©IDOSR PUBLICATIONS International Digital Organization for Scientific Research

IDOSR JOURNAL OF APPLIED SCIENCES 11(2):35-41, 2026 ISSN: 2550-7931

https://doi.org/10.59298/IDOSRJAS/2026/1123541 IDOSRJAS11200

The Prostate-Liver-Immune Axis: Shared Pathways in BPH, Hepatotoxicity, and Systemic Inflammation

Mpora Kakwanzi Evelyn

Department of Pharmacognosy Kampala International University Uganda
Email: evelyne.mpora@studwc.kiu.ac.ug

ABSTRACT

Emerging evidence indicates that the prostate, liver, and immune system are linked through common molecular and cellular pathways that influence disease development beyond organ-specific boundaries. Traditionally, benign prostatic hyperplasia (BPH) has been viewed as an isolated urological condition of aging males, while hepatotoxicity is considered a consequence of toxic exposures and metabolic stress. However, systemic inflammation, immune dysregulation, metabolic alterations, and redox imbalance contribute to both prostate enlargement and liver injury. Shared mechanisms include chronic low-grade inflammation, cytokine networks, oxidative stress, influence of metabolic syndrome, gut–liver–prostate interactions, and roles of innate and adaptive immunity. Understanding the prostate–liver–immune axis provides a unified framework to explain overlapping risk factors and comorbidities, with implications for early diagnosis, integrated therapeutic strategies, and biomarker discovery. Addressing systemic inflammatory drivers rather than isolated organ pathology may improve outcomes in patients with co-existing BPH and liver dysfunction.

Keywords: prostate–liver axis; benign prostatic hyperplasia; hepatotoxicity; systemic inflammation; immunometabolism

INTRODUCTION

Benign prostatic hyperplasia (BPH) and hepatotoxicity appear, at first glance, to be distinct clinical entities affecting different organs[1-4]. Yet, both conditions often share similar risk factors, including age, metabolic syndrome, systemic inflammation, and exposures that perturb immune homeostasis. BPH is a non-malignant enlargement of the prostate that can lead to lower urinary tract symptoms, while hepatotoxicity refers to liver injury resulting from drugs, toxins, or metabolic disturbances[5-7]. Systemic inflammation and immune signaling interconnect these conditions, suggesting the existence of a biologically relevant prostate–liver–immune axis[8-9]. concept of an organ axis describes bidirectional communication between organs mediated by circulating factors (cytokines, hormones, metabolites), shared immune pathways, and metabolic networks. In this review, we explore evidence supporting the liver's influence on prostate health and vice versa through immunometabolic and inflammatory pathways. We also examine how disruptions in redox balance, innate and adaptive immune responses, and gut-derived signals contribute to disease progression in both tissues[10-15]. Recognizing these shared mechanisms fosters integrated approaches to prevention, diagnosis, and therapy that go beyond single-organ treatment.

2. Benign Prostatic Hyperplasia: Immune and Metabolic Underpinnings

2.1 Clinical Features and Pathophysiology

Benign prostatic hyperplasia (BPH) is one of the most common urological disorders affecting aging men, with prevalence rising sharply after the fifth decade of life[16-18]. Clinically, BPH manifests with lower urinary tract symptoms, including urinary frequency, nocturia, weak stream, and incomplete bladder emptying, which can significantly impair quality of life. Pathologically, BPH is characterized by hyperplasia of both stromal and epithelial cells, predominantly in the transition zone of the prostate, leading to progressive enlargement and potential obstruction of the urethra[19-24]. While hormonal regulation, particularly by androgens such as dihydrotestosterone (DHT), has long been considered central to BPH pathogenesis, hormonal factors alone cannot fully account for variability in disease onset, progression, or severity[25-29]. Increasing evidence highlights that BPH arises from a multifactorial process integrating hormonal, immune, and metabolic influences, suggesting that systemic factors beyond local androgen signaling are crucial contributors to prostate enlargement.

2.2 Inflammation in BPH

Chronic inflammation has emerged as a key driver of prostatic hyperplasia. Histological studies consistently demonstrate infiltration of BPH tissue by immune cells, including macrophages, T lymphocytes, and mast cells [30-34]. These cells secrete a variety of pro-inflammatory and pro-fibrotic cytokines, such as interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-α (TNF-α), and transforming growth factor-β (TGF-β). These cytokines stimulate stromal fibroblast proliferation, extracellular matrix deposition, and tissue remodeling, creating a microenvironment conducive to sustained cellular proliferation and organ enlargement[35-38]. Persistent inflammatory signaling also contributes to oxidative stress, DNA damage, and senescence-associated secretory phenotypes in epithelial and stromal cells, further amplifying prostatic growth.

2.3 Metabolic Syndrome and Prostatic Growth

Metabolic syndrome significantly influences the development and progression of BPH. Central obesity, dyslipidemia, insulin resistance, and hypertension collectively create a pro-inflammatory and pro-proliferative systemic milieu[10,39-43]. Hyperinsulinemia and elevated insulin-like growth factor levels act as mitogens in prostatic tissue, enhancing stromal and epithelial cell proliferation. Adipokines, including leptin and adiponectin, modulate cell growth, angiogenesis, and inflammatory signaling within the prostate[44-48]. Additionally, systemic insulin resistance promotes chronic low-grade inflammation, which interacts with local prostatic immune responses to accelerate tissue remodeling and hyperplasia[49-51]. This integrated immune-metabolic network helps explain the strong association between metabolic disorders and increased BPH risk and severity.

3. Hepatotoxicity: Immune and Metabolic Contributors

3.1 Overview of Hepatotoxicity

Hepatotoxicity refers to liver injury due to chemical exposures (drugs, herbals, toxins), viral infections, or metabolic stress[52-55]. Liver injury ranges from asymptomatic elevations in liver enzymes to acute liver failure. The liver's central role in detoxification exposes it to a constant influx of xenobiotics, reactive metabolites, and microbial products from the gut, making it a hub for immune and metabolic integration.

3.2 Immune-Mediated Liver Injury

Like BPH, chronic inflammation is fundamental in many forms of liver injury. Kupffer cells (liver-resident macrophages), recruited monocyte-derived macrophages, and lymphocytes produce pro-inflammatory cytokines in response to hepatocellular stress[56-59]. Cytokine signaling through NF-κB and JAK/STAT pathways propagates inflammatory cascades that can progress to steatohepatitis and fibrosis.

3.3 Metabolic Stress and Oxidative Injury

Metabolic conditions such as obesity and type 2 diabetes predispose individuals to non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH)[15,60]. Excess lipid accumulation in hepatocytes leads to oxidative stress, endoplasmic reticulum (ER) stress, and mitochondrial dysfunction[61]. Reactive oxygen species (ROS) not only damage hepatocellular components but also act as signaling molecules that activate immune cells.

4. Shared Mechanisms Linking Prostate and Liver Pathology

4.1 Chronic Low-Grade Inflammation

Chronic low-grade inflammation is a unifying feature of BPH and hepatotoxic processes[17]. Systemic inflammatory mediators such as IL-6 and TNF-α, elevated in metabolic syndrome and age-related immune dysregulation, can affect multiple organs[18]. IL-6, for example, promotes hepatic acute phase responses and

supports prostatic cell proliferation. TNF-α contributes to insulin resistance and exacerbates inflammatory loops in both liver and prostate tissues[19].

4.2 Oxidative Stress and Redox Imbalance

Oxidative stress, defined by an imbalance between ROS production and antioxidant defenses, underlies many chronic diseases[20]. In the prostate, ROS contribute to DNA damage and pro-inflammatory signaling, promoting stromal proliferation. In the liver, oxidative stress from metabolic overload or toxic metabolites triggers immune activation and cell death[21]. Shared redox pathways involve activation of transcription factors such as NF-κB and AP-1, linking oxidative signals to inflammatory gene expression.

4.3 Innate Immune Activation and Pattern Recognition

Pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs) detect microbial and damage-associated molecular patterns (DAMPs)[22]. Activation of TLRs on liver and prostate cells triggers pro-inflammatory signaling. Gut-derived microbial products that reach systemic circulation due to increased intestinal permeability can stimulate TLRs in distant organs, creating a gut-liver-prostate axis of immune activation[23].

5. Metabolic Syndrome: A Central Driver of Systemic Inflammation

5.1 Insulin Resistance and Cytokine Networks

Metabolic syndrome drives systemic inflammation through adipose tissue-derived cytokines (adipokines), free fatty acids, and altered glucose metabolism[24]. Insulin resistance is associated with elevated circulating levels of IL-6, TNF-α, and C-reactive protein, which influence tissue inflammation in both liver and prostate[25]. These cytokines not only modulate immune cell activity but also interfere with insulin signaling, creating a feed-forward loop of metabolic and immune dysregulation.

5.2 Hormonal Dysregulation and Steroid Metabolism

Sex steroids such as testosterone and estrogen are altered in metabolic syndrome and influence both hepatic and prostatic biology[26]. Obesity and insulin resistance can lead to increased aromatization of androgens to estrogens, which affects prostate growth. In the liver, altered steroid metabolism affects bile acid synthesis and systemic energy balance, maintaining a cycle of metabolic stress.

5.3 Lipotoxicity and Cellular Stress

Free fatty acids and toxic lipid species accumulate in liver and peripheral tissues in metabolic syndrome. Lipotoxicity induces ER stress and mitochondrial dysfunction, thereby increasing ROS production and cytokine release [27]. These changes drive macrophage activation and further amplify inflammation that affects distant organs such as the prostate.

6. Gut–Liver–Prostate Interactions

6.1 Microbiome and Immune Modulation

The gut microbiome influences systemic immune tone through metabolite production (short-chain fatty acids, bile acids) and modulation of intestinal permeability[28]. Dysbiosis increases translocation of microbial products like lipopolysaccharide (LPS), which activate TLRs on hepatic and prostatic cells, promoting inflammation[29]. Emerging evidence suggests that microbial metabolites may directly influence prostate cell proliferation and immune responses.

6.2 Bile Acids as Signaling Molecules

Bile acids, produced by the liver and modified by gut bacteria, act as signaling molecules regulating metabolic and immune pathways through receptors such as FXR and TGR5[30]. Altered bile acid profiles in metabolic syndrome can influence systemic inflammation, glucose metabolism, and cellular stress responses, linking liver metabolism with remote organ effects.

7. Clinical Implications

7.1 Comorbidity of BPH and Liver Disease

Epidemiological data suggest increased prevalence of BPH and lower urinary tract symptoms in individuals with metabolic syndrome and chronic liver disorders[31]. While causal relationships remain under investigation, overlapping risk factors such as obesity and systemic inflammation likely contribute to this comorbidity.

7.2 Biomarkers Across Organs

Circulating markers of inflammation (IL-6, TNF-α), oxidative stress (malondialdehyde, 8-isoprostane), and metabolic dysfunction (insulin, adiponectin) may serve as shared biomarkers for liver and prostate pathology[32]. Integrated biomarker panels could enhance early detection of systemic inflammatory states affecting multiple organs.

7.3 Therapeutic Strategies Targeting the Axis

Treating underlying systemic inflammation and metabolic dysfunction may yield benefits for both BPH and liver health[33]. Lifestyle interventions (diet, exercise) reduce adiposity, improve insulin sensitivity, and decrease systemic cytokines[34]. Pharmacologic agents targeting insulin resistance (metformin), inflammation (NSAIDs with caution), or oxidative stress (antioxidants) may attenuate disease progression in both organs[35]. Modulation of the gut microbiome through diet, probiotics, or prebiotics represents an emerging approach.

8. Future Directions and Research Priorities

Further research is needed to clarify the mechanistic links in the prostate–liver–immune axis[36]. Longitudinal studies assessing how metabolic syndrome and systemic inflammation mediate comorbid prostate and liver disease will inform prevention and management[37]. Multi-omics approaches (genomics, transcriptomics, metabolomics, microbiomics) can identify shared pathways and therapeutic targets[38]. Clinical trials evaluating integrated therapies that address systemic drivers rather than isolated organ symptoms are warranted.

CONCLUSION

The prostate–liver–immune axis highlights the interconnected nature of chronic diseases affecting different organs. Shared pathways involving immune activation, metabolic dysfunction, oxidative stress, and gut-derived signals underlie both BPH and liver injury. Embracing a systems-level view that transcends organ boundaries may improve diagnosis, risk stratification, and treatment strategies, ultimately leading to better outcomes for patients with co-occurring inflammatory and metabolic disorders.

REFERENCES

1. Robert I. Uroko., Charles N. Chukwu., Simeon I. Egba1., Fatima A. Adamude andJoy C. Ajuzie (2020) Combined ethanol extract of Funtumia africana and Abutilon mauritianium leaves improves the lipid profile and kidney function indices of benign prostatic hyperplasia in rats. Acta Sci. Pol. Technol. Aliment. 19(4): 395-4045
2. Obeagu, E.I., Obeagu, G.U. and Ugwu, O. P. C. Benign Prostatic Hyperplasia: A Review. *Eurasian Experiment Journal of Public Health (EEJPH).* 2023; 4(1): 1-3.
3. Ibiam, U. A., Uti, D. E., Ejeogo, C. C., Orji, O. U., Aja, P. M., Ezeaani, N. N., Chukwu, C., AlokeC., Chinedum, K. E., Agu, P. and Nwobodo, V.In Vivo and in Silico Assessment of Ameliorative Effects of Xylopia aethiopica on Testosterone Propionate-Induced Benign Prostatic Hyperplasia. *Pharmaceut Fronts.* 2023;5: e64–e76.DOI:10.1055/s-0043-1768477
4. Oseni SO, Naar C, Pavlović M, Asghar W, Hartmann JX, Fields GB, Esiobu N, Kumi-Diaka J. The Molecular Basis and Clinical Consequences of Chronic Inflammation in Prostatic Diseases: Prostatitis, Benign Prostatic Hyperplasia, and Prostate Cancer. Cancers. 2023; 15(12):3110. https://doi.org/10.3390/cancers15123110
5. Uroko Robert Ikechukwu, Fatima Amin Adamude, Egba Simeon Ikechukwu, Chinedu Paulinus Nwuke, Chidinma Lilian Asadu and Peter Anyaorah (2021) Effect of combined ethanol extract of Funtumia Africana and Abutilon mauritanium leaves on prostate biomarkers and serum mineral levels in prostatic hyperplasia induced in rats. J. Renal Endocrinol 7:e06
6. IR Uroko, F A Adamude, S I Egba, C N Chukwu, C L Asadu, E C Okwara (2020) Effects of combined ethanol extract of Funtumia africana and Abutilon mauritianum leaves (FAAM) on liver function indices of benign prostatic hyperplasia (BPH) induced rats, Herba Polonica,66 (3): 24-35
7. Edyedu I, Ugwu OP, Ugwu CN, Alum EU, Eze VHU, Basajja M, Ugwu JN, Ogenyi FC, Ejemot-Nwadiaro RI, Okon MB, Egba SI, Uti DE, Aja PM. The role of pharmacological interventions in managing urological complications during pregnancy and childbirth: A review. Medicine (Baltimore). 2025 Feb 14;104(7):e41381. doi: 10.1097/MD.0000000000041381. PMID: 39960970; PMCID: PMC11835077.
8. Uhuo EN, Egba SI, Obike CA, Anyiam PN, Alaebo PO, Okeke PM, et al. Combined extracts of Syzygium aromaticum (Clove) and Xylopia aethiopica (Negro pepper) seeds inhibit testosterone propionate-induced benign prostatic hyperplasia in Wistar rats. All Life [Internet]. 2024 Dec 5;17(1). Available from:https://www.tandfonline.com/doi/epdf/10.1080/26895293.2024.2435277
9. Ibiam U. A., Ejeogo, C.C., Orji, O. U. Aja, P. M., Ezeani, N. N., Chukwu, C., Aloke, C., Itodo, M. O., Agada, S. A., Umoru, G. U., Obeten, U. N., Nwobodo, V. O. G., Nwadum, S. K., Udoudoh, M. P. Xylopia aethiopica Attenuates Oxidative Stress and Hepatorenal Damage in Testosterone Propionate-Induced

Benign Prostatic Hyperplasia in Rats. Journal of Health and Allied Sciences. 2024, 01: 1-148. https://doi.org/10.1055/s-0043-1777836.

10. Hamooya BM, Siame L, Muchaili L, Masenga SK, Kirabo A. Metabolic syndrome: epidemiology, mechanisms, and current therapeutic approaches. Front Nutr. 2025 Sep 3;12:1661603. doi: 10.3389/fnut.2025.1661603. PMID: 40969606; PMCID: PMC12441046.
11. Choi W, Woo GH, Kwon T-H, Jeon J-H. Obesity-Driven Metabolic Disorders: The Interplay of Inflammation and Mitochondrial Dysfunction. International Journal of Molecular Sciences. 2025; 26(19):9715. https://doi.org/10.3390/ijms26199715
12. Banerjee D, Mani A. Obesity's systemic impact: exploring molecular and physiological links to diabetes, cardiovascular disease, and heart failure. Front Endocrinol (Lausanne). 2025 Nov 7;16:1681766. doi: 10.3389/fendo.2025.1681766. PMID: 41282294; PMCID: PMC12634369.
13. Francis P, Navarro VJ. Drug-Induced Hepatotoxicity. [Updated 2024 Sep 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557535/
14. Nguyen-Lefebvre AT, Horuzsko A. Kupffer Cell Metabolism and Function. J Enzymol Metab. 2015;1(1):101. Epub 2015 Aug 14. PMID: 26937490; PMCID: PMC4771376.
15. Akter S. Non-alcoholic Fatty Liver Disease and Steatohepatitis: Risk Factors and Pathophysiology. Middle East J Dig Dis. 2022 Apr;14(2):167-181. doi: 10.34172/mejdd.2022.270. Epub 2022 Apr 30. PMID: 36619154; PMCID: PMC9489315.
16. Kosmalski M, Śliwińska A, Drzewoski J. Non-Alcoholic Fatty Liver Disease or Type 2 Diabetes Mellitus—The Chicken or the Egg Dilemma. Biomedicines. 2023; 11(4):1097. https://doi.org/10.3390/biomedicines11041097
17. Inamura S, Terada N. Chronic inflammation in benign prostatic hyperplasia: Pathophysiology and treatment options. Int J Urol. 2024 Sep;31(9):968-974. doi: 10.1111/iju.15518. Epub 2024 Jun 27. PMID: 38934050; PMCID: PMC11524144.
18. Chughtai B, Lee R, Te A, Kaplan S. Role of inflammation in benign prostatic hyperplasia. Rev Urol. 2011;13(3):147-50. PMID: 22110398; PMCID: PMC3221555.
19. Kaltsas A, Giannakas T, Stavropoulos M, Kratiras Z, Chrisofos M. Oxidative Stress in Benign Prostatic Hyperplasia: Mechanisms, Clinical Relevance and Therapeutic Perspectives. Diseases. 2025; 13(2):53. https://doi.org/10.3390/diseases13020053
20. 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
21. 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
22. Chen R, Zou J, Chen J, Zhong X, Kang R, Tang D. Pattern recognition receptors: function, regulation and therapeutic potential. Signal Transduct Target Ther. 2025 Jul 11;10(1):216. doi: 10.1038/s41392-025-02264-1. PMID: 40640149; PMCID: PMC12246121.
23. Wicherska-Pawłowska K, Wróbel T, Rybka J. Toll-Like Receptors (TLRs), NOD-Like Receptors (NLRs), and RIG-I-Like Receptors (RLRs) in Innate Immunity. TLRs, NLRs, and RLRs Ligands as Immunotherapeutic Agents for Hematopoietic Diseases. Int J Mol Sci. 2021 Dec 13;22(24):13397. doi: 10.3390/ijms222413397. PMID: 34948194; PMCID: PMC8704656.
24. Swarup S, Ahmed I, Grigorova Y, et al. Metabolic Syndrome. [Updated 2024 Mar 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459248/
25. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. Nat Rev Immunol. 2011 Feb;11(2):85-97. doi: 10.1038/nri2921. Epub 2011 Jan 21. PMID: 21252989; PMCID: PMC3518031.
26. Kasarinaite A, Sinton M, Saunders PTK, Hay DC. The Influence of Sex Hormones in Liver Function and Disease. Cells. 2023 Jun 11;12(12):1604. doi: 10.3390/cells12121604. PMID: 37371074; PMCID: PMC10296738.
27. Römer A, Linn T, Petry SF. Lipotoxic Impairment of Mitochondrial Function in β-Cells: A Review. Antioxidants (Basel). 2021 Feb 15;10(2):293. doi: 10.3390/antiox10020293. PMID: 33672062; PMCID:

PMC7919463.

28. Nwali, B. U., Aniokete, U. C., Emeruwa, A. P., Obasi, D. C., Okoroh, P. N., … & Aja, P. M. (2025). Gut microbiota at the crossroads of food additives, pollutants, and chronic disease risk. Toxicology and Environmental Health Sciences. https://doi.org/10.1007/s13530-025-00295-3
29. Ugwu, O.P., Okon, M.B., Alum, E.U., Ugwu, C.N., Anyanwu, E.G., Mariam, B., et al. (2025). Unveiling the therapeutic potential of the gut microbiota-brain axis: Novel insights and clinical applications in neurological disorders. Medicine (Baltimore). 2025 Jul 25;104(30):e43542. doi: 10.1097/MD.0000000000043542.
30. Edeh, O.F., Ainbeyoona, C. Unveiling the microbial orchestra: exploring the role of microbiota in cancer development and treatment. *Discov Onc* **16**, 646 (2025). https://doi.org/10.1007/s12672-025-02352-2
31. He J, Pan X, Liu D, Li J, Li Y, Wang Y, Guo J, Zhou Q, Zhou L, Wang L. Association between metabolic syndrome and risk of benign prostatic hyperplasia: a prospective cohort study of 163 975 participants. J Glob Health. 2025 Oct 3;15:04275. doi: 10.7189/jogh.15.04275. PMID: 41039869; PMCID: PMC12491909.
32. Manna P, Jain SK. Obesity, Oxidative Stress, Adipose Tissue Dysfunction, and the Associated Health Risks: Causes and Therapeutic Strategies. Metab Syndr Relat Disord. 2015 Dec;13(10):423-44. doi: 10.1089/met.2015.0095. PMID: 26569333; PMCID: PMC4808277.
33. Aja, P.M, Agu, P.C., Musyoka, A.M., Ngwueche, W., Odo, J.U., Alum, E.U., et al. Integrative Approaches to Prostate Disease Management: Nutrition, Exercise, and Lifestyle Modifications. *American Journal of Men's Health*. 2025;19(3). doi:10.1177/15579883251344571
34. Zhang F, Li W. Impact of fatty liver index and metabolic dysfunction-associated steatotic liver disease on the risk of benign prostatic hyperplasia in older male adults. BMC Geriatr. 2025 Aug 14;25(1):625. doi: 10.1186/s12877-025-06314-9. PMID: 40813969; PMCID: PMC12351979.
35. Csikós E, Horváth A, Ács K, Papp N, Balázs VL, Dolenc MS, Kenda M, Kočevar Glavač N, Nagy M, Protti M, et al. Treatment of Benign Prostatic Hyperplasia by Natural Drugs. Molecules. 2021; 26(23):7141. https://doi.org/10.3390/molecules26237141
36. Xu Y, An L, Xie J, Luo C, Huang X, Luo G. The gut-prostate axis in benign prostatic hyperplasia: systematic review of microbial dysbiosis and pathogenic mechanisms. BMC Urol. 2026 Feb 2;26(1):26. doi: 10.1186/s12894-025-02003-2. PMID: 41629891; PMCID: PMC12866195.
37. Matsushita M, Fujita K, Hatano K, De Velasco MA, Tsujimura A, Uemura H, Nonomura N. Emerging Relationship between the Gut Microbiome and Prostate Cancer. World J Mens Health. 2023 Oct;41(4):759-768. doi: 10.5534/wjmh.220202. Epub 2023 Feb 22. PMID: 36876743; PMCID: PMC10523130.
38. Singh S, Kriti M, Catanzaro R, Marotta F, Malvi M, Jain A, Verma V, Nagpal R, Tiwari R, Kumar M. Deciphering the Gut–Liver Axis: A Comprehensive Scientific Review of Non-Alcoholic Fatty Liver Disease. Livers. 2024; 4(3):435-454. https://doi.org/10.3390/livers4030032
39. Ugwu OPC, Offor CE, Ibiam UA, Ezugwu AL, Uraku AJ, Igwe CN. The effect of ethanol extract of Jatropha curcas on renal markers of chloroform intoxicated albino Wistar rats. Eur J Biol Sci. 2015;7(1):21–5.
40. Ugwu OPC, Nzubechukwu E. The effect of ethanol leaf extract of Jatropha curcas on chloroform-induced hepatotoxicity in albino rats. Glob J Biotechnol Biochem. 2015;10:11–5.
41. Ugwu OPC, Onwe SC, Okon MB. The effect of methanol extract of Rauwolfia vomitoria on lipid profile of chloroform-intoxicated Wistar albino rats. IAA J Sci Res. 2022;8(1):73–82.
42. Ugwu OPC. The effect of ethanol leaf extract of Rauwolfia vomitoria on hepatic markers of chloroform-intoxicated albino Wistar rats. IAA J Appl Sci. 2022;8(1):87–97.
43. Ugwu OPC. The effect of methanol leaf extract of Rauwolfia vomitoria on hepatic markers of chloroform-intoxicated albino Wistar rats. IAA J Appl Sci. 2022;8(1):76–86.
44. Aja PM, Ezeudeh NL, Umahi BG, Ugwu OPC, Enechi OC, Edwin N, et al. Hepato-protective effect of ethanol extract of Pterocarpus santalinoides leaf on carbon tetrachloride (CCl4)-induced albino rats. Caribb J Sci Technol. 2016;4(1):882–95.
45. Enechi OC, Okpe CC, Ibe GN, Omeje KO, Ugwu OPC. Effect of Buchholzia coriacea methanol extract on haematological indices and liver function parameters in Plasmodium berghei-infected mice. Global Vet. 2016;16(1):57–66.

46. Offor CE, Ibiam UA, Ugwu PC, Nwankwo H. The effect of ethanol leaf-extract of Gmelina arborea on liver enzymes. Eur J Biol Sci. 2015;7(1):38–40.
47. Offor CE, Nwankwegu NJ, Ugwu OPC, Aja PM. The effects of ethanol leaf-extract of Pterocarpus santalinoides on liver enzymes of albino rats. 2015.
48. Aja PM, Udeh SMC, Opajobi AO, Uzuegbu UE, Alum EU, Edwin N, et al. Hepato-protective effect of aqueous leaf-extract of Talinum triangulare in monosodium glutamate (MSG)-induced hepatic damage in albino rats. Indo Am J Pharm Sci. 2017;4(2):464–70.
49. Alum EU, Umoru GU, Uti DE, Aja PM, Ugwu OPC, Orji OU, et al. Hepato-protective effect of ethanol leaf extract of Datura stramonium in alloxan-induced diabetic albino rats. J Chem Soc Nigeria. 2022;47(5).
50. Orji OU, Ibiam UA, Aja PM, Uraku AJ, Inya-Agha OR, Ugwu OPC, et al. Hepatoprotective activity of ethanol extract of Vernonia ambigua against carbon tetrachloride-induced hepatotoxicity in albino rats. IOSR J Dent Med Sci. 2015;14:22–9.
51. Orji OU, Ibiam UA, Aloke C, Awoke J, Aja PM, Ugwu OPC, et al. Biochemical assessment of ethanol leaf extract of Cnidoscolus aconitifolius on liver integrity of albino rat treated with lead. Glob J Pharmacol. 2016;10(4):108–13.
52. Ezekwe CI, Ada AC, Ugwu OPC. Effects of methanol extract of Parkia biglobosa stem bark on the liver and kidney functions of albino rats. 2013.
53. Ezekwe CI, Uzomba CR, Ugwu OPC. The effect of methanol extract of Talinum triangulare (water leaf) on the hematology and some liver parameters of experimental rats. Glob J Biotechnol Biochem. 2013;8(2):51–60.
54. Ugwu OPC, Amasiorah VI. Review on health implications, benefits and biochemistry of alcohol intoxication. INOSR Exp Sci. 2020;6(1):62–74.
55. Ezeonwumelu JOC, Uhama KC, Ugwu OPC, Alum EU. Comprehensive overview of alcohol intoxication: mechanisms, effects, and interventions. Res Invent J Sci Exp Sci. 2024;3(1):12–20.
56. Nwaka AC, Ikechi-Agba MC, Ugwu OPC, Igwenyi IO, Agbafor KN, Ibiam UA, et al. The effects of ethanol extracts of Jatropha curcas on some hematological parameters of chloroform intoxicated rats. Am-Eur J Sci Res. 2015;10(1):45–9.
57. Ibiam UA, Ama-Udu, Egwu OA, Ugwu OPC. The protective effect of Gongronema latifolium leaf extract against hepatotoxicity of Rambo and Baygon mosquito coil smoke respectively in albino rats. World Appl Sci J. 2015;33(12):1915–22.
58. Odo CE, Nwodo OFC, Joshua PE, Ubani CS, Etim OE, Ugwu OPC. Effects of Bonny light crude oil on anti-oxidative enzymes and total proteins in Wistar rats. Afr J Biotechnol. 2012;11(98):16455–60.
59. Ozioko PC, Ugwu OPC, Chilaka FC, Ude S. Purification and characterization of beta-D-glucosidase from the digestive tracts of an African Giant Snail (Achatina achatina). 2013.
60. Ozioko PC, Ikeyi AP, Ugwu OPC. Review article: cellulases, their substrates, activity and assay methods. Exp. 2013;12(2):778–85.
61. Ezekwe CI, Okorie A, Ugwu OPC, Nwodo OFC, Egba SC. Blood pressure lowering effect of extract of Gongronema latifolium. Res J Pharm Biol Chem Sci. 2014;5(2):952.

CITE AS: Mpora Kakwanzi Evelyn (2026). The Prostate-Liver-Immune Axis: Shared Pathways in BPH, Hepatotoxicity, and Systemic Inflammation. IDOSR JOURNAL OF APPLIED SCIENCES 11(2):35-41. https://doi.org/10.59298/IDOSRJAS/2026/1123541