EURASIAN EXPERIMENT JOURNAL OF SCIENTIFIC AND APPLIED RESEARCH
(EEJSAR) ISSN: 2992-4146
©EEJSAR Publications Volume 7 Issue 3 2025

Metabolic Syndrome and BPH: A Shared Pathophysiology or Coincidence?

Mugisha Emmanuel K.

Faculty of Science and Technology Kampala International University Uganda

ABSTRACT

Benign prostatic hyperplasia (BPH) is a common condition among aging men, characterized by the enlargement of the prostate gland, leading to lower urinary tract symptoms (LUTS). Metabolic syndrome (MetS), a cluster of metabolic disorders including obesity, insulin resistance, hypertension, and dyslipidemia, has been increasingly associated with BPH. The overlapping risk factors and mechanistic pathways suggest a potential shared pathophysiology between the two conditions rather than a mere coincidence. This review explores the epidemiological evidence linking MetS with BPH, the underlying molecular and biochemical pathways, and potential therapeutic implications. We discuss the role of chronic inflammation, hormonal dysregulation, oxidative stress, and endothelial dysfunction as common contributors to both conditions. Additionally, we evaluate lifestyle and pharmacological interventions targeting MetS and their impact on BPH progression. Understanding the interplay between MetS and BPH may provide new avenues for prevention and treatment strategies for men at risk.

Keywords: Metabolic syndrome, benign prostatic hyperplasia, insulin resistance, obesity, inflammation, oxidative stress, hormonal dysregulation, endothelial dysfunction

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a progressive, non-malignant enlargement of the prostate gland that commonly affects aging men[1, 2]. It is a major cause of lower urinary tract symptoms (LUTS), which can lead to significant discomfort, impair daily activities, and reduce overall quality of life. BPH prevalence increases with age, affecting approximately 50% of men in their 50s and up to 90% of those in their 80s[3]. The condition is often managed with lifestyle modifications, pharmacological interventions, or surgical procedures, depending on the severity of symptoms and patient preference[4]. Traditionally, BPH has been attributed to hormonal imbalances, particularly the age-related increase in dihydrotestosterone (DHT) and alterations in androgen-estrogen ratios[5]. However, accumulating evidence suggests that metabolic disturbances may play a crucial role in its pathophysiology. Among these metabolic disturbances, metabolic syndrome (MetS) has emerged as a significant risk factor for BPH development and progression[6]. MetS is a cluster of interrelated metabolic abnormalities, including central obesity, insulin resistance, hypertension, dyslipidemia, and pro-inflammatory states, which together increase the risk of cardiovascular diseases, type 2 diabetes, and other metabolic disorders[7]. Recent epidemiological and clinical studies indicate a strong association between MetS and BPH, suggesting that these two conditions may share common pathophysiological mechanisms[8]. Several molecular pathways, including chronic inflammation, oxidative stress, hormonal dysregulation, and endothelial dysfunction, have been proposed as potential links between MetS and BPH. These shared mechanisms raise the question of whether MetS directly contributes to BPH progression or if their coexistence is merely coincidental due to common risk factors such as aging and sedentary lifestyle[9]. The link between MetS and BPH is complex and multifactorial. Several key mechanisms have been proposed to explain this association, including chronic inflammation, oxidative stress, hormonal imbalances, and vascular dysfunction. Chronic low-grade inflammation is a hallmark of both MetS and BPH[10]. Adipose tissue, particularly visceral fat, is a major source of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β). These cytokines contribute to systemic inflammation and may promote prostatic tissue proliferation[11, 12]. Histopathological studies have revealed that prostate tissues from BPH patients often exhibit inflammatory infiltrates, suggesting a role for immune-mediated mechanisms in disease progression. Inflammatory pathways activate nuclear factor-kappa B (NF-κB) signaling, which enhances the expression of growth factors such as transforming growth factor-beta (TGF-β) and vascular endothelial growth factor (VEGF), leading to increased stromal proliferation and fibrosis in the prostate[13]. Oxidative stress,

characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, is another crucial link between MetS and BPH. Hyperglycemia, insulin resistance, and dyslipidemia in MetS contribute to excessive ROS generation, which damages cellular structures and promotes prostatic hyperplasia. Studies have shown that oxidative stress markers, such as malondialdehyde (MDA) and superoxide dismutase (SOD), are elevated in both MetS and BPH patients[14, 15]. ROS also activate inflammatory pathways and contribute to mitochondrial dysfunction, further exacerbating prostate tissue remodeling and fibrosis. Vascular dysfunction is a key component of MetS that may contribute to BPH pathogenesis. Hypertension and dyslipidemia impair endothelial function, reducing nitric oxide (NO) availability and leading to vascular insufficiency. Since the prostate is highly vascularized, compromised blood flow may result in ischemia, tissue hypoxia, and compensatory stromal proliferation[16]. This mechanism is similar to what is observed in erectile dysfunction, another condition closely associated with MetS[17]. The growing body of evidence supporting a link between MetS and BPH highlights the importance of a holistic approach to managing prostatic enlargement in aging men. Shared pathophysiological mechanisms, including chronic inflammation, oxidative stress, hormonal imbalances, and vascular dysfunction, suggest that metabolic interventions may offer new therapeutic opportunities[17]. Future research should focus on elucidating causal relationships and identifying targeted treatment strategies that address both MetS and BPH simultaneously.

Epidemiological Evidence Linking Metabolic Syndrome and BPH

Several population-based studies suggest a significant correlation between MetS and BPH. Research indicates that men with MetS have a higher prevalence and severity of BPH compared to those without MetS. The individual components of MetS—particularly obesity, insulin resistance, and hypertension—have been independently associated with prostate enlargement and LUTS severity[18, 19].

Obesity and BPH: Central obesity, marked by increased visceral fat, is a major risk factor for BPH. Adipose tissue functions as an endocrine organ, secreting pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukins, which may contribute to prostate tissue inflammation and hyperplasia[11, 19].

Insulin Resistance and BPH: Insulin resistance, a hallmark of MetS, may drive BPH progression through hyperinsulinemia, leading to increased insulin-like growth factor (IGF-1) signaling, which promotes prostatic stromal and epithelial proliferation[20].

Hypertension and BPH: Endothelial dysfunction and chronic vascular changes associated with hypertension may impair prostatic blood flow, inducing tissue hypoxia and oxidative stress, further exacerbating BPH[21].

Dyslipidemia and BPH: Altered lipid metabolism, characterized by elevated LDL and reduced HDL levels, contributes to chronic inflammation and oxidative stress, creating a conducive environment for prostatic hyperplasia[22].

Pathophysiological Mechanisms Connecting Metabolic Syndrome and BPH

Chronic Inflammation

Inflammation serves as a crucial link between metabolic syndrome (MetS) and benign prostatic hyperplasia (BPH), contributing to the pathophysiological mechanisms that exacerbate lower urinary tract symptoms (LUTS)[23]. MetS is characterized by a cluster of metabolic abnormalities, including insulin resistance, central obesity, dyslipidemia, and hypertension[23]. These factors promote a state of chronic low-grade inflammation, which plays a pivotal role in the initiation and progression of BPH. Elevated levels of pro-inflammatory cytokines and mediators such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) have been detected in both MetS and BPH, indicating a shared inflammatory pathway[24]. One of the primary mechanisms through which inflammation contributes to BPH involves the activation of immune cells and the release of inflammatory cytokines. In MetS, adipose tissue dysfunction leads to an increased release of pro-inflammatory adipokines, which in turn enhance systemic inflammation[25]. This inflammatory milieu fosters the recruitment of immune cells such as macrophages and T lymphocytes to the prostate tissue, where they promote oxidative stress, cellular proliferation, and fibrosis. The persistent presence of inflammatory mediators within the prostate microenvironment results in tissue remodeling, excessive stromal proliferation, and glandular hyperplasia, ultimately leading to increased prostate volume and the exacerbation of LUTS[25]. Chronic inflammation also disrupts normal androgen signaling, further exacerbating prostate enlargement. IL-6 and TNF-α are known to modulate androgen receptor (AR) activity, altering the balance between epithelial and stromal cell growth in the prostate.[26] This dysregulation contributes to the development of hyperplastic nodules, a hallmark of BPH. Additionally, inflammatory cytokines induce the expression of growth factors such as transforming growth factor-beta (TGF-β) and fibroblast growth factor (FGF), both of which play a role in stromal expansion and fibrosis. Over time, this leads to reduced elasticity of the prostate and surrounding tissues, further worsening urinary obstruction and LUTS[26]. Another significant consequence of inflammation in BPH is its impact on vascular function. Patients with MetS often exhibit endothelial dysfunction and impaired microcirculation due to systemic inflammation and oxidative stress. In the prostate, these vascular abnormalities reduce blood flow, leading to ischemic injury and subsequent hypoxia.[27] Hypoxia-inducible factors (HIFs) are activated in response to reduced oxygen availability, further

stimulating the expression of inflammatory cytokines and growth factors that promote prostate cell proliferation and fibrosis. This creates a vicious cycle in which inflammation, hypoxia, and tissue remodeling continuously reinforce each other, exacerbating BPH progression[27]. The interplay between inflammation, metabolic dysfunction, and prostate growth highlights the need for therapeutic interventions that target inflammatory pathways. Anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs) and cytokine inhibitors, have shown potential in reducing prostate inflammation and alleviating LUTS in BPH patients with MetS[28]. Lifestyle modifications, including weight loss, dietary changes, and increased physical activity, can also help mitigate inflammation and improve overall metabolic health, thereby reducing the severity of BPH symptoms. In sum, chronic inflammation acts as a critical link between MetS and BPH by promoting immune cell infiltration, oxidative stress, androgen dysregulation, and vascular dysfunction. Understanding these mechanisms can aid in the development of targeted therapies that address both metabolic and inflammatory aspects of BPH, ultimately improving patient outcomes.[28].

Hormonal Dysregulation

Metabolic Syndrome (MetS) is a cluster of conditions that includes central obesity, insulin resistance, dyslipidemia, and hypertension, all of which contribute to systemic inflammation and endocrine disruption[29]. One of the key consequences of MetS is its impact on hormonal balance, particularly the dysregulation of androgens and estrogens, which play a crucial role in the development and progression of benign prostatic hyperplasia (BPH). Several mechanisms link MetS to hormonal imbalances that contribute to prostatic enlargement and increased risk of lower urinary tract symptoms (LUTS) in aging men[29].

Testosterone and Dihydrotestosterone (DHT)

Testosterone, the primary male androgen, is essential for normal prostate growth and function. However, its levels naturally decline with age, which is often accompanied by compensatory hormonal shifts that can impact prostatic tissue[30]. Despite lower systemic testosterone levels in older men, the enzyme 5α-reductase, which is highly expressed in prostatic tissue, converts testosterone into its more potent metabolite, dihydrotestosterone (DHT). DHT is a crucial driver of prostatic cell proliferation, and evidence suggests that in obese individuals with MetS, the increased activity of 5α-reductase leads to an excessive accumulation of DHT within the prostate[30]. This localized elevation of DHT, despite systemic testosterone decline, may promote prostate enlargement and the progression of BPH. Additionally, insulin resistance, a hallmark of MetS, has been shown to reduce sex hormone-binding globulin (SHBG) levels. SHBG binds to circulating androgens, regulating their bioavailability. Lower SHBG levels in individuals with MetS result in increased levels of free testosterone, which is more readily converted to DHT, further exacerbating prostate overgrowth[30].

Estrogenic Influence and the Androgen-to-Estrogen Ratio

Adipose tissue is a key endocrine organ that influences sex hormone metabolism. In individuals with MetS and obesity, adipose tissue expresses higher levels of aromatase, the enzyme responsible for converting androgens into estrogens[31]. This leads to an altered androgen-to-estrogen ratio, favoring estrogen dominance. Estrogens, particularly estradiol (E2), have been implicated in the pathogenesis of BPH by promoting stromal proliferation and fibrotic changes within the prostate. Studies suggest that estrogenic signaling within the prostate is mediated through estrogen receptor alpha (ERα) and estrogen receptor beta (ERβ). While ERβ has been associated with anti-proliferative and anti-inflammatory effects, ERα activation is linked to prostatic hyperplasia and inflammation. Increased estrogen levels due to obesity and MetS may enhance ERα-mediated pathways, driving fibroblast proliferation and extracellular matrix deposition in the prostate, ultimately contributing to prostate enlargement and LUTS[32].

Leptin and Adipokines in Prostate Growth

Leptin, an adipokine primarily produced by adipose tissue, plays a critical role in energy homeostasis and metabolic regulation. However, in the context of obesity and MetS, leptin levels are chronically elevated, leading to leptin resistance. Elevated leptin levels have been shown to exert mitogenic effects on prostate cells by stimulating proliferative and inflammatory pathways[33]. Leptin activates key signaling pathways, including Janus kinase/signal transducer and activator of transcription (JAK/STAT), phosphoinositide 3-kinase (PI3K), and mitogen-activated protein kinase (MAPK), all of which contribute to increased prostate cell proliferation[33]. Additionally, leptin-induced inflammation promotes the release of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), further exacerbating BPH pathogenesis. MetS-induced hormonal imbalances, particularly increased DHT accumulation, altered estrogenic signaling, and elevated adipokines, create a pro-growth and pro-inflammatory environment in the prostate[34]. Understanding these mechanisms highlights the importance of metabolic health in mitigating the risk and progression of BPH, offering potential therapeutic targets for intervention.

Oxidative Stress and Endothelial Dysfunction

Metabolic syndrome (MetS) is associated with heightened oxidative stress due to excessive reactive oxygen species (ROS) production, which plays a crucial role in the pathophysiology of benign prostatic hyperplasia (BPH). ROS accumulation in the prostate leads to several pathological changes, including[35, 36]:

a. DNA Damage and Aberrant Cellular Proliferation

Oxidative stress-induced DNA damage occurs through the oxidation of nucleotides, strand breaks, and mutations in key regulatory genes. This genomic instability may activate oncogenic pathways while impairing tumor suppressor functions, thereby promoting uncontrolled cellular proliferation. In BPH, increased cell division leads to the expansion of the prostate gland, contributing to urethral compression and lower urinary tract symptoms (LUTS). Moreover, chronic inflammation associated with MetS exacerbates ROS generation, further stimulating epithelial and stromal cell proliferation via pathways such as NF-κB and PI3K/Akt.

b. Microvascular Dysfunction and Tissue Hypoxia

ROS overproduction reduces nitric oxide (NO) bioavailability, a critical molecule for vascular homeostasis. NO depletion leads to endothelial dysfunction, resulting in diminished blood flow to the prostate and the development of microvascular insufficiency. Hypoxia-inducible factor-1α (HIF-1α) is upregulated under these conditions, promoting angiogenesis and fibrosis, which further aggravate prostatic enlargement. Additionally, chronic hypoxia fosters an inflammatory microenvironment, accelerating the progression of BPH through increased fibroblast proliferation and extracellular matrix deposition.

c. Inflammatory Signaling and Fibrosis

Persistent oxidative stress in the prostate triggers the release of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and transforming growth factor-beta (TGF-β). These cytokines enhance fibroblast activation and extracellular matrix remodeling, leading to stromal expansion and fibrosis—hallmarks of advanced BPH. Furthermore, oxidative stress-mediated activation of cyclooxygenase-2 (COX-2) contributes to chronic inflammation, exacerbating oxidative damage and sustaining a pro-proliferative environment.

d. Mitochondrial Dysfunction and Apoptosis Resistance

ROS-driven mitochondrial dysfunction disrupts normal apoptotic signaling, allowing hyperplastic prostatic cells to evade programmed cell death. Dysfunctional mitochondria contribute to metabolic reprogramming, favoring glycolysis over oxidative phosphorylation, a hallmark of proliferative disorders. This metabolic shift supports continued prostatic growth despite the presence of cellular stressors.

d. Hormonal Dysregulation and Androgen Receptor Activation

Oxidative stress alters steroid hormone metabolism, leading to increased conversion of testosterone to dihydrotestosterone (DHT) via upregulation of 5α-reductase. DHT, a potent androgen, drives prostate cell proliferation through androgen receptor (AR) activation. Additionally, ROS-induced lipid peroxidation generates bioactive aldehydes such as 4-hydroxynonenal (4-HNE), which can enhance AR signaling, further promoting prostatic hyperplasia. The interplay between oxidative stress, inflammation, and metabolic dysregulation in MetS creates a pro-proliferative and hypoxic microenvironment in the prostate, accelerating BPH progression. Targeting oxidative stress through antioxidant therapies, NO modulation, and metabolic interventions may provide novel strategies for mitigating prostatic enlargement in individuals with MetS.

Autonomic Nervous System Dysregulation

Metabolic syndrome (MetS) has been closely associated with increased sympathetic nervous system (SNS) activity, which plays a crucial role in the pathophysiology of lower urinary tract symptoms (LUTS), particularly in benign prostatic hyperplasia (BPH).[37] The heightened SNS activity observed in MetS patients leads to excessive stimulation of α-adrenergic receptors in the prostate and bladder neck, resulting in increased prostatic smooth muscle tone[37]. This heightened muscle tone exacerbates urinary symptoms such as weak stream, nocturia, and increased voiding difficulty. Sympathetic hyperactivity also contributes to bladder outlet obstruction (BOO), a major factor in the progression of LUTS associated with BPH. BOO arises due to both static and dynamic components—where the static component involves prostatic enlargement compressing the urethra, and the dynamic component is mediated by increased adrenergic signaling that leads to excessive contraction of the prostate and bladder neck[38]. This results in increased urethral resistance, impaired bladder emptying, and secondary changes in bladder function such as detrusor overactivity or impaired contractility. Moreover, chronic SNS overactivity in MetS is linked to systemic inflammation, oxidative stress, and endothelial dysfunction, which may further exacerbate LUTS by promoting prostatic hyperplasia, fibrosis, and reduced nitric oxide-mediated relaxation of the smooth muscle. This interplay between metabolic dysregulation and autonomic dysfunction highlights the complex mechanisms through which MetS worsens[38] BPH-related urinary symptoms, emphasizing the need for therapeutic strategies targeting both metabolic control and sympathetic inhibition to alleviate LUTS in affected individuals.

Therapeutic Implications and Management Strategies

Lifestyle Interventions

Given the strong association between MetS and BPH, lifestyle modifications may serve as a primary strategy for mitigating BPH risk and progression:

Weight Loss: Reducing central obesity through dietary changes and exercise decreases inflammatory markers and improves insulin sensitivity, potentially alleviating BPH severity.

Exercise: Regular physical activity enhances endothelial function, reduces sympathetic activity, and modulates hormone levels, beneficially impacting both MetS and BPH.

Dietary Modifications: A Mediterranean diet, rich in polyphenols and healthy fats, may reduce oxidative stress and inflammation, slowing BPH progression.

Pharmacological Approaches

Several pharmacological agents targeting MetS components may also influence BPH development:

Statins: Cholesterol-lowering statins possess anti-inflammatory and antioxidant properties, which may attenuate BPH progression.

Metformin: As an insulin-sensitizing agent, metformin reduces hyperinsulinemia and IGF-1 activity, potentially impeding prostatic growth.

Antihypertensives: Certain antihypertensive agents, such as angiotensin receptor blockers (ARBs), exhibit anti-inflammatory effects that could benefit BPH management.

5-Alpha Reductase Inhibitors (5-ARIs): Drugs like finasteride and dutasteride inhibit DHT synthesis, mitigating BPH symptoms, although their effects may be influenced by underlying MetS.

CONCLUSION

The growing body of evidence supports the notion that MetS and BPH share common pathophysiological mechanisms, including chronic inflammation, hormonal imbalances, oxidative stress, and endothelial dysfunction. This interplay suggests that BPH in men with MetS is more than a coincidence, highlighting the need for integrated management strategies. Addressing MetS through lifestyle and pharmacological interventions may not only mitigate cardiovascular risks but also serve as a novel approach to preventing or alleviating BPH progression. Future research should explore targeted therapies that simultaneously address both conditions, improving patient outcomes.

Future Directions

Further studies are needed to:

i. Elucidate the molecular mechanisms linking MetS to BPH at the genetic and epigenetic levels.
ii. Develop novel therapeutic agents targeting shared metabolic and inflammatory pathways.
iii. Investigate the long-term effects of MetS interventions on BPH progression through large-scale, longitudinal studies.

REFERENCES

1. Gravas, S., Oelke, M.: Current status of 5α-reductase inhibitors in the management of lower urinary tract symptoms and BPH. World J Urol. 28, 9–15 (2010). https://doi.org/10.1007/s00345-009-0493-y
2. Ibiam, U.A., Uti, D.E., Ejeogo, C.C., Orji, O.U., Aja, P.M., Nwamaka, E.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 NU. 14, 477–485 (2024). https://doi.org/10.1055/s-0043-1777836
3. Roehrborn, C., Heaton, J.P.W.: Medical Management for BPH: The Role of Combination Therapy. European Urology Supplements. 5, 716–721 (2006). https://doi.org/10.1016/j.eursup.2006.06.010
4. Guadagnoli, L., Simons, M., McGarva, J., Taft, T.H., van Tilburg, M.A.L.: Improving Patient Adherence to Lifestyle Changes for the Management of Gastroesophageal Reflux. Patient Prefer Adherence. 16, 897–909 (2022). https://doi.org/10.2147/PPA.S356466
5. Patel, R., Sina, R.E., Keyes, D.: Lifestyle Modification for Diabetes and Heart Disease Prevention. In: StatPearls. StatPearls Publishing, Treasure Island (FL) (2025)
6. Ngai, H.-Y., Yuen, K.-K.S., Ng, C.-M., Cheng, C.-H., Chu, S.-K.P.: Metabolic syndrome and benign prostatic hyperplasia: An update. Asian J Urol. 4, 164–173 (2017). https://doi.org/10.1016/j.ajur.2017.05.001
7. Fahed, G., Aoun, L., Bou Zerdan, M., Allam, S., Bou Zerdan, M., Bouferraa, Y., Assi, H.I.: Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. Int J Mol Sci. 23, 786 (2022). https://doi.org/10.3390/ijms23020786
8. Fu, X., Wang, Y., Lu, Y., Liu, J., Li, H.: Association between metabolic syndrome and benign prostatic hyperplasia: The underlying molecular connection. Life Sciences. 358, 123192 (2024). https://doi.org/10.1016/j.lfs.2024.123192
9. Calogero, A.E., Burgio, G., Condorelli, R.A., Cannarella, R., La Vignera, S.: Epidemiology and risk factors of lower urinary tract symptoms/benign prostatic hyperplasia and erectile dysfunction. The Aging Male. 22, 12–19 (2019). https://doi.org/10.1080/13685538.2018.1434772
10. Agrawal, A.: Metabolic syndrome and BPH: What do we know? Med J Armed Forces India. 73, 102–103 (2017). https://doi.org/10.1016/j.mjafi.2016.11.011
11. Uti, D.E., Atangwho, I.J., Omang, W.A., Alum, E.U., Obeten, U.N., Udeozor, P.A., Agada, S.A., Bawa, I., Ogbu, C.O.: Cytokines as key players in obesity low grade inflammation and related complications. Obesity Medicine. 54, 100585 (2025). https://doi.org/10.1016/j.obmed.2025.100585

12. Gratal, P., Lamuedra, A., Medina, J.P., Bermejo-Álvarez, I., Largo, R., Herrero-Beaumont, G., Mediero, A.: Purinergic System Signaling in Metainflammation-Associated Osteoarthritis. Front. Med. 7, (2020). https://doi.org/10.3389/fmed.2020.00506
13. Song, G., Tong, J., Wang, Y., Li, Y., Liao, Z., Fan, D., Fan, X.: Nrf2-mediated macrophage function in benign prostatic hyperplasia: Novel molecular insights and implications. Biomedicine & Pharmacotherapy. 167, 115566 (2023). https://doi.org/10.1016/j.biopha.2023.115566
14. Masenga, S.K., Kabwe, L.S., Chakulya, M., Kirabo, A.: Mechanisms of Oxidative Stress in Metabolic Syndrome. Int J Mol Sci. 24, 7898 (2023). https://doi.org/10.3390/ijms24097898
15. Tangvarasittichai, S.: Oxidative stress, insulin resistance, dyslipidemia and type 2 diabetes mellitus. World J Diabetes. 6, 456–480 (2015). https://doi.org/10.4239/wjd.v6.i3.456
16. Raee, P., Tan, S.C., Najafi, S., Zandsalimi, F., Low, T.Y., Aghamiri, S., Fazeli, E., Aghapour, M., Mofarahe, Z.S., Heidari, M.H., Fathabadi, F.F., Abdi, F., Asouri, M., Ahmadi, A.A., Ghanbarian, H.: Autophagy, a critical element in the aging male reproductive disorders and prostate cancer: a therapeutic point of view. Reproductive Biology and Endocrinology. 21, 88 (2023). https://doi.org/10.1186/s12958-023-01134-1
17. Han, Z., Ma, K., Tao, H., Liu, H., Zhang, J., Sai, X., Li, Y., Chi, M., Nian, Q., Song, L., Liu, C.: A Deep Insight Into Regulatory T Cell Metabolism in Renal Disease: Facts and Perspectives. Front. Immunol. 13, (2022). https://doi.org/10.3389/fimmu.2022.826732
18. Daher, M., Saqer, T., Jabr, M., Al-Mousa, S.: Benign prostatic hyperplasia and metabolic syndrome; prevalence and association: a cross-sectional study in Syria. BMC Urology. 23, 187 (2023). https://doi.org/10.1186/s12894-023-01365-9
19. Omran, A., Leca, B.M., Oštarijaš, E., Graham, N., Da Silva, A.S., Zaïr, Z.M., Miras, A.D., le Roux, C.W., Vincent, R.P., Cardozo, L., Dimitriadis, G.K.: Metabolic syndrome is associated with prostate enlargement: a systematic review, meta-analysis, and meta-regression on patients with lower urinary tract symptom factors. Therapeutic Advances in Endocrinology. 12, 20420188211066210 (2021). https://doi.org/10.1177/20420188211066210
20. Wang, Z., Olumi, A.F.: Diabetes, growth hormone-insulin-like growth factor pathways and association to benign prostatic hyperplasia. Differentiation. 82, 261–271 (2011). https://doi.org/10.1016/j.diff.2011.04.004
21. Kaltsas, A., Giannakas, T., Stavropoulos, M., Kratiras, Z., Chrisofos, M.: Oxidative Stress in Benign Prostatic Hyperplasia: Mechanisms, Clinical Relevance and Therapeutic Perspectives. Diseases. 13, 53 (2025). https://doi.org/10.3390/diseases13020053
22. Parsons, J.K., Bergstrom, J., Barrett-Connor, E.: Lipids, Lipoproteins, and Risk of Benign Prostatic Hyperplasia in Community Dwelling Men. BJU Int. 101, 313–318 (2008). https://doi.org/10.1111/j.1464-410X.2007.07332.x
23. He, Q., Wang, Z., Liu, G., Daneshgari, F., MacLennan, G.T., Gupta, S.: Metabolic Syndrome, Inflammation and Lower Urinary Tract Symptoms – Possible Translational Links. Prostate Cancer Prostatic Dis. 19, 7–13 (2016). https://doi.org/10.1038/pcan.2015.43
24. Naiyila, X., Li, J., Huang, Y., Chen, B., Zhu, M., Li, J., Chen, Z., Yang, L., Ai, J., Wei, Q., Liu, L., Cao, D.: A Novel Insight into the Immune-Related Interaction of Inflammatory Cytokines in Benign Prostatic Hyperplasia. J Clin Med. 12, 1821 (2023). https://doi.org/10.3390/jcm12051821
25. Xia, B.-W., Zhao, S.-C., Chen, Z.-P., Chen, C., Liu, T.-S., Yang, F., Yan, Y.: The underlying mechanism of metabolic syndrome on benign prostatic hyperplasia and prostate volume. The Prostate. 80, 481–490 (2020). https://doi.org/10.1002/pros.23962
26. Archer, M., Dogra, N., Kyprianou, N.: Inflammation as a Driver of Prostate Cancer Metastasis and Therapeutic Resistance. Cancers (Basel). 12, 2984 (2020). https://doi.org/10.3390/cancers12102984
27. Wong, L., Gipp, J., Carr, J., Loftus, C., Benck, M., Lee, S., Mehta, V., Vezina, C., Bushman, W.: Prostate Angiogenesis in Development and Inflammation. Prostate. 74, 346–358 (2014). https://doi.org/10.1002/pros.22751
28. Hatano, K., Fujita, K., Nonomura, N.: Application of Anti-Inflammatory Agents in Prostate Cancer. J Clin Med. 9, 2680 (2020). https://doi.org/10.3390/jcm9082680
29. Kassi, E., Pervanidou, P., Kaltsas, G., Chrousos, G.: Metabolic syndrome: definitions and controversies. BMC Medicine. 9, 48 (2011). https://doi.org/10.1186/1741-7015-9-48
30. Welén, K., Damber, J.-E.: Androgens, aging, and prostate health. Rev Endocr Metab Disord. 23, 1221–1231 (2022). https://doi.org/10.1007/s11154-022-09730-z
31. Mair, K.M., Gaw, R., MacLean, M.R.: Obesity, estrogens and adipose tissue dysfunction – implications for pulmonary arterial hypertension. Pulm Circ. 10, 2045894020952019 (2020). https://doi.org/10.1177/2045894020952023
32. Cannarella, R., Condorelli, R.A., Barbagallo, F., La Vignera, S., Calogero, A.E.: Endocrinology of the Aging Prostate: Current Concepts. Front Endocrinol (Lausanne). 12, 554078 (2021). https://doi.org/10.3389/fendo.2021.554078

33. Picó, C., Palou, M., Pomar, C.A., Rodríguez, A.M., Palou, A.: Leptin as a key regulator of the adipose organ. Rev Endocr Metab Disord. 23, 13–30 (2022). https://doi.org/10.1007/s11154-021-09687-5
34. Dessie, G., Ayelign, B., Akalu, Y., Shibabaw, T., Molla, M.D.: Effect of Leptin on Chronic Inflammatory Disorders: Insights to Therapeutic Target to Prevent Further Cardiovascular Complication. Diabetes Metab Syndr Obes. 14, 3307–3322 (2021). https://doi.org/10.2147/DMSO.S321311
35. Wang, Z., Mao, Q., Yuan, Y., Wang, C., Wei, H.: Shuangshi Tonglin Capsule treats benign prostatic hyperplasia through the ROS/NLRP3 signaling pathway. Int Urol Nephrol. 56, 1259–1271 (2024). https://doi.org/10.1007/s11255-023-03874-w
36. Masenga, S.K., Kabwe, L.S., Chakulya, M., Kirabo, A.: Mechanisms of Oxidative Stress in Metabolic Syndrome. International Journal of Molecular Sciences. 24, 7898 (2023). https://doi.org/10.3390/ijms24097898
37. Bahar Arıcan Tarım, E.Ç.: Metabolic Syndrome and Benign Prostatic Hyperplasia/Which Component of Metabolic Syndrome Is Related to Benign Prostatic Hyperplasia?, https://jurolsurgery.org/articles/metabolic-syndrome-and-benign-prostatic-hyperplasiawhich-component-of-metabolic-syndrome-is-related-to-benign-prostatic-hyperplasia/doi/jus.galenos.2023.2022.0081
38. Komninos, C., Mitsogiannis, I.: Obstruction-induced alterations within the urinary bladder and their role in the pathophysiology of lower urinary tract symptomatology. Can Urol Assoc J. 8, E524–E530 (2014). https://doi.org/10.5489/cuaj.1636

CITE AS: Mugisha Emmanuel K. (2025). Metabolic Syndrome and BPH: A Shared Pathophysiology or Coincidence?. EURASIAN EXPERIMENT JOURNAL OF SCIENTIFIC AND APPLIED RESEARCH, 7(3):15-21