OPEN ACCESS
©NIJRMS ONLINE ISSN: 2992-5460
Publications 2025 PRINT ISSN: 2992-6041

NEWPORT INTERNATIONAL JOURNAL OF RESEARCH IN MEDICAL SCIENCES (NIJRMS)

Volume 7 Issue 2 Page 46-51, 2026

https://doi.org/10.59298/NIJRMS/2026/7.2.4651

Immunotoxicity of Common Therapeutics in Chronic Diseases: Mechanistic and Safety Considerations

Chelimo Faith Rebecca

Department of Clinical Medicine and Dentistry Kampala International University Uganda
Email: rebecca.chelimo@studwc.kiu.ac.ug

ABSTRACT

Therapeutics used to manage chronic diseases-rheumatologic, metabolic, cardiovascular, infectious, and oncologic-carry diverse immunological risks that can undermine patient safety and long-term outcomes. Immunotoxicity encompasses immunosuppression and infection risk, hypersensitivity and allergic reactions, immune-mediated organ damage (autoimmunity), hematologic immune injury, and aberrant inflammatory responses such as cytokine release syndrome. Mechanisms range from direct cytotoxic effects on immune cells and off-target kinase inhibition to immunogenicity of biologic agents, Fc-mediated complement activation, formation of drug-protein haptens, and dysregulation of immune checkpoints. This review synthesizes mechanistic pathways, illustrates representative classes of therapeutics (conventional immunosuppressants, biologic DMARDs and monoclonal antibodies, small-molecule kinase inhibitors, hormone and metabolic therapies, and advanced cellular therapies), and describes common clinical phenotypes. It then outlines risk factors, pre-treatment assessment, monitoring strategies, and management approaches to mitigate harm, including screening for latent infections, vaccination strategies, therapeutic drug monitoring, and stewardship of immunomodulatory regimens. Finally, it highlights gaps in preclinical immunotoxicity testing, the role of pharmacogenomics, and priorities for future research and regulatory harmonization. Clinicians and drug developers must integrate mechanistic understanding with patient-centred monitoring to balance therapeutic benefit against immunologic risk.

Keywords: Immunotoxicity; biologics; immunosuppression; adverse immune effects; safety monitoring

INTRODUCTION

Chronic diseases remain the leading drivers of global morbidity, affecting populations across all age groups and socioeconomic settings [1]. Their management often requires prolonged or lifelong pharmacotherapy, frequently involving agents that modulate inflammation, metabolism, immunity, or cellular signaling [2]. As therapeutic options have expanded, especially with the growth of biologics and targeted small molecules, an increasingly complex spectrum of unintended immune effects has become apparent. These unintended effects, broadly classified as immunotoxicity, encompass a range of dysfunctional immune responses that arise directly from drug mechanisms or indirectly through immune dysregulation [3]. Immunotoxicity can present in many forms, including heightened susceptibility to common or opportunistic infections, reactivation of latent pathogens such as tuberculosis or hepatitis viruses, hypersensitivity and allergic reactions, drug-induced autoimmunity involving multiple organ systems, bone-marrow suppression with associated cytopenias, or paradoxical inflammatory syndromes that mimic or exacerbate underlying disease [4]. Because these adverse immune outcomes often overlap clinically with disease progression, treatment failure, or comorbid conditions, they present diagnostic challenges in routine practice [5]. The diversity of mechanisms underlying these events further complicates evaluation, making it essential for clinicians and regulatory scientists to develop an integrated framework that combines mechanistic understanding with practical strategies for prevention, detection, and management [6]. Such a framework is particularly important as many chronic disease therapies exert both therapeutic and potentially harmful effects on the immune system, requiring continual risk–benefit assessment throughout the treatment course [7].

Mechanisms of Immunotoxicity

Understanding the mechanisms that drive immunotoxicity is critical for predicting patient risk and guiding safer therapeutic use [8]. A central pathway involves direct cytotoxicity to immune cells. Many classic cytotoxic drugs, including certain chemotherapeutics, purine analogues, and antimetabolites, impair bone-marrow progenitors or circulating lymphocytes [9]. This leads to neutropenia, lymphopenia, and reduced antigen-presenting capacity, all of which weaken host defenses and predispose to infections. A second mechanistic category involves off-target kinase or intracellular signaling inhibition [10]. Targeted inhibitors such as those directed against JAK, BTK, or PI3K pathways disrupt key signaling modules required for immune-cell development, cytokine signaling, and functional coordination [11]. As a result, patients may develop broad immunodeficiency, altered cytokine production, or impaired humoral responses despite receiving agents designed to improve disease control. Another major contributor to immunotoxicity is drug immunogenicity [12]. Biologic therapeutics, including monoclonal antibodies and recombinant fusion proteins, may be recognized as foreign by the immune system, prompting the formation of anti-drug antibodies. These antibodies can neutralize therapeutic effects, create circulating immune complexes that deposit within tissues, or cause immediate hypersensitivity reactions through IgE- or IgG-mediated pathways [13]. The effector functions of Fc regions in monoclonal antibodies further influence immunotoxic potential. Engagement of Fc gamma receptors or activation of complement pathways can induce targeted cell depletion, complement-mediated tissue damage, or systemic inflammatory responses, particularly when Fc engineering is incomplete or when patient-specific immune factors amplify these interactions. Small-molecule drugs may act through haptenization, in which the parent drug or a reactive metabolite covalently binds to host proteins [14]. These drug–protein adducts behave as neoantigens that activate T cell-mediated immune responses, producing clinical manifestations ranging from mild maculopapular rashes to severe cutaneous adverse reactions such as Stevens–Johnson syndrome or toxic epidermal necrolysis [15]. Additional pathways involve modulation of immune checkpoints. Therapies that inhibit inhibitory pathways such as PD-1, PD-L1, or CTLA-4 remove restraints on immune activation, increasing antitumor immunity but also breaking peripheral tolerance [16]. Consequent autoimmune toxicities may affect endocrine glands, the gastrointestinal tract, lungs, skin, or myocardium, often emerging weeks to months after therapy initiation [17].

Some modern therapies also provoke excessive cytokine production. Cellular therapies such as CAR-T cells and bispecific T-cell engagers can induce overwhelming immune activation and cytokine release syndrome, characterized by fever, hypotension, organ dysfunction, and laboratory evidence of systemic inflammation [18]. Finally, alterations in the microbiome or epithelial barriers contribute to immunotoxic risks. Drugs that significantly disrupt microbial communities, including broad-spectrum antibiotics or agents that affect gastric acidity, can modify host–microbe interactions, impair mucosal immunity, and predispose to opportunistic infections while altering systemic immune tone [19]. Taken together, these mechanistic categories highlight the multifaceted ways in which chronic disease therapeutics can derail immune homeostasis.

Representative Therapeutic Classes and Their Immunotoxic Profiles

Conventional immunosuppressants

Conventional immunosuppressive agents remain foundational treatments for many chronic inflammatory and autoimmune diseases, yet their broad effects on immune-cell populations make them significant contributors to immunotoxicity [20]. Corticosteroids suppress multiple immune pathways, inducing lymphocyte apoptosis, reducing macrophage and dendritic cell activation, and downregulating inflammatory cytokines [21]. While effective for rapid symptom control, prolonged use impairs innate immunity, diminishes mucosal barrier function, delays wound healing, and weakens vaccine responses [22]. Antimetabolites such as methotrexate, azathioprine, and mycophenolate mofetil inhibit nucleotide synthesis and lymphocyte proliferation, producing dose-dependent marrow suppression, lymphopenia, and susceptibility to bacterial, viral, and opportunistic infections, including Pneumocystis, cytomegalovirus, and varicella-zoster virus [23]. Drug–drug combinations can intensify these effects, amplifying immunosuppression beyond what is expected from individual agents [24].

Biologic disease-modifying agents and monoclonal antibodies

Biologic therapies have transformed the treatment landscape for rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, and other chronic immune-mediated disorders [25]. However, by selectively neutralizing cytokines or depleting specific immune-cell subsets, these agents introduce characteristic immunotoxicity profiles [26]. Tumor necrosis factor inhibitors, for example, impair granuloma maintenance, increasing the likelihood of reactivation of latent tuberculosis and raising susceptibility to invasive fungal infections [27]. IL-6 receptor blockade can blunt acute-phase responses and mask early signs of infection, complicating clinical detection. B–cell–targeting agents such as anti-CD20 monoclonal antibodies cause prolonged B-cell depletion, reducing

ONLINE ISSN: 2992-5460
PRINT ISSN: 2992-6041

antibody production, diminishing vaccine responsiveness, and predisposing patients to hypogammaglobulinemia and recurrent sinopulmonary infections [28]. Immunogenicity remains an ongoing challenge, as anti-drug antibody formation can reduce therapeutic efficacy or precipitate infusion reactions.

Small-molecule kinase inhibitors and targeted agents

Targeted kinase inhibitors modulate intracellular signaling pathways central to immune-cell survival, differentiation, and communication [29]. JAK inhibitors broadly impair cytokine signaling across multiple hematopoietic lineages, contributing to viral reactivation particularly herpes zoster, and increasing risk of serious bacterial infections [30]. Some agents have also been associated with lymphopenia and changes in lipid metabolism that may compound cardiovascular risk. PI3K inhibitors, used in oncology and hematologic malignancies, can produce pronounced immune dysregulation, manifesting as autoimmune-like colitis, hepatotoxicity, and noninfectious pneumonitis [31]. Many kinase inhibitors also cause cytopenias by suppressing marrow progenitors, further compounding infection risk.

Metabolic and hormonal therapies with immune effects

Although metabolic therapies such as SGLT2 inhibitors and GLP-1 receptor agonists are not primarily immunomodulators, emerging evidence suggests they influence inflammation and host defense indirectly through metabolic–immune crosstalk [32]. Their immunotoxic potential remains relatively low, but some hormonal therapies, including older corticosteroid-sparing agents or androgen-modulating therapies, can shift leukocyte distributions, influence susceptibility to certain pathogens, or alter inflammatory tone [33].

Advanced cellular therapies and immune stimulants

Advanced immunotherapies provide powerful disease control but carry the highest risk of acute immune-mediated toxicity [34]. CAR-T cell therapies and bispecific T-cell engagers induce vigorous immune activation, frequently leading to cytokine release syndrome and immune effector cell–associated neurotoxicity syndrome [35]. While these toxicities are often reversible with early intervention, they pose substantial clinical risk. Conversely, immune stimulants and therapeutic vaccines used in chronic infections or malignancy can destabilize immune tolerance and precipitate autoimmune flare-ups in predisposed individuals.

Future Directions and Research Priorities

Advancing the safety of therapeutics used in chronic diseases requires a multifaceted research agenda that spans basic science, translational modeling, and real-world evidence [36]. A major priority is the development of validated biomarkers capable of predicting individual susceptibility to immunotoxicity and detecting early immune injury before clinical deterioration occurs. Such markers may include soluble cytokines, immune-cell transcriptomic signatures, metabolic indicators of immune activation, or genetic variants influencing drug metabolism and immune regulation [37]. Parallel to biomarker discovery is the need for improved in vitro and ex vivo platforms that more accurately recapitulate human immunology. Humanized organoid systems, microfluidic immune–tissue interfaces, and machine-learning–driven cytokine assays offer promise for predicting cytokine release potential, immunogenicity, and immune-cell depletion profiles [38]. Longitudinal real-world datasets are essential to capture rare but severe immune-mediated events that clinical trials are underpowered to detect. Integrating electronic health records, pharmacovigilance databases, and patient-reported outcomes can refine risk estimates and guide post-marketing safety policies [39]. Future therapies should also focus on restoring immune competence using targeted and reversible approaches that avoid broad immunosuppression, such as selective cytokine pathway tuning or precision microbial interventions informed by microbiome profiling. Finally, engineering biologics with reduced immunogenicity and optimized Fc architecture remains a key translational goal to minimize off-target immune activation while preserving therapeutic potency.

CONCLUSION

Immunotoxicity is a multifaceted safety challenge across therapeutics used for chronic diseases. Clinicians must balance therapeutic benefit with potential immune harms through pre-treatment screening, vaccination, targeted monitoring, and timely management of adverse immune events. Drug developers and regulators should prioritize improved preclinical immunotoxicity assays, post-marketing surveillance, and personalized risk stratification. With coordinated clinical vigilance and continued mechanistic research, it is possible to optimize efficacy while minimizing the immunologic costs of chronic disease therapy.

REFERENCES

1. Hacker K. The Burden of Chronic Disease. Mayo Clin Proc Innov Qual Outcomes. 2024 Jan 20;8(1):112-119. doi: 10.1016/j.mayocpiqo.2023.08.005. Erratum in: Mayo Clin Proc Innov Qual Outcomes. 2024 Dec 13;9(1):100588. doi: 10.1016/j.mayocpiqo.2024.11.005. PMID: 38304166; PMCID: PMC10830426.

ONLINE ISSN: 2992-5460
PRINT ISSN: 2992-6041

2. Obeagu, E. I., Obeagu, G. U., **Alum, E. U.** and Ugwu, O. P. C. Persistent Immune Activation and Chronic Inflammation: Unraveling Their Impact on Anemia in HIV Infection. *INOSR Experimental Sciences*. 2023; 12(3):73-84. https://doi.org/10.59298/INOSRES/2023/7.3.21322
3. Leite Â. Chronic Illnesses: Varied Health Patterns and Mental Health Challenges. Healthcare. 2025; 13(12):1396. https://doi.org/10.3390/healthcare13121396
4. Obeagu, E. I., Obeagu, G. U., **Alum, E. U.** and Ugwu, O. P. C. Advancements in Immune Augmentation Strategies for HIV Patients. *IAA Journal of Biological Sciences*. 2023; 11(1):1-11.
1. https://doi.org/10.59298/IAAJB/2023/1.2.23310
5. Bou Zerdan M, Moussa S, Atoui A, Assi HI. Mechanisms of Immunotoxicity: Stressors and Evaluators. International Journal of Molecular Sciences. 2021; 22(15):8242. https://doi.org/10.3390/ijms22158242
6. Mennella C, Maniscalco U, De Pietro G, Esposito M. Ethical and regulatory challenges of AI technologies in healthcare: A narrative review. Heliyon. 2024 Feb 15;10(4):e26297. doi: 10.1016/j.heliyon.2024.e26297. PMID: 38384518; PMCID: PMC10879008.
7. Niazi SK. Bridging the Regulatory Divide: A Dual-Pathway Framework Using SRA Approvals and AI Evaluation to Ensure Drug Quality in Developing Countries. Pharmaceuticals. 2025; 18(7):1024. https://doi.org/10.3390/ph18071024
8. Germolec DR, Lebrec H, Anderson SE, Burleson GR, Cardenas A, Corsini E, Elmore SE, Kaplan BLF, Lawrence BP, Lehmann GM, Maier CC, McHale CM, Myers LP, Pallardy M, Rooney AA, Zeise L, Zhang L, Smith MT. Consensus on the Key Characteristics of Immunotoxic Agents as a Basis for Hazard Identification. Environ Health Perspect. 2022 Oct;130(10):105001. doi: 10.1289/EHP10800. Epub 2022 Oct 6. PMID: 36201310; PMCID: PMC9536493.
9. Aja O. A., Egba S. I., Omoboyowa D. A., Odo C. E., Vining-Ogu I. C., Oko F. O (2020) Anti-anaemic and immunomodulatory potentials of aqueous, chloroform and methanol leaf extracts of *whitfieldia lateritia* on 2, 4-dinitrophenylhydrazine induced anaemia in rats. World Journal of Pharmacy Research 2020; 9(10): 44-58
10. Narayanan J, Tamilanban T, Kumar PS, Guru A, Muthupandian S, Kathiravan MK, Arockiaraj J. Role and mechanistic actions of protein kinase inhibitors as an effective drug target for cancer and COVID. Arch Microbiol. 2023 May 17;205(6):238. doi: 10.1007/s00203-023-03559-z. PMID: 37193831; PMCID: PMC10188327.
11. Li J, Gong C, Zhou H, Liu J, Xia X, Ha W, Jiang Y, Liu Q, Xiong H. Kinase Inhibitors and Kinase-Targeted Cancer Therapies: Recent Advances and Future Perspectives. International Journal of Molecular Sciences. 2024; 25(10):5489. https://doi.org/10.3390/ijms25105489
12. Shim JV, Chun B, van Hasselt JGC, Birtwistle MR, Saucerman JJ, Sobie EA. Mechanistic Systems Modeling to Improve Understanding and Prediction of Cardiotoxicity Caused by Targeted Cancer Therapeutics. Front Physiol. 2017 Sep 8;8:651. doi: 10.3389/fphys.2017.00651. PMID: 28951721; PMCID: PMC5599787.
13. Vogel M, Engeroff P. A Comparison of Natural and Therapeutic Anti-IgE Antibodies. Antibodies. 2024; 13(3):58. https://doi.org/10.3390/antib13030058
14. Aja O. A., Egba S. I., Uhuo Emmanuel Nnaemeka, Alaebo Prince Ogocukwu, Mba Obinna Joseph, and Oriaku Chinwe Edith. Hepatoprotective potentials of aqueous chloroform and methanol leaf extracts Whitfieldia lateritia 2, 4-dinitrophenylhydrazine induced anaemia in rats. Bio-research and Biotechnology, 2022; 20(2) 1434-1445
15. Johnson DS, Weerapana E, Cravatt BF. Strategies for discovering and derisking covalent, irreversible enzyme inhibitors. Future Med Chem. 2010 Jun;2(6):949-64. doi: 10.4155/fmc.10.21. PMID: 20640225; PMCID: PMC2904065.
16. Wojtukiewicz MZ, Rek MM, Karpowicz K, Górska M, Polityńska B, Wojtukiewicz AM, Moniuszko M, Radziwon P, Tucker SC, Honn KV. Inhibitors of immune checkpoints-PD-1, PD-L1, CTLA-4-new opportunities for cancer patients and a new challenge for internists and general practitioners. Cancer Metastasis Rev. 2021 Sep;40(3):949-982. doi: 10.1007/s10555-021-09976-0. Epub 2021 Jul 8. PMID: 34236546; PMCID: PMC8556173.
17. Nagahara K, Arikawa T, Oomizu S, Kontani K, Nobumoto A, Tateno H, Watanabe K, Niki T, Katoh S, Miyake M, Nagahata S, Hirabayashi J, Kuchroo VK, Yamauchi A, Hirashima M. Galectin-9 increases Tim-3+ dendritic cells and CD8+ T cells and enhances antitumor immunity via galectin-9-Tim-3 interactions. J Immunol. 2008 Dec 1;181(11):7660-9. doi: 10.4049/jimmunol.181.11.7660. PMID: 19017954; PMCID: PMC5886706.
18. Delmas D, Hermetet F, Aires V. PD-1/PD-L1 Checkpoints and Resveratrol: A Controversial New Way

for a Therapeutic Strategy. Cancers. 2021; 13(18):4509. https://doi.org/10.3390/cancers13184509

19. Patangia DV, Anthony Ryan C, Dempsey E, Paul Ross R, Stanton C. Impact of antibiotics on the human microbiome and consequences for host health. Microbiologyopen. 2022 Feb;11(1):e1260. doi: 10.1002/mbo3.1260. PMID: 35212478; PMCID: PMC8756738.
20. Alum, E. U. and Ugwu, O. P. C. Nutritional Strategies for Rheumatoid Arthritis: Exploring Pathways to Better Management. *INOSR Scientific Research.* 2023; 10(1):18-26. https://doi.org/10.59298/INOSRSR/2023/3.2.47322
21. Xu Z, Chu M. Advances in Immunosuppressive Agents Based on Signal Pathway. Front Pharmacol. 2022 May 26;13:917162. doi: 10.3389/fphar.2022.917162. PMID: 35694243; PMCID: PMC9178660.
22. Egba, S. I., Ikechukwu, G. C and Njoku, O U. Aqueous extracts of *Telfairia occidentalis* leaf reverses pyrogallol induced leucopenia and stimulates the immune system in wistar albino rats. *Journal of Chemical and Pharmaceutical. Research*, 2013; 5(4): 149-153
23. Ibiam, U. A. and Ugwu, O. P. C. A Comprehensive Review of Treatment Approaches and Perspectives for Management of Rheumatoid Arthritis. *INOSR Scientific Research.* 2023; 10(1):12-17. https://doi.org/10.59298/INOSRSR/2023/2.2.13322
24. Blagova O, Rud' R, Kogan E, Zaitsev A, Nedostup A. Comparative Efficacy and Safety of Mycophenolate Mofetil and Azathioprine in Combination with Corticosteroids in the Treatment of Lymphocytic Myocarditis. Journal of Clinical Medicine. 2023; 12(15):4913. https://doi.org/10.3390/jcm12154913
25. Ibiam, U. A., Ugwuja, E. I., Aja, P. M., Igwenyi, I. O., Offor, C. E., Orji, O. U., Ezeani N. N, Ugwu, O. P. C., Aloke, C., Egwu, C. O. Antioxidant Effect of *Buchholzia coriacea* Ethanol Leaf Extract and Fractions on Freund's Adjuvant-induced Arthritis in Albino Rats: A Comparative Study. *Slovenian Veterinary Research.* 2022; 59 (1): 31–45. doi: 10.26873/svr-1150-2022.
26. Aloke, C., Ibiam, U. A., Obasi, N. A., Orji, O. U., Ezeani, N. N., Aja, P. M. and Mordi, J. C. Effect of ethanol and aqueous extracts of seed pod of Copaifera salikounda (Heckel) on complete Freund's adjuvant-induced rheumatoid arthritis in rats. J Food Biochem. 2019 Jul;43(7):e12912. doi: 10.1111/jfbc.12912. Epub 2019 May 23. PMID: 31353723.
27. Alıravcı ID, Mutlu P, Oymak S, Guney UI, Keskin O. Risk of Latent Tuberculosis Infection Reactivation in Patients Treated with Tumor Necrosis Factor Antagonists: A Five-Year Retrospective Study. Trop Med Infect Dis. 2025 Jul 7;10(7):190. doi: 10.3390/tropicalmed10070190. PMID: 40711067; PMCID: PMC12300220.
28. Ali T, Kaitha S, Mahmood S, Ftesi A, Stone J, Bronze MS. Clinical use of anti-TNF therapy and increased risk of infections. Drug Healthc Patient Saf. 2013;5:79-99. doi: 10.2147/DHPS.S28801. Epub 2013 Mar 28. PMID: 23569399; PMCID: PMC3615849.
29. Ott PA, Adams S. Small-molecule protein kinase inhibitors and their effects on the immune system: implications for cancer treatment. Immunotherapy. 2011 Feb;3(2):213-27. doi: 10.2217/imt.10.99. PMID: 21322760; PMCID: PMC4009988.
30. Filippone A, Mannino D, Casili G, Lanza M, Paterniti I, Cuzzocrea S, Capra AP, Colarossi L, Giuffrida D, Lombardo SP, et al. Protein Kinase Inhibitors as a New Target for Immune System Modulation and Brain Cancer Management. International Journal of Molecular Sciences. 2022; 23(24):15693. https://doi.org/10.3390/ijms232415693
31. Scott SS, Greenlee AN, Matzko A, Stein M, Naughton MT, Zaramo TZ, Schwendeman EJ, Mohammad SJ, Diallo M, Revan R, Shimmin G, Tarun S, Ferrall J, Ho TH, Smith SA. Intracellular Signaling Pathways Mediating Tyrosine Kinase Inhibitor Cardiotoxicity. Heart Fail Clin. 2022 Jul;18(3):425-442. doi: 10.1016/j.hfc.2022.02.003. Epub 2022 Jun 1. PMID: 35718417; PMCID: PMC10391230.
32. Santulli G, Mone P, Varzideh F. GLP-1 receptor agonists and SGLT2 inhibitors: new anti-aging tools? Future Cardiol. 2025 Jan;21(1):5-8. doi: 10.1080/14796678.2024.2433381. Epub 2024 Nov 26. PMID: 39589856; PMCID: PMC11812426.
33. Schönberger E, Mihaljević V, Steiner K, Šarić S, Kurevija T, Majnarić LT, Bilić Ćurčić I, Canecki-Varžić S. Immunomodulatory Effects of SGLT2 Inhibitors-Targeting Inflammation and Oxidative Stress in Aging. Int J Environ Res Public Health. 2023 Aug 29;20(17):6671. doi: 10.3390/ijerph20176671. PMID: 37681811; PMCID: PMC10487537.
34. Casagrande S, Sopetto GB, Bertalot G, Bortolotti R, Racanelli V, Caffo O, Giometto B, Berti A, Veccia A. Immune-Related Adverse Events Due to Cancer Immunotherapy: Immune Mechanisms and Clinical Manifestations. Cancers (Basel). 2024 Apr 8;16(7):1440. doi: 10.3390/cancers16071440. PMID: 38611115; PMCID: PMC11011060.
35. Savino A, Rossi A, Fagiuoli S, Invernizzi P, Gerussi A, Viganò M. Hepatotoxicity in Cancer

ONLINE ISSN: 2992-5460
PRINT ISSN: 2992-6041

Immunotherapy: Diagnosis, Management, and Future Perspectives. Cancers. 2025; 17(1):76. https://doi.org/10.3390/cancers17010076

36. Marques L, Costa B, Pereira M, Silva A, Santos J, Saldanha L, Silva I, Magalhães P, Schmidt S, Vale N. Advancing Precision Medicine: A Review of Innovative In Silico Approaches for Drug Development, Clinical Pharmacology and Personalized Healthcare. Pharmaceutics. 2024; 16(3):332. https://doi.org/10.3390/pharmaceutics16030332
37. Chengyu Z, Xueyan H, Ying F. Research on disease management of chronic disease patients based on digital therapeutics: A scoping review. Digit Health. 2024 Nov 8;10:20552076241297064. doi: 10.1177/20552076241297064. PMID: 39525556; PMCID: PMC11544657.
38. Wang Q, Yuan F, Zuo X, Li M. Breakthroughs and challenges of organoid models for assessing cancer immunotherapy: a cutting-edge tool for advancing personalised treatments. Cell Death Discov. 2025 May 7;11(1):222. doi: 10.1038/s41420-025-02505-w. PMID: 40335487; PMCID: PMC12059183.
39. Bogoslowski A, An M, Penninger JM. Incorporating Immune Cells into Organoid Models: Essential for Studying Human Disease. Organoids. 2023; 2(3):140-155. https://doi.org/10.3390/organoids2030011

CITE AS: Chelimo Faith Rebecca. (2026). Immunotoxicity of Common Therapeutics in Chronic Diseases: Mechanistic and Safety Considerations. NEWPORT INTERNATIONAL JOURNAL OF RESEARCH IN MEDICAL SCIENCES, 7(2):46-51 https://doi.org/10.59298/NIJRMS/2026/7.2.4651