NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES (NIJSES)

Volume 7 Issue 2 Page 95-100, 2026

ONLINE ISSN:2992-5819 PRINT ISSN:2992-6149

Open Access

https://doi.org/10.59298/NIJSES/2026/72.95100

Oxidative Stress in Drug-Induced Liver Injury: Mechanistic Pathways and Modifying Factors

Ahereza Prissy

Department of Pharmacy Kampala International University Uganda
Email: prissy.ahereza@studwc.kiu.ac.ug

ABSTRACT

Drug-induced liver injury (DILI) is a leading cause of acute liver failure and a major barrier in drug development and clinical therapeutics. Among the diverse mechanisms implicated in DILI, oxidative stress plays a central role in initiating and propagating hepatocellular damage. Oxidative stress results from an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defenses, leading to macromolecular damage, mitochondrial dysfunction, and cell death. A wide range of drugs, including acetaminophen, antibiotics, antiepileptics, and anticancer agents, induce oxidative stress through metabolic activation, redox cycling, and disruption of mitochondrial electron transport. Importantly, individual susceptibility to oxidative damage is influenced by genetic polymorphisms, age, sex, nutritional status, co-medications, and underlying liver disease. Additionally, sex hormones, gut microbiota composition, and environmental exposures modify redox pathways and DILI risk. Understanding how oxidative stress integrates with immune responses, inflammation, and cell death pathways provides insight into mechanisms of liver injury and suggests therapeutic targets. This review synthesizes current knowledge of oxidative stress in DILI pathogenesis, highlights key modifying factors, and discusses potential antioxidant-based interventions for the prevention and mitigation of liver injury.

Keywords: Drug-induced liver injury, oxidative stress, reactive oxygen species, mitochondria, antioxidant defense

INTRODUCTION

Drug-induced liver injury (DILI) refers to liver damage caused by prescription medications, over-the-counter drugs, herbal products, and dietary supplements [1]. DILI encompasses a spectrum from asymptomatic elevations in liver enzymes to fulminant hepatic failure necessitating liver transplantation. It accounts for a significant proportion of acute liver injury cases in clinical practice and remains a leading reason for regulatory actions, drug withdrawals, and restricted approvals[2]. Although the mechanisms of DILI are multifactorial, oxidative stress is recognized as a central initiating event in many forms of injury[3]. Oxidative stress arises when reactive oxygen species (ROS) and reactive nitrogen species (RNS) overwhelm cellular antioxidant defenses. ROS, including superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (•OH), are normally generated during mitochondrial respiration and drug metabolism[4]. Under physiological conditions, antioxidant systems—such as glutathione, superoxide dismutase (SOD), catalase, and peroxiredoxins—neutralize ROS and maintain redox balance[5]. In DILI, drugs or their metabolites disrupt redox homeostasis by increasing ROS production or depleting antioxidants, leading to direct oxidative damage to lipids, proteins, and DNA[6]. Oxidative stress also propagates secondary injury through mitochondrial dysfunction, activation of stress signaling pathways, and promotion of inflammatory responses[7]. The severity and outcome of DILI depend not only on the intrinsic toxicity of the drug but also on host factors that influence redox capacity and stress responses.

This review details the mechanistic pathways by which oxidative stress contributes to DILI, describes modifying factors that influence susceptibility, and discusses emerging strategies to mitigate oxidative injury.

2. Sources and Consequences of Oxidative Stress in the Liver

2.1 Endogenous and Exogenous ROS Production

Reactive oxygen species in hepatocytes arise from both endogenous metabolic processes and exogenous drug-related insults. Under physiological conditions, mitochondria are the primary endogenous source of ROS, generated as byproducts of electron transport during oxidative phosphorylation[8]. Limited electron leakage at complexes I and III leads to superoxide formation, which is normally neutralized by mitochondrial antioxidant systems[9]. During drug-induced toxic stress, however, mitochondrial dysfunction increases electron leakage, overwhelming antioxidant defenses and resulting in excessive ROS accumulation. Drug metabolism represents a major exogenous contributor to oxidative stress in the liver[10]. Cytochrome P450 enzymes, particularly CYP2E1, CYP1A2, and CYP3A4, are heavily involved in the bioactivation of xenobiotics. Many drugs are converted into reactive intermediates that participate in redox cycling, continuously generating ROS while depleting intracellular antioxidants such as glutathione[11]. In addition, activation of hepatic immune responses contributes to oxidative stress. Kupffer cells and recruited inflammatory cells produce ROS as part of innate defense mechanisms[12]. While this response is protective in acute injury, sustained or excessive ROS release extends damage to surrounding hepatocytes and amplifies liver injury.

2.2 Oxidative Damage to Macromolecules

Excessive ROS directly damages essential cellular macromolecules, leading to loss of hepatocellular integrity[13]. Lipid peroxidation targets polyunsaturated fatty acids in cellular membranes, generating reactive aldehydes that disrupt membrane structure and intracellular signaling[14]. Protein oxidation modifies amino acid side chains, impairing enzyme activity and structural stability, particularly in metabolic and mitochondrial proteins[15]. ROS also induce DNA base modifications and strand breaks, triggering apoptosis or cellular senescence. Collectively, these events destabilize cellular homeostasis and activate hepatocellular death pathways that underpin drug-induced liver injury.

3. Mechanisms of ROS Generation in DILI

3.1 Drug Metabolism and Redox Cycling

Many drugs undergo phase I metabolism in the liver via cytochrome P450 enzymes, producing reactive metabolites that can directly or indirectly generate reactive oxygen species[16]. These metabolites may form covalent adducts with cellular proteins or lipids, impairing their function, or participate in redox cycling, a process in which repeated oxidation and reduction reactions continuously produce ROS[17]. Redox cycling amplifies oxidative stress and disrupts the delicate balance between ROS generation and antioxidant defenses.

Acetaminophen is the prototypical example of metabolism-driven oxidative stress. At therapeutic doses, it is mainly metabolized through glucuronidation and sulfation and safely eliminated[17]. In overdose, however, cytochrome P450 enzymes convert acetaminophen to N-acetyl-p-benzoquinone imine, a highly reactive intermediate[18]. This metabolite binds to cellular proteins and depletes glutathione, the primary intracellular antioxidant, leading to unchecked ROS accumulation. The resulting oxidative burden causes mitochondrial injury and activates cell death pathways. Other drugs, including isoniazid, diclofenac, and methotrexate, similarly generate reactive intermediates that disturb redox homeostasis and sensitize hepatocytes to oxidative damage.

3.2 Mitochondrial Dysfunction

Mitochondria serve as both a major source and target of ROS in drug-induced liver injury[19]. Certain hepatotoxic drugs impair electron transport chain function, causing electron leakage and elevated ROS production[20]. This leads to loss of mitochondrial membrane potential, opening of the mitochondrial permeability transition pore, and release of cytochrome c and other pro-apoptotic factors[21]. Mitochondrial dysfunction also shifts metabolism toward anaerobic glycolysis, depleting ATP and triggering apoptosis or necrosis depending on injury severity[22]. Cumulative mitochondrial damage amplifies oxidative stress, establishing a vicious cycle of ROS production and hepatocellular injury[23].

4. Oxidative Stress-Mediated Cell Death Pathways

Oxidative stress triggers hepatocellular death through several interrelated mechanisms, each contributing to the progression and severity of drug-induced liver injury.

4.1 Apoptosis

Reactive oxygen species activate stress-responsive kinases, including c-Jun N-terminal kinase (JNK) and p38 MAPK, which initiate pro-apoptotic signaling cascades[24]. Oxidative damage compromises mitochondrial membrane integrity, facilitating the release of cytochrome c into the cytosol[25]. Cytochrome c then engages the apoptotic protease activating factor-1 (Apaf-1) complex, leading to caspase activation and programmed cell death[26]. Apoptosis is a controlled process that limits inflammatory spillover, but extensive apoptotic loss of hepatocytes contributes significantly to liver dysfunction and parenchymal depletion.

4.2 Necrosis

When oxidative damage exceeds cellular repair capacity and ATP levels are depleted, necrosis becomes the predominant mode of cell death[27]. Necrotic hepatocytes release damage-associated molecular patterns (DAMPs), which amplify local inflammation by activating Kupffer cells and recruiting immune cells[28]. This feed-forward loop extends tissue injury beyond the initially affected hepatocytes, contributing to widespread liver damage.

4.3 Ferroptosis

Ferroptosis is an iron-dependent form of cell death driven by lipid peroxidation[29]. In the context of oxidative stress, impaired glutathione peroxidase 4 activity leads to accumulation of lipid ROS, triggering ferroptotic hepatocyte death[30]. Recent studies indicate that ferroptosis plays a significant role in certain drug-induced liver injuries, particularly those involving iron overload or compromised antioxidant defenses, linking redox imbalance directly to cell demise[31]. Collectively, these oxidative stress-mediated pathways interact dynamically, determining the extent, severity, and reversibility of hepatocellular injury.

5. Immune Activation and Oxidative Stress

Oxidative stress interacts closely with innate and adaptive immune responses in DILI[32]. Hepatocellular damage releases DAMPs that activate Kupffer cells and recruit neutrophils and monocytes[33]. These immune cells generate additional ROS via NADPH oxidase, amplifying oxidative injury. Pro-inflammatory cytokines such as TNF-α and IL-1β upregulate inducible nitric oxide synthase (iNOS), increasing production of nitric oxide (NO) and reactive nitrogen species (RNS)[34]. Peroxynitrite (formed by NO and superoxide reaction) is highly reactive and exacerbates oxidative damage[35]. This bidirectional relationship between oxidative stress and inflammation creates a feed-forward loop that escalates liver injury.

6. Biomarkers of Oxidative Stress in DILI

The identification of reliable biomarkers for oxidative stress in drug-induced liver injury is crucial for early detection, monitoring disease progression, and guiding therapeutic interventions[36]. Among the most studied markers are serum malondialdehyde and 4-hydroxynonenal, which reflect lipid peroxidation and indicate damage to cellular membranes. Protein carbonyls serve as markers of protein oxidation, providing insight into the functional impairment of enzymes and structural proteins within hepatocytes[37]. The ratio of reduced to oxidized glutathione (GSH/GSSG) is a sensitive indicator of cellular redox status, with a decreased ratio signaling heightened oxidative stress. Circulating mitochondrial DNA, released from damaged hepatocytes, reflects mitochondrial injury and may serve as an early warning of hepatocellular dysfunction. Combining these oxidative stress biomarkers with conventional liver function tests and clinical assessment enhances the ability to stratify patients by risk, monitor progression, and potentially identify individuals who may benefit from antioxidant or protective interventions. This integrative approach holds promise for improving the management and prognosis of DILI.

7. Future Perspectives

Future research on oxidative stress in drug-induced liver injury should focus on comprehensive, integrative approaches to improve understanding and patient care. Systems biology techniques can map complex redox pathways and their interactions with drug metabolism, immune responses, and cell death mechanisms[38]. Development of integrative biomarker panels that combine oxidative, inflammatory, and genetic indicators may enhance risk prediction and early detection of DILI. Personalized medicine frameworks incorporating host-specific factors, such as genetic polymorphisms, age, sex, and comorbidities, can guide individualized prevention strategies[39]. Well-designed clinical trials evaluating targeted antioxidant therapies and mitochondria-protective interventions across diverse drug classes are also needed to translate mechanistic insights into effective clinical management[40]. Together, these strategies will advance prevention, early diagnosis, and therapeutic interventions for DILI.

CONCLUSION

Oxidative stress is a fundamental mechanistic pathway in drug-induced liver injury. Through direct macromolecular damage, mitochondrial dysfunction, activation of cell death pathways, and amplification of inflammation, ROS and RNS drive hepatocellular injury across many drug classes. Individual susceptibility to oxidative DILI is shaped by genetic, physiological, and environmental modifiers that influence redox balance. While antioxidant therapies like N-acetylcysteine have transformed the treatment of acetaminophen toxicity, broader strategies targeting oxidative stress and its downstream effects hold promise for improving outcomes in DILI. A deeper mechanistic understanding integrated with clinical biomarkers and personalized risk assessment will pave the way for more effective prevention and intervention.

REFERENCES

1. David S, Hamilton JP. Drug-induced Liver Injury. US Gastroenterol Hepatol Rev. 2010 Jan 1;6:73-80. PMID: 21874146; PMCID: PMC3160634.
2. Hosack T, Damry D, Biswas S. Drug-induced liver injury: a comprehensive review. Therap Adv Gastroenterol. 2023 Mar 21;16:17562848231163410. doi: 10.1177/17562848231163410. PMID:

36968618; PMCID: PMC10031606.
3. Chalasani N, Fontana RJ, Bonkovsky HL, Watkins PB, Davern T, Serrano J, Yang H, Rochon J; Drug Induced Liver Injury Network (DILIN). Causes, clinical features, and outcomes from a prospective study of drug-induced liver injury in the United States. Gastroenterology. 2008 Dec;135(6):1924-34, 1934.e1-4. doi: 10.1053/j.gastro.2008.09.011. Epub 2008 Sep 17. PMID: 18955056; PMCID: PMC3654244.
4. Ogugua, Victor N., Njoku, Obioma U., Egba, Simeon I., Uroko, Robert I and Ignatius Glory. In vitro study of nutritional and antioxidant properties of methanol extract of *Nauclea latifolia* root bark. Biomedical Research, 2018; 29(21): 3766-3773
5. 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
6. Daraghmeh DN, Karaman R. The Redox Process in Red Blood Cells: Balancing Oxidants and Antioxidants. Antioxidants. 2025; 14(1):36. https://doi.org/10.3390/antiox14010036
7. 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
8. Di Meo S, Reed TT, Venditti P, Victor VM. Role of ROS and RNS Sources in Physiological and Pathological Conditions. Oxid Med Cell Longev. 2016;2016:1245049. doi: 10.1155/2016/1245049. Epub 2016 Jul 12. PMID: 27478531; PMCID: PMC4960346.
9. Aranda-Rivera AK, Cruz-Gregorio A, Arancibia-Hernández YL, Hernández-Cruz EY, Pedraza-Chaverri J. RONS and Oxidative Stress: An Overview of Basic Concepts. Oxygen. 2022; 2(4):437-478. https://doi.org/10.3390/oxygen2040030
10. Kowalczyk P, Sulejczak D, Kleczkowska P, Bukowska-Ośko I, Kucia M, Popiel M, Wietrak E, Kramkowski K, Wrzosek K, Kaczyńska K. Mitochondrial Oxidative Stress-A Causative Factor and Therapeutic Target in Many Diseases. Int J Mol Sci. 2021 Dec 13;22(24):13384. doi: 10.3390/ijms222413384. PMID: 34948180; PMCID: PMC8707347.
11. 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
12. Manful CF, Fordjour E, Subramaniam D, Sey AA, Abbey L, Thomas R. Antioxidants and Reactive Oxygen Species: Shaping Human Health and Disease Outcomes. International Journal of Molecular Sciences. 2025; 26(15):7520. https://doi.org/10.3390/ijms26157520
13. Sharma P, Nandave M, Nandave D, Yadav S, Vargas-De-La-Cruz C, Singh S, Tandon R, Ramniwas S, Behl T. Reactive oxygen species (ROS)-mediated oxidative stress in chronic liver diseases and its mitigation by medicinal plants. Am J Transl Res. 2023 Nov 15;15(11):6321-6341. PMID: 38074830; PMCID: PMC10703659.
14. Alum, E.U., Uti, D.E. & Offor, C.E. 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
15. Zhang Z, Yang H, Han F, Guo P. Reactive Oxygen Species as Key Molecules in the Pathogenesis of Alcoholic Fatty Liver Disease and Nonalcoholic Fatty Liver Disease: Future Perspectives. Curr Issues Mol Biol. 2025 Jun 17;47(6):464. doi: 10.3390/cimb47060464. PMID: 40699863; PMCID: PMC12191510.
16. Zhao M, Ma J, Li M, Zhang Y, Jiang B, Zhao X, Huai C, Shen L, Zhang N, He L, Qin S. Cytochrome P450 Enzymes and Drug Metabolism in Humans. Int J Mol Sci. 2021 Nov 26;22(23):12808. doi: 10.3390/ijms222312808. PMID: 34884615; PMCID: PMC8657965.
17. Iacopetta D, Ceramella J, Catalano A, Scali E, Scumaci D, Pellegrino M, Aquaro S, Saturnino C, Sinicropi MS. Impact of Cytochrome P450 Enzymes on the Phase I Metabolism of Drugs. Applied Sciences. 2023; 13(10):6045. https://doi.org/10.3390/app13106045
18. Mazaleuskaya LL, Sangkuhl K, Thorn CF, FitzGerald GA, Altman RB, Klein TE. PharmGKB summary: pathways of acetaminophen metabolism at the therapeutic versus toxic doses. Pharmacogenet Genomics. 2015 Aug;25(8):416-26. doi: 10.1097/FPC.0000000000000150. PMID: 26049587; PMCID: PMC4498995.
19. McGill MR, Jaeschke H. Metabolism and disposition of acetaminophen: recent advances in relation to hepatotoxicity and diagnosis. Pharm Res. 2013 Sep;30(9):2174-87. doi: 10.1007/s11095-013-1007-6. Epub 2013 Mar 6. PMID: 23462933; PMCID: PMC3709007.
20. Mihajlovic M, Vinken M. Mitochondria as the Target of Hepatotoxicity and Drug-Induced Liver Injury: Molecular Mechanisms and Detection Methods. Int J Mol Sci. 2022 Mar 18;23(6):3315. doi: 10.3390/ijms23063315. PMID: 35328737; PMCID: PMC8951158.

21. Allameh A, Niayesh-Mehr R, Aliarab A, Sebastiani G, Pantopoulos K. Oxidative Stress in Liver Pathophysiology and Disease. Antioxidants (Basel). 2023 Aug 22;12(9):1653. doi: 10.3390/antiox12091653. PMID: 37759956; PMCID: PMC10525124.
22. Rao VK, Carlson EA, Yan SS. Mitochondrial permeability transition pore is a potential drug target for neurodegeneration. Biochim Biophys Acta. 2014 Aug;1842(8):1267-72. doi: 10.1016/j.bbadis.2013.09.003. Epub 2013 Sep 18. PMID: 24055979; PMCID: PMC3991756.
23. Nesci S, Rubattu S. Mitochondrial Permeability Transition Pore: The Cardiovascular Disease's Molecular Achilles Heel. Biomedicines. 2025; 13(12):3014. https://doi.org/10.3390/biomedicines13123014
24. Guo X, Chen S, Zhang Z, Dobrovolsky VN, Dial SL, Guo L, Mei N. Reactive oxygen species and c-Jun N-terminal kinases contribute to TEMPO-induced apoptosis in L5178Y cells. Chem Biol Interact. 2015 Jun 25;235:27-36. doi: 10.1016/j.cbi.2015.04.009. Epub 2015 Apr 13. PMID: 25882087; PMCID: PMC5901690.
25. 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
26. Pan J, Chang Q, Wang X, Son Y, Zhang Z, Chen G, Luo J, Bi Y, Chen F, Shi X. Reactive oxygen species-activated Akt/ASK1/p38 signaling pathway in nickel compound-induced apoptosis in BEAS 2B cells. Chem Res Toxicol. 2010 Mar 15;23(3):568-77. doi: 10.1021/tx9003193. PMID: 20112989; PMCID: PMC2838407.
27. Vardar Acar N, Özgül RK. The bridge between cell survival and cell death: reactive oxygen species-mediated cellular stress. EXCLI J. 2023 Jun 22;22:520-555. doi: 10.17179/excli2023-6221. PMID: 37534225; PMCID: PMC10390897.
28. Olufunmilayo EO, Gerke-Duncan MB, Holsinger RMD. Oxidative Stress and Antioxidants in Neurodegenerative Disorders. Antioxidants. 2023; 12(2):517. https://doi.org/10.3390/antiox12020517
29. Akwari, A.A., Okoroh, P.N., Aniokete, U.C., Abba, J.N. Phytochemicals as modulators of ferroptosis: a novel therapeutic avenue in cancer and neurodegeneration. *Mol Biol Rep* **52**, 636 (2025). https://doi.org/10.1007/s11033-025-10752-4
30. Artusi I, Rubin M, Cravin G, Cozza G. Ferroptosis in Human Diseases: Fundamental Roles and Emerging Therapeutic Perspectives. Antioxidants. 2025; 14(12):1411. https://doi.org/10.3390/antiox14121411
31. Zhu W, Fu L, Cui Y, Tang Y, Liu K, Shi L, Gao Y, Li M, Huang L. Ferroptosis in liver fibrosis and its potential intervention strategy. Cell Biol Toxicol. 2025 Nov 25;41(1):152. doi: 10.1007/s10565-025-10111-w. PMID: 41286189; PMCID: PMC12644137.
32. Kozlov EM, Ivanova E, Grechko AV, Wu WK, Starodubova AV, Orekhov AN. Involvement of Oxidative Stress and the Innate Immune System in SARS-CoV-2 Infection. Diseases. 2021 Feb 24;9(1):17. doi: 10.3390/diseases9010017. PMID: 33668325; PMCID: PMC8005963.
33. Vona R, Pallotta L, Cappelletti M, Severi C, Matarrese P. The Impact of Oxidative Stress in Human Pathology: Focus on Gastrointestinal Disorders. Antioxidants. 2021; 10(2):201. https://doi.org/10.3390/antiox10020201
34. Uroko Robert Ikechukwu., Agbafor Amarachi, Uchenna Oluomachi Nancy, Achi Ngozi Kalu, Egba Simeon Ikechukwu**,** Nweje-Anyalowu Paul Chukwuemaka and Ngwu Ogochukwu Rita. Evaluation of Antioxidant Activity of Aqueous Extracts of Palm Friuts *(Elaeis guineensis)* Asian Journal of Biochemistry, 2017; 12: 49-57
35. Aboderin FI, Oduola T, Davison GM, Oguntibeju OO. A Review of the Relationship between the Immune Response, Inflammation, Oxidative Stress, and the Pathogenesis of Sickle Cell Anaemia. Biomedicines. 2023; 11(9):2413. https://doi.org/10.3390/biomedicines11092413
36. Uroko, Robert I.., Adamude, Fatima A.., Egba, Simeon I., Ani, Chijioke C and 1 Ekpenyong, James E. Hepatoprotective Effects of Methanol Extract of *Acanthus montanus* (*acanthaceae*) Leaves on Acetaminophen Induced Liver Injury in Rats. *Pharmacologyonline*, 2020; 1: 248-260
37. Ukpabi-Ugo Jacinta Chigozie., Monanu, Michael Okechukwu., Patrick-Iwuanyanwu, Kingsley and Egbachukwu Simeon Ikechukwu. Potential hepatoprotective effect of different solvent fractions of *Ocimum gratissimum* (O G) in a paracetamol-induced hepatotoxicity in Wistar albino rats. *ScopeMed* 2016; 5(1): 10-16
38. Wang RS, Oldham WM, Maron BA, Loscalzo J. Systems Biology Approaches to Redox Metabolism in Stress and Disease States. Antioxid Redox Signal. 2018 Oct 1;29(10):953-972. doi: 10.1089/ars.2017.7256. Epub 2017 Dec 20. PMID: 29121773; PMCID: PMC6104248.
39. Feelisch M, Cortese-Krott MM, Santolini J, Wootton SA, Jackson AA. Systems redox biology in health and disease. EXCLI J. 2022 Mar 21;21:623-646. doi: 10.17179/excli2022-4793. PMID: 35721574;

PMCID: PMC9203981.
40. Alhaj Sulaiman A, Katanaev VL. Beyond Antioxidants: How Redox Pathways Shape Cellular Signaling and Disease Outcomes. Antioxidants. 2025; 14(9):1142. https://doi.org/10.3390/antiox14091142

CITE AS: Ahereza Prissy (2026). Oxidative Stress in Drug-Induced Liver Injury: Mechanistic Pathways and Modifying Factors. NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES, 7(2):95-100.
https://doi.org/10.59298/NIJSES/2026/72.95100