24. doi: 10.1161/CIRCULATIONAHA.108.771170. PMID: 19917895; PMCID: PMC2782878.
34. Mayadas TN, Tsokos GC, Tsuboi N. Mechanisms of immune complex-mediated neutrophil recruitment and tissue injury. Circulation. 2009 Nov 17;120(20):2012-24. doi: 10.1161/CIRCULATIONAHA.108.771170. PMID: 19917895; PMCID: PMC2782878.
35. MohanKumar SMJ, Murugan A, Palaniyappan A, MohanKumar PS. Role of cytokines and reactive oxygen species in brain aging. Mech Ageing Dev. 2023 Sep;214:111855. doi: 10.1016/j.mad.2023.111855. Epub 2023 Aug 3. PMID: 37541628; PMCID: PMC10528856.
36. Ashok A, Andrabi SS, Mansoor S, Kuang Y, Kwon BK, Labhasetwar V. Antioxidant Therapy in Oxidative Stress-Induced Neurodegenerative Diseases: Role of Nanoparticle-Based Drug Delivery Systems in Clinical Translation. Antioxidants. 2022; 11(2):408. https://doi.org/10.3390/antiox11020408
37. Siuchnińska H, Minarowska A, Wasilewska E. Beyond Joints: Neuropsychiatric Benefits of TNF-α and IL-6 Inhibitors in Rheumatoid Arthritis—Narrative Review. International Journal of Molecular Sciences. 2025; 26(17):8361. https://doi.org/10.3390/ijms26178361
38. Zhang Z, Xu Q, Huang L. B cell depletion therapies in autoimmune diseases: Monoclonal antibodies or chimeric antigen receptor-based therapy? Front Immunol. 2023 Feb 10;14:1126421. doi: 10.3389/fimmu.2023.1126421. PMID: 36855629; PMCID: PMC9968396.

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