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Oxidative Stress-Driven Prostatic Remodeling: Pathogenic Pathways and Therapeutic Targets

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ABSTRACT

Oxidative stress has emerged as a central pathogenic mechanism in prostatic remodeling, particularly in benign prostatic hyperplasia (BPH) and other age-associated prostate disorders. Characterized by an imbalance between reactive oxygen species generation and antioxidant defense capacity, oxidative stress contributes to chronic inflammation, cellular senescence, extracellular matrix remodeling, and dysregulated epithelial–stromal interactions within the prostate. Accumulating experimental and clinical evidence indicates that sustained redox imbalance promotes prostatic enlargement through activation of redox-sensitive signaling pathways, including nuclear factor kappa B, mitogen-activated protein kinases, and transforming growth factor beta signaling. These pathways drive fibroblast-to-myofibroblast differentiation, smooth muscle hypercontractility, and excessive matrix deposition, collectively leading to structural and functional alterations of prostatic tissue. Moreover, oxidative stress intersects with metabolic dysfunction, hormonal imbalance, and immune dysregulation, amplifying disease progression and symptom severity. This review synthesizes current knowledge on the sources of oxidative stress in the prostate, delineates the molecular mechanisms linking redox imbalance to tissue remodeling, and evaluates emerging therapeutic strategies targeting oxidative and inflammatory pathways. Understanding oxidative stress–driven prostatic remodeling offers opportunities for novel interventions aimed at slowing disease progression and improving clinical outcomes.

Keywords: oxidative stress, prostatic remodeling, benign prostatic hyperplasia, inflammation, redox signaling

INTRODUCTION

Prostatic remodeling is a dynamic process involving coordinated changes in epithelial proliferation, stromal expansion, smooth muscle tone, and extracellular matrix organization[1]. While physiological remodeling is essential for tissue maintenance, pathological remodeling underlies conditions such as benign prostatic hyperplasia (BPH), which affects a majority of aging men[2]. Traditionally, androgenic stimulation and aging have been viewed as the primary drivers of BPH; however, increasing evidence implicates oxidative stress as a critical contributor to prostatic structural and functional alterations[3]. Oxidative stress refers to a sustained imbalance between reactive oxygen species (ROS) production and antioxidant defense mechanisms. In the prostate, ROS are generated from multiple sources, including mitochondrial respiration, inflammatory cell infiltration, metabolic dysregulation, and enzymatic systems such as NADPH oxidases[4]. When inadequately neutralized, ROS induce cellular damage, activate redox-sensitive signaling pathways, and promote chronic inflammation. These events collectively create a microenvironment conducive to pathological tissue remodeling[5]. This review examines the role of oxidative stress in driving prostatic remodeling, focusing on molecular pathways that link redox imbalance

to cellular proliferation, fibrosis, and smooth muscle dysfunction. Additionally, potential therapeutic strategies targeting oxidative stress and its downstream effects are discussed.

Sources of Oxidative Stress in the Prostate

Mitochondrial Dysfunction

Mitochondria represent a primary endogenous source of reactive oxygen species through electron leakage from the electron transport chain during oxidative phosphorylation[6]. Under physiological conditions, mitochondrial antioxidant systems tightly regulate ROS production. However, aging, androgen imbalance, and metabolic disturbances such as insulin resistance impair mitochondrial efficiency, leading to excessive superoxide generation[7]. In prostatic epithelial and stromal cells, mitochondrial dysfunction is associated with reduced ATP production, altered metabolic reprogramming, and accumulation of oxidative damage[8]. These changes activate stress-responsive signaling pathways, including mitogen-activated protein kinases and redox-sensitive transcription factors, which favor cellular proliferation and resistance to apoptosis[9]. Progressive mitochondrial DNA damage further amplifies ROS production, establishing a self-perpetuating cycle that promotes hyperplastic growth and tissue remodeling within the prostate.

Inflammation-Associated ROS Production

Chronic inflammation is a defining feature of many prostatic disorders and constitutes a major extrinsic source of oxidative stress[10]. Infiltrating immune cells such as macrophages, neutrophils, and lymphocytes, generate large amounts of ROS as part of their antimicrobial defense mechanisms[11]. In the prostate, persistent inflammatory infiltrates maintain sustained ROS production even in the absence of overt infection[12]. This prolonged oxidative burden damages surrounding epithelial and stromal cells, stimulates cytokine and chemokine release, and perpetuates immune cell recruitment[13]. The resulting inflammatory–oxidative microenvironment reinforces tissue injury and drives aberrant wound-healing responses that favor stromal expansion and fibrosis.

Enzymatic Sources of ROS

Several enzyme systems contribute directly to ROS generation in prostatic tissue. NADPH oxidases are particularly important, as they produce superoxide in a regulated but inducible manner[14]. Upregulation of specific NADPH oxidase isoforms has been documented in hyperplastic prostate tissue, linking these enzymes to redox-driven proliferation and fibrotic remodeling[15]. Additional contributors include xanthine oxidase and uncoupled nitric oxide synthase, both of which enhance oxidative stress under pathological conditions[16].

Oxidative Stress and Cellular Damage

Oxidative stress exerts its pathological effects largely through damage to lipids, proteins, and nucleic acids[17]. ROS readily initiate lipid peroxidation by attacking polyunsaturated fatty acids within cellular membranes, generating reactive aldehydes such as malondialdehyde and 4-hydroxynonenal[18]. These secondary products further propagate oxidative injury and modulate signaling pathways that regulate cell growth, inflammation, and apoptosis[19]. Elevated lipid peroxidation markers have been consistently detected in hyperplastic prostatic tissues and correlate with disease severity.

In parallel, oxidative stress induces DNA strand breaks, base modifications, and protein oxidation, leading to genomic instability and altered cellular function. In prostatic cells, oxidative DNA damage often activates repair and survival pathways rather than apoptosis, paradoxically promoting cellular persistence and proliferation[20]. This imbalance between damage and appropriate cell elimination contributes to progressive hyperplasia and pathological prostatic remodeling.

Redox-Sensitive Signaling Pathways in Prostatic Remodeling

Nuclear Factor Kappa B Signaling

Nuclear factor kappa B is a key transcription factor activated by oxidative stress. ROS-mediated activation of this pathway promotes expression of pro-inflammatory cytokines, chemokines, and growth factors within the prostate[21]. Sustained nuclear factor kappa B signaling fosters chronic inflammation, enhances stromal cell proliferation, and inhibits apoptotic pathways, thereby driving tissue remodeling.

Mitogen-Activated Protein Kinase Pathways

Mitogen-activatare activated by oxidative stimulied protein kinase cascades, including extracellular signal-regulated kinase, c-Jun N-terminal kinase, and p38 pathways[22]. These pathways regulate cell proliferation, differentiation, and stress responses. In prostatic stromal and epithelial cells, oxidative stress–induced mitogen-activated protein kinase activation promotes hyperplastic growth and resistance to apoptotic signals.

Transforming Growth Factor Beta Signaling

Transforming growth factor beta plays a dual role in tissue homeostasis and fibrosis. Oxidative stress enhances transforming growth factor beta signaling, leading to fibroblast activation and myofibroblast differentiation[23]. These cells produce excessive extracellular matrix components, resulting in stromal fibrosis and increased tissue stiffness characteristic of prostatic remodeling.

Stromal–Epithelial Interactions and Fibrosis

Prostatic remodeling is driven not only by epithelial proliferation but also by stromal expansion and fibrosis[24]. Oxidative stress disrupts stromal–epithelial crosstalk by altering paracrine signaling mechanisms. Stromal cells exposed to oxidative stress secrete growth factors and cytokines that stimulate epithelial proliferation, while epithelial cells contribute to stromal activation through redox-sensitive mediators[25]. Fibrosis, characterized by excessive collagen deposition and altered matrix composition, increases prostatic volume and impairs tissue compliance[26]. Oxidative stress promotes fibrotic remodeling by activating profibrotic signaling pathways and suppressing matrix degradation enzymes, leading to progressive structural alteration of the prostate.

Oxidative Stress, Smooth Muscle Tone, and Functional Remodeling

In addition to driving structural alterations, oxidative stress plays a critical role in the functional remodeling of the prostate, particularly through its effects on smooth muscle tone[27]. Reactive oxygen species influence smooth muscle contractility by disrupting intracellular calcium homeostasis and impairing nitric oxide–mediated relaxation pathways[28]. Under physiological conditions, nitric oxide promotes smooth muscle relaxation by activating cyclic guanosine monophosphate signaling. However, elevated oxidative stress reduces nitric oxide bioavailability through direct scavenging and by promoting endothelial nitric oxide synthase uncoupling, resulting in diminished relaxant capacity[29]. Concurrently, oxidative stress enhances calcium sensitization and activates pro-contractile signaling pathways, leading to increased smooth muscle tone. This heightened contractility contributes directly to bladder outlet obstruction and exacerbation of lower urinary tract symptoms, even in the absence of marked prostate enlargement. Consequently, oxidative stress serves as a mechanistic bridge linking structural remodeling with functional obstruction in prostatic disease.

Interaction with Aging and Metabolic Dysfunction

Aging is accompanied by a progressive decline in endogenous antioxidant defenses and increased production of reactive oxygen species, rendering the prostate particularly vulnerable to oxidative injury[30]. Mitochondrial inefficiency, cellular senescence, and chronic low-grade inflammation further amplify redox imbalance with advancing age[31]. Metabolic disorders such as obesity, insulin resistance, and diabetes intensify oxidative stress through persistent hyperglycemia, abnormal lipid metabolism, and systemic inflammation[32]. These metabolic disturbances promote excessive ROS generation and impair antioxidant systems, accelerating both structural and functional prostatic remodeling. The convergence of aging and metabolic dysfunction creates a sustained pro-oxidant environment that drives disease progression and worsens symptom severity, underscoring oxidative stress as a unifying pathogenic mechanism.

Therapeutic Targets in Oxidative Stress–Driven Prostatic Remodeling

Targeting oxidative stress offers promising opportunities for therapeutic intervention in prostatic remodeling[33]. Antioxidant-based strategies, including dietary polyphenols, vitamins, and agents that enhance endogenous antioxidant systems, have shown potential to reduce ROS burden and suppress redox-sensitive signaling. However, inconsistent clinical outcomes highlight the importance of appropriate patient selection and dosing strategies. Anti-inflammatory interventions represent an additional approach, as reducing inflammatory cytokine production can indirectly attenuate oxidative damage and fibrotic progression[34]. More recently, attention has focused on selectively targeting redox-sensitive signaling pathways, such as nuclear factor kappa B and transforming growth factor beta pathways, which regulate inflammation, fibrosis, and cellular proliferation[35]. Modulating these pathways may provide more precise control of pathological remodeling while preserving essential physiological functions.

Enhancing Endogenous Antioxidant Defenses

Activation of endogenous antioxidant systems, such as those regulated by nuclear factor erythroid 2–related factor 2, represents a novel approach to restoring redox balance[36]. Enhancing intrinsic defense mechanisms may provide sustained protection against oxidative stress–induced tissue remodeling.

Future Perspectives

Advancing understanding of oxidative stress–driven prostatic remodeling requires integrative research approaches combining molecular biology, clinical studies, and translational models[37]. Identification of reliable oxidative stress biomarkers may enable early detection of disease progression and therapeutic response. Furthermore, personalized strategies targeting redox imbalance in high-risk populations may improve long-term management of prostatic disorders.

CONCLUSION

Oxidative stress is a central driver of pathological prostatic remodeling, linking aging, inflammation, and metabolic dysfunction to structural and functional changes in prostatic tissue. Through activation of redox-sensitive signaling pathways, oxidative stress promotes epithelial proliferation, stromal fibrosis, and smooth muscle dysfunction, contributing to disease progression and symptom burden. Targeting oxidative stress and its downstream effects offers a promising framework for novel therapeutic interventions. Continued exploration of redox biology in the prostate is essential for developing effective, mechanism-based strategies to prevent and treat prostatic remodeling disorders.

REFERENCES

1. Di Carlo E, Sorrentino C. The multifaceted role of the stroma in the healthy prostate and prostate cancer. J Transl Med. 2024 Sep 5;22(1):825. doi: 10.1186/s12967-024-05564-2. PMID: 39238004; PMCID: PMC11378418.
2. Vecchiotti D, Clementi L, Cornacchia E, Di Vito Nolfi M, Verzella D, Capece D, Zazzeroni F, Angelucci A. Evidence of the Link between Stroma Remodeling and Prostate Cancer Prognosis. Cancers. 2024; 16(18):3215. https://doi.org/10.3390/cancers16183215
3. 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
4. Levesque C, Nelson PS. Cellular Constituents of the Prostate Stroma: Key Contributors to Prostate Cancer Progression and Therapy Resistance. Cold Spring Harb Perspect Med. 2018 Aug 1;8(8):a030510. doi: 10.1101/cshperspect.a030510. PMID: 28490538; PMCID: PMC5681439.
5. Schauer IG, Rowley DR. The functional role of reactive stroma in benign prostatic hyperplasia. Differentiation. 2011 Nov-Dec;82(4-5):200-10. doi: 10.1016/j.diff.2011.05.007. Epub 2011 Jun 12. PMID: 21664759; PMCID: PMC3179838.
6. Kausar S, Wang F, Cui H. The Role of Mitochondria in Reactive Oxygen Species Generation and Its Implications for Neurodegenerative Diseases. Cells. 2018 Dec 17;7(12):274. doi: 10.3390/cells7120274. PMID: 30563029; PMCID: PMC6316843.
7. Bolisetty S, Jaimes EA. Mitochondria and Reactive Oxygen Species: Physiology and Pathophysiology. International Journal of Molecular Sciences. 2013; 14(3):6306-6344. https://doi.org/10.3390/ijms14036306
8. 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
9. 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.
10. Kaltsas A, Giannakas T, Stavropoulos M, Kratiras Z, Chrisofos M. Oxidative Stress in Benign Prostatic Hyperplasia: Mechanisms, Clinical Relevance and Therapeutic Perspectives. Diseases. 2025 Feb 11;13(2):53. doi: 10.3390/diseases13020053. PMID: 39997060; PMCID: PMC11854834.
11. 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
12. 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.
13. Paulis G. Inflammatory mechanisms and oxidative stress in prostatitis: the possible role of antioxidant therapy. Res Rep Urol. 2018 Sep 17;10:75-87. doi: 10.2147/RRU.S170400. PMID: 30271757; PMCID: PMC6149977.
14. Cipriano A, Viviano M, Feoli A, Milite C, Sarno G, Castellano S, Sbardella G. NADPH Oxidases: From Molecular Mechanisms to Current Inhibitors. J Med Chem. 2023 Sep 14;66(17):11632-11655. doi: 10.1021/acs.jmedchem.3c00770. Epub 2023 Aug 31. PMID: 37650225; PMCID: PMC10510401.
15. Vilchis-Landeros MM, Matuz-Mares D, Vázquez-Meza H. Regulation of Metabolic Processes by Hydrogen Peroxide Generated by NADPH Oxidases. Processes. 2020; 8(11):1424. https://doi.org/10.3390/pr8111424
16. Prieto-Bermejo R, Hernández-Hernández A. The Importance of NADPH Oxidases and Redox Signaling in Angiogenesis. Antioxidants (Basel). 2017 May 13;6(2):32. doi: 10.3390/antiox6020032. PMID: 28505091; PMCID: PMC5488012.
17. Pizzino G, Irrera N, Cucinotta M, Pallio G, Mannino F, Arcoraci V, Squadrito F, Altavilla D, Bitto A. Oxidative Stress: Harms and Benefits for Human Health. Oxid Med Cell Longev. 2017;2017:8416763. doi: 10.1155/2017/8416763. Epub 2017 Jul 27. PMID: 28819546; PMCID: PMC5551541.
18. 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
19. García-Sánchez A, Miranda-Díaz AG, Cardona-Muñoz EG. The Role of Oxidative Stress in

Physiopathology and Pharmacological Treatment with Pro- and Antioxidant Properties in Chronic Diseases. Oxid Med Cell Longev. 2020 Jul 23;2020:2082145. doi: 10.1155/2020/2082145. PMID: 32774665; PMCID: PMC7396016.

20. Ibiam U. A., Uti, D. E., 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.

21. Song Y, Hou Z, Zhu L, Chen Y, Li J. Oxidative stress as a catalyst in prostate cancer progression: unraveling molecular mechanisms and exploring therapeutic interventions. Discov Oncol. 2025 Apr 3;16(1):457. doi: 10.1007/s12672-025-02245-4. PMID: 40178629; PMCID: PMC11968600.
22. Kostenko S, Dumitriu G, Lægreid KJ, Moens U. Physiological roles of mitogen-activated-protein-kinase-activated p38-regulated/activated protein kinase. World J Biol Chem. 2011 May 26;2(5):73-89. doi: 10.4331/wjbc.v2.i5.73. PMID: 21666810; PMCID: PMC3110898.
23. Toullec A, Gerald D, Despouy G, Bourachot B, Cardon M, Lefort S, Richardson M, Rigaill G, Parrini MC, Lucchesi C, Bellanger D, Stern MH, Dubois T, Sastre-Garau X, Delattre O, Vincent-Salomon A, Mechta-Grigoriou F. Oxidative stress promotes myofibroblast differentiation and tumour spreading. EMBO Mol Med. 2010 Jun;2(6):211-30. doi: 10.1002/emmm.201000073. PMID: 20535745; PMCID: PMC3377319.
24. Xiang J, Zheng Y, Chen D, Zeng Y, Zhang J, Chang D, Chang C. The pathogenesis of benign prostatic hyperplasia and the roles of Prdx3, oxidative stress, pyroptosis and autophagy:a review. Front Oncol. 2025 Aug 5;15:1579539. doi: 10.3389/fonc.2025.1579539. PMID: 40837016; PMCID: PMC12361133.
25. Acheva A, Haghdoost S, Sollazzo A, Launonen V, Kämäräinen M. Presence of Stromal Cells Enhances Epithelial-to-Mesenchymal Transition (EMT) Induction in Lung Bronchial Epithelium after Protracted Exposure to Oxidative Stress of Gamma Radiation. Oxid Med Cell Longev. 2019 Sep 8;2019:4120379. doi: 10.1155/2019/4120379. PMID: 31583039; PMCID: PMC6754954.
26. 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
27. 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
28. Shukla S, Srivastava JK, Shankar E, Kanwal R, Nawab A, Sharma H, Bhaskaran N, Ponsky LE, Fu P, MacLennan GT, et al. Oxidative Stress and Antioxidant Status in High-Risk Prostate Cancer Subjects. Diagnostics. 2020; 10(3):126. https://doi.org/10.3390/diagnostics10030126
29. Biesiadecki M, Mołoń M, Balawender K, Kobylińska Z, Galiniak S. Shedding light on the shadows: oxidative stress and its pivotal role in prostate cancer progression. Front Oncol. 2024 May 7;14:1393078. doi: 10.3389/fonc.2024.1393078. Erratum in: Front Oncol. 2024 Aug 20;14:1468582. doi: 10.3389/fonc.2024.1468582. PMID: 38774418; PMCID: PMC11106406.
30. Maldonado E, Morales-Pison S, Urbina F, Solari A. Aging Hallmarks and the Role of Oxidative Stress. Antioxidants (Basel). 2023 Mar 6;12(3):651. doi: 10.3390/antiox12030651. PMID: 36978899; PMCID: PMC10044767.
31. Ogbodo John Onyebuchi, Chinazom Precious Agbo, Ugoci Olivia Njoku, Martins Obinna Ogugofor, Egba Simeon Ikechukwu, Stella Amarachi Ihim, Adaeze Chidiebere Echezona Kenneth Chibuike Brendan, Aman Babanrao Upaganlawar, and ChandrashekarDevidas Upasani (2021) Alzheimer's Disease: Pathogenesis and Therapeutic Interventions, Current Aging Science, 21:1-25.
32. Izah, S.C., Betiang, P.A., Ainebyoona, C., Uti, D.E., Echegu, D.A. The Ketogenic Diet in Obesity Management: Friend or Foe?. *Cell Biochem Biophys* (2025). https://doi.org/10.1007/s12013-025-01878-0
33. Manful CF, Fordjour E, Ikumoinein E, Abbey L, Thomas R. Therapeutic Strategies Targeting Oxidative Stress and Inflammation: A Narrative Review. BioChem. 2025; 5(4):35. https://doi.org/10.3390/biochem5040035
34. Steen EH, Wang X, Balaji S, Butte MJ, Bollyky PL, Keswani SG. The Role of the Anti-Inflammatory Cytokine Interleukin-10 in Tissue Fibrosis. Adv Wound Care (New Rochelle). 2020 Apr 1;9(4):184-198. doi: 10.1089/wound.2019.1032. Epub 2020 Feb 7. PMID: 32117582; PMCID: PMC7047112.
35. Bezerra FS, Lanzetti M, Nesi RT, Nagato AC, Silva CPE, Kennedy-Feitosa E, Melo AC, Cattani-Cavalieri I, Porto LC, Valenca SS. Oxidative Stress and Inflammation in Acute and Chronic Lung Injuries. Antioxidants (Basel). 2023 Feb 21;12(3):548. doi: 10.3390/antiox12030548. PMID: 36978796; PMCID:

PMC10045332.
36. Obasi, D.C., Abba, J.N., Aniokete, U.C., Okoroh, P.N. Endogenous Plant signals and human Health: Molecular mechanisms, ecological functions, and therapeutic Prospects. Biochemistry and Biophysics Reports, 43(2025):102114. https://doi.org/10.1016/j.bbrep.2025.102114
37. Aja PM, Agu PC, Musyoka AM, Ngwueche W, Odo JU, Alum EU, Fasogbon IV, Herbert M, Daniel Eze E. Integrative Approaches to Prostate Disease Management: Nutrition, Exercise, and Lifestyle Modifications. Am J Mens Health. 2025 May-Jun;19(3):15579883251344571. doi: 10.1177/15579883251344571. Epub 2025 Jun 23. PMID: 40548677; PMCID: PMC12185962.

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