NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES (NIJSES)

Volume 7 Issue 2 Page 10-15, 2026

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

Open Access

https://doi.org/10.59298/NIJSES/2026/72.1015

Immune Profiling in BPH: Understanding Chronic Inflammation as a Driver of Prostate Enlargement

Abaho Areeba Fortunate

Department of Pharmacy Kampala International University Uganda
Email address: fortunate.abaho@studwc.kiu.ac.ug

ABSTRACT

Benign prostatic hyperplasia (BPH) is a highly prevalent age-associated condition characterized by progressive enlargement of the prostate and lower urinary tract symptoms that significantly impair quality of life. Traditionally regarded as a hormonally driven disorder, BPH is now increasingly recognized as a chronic inflammatory disease in which immune dysregulation plays a central pathogenic role. Immune profiling studies of prostatic tissue, peripheral blood, and expressed prostatic secretions have consistently demonstrated persistent infiltration of innate and adaptive immune cells, accompanied by sustained cytokine and chemokine production. This chronic inflammatory milieu promotes epithelial and stromal proliferation, extracellular matrix remodeling, smooth muscle hypercontractility, and fibrotic changes that collectively drive prostate enlargement and functional obstruction. Emerging evidence also highlights the interaction between immune activation, oxidative stress, cellular senescence, aging, and metabolic dysfunction as critical amplifiers of inflammatory signaling in BPH. Importantly, interindividual variability in immune signatures correlates with prostate volume, symptom severity, and therapeutic responsiveness, underscoring the clinical relevance of immune heterogeneity. This section synthesizes current knowledge on immune cell composition, inflammatory mediators, and mechanistic pathways linking chronic inflammation to prostatic remodeling. Understanding immune profiling in BPH provides a framework for redefining disease pathogenesis and supports the development of targeted immunomodulatory strategies as adjuncts or alternatives to conventional therapies.

Keywords: Benign prostatic hyperplasia; chronic inflammation; immune profiling; cytokines; prostate remodeling

INTRODUCTION

Benign prostatic hyperplasia is one of the most prevalent nonmalignant disorders affecting aging men, with incidence and clinical impact increasing markedly after the fifth decade of life [1]. Although androgen signaling and age-related hormonal changes remain fundamental contributors to prostatic growth, these factors alone do not adequately explain the wide variability observed in disease onset, progression, symptom severity, or therapeutic response [2]. Increasing evidence now implicates chronic inflammation as a central pathogenic mechanism that integrates hormonal imbalance, metabolic dysfunction, oxidative stress, and progressive tissue remodeling in BPH [3]. Early histopathological studies consistently reported inflammatory cell infiltrates in hyperplastic prostate tissue, yet these findings were long regarded as incidental or secondary to tissue enlargement. Advances in immune profiling methodologies, including immunohistochemistry, flow cytometry, transcriptomic analyses, and multiplex cytokine assays, have fundamentally reshaped this interpretation [4]. These approaches have revealed structured and persistent immune cell populations within the prostate, accompanied by sustained production of pro-inflammatory mediators. Importantly, the extent of inflammation correlates with prostate volume, symptom burden, and risk of disease progression. Current understanding recognizes inflammation in BPH as an active,

organized, and self-perpetuating process that directly influences epithelial and stromal proliferation, extracellular matrix remodeling, and smooth muscle tone [5]. This paradigm shift reframes BPH as an immune-mediated disorder rather than a purely hormonally driven condition [6]. Appreciating immune dysregulation as a primary driver of pathogenesis provides a mechanistic framework for disease heterogeneity and highlights novel opportunities for targeted, inflammation-focused therapeutic strategies [7].

2. Immune Cell Infiltration in the Hyperplastic Prostate

2.1 Adaptive Immune Cells

Adaptive immune responses play a dominant role in the inflammatory landscape of BPH. $CD4^+$ T lymphocytes constitute the most abundant immune cell population within hyperplastic prostatic tissue [8]. Phenotypic analyses reveal a skewing toward pro-inflammatory T helper subsets, particularly Th1 and Th17 cells [9]. Th1 cells secrete interferon-γ and tumor necrosis factor-α, which activate stromal fibroblasts, enhance antigen presentation, and sustain macrophage activation. Th17 cells produce interleukin-17, a cytokine strongly implicated in chronic inflammatory and fibrotic disorders, promoting recruitment of neutrophils and amplification of local cytokine networks[10]. $CD8^+$ cytotoxic T cells are also present but appear less prominent than $CD4^+$ subsets. Their contribution may involve epithelial injury and antigen release, perpetuating immune activation [11]. B cells and plasma cells are detected in a subset of patients, suggesting localized humoral immune responses and potential autoantibody production against prostatic antigens [12]. Regulatory T cells, which normally suppress excessive immune activation, are detectable in BPH tissue but often insufficient in number or functional capacity to counterbalance ongoing inflammation [13]. This imbalance favors chronic immune activation rather than resolution, reinforcing the concept of immune dysregulation in BPH.

2.2 Innate Immune Cells

Innate immune cells are equally critical in shaping the inflammatory microenvironment [14]. Macrophages are abundant within the prostatic stroma and display a mixed activation phenotype. Rather than conforming strictly to pro-inflammatory or anti-inflammatory states, these macrophages secrete both inflammatory cytokines, such as interleukin-6 and interleukin-1β, and profibrotic growth factors, including transforming growth factor-β [15]. This dual functionality enables macrophages to simultaneously sustain inflammation and drive tissue remodeling. Mast cells are another prominent innate immune population in BPH [16]. They localize preferentially to stromal regions and perivascular areas, where they release histamine, proteases, cytokines, and angiogenic factors [17]. Mast cell mediators enhance smooth muscle contraction, vascular permeability, and fibroblast activation, linking immune activation directly to lower urinary tract symptoms [18]. Neutrophils and dendritic cells are less abundant but contribute to antigen presentation and early inflammatory signaling, particularly during episodes of acute exacerbation superimposed on chronic inflammation.

3. Cytokine and Chemokine Networks Sustaining Chronic Inflammation

Chronic inflammation in benign prostatic hyperplasia is sustained by a tightly regulated but persistent network of cytokines and chemokines that orchestrate immune cell recruitment, activation, and survival within the prostatic microenvironment [19]. Among these mediators, interleukin-6 plays a pivotal role and is consistently elevated in hyperplastic prostate tissue. Increased interleukin-6 levels correlate with prostate volume, inflammatory burden, and severity of lower urinary tract symptoms [20]. Through activation of downstream signaling pathways, interleukin-6 promotes epithelial cell proliferation, stromal expansion, and resistance to apoptosis, directly linking inflammatory signaling to tissue growth [21]. Interleukin-8 serves as a key chemotactic and pro-angiogenic factor, facilitating recruitment of neutrophils and promoting vascular remodeling that supports sustained immune infiltration [22]. Additional chemokines, including CCL2 and CXCL12, enhance the migration and retention of monocytes, macrophages, and lymphocytes within the prostate, reinforcing immune cell accumulation and local inflammatory signaling. Tumor necrosis factor-α and interleukin-1β further amplify inflammatory responses by activating transcription factors that drive expression of secondary cytokines, chemokines, and adhesion molecules [23]. Crucially, these inflammatory mediators function within interconnected signaling loops rather than as isolated factors. Cytokine-driven immune cell activation leads to further mediator release, perpetuating inflammation even in the absence of an active infectious trigger [24]. This self-sustaining cytokine and chemokine network is characteristic of chronic inflammatory disorders and represents a defining pathogenic feature of progressive benign prostatic hyperplasia.

4. Mechanistic Links Between Inflammation and Prostatic Remodeling

4.1 Stromal and Epithelial Proliferation

One of the most direct consequences of chronic immune activation in benign prostatic hyperplasia is sustained stimulation of stromal and epithelial cell proliferation. Pro-inflammatory cytokines activate mitogenic and survival signaling pathways within prostatic cells, resulting in increased cell cycle progression, resistance to apoptosis, and expansion of both stromal and epithelial compartments [25]. Stromal fibroblasts are particularly responsive to inflammatory cues and undergo phenotypic differentiation into myofibroblasts [26]. These cells exhibit enhanced

contractile properties and secrete large amounts of extracellular matrix components and growth factors, thereby amplifying local tissue growth [27]. Stromal expansion not only increases prostate volume but also alters the biomechanical environment of the gland. Epithelial cells likewise respond to chronic inflammatory signaling by increasing proliferative activity and displaying altered differentiation patterns [28]. Persistent immune-mediated injury and repair disrupt normal epithelial–stromal communication, leading to dysregulated paracrine signaling that further promotes hyperplasia [29]. This reciprocal interaction between inflammatory cells, stroma, and epithelium establishes a self-sustaining cycle of tissue growth and remodeling.

4.2 Extracellular Matrix Remodeling and Fibrosis

Chronic inflammation also drives pathological extracellular matrix remodeling. Sustained release of transforming growth factor-β and other profibrotic mediators stimulates excessive deposition of collagen, fibronectin, and other matrix proteins [30]. Progressive fibrosis increases tissue stiffness and reduces compliance, altering the structural integrity of the prostate and contributing to luminal narrowing and urethral compression.

4.3 Smooth Muscle Tone and Functional Obstruction

Beyond structural remodeling, inflammatory mediators profoundly influence smooth muscle tone. Cytokines and mast cell–derived factors enhance smooth muscle contractility and impair relaxation, increasing urethral resistance independent of prostate size [31]. This functional component helps explain the weak correlation between prostate volume and symptom severity, highlighting inflammation as a key determinant of lower urinary tract dysfunction in BPH.

5. Interaction with Oxidative Stress, Aging, and Cellular Senescence

Chronic inflammation in benign prostatic hyperplasia is closely linked to oxidative stress and aging-related biological processes, forming a self-perpetuating cycle that drives disease progression [32]. Activated immune cells within the hyperplastic prostate generate reactive oxygen species as part of sustained inflammatory responses. When antioxidant defenses are overwhelmed, excess reactive oxygen species cause oxidative damage to cellular macromolecules, impairing normal epithelial and stromal cell function [33]. Oxidative stress also activates redox-sensitive signaling pathways that enhance transcription of pro-inflammatory cytokines and chemokines, thereby reinforcing immune activation and tissue remodeling. Aging further intensifies this pathogenic loop through immunosenescence and the accumulation of senescent cells in prostatic tissue [34]. Senescent epithelial and stromal cells acquire a pro-inflammatory secretory phenotype, characterized by continuous release of cytokines, growth factors, and matrix-modifying enzymes [35]. These factors promote immune cell recruitment, fibroblast activation, and extracellular matrix remodeling. Consequently, aging actively shapes and sustains the inflammatory microenvironment of the prostate, rather than simply increasing susceptibility to benign prostatic hyperplasia.

6. Therapeutic Implications and Future Directions

Recognition of chronic inflammation as a central driver of benign prostatic hyperplasia expands therapeutic possibilities beyond conventional approaches targeting androgen signaling and smooth muscle tone. Anti-inflammatory agents, cytokine-modulating therapies, and mast cell stabilizers offer potential to directly suppress immune-mediated tissue remodeling and functional obstruction [36]. Strategies aimed at restoring immune balance, including modulation of macrophage activation and reduction of pro-inflammatory T cell responses, may be particularly beneficial in patients with prominent inflammatory profiles [37]. In parallel, interventions targeting oxidative stress and metabolic dysfunction, such as lifestyle modification and antioxidant-supportive therapies, may indirectly reduce inflammatory burden and disease progression [38]. Future research should prioritize the integration of immune profiling into clinical evaluation, allowing stratification of patients based on inflammatory signatures and risk of progression. Longitudinal and interventional studies are needed to determine whether early targeting of inflammation can modify the natural history of BPH, improve symptom control, and reduce reliance on invasive surgical interventions.

CONCLUSION

Immune profiling has fundamentally reshaped our understanding of benign prostatic hyperplasia, revealing chronic inflammation as a central driver of prostate enlargement and functional impairment. Persistent infiltration of immune cells, sustained cytokine signaling, and interactions with oxidative stress and aging converge to promote pathological remodeling of the prostate. Appreciating the immune dimension of BPH not only clarifies disease mechanisms but also highlights novel opportunities for targeted, patient-specific interventions. As immune profiling technologies advance, they are poised to play an increasingly important role in the diagnosis, prognosis, and management of BPH.

REFERENCES

1. Ng M, Leslie SW, Baradhi KM. Benign Prostatic Hyperplasia. [Updated 2024 Oct 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK558920/

2. Uroko Robert Ikechukwu, Fatima Amin Adamude, Egba Simeon Ikechukwu, Chinedu Paulinus Nwuke, Chidinma Lilian Asadu and Peter Anyaorah. 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 2021; 7:e06
3. IR Uroko, F A Adamude, S I Egba, C N Chukwu, C L Asadu, E C Okwara. 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,2020; 66 (3): 24-35
4. Robert I. Uroko., Charles N. Chukwu., Simeon I. Egba., Fatima A. Adamude andJoy C. Ajuzie 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. 2020; 19(4): 395-404
5. 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.
6. 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
7. Hata J, Harigane Y, Matsuoka K, Akaihata H, Yaginuma K, Meguro S, Hoshi S, Sato Y, Ogawa S, Uemura M, et al. Mechanism of Androgen-Independent Stromal Proliferation in Benign Prostatic Hyperplasia. International Journal of Molecular Sciences. 2023; 24(14):11634. https://doi.org/10.3390/ijms241411634
8. Ahmed R, Lozano LE, Anastasio A, Lofek S, Mastelic-Gavillet B, Navarro Rodrigo B, Nguyen S, Dartiguenave F, Rodrigues-Dias SC, Cesson V, Valério M, Roth B, Kandalaft LE, Redchenko I, Hill AVS, Harari A, Romero P, Derré L, Viganó S. Phenotype and Reactivity of Lymphocytes Expanded from Benign Prostate Hyperplasic Tissues and Prostate Cancer. Cancers (Basel). 2023 Jun 8;15(12):3114. doi: 10.3390/cancers15123114. PMID: 37370724; PMCID: PMC10296351.
9. Dang B, Liang L, Li Z, Luo J, Zhong S. Bolstering CD8+ T Cells' Antitumor Immunity: A Promising Strategy to Improve the Response to Advanced Prostate Cancer Treatment. Biology. 2025; 14(5):544. https://doi.org/10.3390/biology14050544
10. Elkahwaji JE. The role of inflammatory mediators in the development of prostatic hyperplasia and prostate cancer. Res Rep Urol. 2012 Dec 31;5:1-10. doi: 10.2147/RRU.S23386. PMID: 24400229; PMCID: PMC3826944.
11. Naiyila X, Li J, Huang Y, Chen B, Zhu M, Li J, Chen Z, Yang L, Ai J, Wei Q, et al. A Novel Insight into the Immune-Related Interaction of Inflammatory Cytokines in Benign Prostatic Hyperplasia. Journal of Clinical Medicine. 2023; 12(5):1821. https://doi.org/10.3390/jcm12051821
12. Jayakrishnan R, Schafer C, Tan SH. Prostate cancer autoantibodies - applications in diagnosis, prognosis, monitoring disease progression and immunotherapy. Am J Clin Exp Urol. 2023 Apr 15;11(2):79-102. PMID: 37168942; PMCID: PMC10165224.
13. Saudi A, Banday V, Zirakzadeh AA, Selinger M, Forsberg J, Holmbom M, Henriksson J, Waldén M, Alamdari F, Aljabery F, Winqvist O, Sherif A. Immune-Activated B Cells Are Dominant in Prostate Cancer. Cancers (Basel). 2023 Feb 1;15(3):920. doi: 10.3390/cancers15030920. PMID: 36765877; PMCID: PMC9913271.
14. Lu C, Liu Y, Ali NM, Zhang B, Cui X. The role of innate immune cells in the tumor microenvironment and research progress in anti-tumor therapy. Front Immunol. 2023 Jan 19;13:1039260. doi: 10.3389/fimmu.2022.1039260. PMID: 36741415; PMCID: PMC9893925.
15. Janeway CA Jr, Travers P, Walport M, et al. Immunobiology: The Immune System in Health and Disease. 5th edition. New York: Garland Science; 2001. Principles of innate and adaptive immunity. Available from: https://www.ncbi.nlm.nih.gov/books/NBK27090/
16. Ibiam U. A., Uti, D. E., Ejeogo, C.C., Orji, O. U. Aja, P. M., Ezeani, N. N., Alum, E. U., 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
17. Dileepan KN, Raveendran VV, Sharma R, Abraham H, Barua R, Singh V, Sharma R, Sharma M. Mast cell-mediated immune regulation in health and disease. Front Med (Lausanne). 2023 Aug 17;10:1213320.

doi: 10.3389/fmed.2023.1213320. PMID: 37663654; PMCID: PMC10470157.
18. Soliman AM, Barreda DR. Acute Inflammation in Tissue Healing. International Journal of Molecular Sciences. 2023; 24(1):641. https://doi.org/10.3390/ijms24010641
19. 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
20. Wang L, Yang JR, Yang LY, Liu ZT. Chronic inflammation in benign prostatic hyperplasia: implications for therapy. Med Hypotheses. 2008;70(5):1021-3. doi: 10.1016/j.mehy.2007.08.022. Epub 2007 Nov 1. PMID: 17935901.
21. Li B, Jones LL, Geiger TL. IL-6 Promotes T Cell Proliferation and Expansion under Inflammatory Conditions in Association with Low-Level RORγt Expression. J Immunol. 2018 Nov 15;201(10):2934-2946. doi: 10.4049/jimmunol.1800016. Epub 2018 Oct 12. PMID: 30315140; PMCID: PMC6324200.
22. Abaurrea A, Araujo AM, Caffarel MM. The Role of the IL-6 Cytokine Family in Epithelial–Mesenchymal Plasticity in Cancer Progression. International Journal of Molecular Sciences. 2021; 22(15):8334. https://doi.org/10.3390/ijms22158334
23. Hirano T. IL-6 in inflammation, autoimmunity and cancer. Int Immunol. 2021 Mar 1;33(3):127-148. doi: 10.1093/intimm/dxaa078. PMID: 33337480; PMCID: PMC7799025.
24. Jarczak D, Nierhaus A. Cytokine Storm—Definition, Causes, and Implications. International Journal of Molecular Sciences. 2022; 23(19):11740. https://doi.org/10.3390/ijms231911740
25. Ibiam, U. A., Uti, D. E., Ejeogo, C. C., Orji, O. U., Aja, P. M., Ezeaani, N. N., **Alum, E. U.,** 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
26. Orchel A, Skrobek J, Kaps A, Paduszyński P, Chodurek E. Current Research on the Control Mechanisms of Cell Survival and Proliferation as Potential Interaction Sites with Pentacyclic Triterpenoids in Ovarian Cancer. International Journal of Molecular Sciences. 2025; 26(23):11622. https://doi.org/10.3390/ijms262311622
27. Grunnet LG, Aikin R, Tonnesen MF, Paraskevas S, Blaabjerg L, Størling J, Rosenberg L, Billestrup N, Maysinger D, Mandrup-Poulsen T. Proinflammatory cytokines activate the intrinsic apoptotic pathway in beta-cells. Diabetes. 2009 Aug;58(8):1807-15. doi: 10.2337/db08-0178. Epub 2009 May 26. PMID: 19470609; PMCID: PMC2712790.
28. Croasdell Lucchini A, Gachanja NN, Rossi AG, Dorward DA, Lucas CD. Epithelial Cells and Inflammation in Pulmonary Wound Repair. Cells. 2021; 10(2):339. https://doi.org/10.3390/cells10020339
29. Peeters PM, Wouters EF, Reynaert NL. Immune Homeostasis in Epithelial Cells: Evidence and Role of Inflammasome Signaling Reviewed. J Immunol Res. 2015;2015:828264. doi: 10.1155/2015/828264. Epub 2015 Aug 19. PMID: 26355424; PMCID: PMC4556877.
30. Frangogiannis N. Transforming growth factor-β in tissue fibrosis. J Exp Med. 2020 Feb 13;217(3):e20190103. doi: 10.1084/jem.20190103. PMID: 32997468; PMCID: PMC7062524.
31. Pattabiraman G, Bell-Cohn AJ, Murphy SF, Mazur DJ, Schaeffer AJ, Thumbikat P. Mast cell function in prostate inflammation, fibrosis, and smooth muscle cell dysfunction. Am J Physiol Renal Physiol. 2021 Oct 1;321(4):F466-F479. doi: 10.1152/ajprenal.00116.2021. Epub 2021 Aug 23. PMID: 34423679; PMCID: PMC8560406.
32. Stewart KL, Lephart ED. Overview of BPH: Symptom Relief with Dietary Polyphenols, Vitamins and Phytochemicals by Nutraceutical Supplements with Implications to the Prostate Microbiome. International Journal of Molecular Sciences. 2023; 24(6):5486. https://doi.org/10.3390/ijms24065486
33. 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.
34. 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
35. Snezhkina AV, Kudryavtseva AV, Kardymon OL, Savvateeva MV, Melnikova NV, Krasnov GS, Dmitriev AA. ROS Generation and Antioxidant Defense Systems in Normal and Malignant Cells. Oxid Med Cell Longev. 2019 Aug 5;2019:6175804. doi: 10.1155/2019/6175804. PMID: 31467634; PMCID:

PMC6701375.
36. Placha D, Jampilek J. Chronic Inflammatory Diseases, Anti-Inflammatory Agents and Their Delivery Nanosystems. Pharmaceutics. 2021 Jan 6;13(1):64. doi: 10.3390/pharmaceutics13010064. PMID: 33419176; PMCID: PMC7825503.
37. Galván-Morales MÁ, Vizuet-de-Rueda JC, Montero-Vargas JM, Teran LM. Role of Mast Cells in Human Health and Disease: Controversies and Novel Therapies. International Journal of Molecular Sciences. 2025; 26(18):8895. https://doi.org/10.3390/ijms26188895
38. Ryan JJ, Morales JK, Falanga YT, Fernando JF, Macey MR. Mast cell regulation of the immune response. World Allergy Organ J. 2009 Oct;2(10):224-32. doi: 10.1097/WOX.0b013e3181c2a95e. Erratum in: World Allergy Organ J. 2010 Jan;3(1):14. PMID: 23283207; PMCID: PMC3650966.

CITE AS: Abaho Areeba Fortunate (2026). Immune Profiling in BPH: Understanding Chronic Inflammation as a Driver of Prostate Enlargement. NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES, 7(2):10-15.
https://doi.org/10.59298/NIJSES/2026/72.1015