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Inflammation and Oxidative Stress in Benign Prostate Hyperplasia: Current Evidence and Emerging Concepts

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ABSTRACT

Benign prostatic hyperplasia (BPH) is a highly prevalent condition in aging men and a major contributor to lower urinary tract symptoms (LUTS). Increasing evidence implicates chronic inflammation and oxidative stress as central drivers of prostate tissue remodeling, stromal–epithelial interactions, and symptomatic progression. This review synthesizes current understandings of the inflammatory and redox biology underpinning BPH, examines key cellular and molecular mechanisms, evaluates candidate biomarkers, and explores therapeutic implications. We highlight how innate and adaptive immune activation, senescence-associated secretory phenotypes (SASP), and reactive oxygen species (ROS)-mediated signaling converge to promote proliferative and fibrotic pathways in the prostate. Novel and emerging areas-including the role of the prostate microbiome, mitochondrial dysfunction, impaired antioxidant responses (e.g., Nrf2 signaling), and immunometabolic reprogramming-offer fresh mechanistic insight and potential therapeutic targets. Finally, we identify gaps in translational evidence and propose priorities for future research, emphasizing the need for longitudinal, mechanism-focused clinical studies and biomarker-guided interventional trials.

Keywords: Benign prostatic hyperplasia, inflammation, oxidative stress, senescence, Nrf2

INTRODUCTION

Benign prostatic hyperplasia (BPH) affects a large proportion of men over the age of 50 and remains one of the most significant contributors to lower urinary tract symptoms (LUTS), urinary obstruction, and age-related morbidity. Its clinical impact extends beyond urinary difficulties, influencing sleep quality, mental health, and overall well-being [1-6]. Traditionally, BPH was viewed primarily as the consequence of age-related hormonal changes-particularly the actions of androgens and dihydrotestosterone-leading to gradual proliferation of both stromal and epithelial elements of the transition zone of the prostate [7-11]. While hormonal regulation remains central, contemporary research has shifted the paradigm toward a more complex model in which chronic inflammation, oxidative stress, and immune–stromal interactions play equally crucial roles [12-18]. These processes shape the prostate's microenvironment, driving hyperplasia, fibrosis, and tissue remodeling. Chronic inflammation generates a persistent milieu of cytokines, chemokines, and immune cells that alter cellular behavior and promote proliferative and fibrotic responses [19-25]. Simultaneously, increased production of reactive oxygen species (ROS) and impaired antioxidant defenses create oxidative stress, which further amplifies inflammatory signaling, damages cellular components, and contributes to genomic instability [26-30]. Together, these interconnected pathways form a self-perpetuating cycle capable of accelerating disease progression [31-38]. Understanding how inflammatory signals and redox imbalance shape prostate biology is essential for developing disease-modifying therapies that extend beyond symptomatic relief and address the underlying drivers of hyperplasia.

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Epidemiologic and clinical associations

Epidemiologic research strongly supports the relevance of inflammation and oxidative stress in the natural history of BPH [39-42]. Histologic studies reveal that a significant proportion-often more than 70%-of prostate specimens from men with BPH show inflammatory infiltrates, including lymphocytes, macrophages, and plasma cells [43-48]. The degree of inflammation frequently correlates with prostate enlargement, International Prostate Symptom Score (IPSS) severity, and risk of acute urinary retention. Molecular analyses further demonstrate upregulation of inflammatory genes and oxidative stress markers in hyperplastic tissue [49-53]. Beyond histology, clinical observations reinforce the link between systemic inflammatory burden and prostate disease. Men with metabolic comorbidities such as obesity, insulin resistance, type 2 diabetes, and metabolic syndrome exhibit a disproportionately higher risk of developing BPH and experiencing more rapid symptom progression [54-58]. These conditions are characterized by chronic low-grade inflammation, increased circulating cytokines, elevated ROS generation, and impaired antioxidant capacity [59-60]. Adipose tissue dysfunction, endotoxemia, and metabolic dysregulation may contribute to a systemic pro-inflammatory state with downstream effects on the prostate [61-68]. Although causality is not fully established, these robust associations provide a compelling epidemiologic rationale to further explore inflammatory and oxidative pathways as potential therapeutic targets for BPH prevention and management.

Cellular players and inflammatory pathways

Innate immunity and macrophages

Macrophages are among the most abundant immune cells infiltrating hyperplastic prostate tissue, and they play a pivotal role in orchestrating inflammation-driven prostate remodeling [13]. Activated macrophages secrete a diverse array of cytokines, including IL-1β, TNF-α, and IL-6, which stimulate stromal cell proliferation, enhance angiogenesis, and modulate the extracellular matrix (ECM). These mediators activate fibroblasts and promote the transition of stromal cells into myofibroblasts, contributing to increased tissue stiffness and fibrosis-key contributors to urinary obstruction[14]. Macrophage polarization across the M1/M2 continuum adds further complexity. M1-like macrophages produce pro-inflammatory cytokines and ROS, perpetuating tissue injury and immune activation. M2-like macrophages, while associated with tissue repair, can paradoxically promote fibrosis and stromal proliferation[15]. In BPH, macrophages often exhibit hybrid or mixed polarization states, simultaneously driving inflammation and wound-healing programs that facilitate hyperplastic growth[16]. Pattern-recognition receptor activation-including Toll-like receptors (TLRs) and NOD-like receptors (NLRs)-is a major initiating factor for macrophage activation within the prostate. Endogenous danger-associated molecular patterns (DAMPs), released from stressed or senescent prostate cells, and exogenous microbial products can trigger these receptors[17]. Such activation leads to inflammasome assembly, enhanced cytokine secretion, and ROS production. Repeated or unresolved activation of these pathways contributes to a chronic inflammatory cycle that sustains macrophage recruitment and activity, thereby reinforcing the pathophysiologic environment that drives BPH progression.

Adaptive immunity and T cells

T-cell infiltration represents a consistent and biologically significant feature of hyperplastic prostate tissue [18]. Among these populations, $CD4^+$ T cells are particularly prominent, and several studies suggest that their phenotype and activation status shape the trajectory of inflammatory and proliferative responses within the gland. Th1 cells release IFN-γ and TNF-α, sustaining chronic inflammatory signaling and enhancing macrophage recruitment and activation [19]. Th17 cells, characterized by secretion of IL-17 and IL-22, play an especially important role in chronic tissue irritation by stimulating stromal cells to produce IL-6, chemokines, and additional pro-inflammatory mediators that propagate leukocyte influx[20]. Increased Th17 activity has been associated with more extensive stromal proliferation and fibrotic remodeling in BPH. Conversely, regulatory T cells (Tregs), which normally restrain excessive immune activation, are often reduced or functionally impaired in BPH tissue[21]. This imbalance between pro-inflammatory Th17 cells and immunosuppressive Tregs creates a microenvironment skewed toward persistent inflammation and tissue remodeling. T-cell–derived cytokines also have direct effects on resident prostate cells[22]. IL-4, IL-17, and IL-22 can stimulate stromal fibroblasts to proliferate and differentiate, while IL-2 and IL-15 may support epithelial cell survival, making them less responsive to apoptotic cues. This cross-communication ultimately reinforces the hyperplastic phenotype by sustaining an environment in which both stromal and epithelial elements receive continuous proliferative and survival signals.

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Stromal-epithelial crosstalk

Stromal-epithelial interaction is a central, dynamic driver of BPH progression [23]. Under inflammatory stimulation, stromal fibroblasts and myofibroblasts release a broad array of mediators-including fibroblast growth factors (FGFs), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), and various chemokines-that shape epithelial behavior and promote glandular expansion [24]. Growth factors enhance epithelial cell proliferation and induce structural alterations in glandular architecture, while TGF-β fosters differentiation of fibroblasts into contractile myofibroblasts[25]. Myofibroblasts contribute substantially to extracellular matrix (ECM) deposition through overproduction of collagen, fibronectin, and proteoglycans, coupled with altered expression of matrix metalloproteinases (MMPs) and their inhibitors (TIMPs)[26]. Inflammatory signals also stimulate stromal cells to generate reactive oxygen species (ROS), amplifying redox stress within the tissue microenvironment[27]. The increased ECM deposition and altered stromal composition lead to tissue stiffness and fibrosis-processes that aggravate urinary obstruction even when prostate size is only moderately increased[28]. As this paracrine loop persists, stromal cells remain activated, epithelial cells continue to proliferate, and mechanical distortion of the urethra worsens lower urinary tract symptoms.

Oxidative stress: sources and molecular consequences

ROS generation and mitochondrial dysfunction

Oxidative stress arises when ROS production exceeds the capacity of cellular antioxidant defenses [29]. In BPH, ROS originate from multiple sources: dysfunctional mitochondria in aging prostate cells, NADPH oxidase activity in both stromal and immune cells, and respiratory bursts from infiltrating leukocytes. Age-related mitochondrial decline, often compounded by metabolic comorbidities such as diabetes and obesity, leads to inefficient oxidative phosphorylation and electron leakage, intensifying ROS output [30]. These reactive species damage DNA, lipids, and proteins, disrupting cellular homeostasis and activating redox-sensitive signaling pathways such as MAPKs, JNK, p38, and NF-κB-key regulators of inflammation, proliferation, and stress responses.

Redox-sensitive transcription factors and antioxidant defenses

ROS act as potent second messengers that activate transcription factors including NF-κB and AP-1, which drive the expression of cytokines, chemokines, and adhesion molecules involved in immune cell recruitment and inflammatory persistence [31]. In contrast, the Nrf2 pathway normally provides a counterbalancing mechanism by inducing antioxidant enzymes such as heme oxygenase-1, glutathione peroxidase, and superoxide dismutase. In BPH, impaired Nrf2 activation or downregulation of its downstream effectors reduces the cell's ability to neutralize ROS, perpetuating oxidative injury[32]. The resulting imbalance between pro-oxidant and antioxidant forces creates a biochemical environment that favors hyperplasia, fibrosis, and chronic inflammation.

Oxidative DNA damage and genomic instability

Persistent oxidative stress inflicts cumulative damage on nuclear and mitochondrial DNA, producing lesions such as 8-oxo-deoxyguanosine and double-strand breaks. These injuries activate DNA damage response pathways that can push cells toward senescence when repair capacity is exhausted [33]. Senescent prostate epithelial and stromal cells develop a senescence-associated secretory phenotype (SASP), releasing IL-6, IL-8, MMPs, and other mediators that reinforce inflammation and matrix remodeling [34]. Alternatively, sublethal DNA damage may promote genomic instability and dysregulated proliferation, further contributing to the hyperplastic process.

The inflammation–oxidative stress nexus

Inflammation and oxidative stress are tightly intertwined in BPH pathogenesis, forming a self-reinforcing loop that drives chronic tissue remodeling. Inflammatory cells such as macrophages and T cells generate ROS during activation; these reactive molecules activate NF-κB and other transcriptional programs that further enhance cytokine production and immune recruitment. ROS also synergize with TGF-β signaling to promote fibrosis and myofibroblast differentiation, thereby exacerbating structural changes in the prostate[35]. Redox-regulated checkpoints influence whether prostate cells undergo apoptosis, senescence, or proliferation, determining the balance between tissue expansion and regression. Collectively, these mechanisms explain how chronic inflammation and oxidative stress jointly fuel the proliferation, ECM remodeling, angiogenesis, and fibrosis characteristic of BPH progression[36].

Therapeutic implications

Anti-inflammatory strategies

Nonsteroidal anti-inflammatory drugs (NSAIDs), selective COX-2 inhibitors, and other anti-inflammatory approaches have shown mixed effects on BPH progression in observational studies. Targeted modulation of cytokines or immune cell function remains exploratory but conceptually attractive [37]. Importantly, broad

immunosuppression is not desirable in this typically benign disorder; precision approaches that dampen pathogenic inflammation without compromising host defense are needed.

Antioxidant approaches and redox modulators

Dietary antioxidants and nutraceuticals (e.g., lycopene, green tea polyphenols, vitamin E) have been studied with variable results. Pharmacologic activation of endogenous antioxidant defenses-most notably via Nrf2 agonism-or inhibition of ROS-producing enzymes (NADPH oxidase inhibitors) represent mechanistic strategies currently under investigation [38]. Restoring mitochondrial health through metabolic or mitophagy-enhancing interventions could reduce ROS at the source.

CONCLUSION

Inflammation and oxidative stress are central, interacting contributors to the pathobiology of BPH. Through a complex network involving immune cells, senescent phenotypes, redox-sensitive signaling, and stromal-epithelial cross-talk, these processes drive proliferation, fibrosis, and symptomatic progression. Emerging areas-microbiome influences, immunometabolic regulation, Nrf2 and mitochondrial therapeutics, and senescence-targeted approaches-offer promising avenues for disease-modifying interventions. Realizing this potential requires targeted translational research, validated biomarkers, and carefully designed clinical trials to move from descriptive associations to precision therapeutics that slow or reverse BPH progression.

REFERENCES

1. Obeagu, E.I., Alum, E.U., 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.https://www.eejournals.org/public/uploads/1687980288_52785ca83cc0c789d8ae.pdf
2. Kalata U, Jarkiewicz M, Pomian A, Zwierzchowska AJ, Horosz E, Majkusiak W, Rutkowska B, Barcz EM. The Influence of Successful Treatment of Stress Urinary Incontinence and Pelvic Organ Prolapse on Depression, Anxiety, and Insomnia—A Prospective Intervention Impact Assessment Study. Journal of Clinical Medicine. 2024; 13(6):1528. https://doi.org/10.3390/jcm13061528
3. Famurewa, A. C., Orji, O. U., Aja, P. M., Nwite, F., Ohuche, S. E., Ukasoanya, S. C., Nnaji, L. O., Joshua, D., Igwe, K. U. and Chima, S. F. Nephroprotective effects of *Datura stramonium* leaves against methotrexate nephrotoxicity via attenuation of oxidative stress-mediated inflammation and apoptosis in rats. *Avicenna Journal of Phytomedicine.* 2023; 13(4): 377-387. doi: 10.22038/ ajp.2023.21903.
4. 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
5. 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
6. Liu H, Wang S, Wang J, Guo X, Song Y, Fu K, Gao Z, Liu D, He W, Yang LL. Energy metabolism in health and diseases. Signal Transduct Target Ther. 2025 Feb 18;10(1):69. doi: 10.1038/s41392-025-02141-x. PMID: 39966374; PMCID: PMC11836267.
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. Boarescu P-M, Boarescu I, Pop RM, Roşian ŞH, Bocşan IC, Rus V, Mada RO, Popa ID, Neagu N, Bulboacă AE, et al. Evaluation of Oxidative Stress Biomarkers, Pro-Inflammatory Cytokines, and Histological Changes in Experimental Hypertension, Dyslipidemia, and Type 1 Diabetes Mellitus. International Journal of Molecular Sciences. 2022; 23(3):1438. https://doi.org/10.3390/ijms23031438
10. Ogbodo John Onyebuchi, Chinazom Precious Agbo, Ugoci Olivia Njoku, Martins Obinna Ogugofor, Egba Simeon Ikechukwu, Stella Amarachi Ihim, Adaeze Chidiebere Echezona Kenneth Chibuike Brendan,

OPEN ACCESS
ONLINE ISSN: 2992-5479
PRINT ISSN: 2992-605X

Aman Babanrao Upaganlawar, and ChandrashekarDevidas Upasani. Alzheimer's Disease: Pathogenesis and Therapeutic Interventions, Current Aging Science, 2021; 21:1-25.

11. Yu W, Tu Y, Long Z, Liu J, Kong D, Peng J, Wu H, Zheng G, Zhao J, Chen Y, Liu R, Li W, Hai C. Reactive Oxygen Species Bridge the Gap between Chronic Inflammation and Tumor Development. Oxid Med Cell Longev. 2022 Jun 28;2022:2606928. doi: 10.1155/2022/2606928. PMID: 35799889; PMCID: PMC9256443.
12. Obeagu, E. I., Obeagu, G. U., **Alum, E. U.** and Ugwu, O. P. C. Persistent Immune Activation and Chronic Inflammation: Unraveling Their Impact on Anemia in HIV Infection. *INOSR Experimental Sciences*. 2023; 12(3):73-84. https://doi.org/10.59298/INOSRES/2023/7.3.21322
13. 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
14. Popovics P, Skalitzky KO, Schroeder E, Jain A, Silver SV, Van Fritz F, Uchtmann KS, Vezina CM, Ricke WA. Steroid hormone imbalance drives macrophage infiltration and Spp1/osteopontin+ foam cell differentiation in the prostate. J Pathol. 2023 Jun;260(2):177-189. doi: 10.1002/path.6074. Epub 2023 Apr 4. PMID: 36825524; PMCID: PMC10231633.
15. 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
16. 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
17. 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.
18. 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
19. 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
20. Aaron-Brooks LM, Sasaki T, Vickman RE, Wei L, Franco OE, Ji Y, Crawford SE, Hayward SW. Hyperglycemia and T Cell infiltration are associated with stromal and epithelial prostatic hyperplasia in the nonobese diabetic mouse. Prostate. 2019 Jun;79(9):980-993. doi: 10.1002/pros.23809. Epub 2019 Apr 18. PMID: 30999385; PMCID: PMC6591734.
21. Seputra KP, Kalim H, Susianti H, Purnomo BB, Al Rasyid H, Purnomo AF. Modulation of T-cell Regulators Associated with Advanced Stage of Prostate Cancer. Acta Inform Med. 2021 Sep;29(3):182-186. doi: 10.5455/aim.2021.29.182-186. PMID: 34759457; PMCID: PMC8563035.
22. Jin X, Lin T, Yang G, Cai H, Tang B, Liao X, Li H, Chen X, Gong L, Xu H, Sun Y, Tan P, Yin J, Ma H, Ai J, Wang K, Wei Q, Yang L, Li H. Use of Tregs as a cell-based therapy via CD39 for benign prostate hyperplasia with inflammation. J Cell Mol Med. 2020 May;24(9):5082-5096. doi: 10.1111/jcmm.15137. Epub 2020 Mar 19. PMID: 32191396; PMCID: PMC7205803.
23. 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.
24. Jiang Q, Han BM, Zhao FJ, Hong Y, Xia SJ. The differential effects of prostate stromal cells derived from different zones on prostate cancer epithelial cells under the action of sex hormones. Asian J Androl. 2011

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ONLINE ISSN: 2992-5479
PRINT ISSN: 2992-605X

Nov;13(6):798-805. doi: 10.1038/aja.2011.22. Epub 2011 Jul 18. PMID: 21765438; PMCID: PMC3739557.

25. 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
26. Ma Y, de Castro Brás LE, Toba H, Iyer RP, Hall ME, Winniford MD, Lange RA, Tyagi SC, Lindsey ML. Myofibroblasts and the extracellular matrix network in post-myocardial infarction cardiac remodeling. Pflugers Arch. 2014 Jun;466(6):1113-27. doi: 10.1007/s00424-014-1463-9. Epub 2014 Feb 13. PMID: 24519465; PMCID: PMC4033805.
27. Młynarska E, Kowalik A, Krajewska A, Krupińska N, Marcinkowska W, Motor J, Przybylak A, Tłustochowicz K, Rysz J, Franczyk B. Inflammaging and Senescence-Driven Extracellular Matrix Remodeling in Age-Associated Cardiovascular Disease. Biomolecules. 2025; 15(10):1452. https://doi.org/10.3390/biom15101452
28. Klingberg F, Hinz B, White ES. The myofibroblast matrix: implications for tissue repair and fibrosis. J Pathol. 2013 Jan;229(2):298-309. doi: 10.1002/path.4104. PMID: 22996908; PMCID: PMC4005341.
29. Harrison DG. Basic science: Pathophysiology: oxidative stress. J Am Soc Hypertens. 2014 Aug;8(8):601-3. doi: 10.1016/j.jash.2014.07.002. Epub 2014 Jul 5. PMID: 25151322; PMCID: PMC4300202.
30. 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
31. Mittal M, Siddiqui MR, Tran K, Reddy SP, Malik AB. Reactive oxygen species in inflammation and tissue injury. Antioxid Redox Signal. 2014 Mar 1;20(7):1126-67. doi: 10.1089/ars.2012.5149. Epub 2013 Oct 22. PMID: 23991888; PMCID: PMC3929010.
32. 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
33. 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
34. Izah SC, Uti DE, Ugwu OP, Betiang PA, Basajja M, Ejemot-Nwadiaro RI. Targeting Cellular Senescence for Healthy Aging: Advances in Senolytics and Senomorphics. Drug Des Devel Ther. 2025 Sep 19;19:8489-8522. doi: 10.2147/DDDT.S543211. PMID: 40994903; PMCID: PMC12456441.
35. Liu RM, Desai LP. Reciprocal regulation of TGF-β and reactive oxygen species: A perverse cycle for fibrosis. Redox Biol. 2015 Dec;6:565-577. doi: 10.1016/j.redox.2015.09.009. Epub 2015 Oct 10. PMID: 26496488; PMCID: PMC4625010.
36. Sampson N, Koziel R, Zenzmaier C, Bubendorf L, Plas E, Jansen-Dürr P, Berger P. ROS signaling by NOX4 drives fibroblast-to-myofibroblast differentiation in the diseased prostatic stroma. Mol Endocrinol. 2011 Mar;25(3):503-15. doi: 10.1210/me.2010-0340. Epub 2011 Jan 27. PMID: 21273445; PMCID: PMC5417274.
37. Yousefpour P, Ni K, Irvine DJ. Targeted modulation of immune cells and tissues using engineered biomaterials. Nat Rev Bioeng. 2023 Feb;1(2):107-124. doi: 10.1038/s44222-022-00016-2. Epub 2023 Jan 30. PMID: 37772035; PMCID: PMC10538251.
38. Kovac S, Angelova PR, Holmström KM, Zhang Y, Dinkova-Kostova AT, Abramov AY. Nrf2 regulates ROS production by mitochondria and NADPH oxidase. Biochim Biophys Acta. 2015 Apr;1850(4):794-801. doi: 10.1016/j.bbagen.2014.11.021. Epub 2014 Dec 5. PMID: 25484314; PMCID: PMC4471129.
39. Ugwu OPC, Alum EU, Okon MB, Aja PM, Obeagu EI, Onyeneke EC. Ethanol root extract and fractions of Sphenocentrum jollyanum abrogate hyperglycaemia and low body weight in streptozotocin-induced diabetic Wistar albino rats. RPS Pharm Pharmacol Rep. 2023;2(2):rqad010.

40. Ugwu OPC, Alum EU, Okon MB, Aja PM, Obeagu EI, Onyeneke EC. Anti-nutritional and gas chromatography-mass spectrometry (GC-MS) analysis of ethanol root extract and fractions of Sphenocentrum jollyanum. RPS Pharm Pharmacol Rep. 2023;2(2):rqad007.

41. Ugwu OPC, Amasiorah VI. The in vitro antioxidant potentials of the crude ethanol root extract and fractions of Sphenocentrum jollyanum. INOSR Appl Sci. 2020;6(1):125–33.

42. Ugwu OPC, Amasiorah VI. The effects of the crude ethanol root extract and fractions of Sphenocentrum jollyanum on hematological indices and glycosylated haemoglobin of streptozotocin-induced diabetic rats. INOSR Sci Res. 2020;6(1):61–74.

43. Ugwu OPC, Amasiorah VI. The effects of crude ethanol root extract and fractions of Sphenocentrum jollyanum on the lipid profile of streptozotocin-induced diabetic Wistar albino rats. IDOSR J Biol Chem Pharm. 2020;5(1):36–46.

44. Ugwu OPC, Amasiorah VI. The in vivo antioxidant potentials of the crude ethanol root extract and fractions of Sphenocentrum jollyanum on oxidative stress indices in streptozotocin-induced diabetic rats. IDOSR J Biol Chem Pharm. 2020;5(1):26–35.

45. Ugwu OPC, Onyeneke EC, Igwenyi IO, Aja PM, Okon MB. The effects of crude ethanol root extract and fractions of Sphenocentrum jollyanum on liver and kidney function parameters of streptozotocin-induced diabetic Wistar albino rats. IAA J Sci Res. 2018;4(1):75–90.

46. Aja PM, Igwenyi IO, Ugwu OPC, Orji OU, Alum EU. Evaluation of anti-diabetic effect and liver function indices of ethanol extracts of Moringa oleifera and Cajanus cajan leaves in alloxan induced diabetic albino rats. Global Vet. 2015;14(3):439–47.

47. Offor CE, Ugwu OPC, Alum EU. The anti-diabetic effect of ethanol leaf-extract of Allium sativum on albino rats. Int J Pharm Med Sci. 2014;4(1):1–3.

48. Ugwu OPC, Alum EU, Uhama KC. Dual burden of diabetes mellitus and malaria: exploring the role of phytochemicals and vitamins in disease management. Res Invent J Res Med Sci. 2024;3(2):38–49.

49. Obeagu EI, Ugwu OPC, Alum EU. Poor glycaemic control among diabetic patients: a review on associated factors. Newport Int J Res Med Sci. 2023;3(1):30–3.

50. Alum EU, Ugwu OPC, Obeagu EI, Ugwu CN, Uti DE, Samson AO, et al. Nutritional care in diabetes mellitus: a comprehensive guide. Int J Innov Appl Res. 2023;11(12):16–25.

51. Alum EU, Ugwu OPC, Obeagu EI, Uti DE, Egba SI, Alum BN. Managing the dual burden: addressing mental health in diabetes care. Elite J Med Sci. 2024;2(6):1–9.

52. Alum EU, Obeagu EI, Ugwu OPC, Alum BN, Arinze ED, Ukaidi CUA. Exploring the differential impacts of intermittent fasting on men and women. Elite J Health Sci. 2024;2(5):37–44.

53. Obeagu EI, Scott GY, Amekpor F, Ugwu OPC, Alum EU. Covid-19 infection and diabetes: a current issue. Int J Innov Appl Res. 2023;11(1):25–30.

54. Alum EU, Ugwu OPC. Beyond pregnancy: understanding the long-term implications of gestational diabetes mellitus. INOSR Sci Res. 2024;11(1):63–71.

55. Ugwu OPC, Ugwu MN, Basajja M, Anyanwu CN, Ogenyi FC, Ugwu CN, et al. Targeting gut microbial signatures to personalize obesity treatment: integrating microbiome-based stratification into precision medicine. Obes Med. 2025;57:100639.

56. Ugwu OPC, Ogenyi FC, Ugwu CN, Ugwu MN. Gut microbiota-derived metabolites as early biomarkers for childhood obesity: a policy commentary from urban African populations. Obes Med. 2025;57:100641.

57. Ugwu OPC, Swase DT, Okon MB, Mujinya R. Traditional East African medicinal plants as modulators of gut microbiota in obesity-associated diabetes: a short communication. Obes Med. 2025;654:00654.

OPEN ACCESS
ONLINE ISSN: 2992-5479
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58. Uti DE, Omang WA, Alum EU, Ugwu OPC, Wokoma MA, Oplekwu RI, et al. Targeting CD44-hyaluronic acid signalling in obesity treatment: insights from small molecules and nanobioconjugates. Nutr Metab Insights. 2026;19:11786388251408961.

59. Uti DE, Omang WA, Alum EU, Atangwho IJ, Ugwu OPC, Wokoma MA, et al. Combined hyaluronic acid nanobioconjugates impair CD44-signaling for effective treatment against obesity: a review of comparison with other actors. Int J Nanomedicine. 2025;20:10101–26.

60. Uti DE, Egbung JE, Alum EU, Bawa I, Ugwu OPC, Egba SI, et al. Nanotechnology-enabled targeted delivery of incretin therapies for the treatment of obesity and metabolic disorders. Obes Med. 2026:100712.

61. Ugwu OPC, Eke MC, Ogenyi FC, Basajja M, Ugwu CN. Could chronic exposure to nocturnal artificial light pollution be an overlooked driver of urban obesity and metabolic syndrome? Med Hypotheses. 2025;205:111819.

62. Alum EU, Izah SC, Uti DE, Ugwu OPC, Betiang PA, Basajja M, et al. The ketogenic diet in obesity management: friend or foe? Cell Biochem Biophys. 2025.

63. Udeh SMC, Nwodo OFC, Yakubu OE, Parker EJ, Egba SI, Omenyi SN, et al. Effects of methanol extract of Gongronema latifolium leaves on glycaemic responses to carbohydrate diets in streptozotocin-induced diabetic rats. J Biol Sci. 2022;22:70–9.

64. Ugwu OPC, Alum EU, Obeagu EI, Okon MB. Effect of ethanol leaf extract of Chromolaena odorata on lipid profile of streptozotocin-induced diabetic Wistar albino rats. IAA J Biol Sci. 2023;10(1):109–17.

65. Ugwu OPC, Alum EU, Obeagu EI, Okon MB. Effect of ethanol leaf extract of Chromolaena odorata on hepatic markers in streptozotocin-induced diabetic Wistar albino rats. IAA J Appl Sci. 2023;9(1):46–56.

66. Aja PM, Ibekwe VI, Ekpono EU, Ugwu PC, Ugwu OPC. Effect of ethanol extract of Cajanus cajan leaf on plasma lipid level in albino rats. Int J Curr Res Acad Rev. 2015;3(1):161–7.

67. Obeagu EI, Ugwu OPC. Overweight and obesity among adolescents: a review. Newport Int J Res Med Sci. 2023;3(1):25–9.

68. Obeagu EI, Ugwu OPC. Insulin self-medication among diabetic patients: a review of associated factors. Newport Int J Res Med Sci. 2023;3(1):21–4.

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