©NIJPP
Publications 2025

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

NEWPORT INTERNATIONAL JOURNAL OF PUBLIC HEALTH AND PHARMACY (NIJPP)

Volume 6 Issue 1 Page 134-140, 2025

https://doi.org/10.59298/NIJPP/2025/61134140

Association between Metabolic Syndrome and Benign Prostatic Hyperplasia

Omeye Francis I.

Faculty of Medicine Kampala International University Uganda

ABSTRACT

Benign prostatic hyperplasia (BPH) is a common condition that affects the prostate gland in aging men, leading to lower urinary tract symptoms (LUTS) that significantly impact quality of life. Metabolic syndrome (MetS), characterized by a cluster of risk factors such as obesity, insulin resistance, dyslipidemia, hypertension, and pro-inflammatory states, is increasingly being recognized as a significant determinant in the progression of various chronic diseases, including prostate disorders. The relationship between MetS and BPH has garnered considerable attention in recent years, suggesting that components of MetS may contribute to the pathophysiology of BPH. This review explores the mechanisms linking MetS with BPH, highlighting the role of insulin resistance, obesity, and inflammation in prostate enlargement. We also examine the clinical implications of this association, including its potential impact on diagnosis, treatment, and prevention of BPH in men with MetS. Understanding these interconnections may provide new avenues for managing BPH in patients with metabolic abnormalities.

Keywords: Benign prostatic hyperplasia, metabolic syndrome, insulin resistance, obesity, dyslipidemia, hypertension, inflammation, lower urinary tract symptoms.

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a prevalent, age-related condition characterized by the non-cancerous enlargement of the prostate gland, leading to a range of lower urinary tract symptoms (LUTS)[1–3]. These symptoms, which include frequent urination, weak urinary stream, and nocturia, significantly affect the quality of life of affected individuals. The incidence of BPH rises with age, making it a growing concern in older men, with studies indicating that a substantial proportion of men over 50 years of age experience varying degrees of prostate enlargement[2, 4, 5]. BPH often coexists with other chronic health conditions, and its impact on daily functioning can lead to substantial morbidity, including urinary retention, recurrent urinary tract infections, and in some cases, the need for surgical intervention. Given the considerable burden of BPH, it is essential to explore modifiable risk factors that contribute to the onset and progression of the disease, aiming to reduce its impact on affected individuals[3, 6, 7].

Metabolic syndrome (MetS) represents a cluster of interrelated metabolic disturbances, including abdominal obesity, insulin resistance, dyslipidemia (marked by elevated triglycerides and low high-density lipoprotein cholesterol), and hypertension[8, 9]. This syndrome is associated with a significantly increased risk of developing cardiovascular diseases, type 2 diabetes, and other chronic disorders. MetS has become a major public health issue, especially as the global population ages and lifestyle-related factors, such as poor diet and sedentary behavior, continue to contribute to its prevalence. More recently, research has begun to uncover a potential link between MetS and BPH. Studies have suggested that the metabolic dysfunctions characteristic of MetS, such as insulin resistance and dyslipidemia, may play a role in the development and exacerbation of prostate enlargement. However, the precise mechanisms underlying this relationship remain complex and are still under investigation[10, 11].

This article aims to provide a comprehensive review of the growing body of evidence examining the association between MetS and BPH. It will explore potential biological mechanisms that could explain this link, including hormonal imbalances, inflammation, and endothelial dysfunction. Additionally, the article will discuss the clinical implications of this association, particularly regarding the management of BPH in patients with MetS. Finally, it will address potential strategies for intervention and prevention, focusing on lifestyle modifications,

OPEN ACCESS
ONLINE ISSN: 2992-5479
PRINT ISSN: 2992-605X
pharmacological treatments, and future research directions to improve the outcomes for patients suffering from both conditions.

Pathophysiology of BPH in the Context of Metabolic Syndrome

Insulin Resistance: Insulin resistance (IR), a central feature of metabolic syndrome (MetS), is increasingly recognized as a key factor in the pathophysiology of benign prostatic hyperplasia (BPH). In IR, the body's ability to respond to insulin is diminished, leading to higher circulating levels of insulin, which, in turn, disrupt normal glucose metabolism. This metabolic dysregulation contributes to an array of cellular and molecular processes that can exacerbate the growth of prostate tissues[12, 13]. One critical pathway involves the insulin-like growth factor (IGF) system. Insulin resistance has been shown to elevate circulating insulin levels, which can enhance the production of IGF-1. IGF-1, a peptide with structural similarities to insulin, plays a pivotal role in regulating cell growth, survival, and differentiation. In prostate cells, increased IGF-1 levels can stimulate the activation of intracellular signaling pathways that promote cell proliferation and inhibit apoptosis[14]. This leads to the uncontrolled growth of prostate cells, contributing to the enlargement of the prostate, a hallmark of BPH. Furthermore, IGF-1 has been implicated in prostate cell hypertrophy (increased cell size), further exacerbating prostate enlargement[15].

In addition to direct hormonal effects, insulin resistance can lead to the accumulation of visceral adipose tissue (VAT), which is metabolically active and releases a variety of bioactive molecules, including pro-inflammatory cytokines. Visceral fat, in particular, has been shown to secrete high levels of adipokines such as TNF-α, IL-6, and leptin, all of which are potent mediators of inflammation[16, 17]. This chronic low-grade inflammation creates an environment that is conducive to prostate tissue remodeling and growth. The pro-inflammatory cytokines released by visceral fat have been shown to influence prostate stromal and epithelial cells, encouraging a local inflammatory response that can contribute to the development and progression of BPH.

Moreover, the interaction between insulin resistance, visceral fat accumulation, and inflammation in BPH may extend beyond the prostate. The systemic effects of insulin resistance, including altered lipid metabolism, can further exacerbate the metabolic imbalances that contribute to the pathophysiology of BPH. These factors highlight the complex interplay between metabolic disturbances and prostate pathology. Thus, insulin resistance, by elevating insulin and IGF-1 levels, along with the accumulation of visceral fat and its associated pro-inflammatory cytokines, creates a cascade of events that not only promote prostate cell proliferation and hypertrophy but also foster a chronic inflammatory environment that accelerates the progression of BPH[18]. This underscores the importance of managing insulin resistance and metabolic abnormalities as potential therapeutic targets in individuals with BPH, particularly in those with underlying metabolic syndrome.

Obesity and Adiposity: Obesity, especially the accumulation of visceral fat, plays a central role in the pathogenesis of Metabolic Syndrome (MetS), a cluster of conditions that increase the risk of heart disease, stroke, and type 2 diabetes. Visceral fat, which surrounds internal organs, is particularly harmful because it is metabolically active and secretes a wide range of bioactive molecules, including adipokines[9, 19]. These adipokines, such as leptin, adiponectin, and resistin, are involved in regulating various physiological processes, including energy balance, immune response, and inflammation. Importantly, these molecules can also influence prostate health, potentially contributing to prostate diseases like benign prostatic hyperplasia (BPH).

Leptin is a well-known adipokine that is primarily involved in regulating energy balance by signaling the brain to suppress appetite and increase energy expenditure. However, elevated levels of leptin, which are commonly seen in obese individuals, can have detrimental effects on prostate tissue. Studies have shown that high leptin levels are associated with increased cell proliferation and may contribute to the development and progression of prostate conditions such as BPH. Leptin can activate various signaling pathways that promote inflammation and cell growth, thereby creating an environment that favors prostate enlargement[20].

On the other hand, adiponectin, another adipokine, has anti-inflammatory and anti-proliferative properties. It generally exerts protective effects on metabolic health by improving insulin sensitivity and reducing fat accumulation. However, in individuals with obesity, adiponectin levels tend to be lower, which further exacerbates the inflammatory state. Reduced levels of adiponectin have been linked to an increased risk of BPH and prostate cancer. Adiponectin helps inhibit cell proliferation and inflammation, so its deficiency may contribute to excessive prostate growth, potentially leading to benign or malignant transformation. Resistin is another adipokine that has been implicated in inflammatory processes. It is typically elevated in obesity and has been shown to promote the release of pro-inflammatory cytokines, further contributing to the chronic low-grade inflammation seen in MetS[21]. This inflammatory milieu can exacerbate prostate growth and may influence the pathophysiology of BPH.

Visceral fat accumulation is also a major source of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which are critical mediators of inflammation. These cytokines are often elevated in obesity and can directly impact prostate tissue by stimulating inflammatory responses, oxidative stress, and abnormal cell growth. TNF-α and IL-6 have been implicated in accelerating prostate tissue remodeling, fostering an environment that promotes both hypertrophy and hyperplasia of prostate cells[22,

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

23]. This inflammatory cascade may not only drive BPH progression but could also contribute to the risk of prostate cancer, as the inflammatory microenvironment can enhance tumor initiation and progression. In sum, the interplay between obesity, visceral fat, and the secretion of adipokines and pro-inflammatory cytokines creates a complex environment that accelerates prostate growth. Elevated leptin, reduced adiponectin, and increased levels of resistin, TNF-α, and IL-6 all contribute to local inflammation and enhanced cell proliferation, thereby increasing the risk of BPH. Understanding these mechanisms could provide valuable insights into potential therapeutic strategies for managing prostate diseases, particularly in obese individuals.

Hypertension and Sympathetic Nervous System Activation: Hypertension, a hallmark of Metabolic Syndrome (MetS), plays a significant role in the pathophysiology of benign prostatic hyperplasia (BPH), a condition characterized by prostate enlargement that often leads to lower urinary tract symptoms (LUTS). The association between hypertension and BPH is complex, involving both vascular and neural mechanisms that can influence prostate health[24, 25].

1. **Increased Smooth Muscle Tone and Vascular Constriction**: Hypertension is characterized by sustained high blood pressure, which can lead to increased smooth muscle tone and vascular constriction, affecting blood flow within various tissues, including the prostate. Chronic hypertension can lead to the thickening of blood vessel walls and a reduction in the blood supply to the prostate. This altered blood flow could promote tissue hypoxia (lack of oxygen), contributing to prostatic tissue remodeling and cell growth, which are key features of BPH. The increased vascular tone within the prostate may also result in an imbalance between growth factors, such as vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF), further promoting prostate enlargement.
2. **Sympathetic Nervous System (SNS) Overactivity**: In MetS, the sympathetic nervous system (SNS) is often overactive, and this increased sympathetic drive can have direct effects on the prostate. The sympathetic fibers release norepinephrine, which binds to α-adrenergic receptors in smooth muscle cells. These receptors, particularly the α1-adrenergic receptors, mediate smooth muscle contraction, leading to increased tone in the prostatic stroma and surrounding smooth muscle. This contraction not only increases the internal pressure of the prostate but can also exacerbate LUTS, including urinary frequency, urgency, and nocturia.

   The SNS also contributes to inflammation and fibrosis, processes that are implicated in the pathogenesis of BPH. Increased adrenergic activity leads to the release of pro-inflammatory cytokines and growth factors that promote prostate cell proliferation and fibrosis, further contributing to prostate enlargement and the worsening of LUTS.
3. **Interaction Between Hypertension and α-Adrenergic Receptor Activity**: The overactivity of the sympathetic nervous system in the context of MetS increases α-adrenergic receptor activity, which has a direct impact on smooth muscle contraction in the prostate. α1-adrenergic receptor antagonists, such as tamsulosin, are commonly used in the treatment of BPH, demonstrating the critical role of adrenergic receptors in prostate physiology. In hypertensive individuals with MetS, the heightened adrenergic stimulation may result in a more pronounced response to α-adrenergic signaling, leading to increased smooth muscle tone and exacerbating the symptoms of BPH.
4. **Exacerbation of LUTS**: The combined effects of hypertension-induced smooth muscle contraction, increased vascular tone, and overactive sympathetic nervous system can lead to a vicious cycle of BPH progression and worsening LUTS. As the prostate enlarges and smooth muscle tone increases, the bladder becomes less efficient in emptying, leading to urinary retention and obstruction. The increased α-adrenergic activity in the prostate further complicates this process by promoting further contraction of the prostatic smooth muscle, worsening urinary flow and increasing the severity of LUTS.

Hypertension in MetS contributes to BPH and LUTS through both vascular mechanisms, such as increased smooth muscle tone and altered blood flow, and neural mechanisms involving excessive sympathetic activation and α-adrenergic receptor stimulation. These pathways highlight the interrelationship between MetS components and their impact on prostate health, offering potential therapeutic targets for managing both hypertension and BPH in affected individuals.

Dyslipidemia and Oxidative Stress: Dyslipidemia, characterized by abnormal lipid levels in the blood, is a hallmark of metabolic syndrome (MetS) and significantly contributes to various pathophysiological processes. In MetS, the imbalance in lipid profiles, particularly elevated triglycerides, decreased high-density lipoprotein (HDL) cholesterol, and increased low-density lipoprotein (LDL) cholesterol, creates a pro-inflammatory and pro-oxidant state that has widespread effects on tissues, including the prostate[26, 27].

1. **Elevated Triglycerides and Low HDL Cholesterol:** Increased triglycerides and low HDL cholesterol are often linked with insulin resistance, obesity, and central adiposity, all of which are core features of MetS. These lipid abnormalities promote endothelial dysfunction and contribute to atherosclerosis, which impairs vascular health. The diminished HDL levels further reduce the body's ability to clear

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

excess cholesterol from peripheral tissues, allowing the accumulation of oxidized LDL. This process creates a cycle of inflammation and oxidative damage, which plays a crucial role in the progression of various diseases, including prostate conditions.

2. **Increased LDL Cholesterol and Oxidative Stress:** Elevated LDL cholesterol, particularly when oxidized, becomes a potent contributor to oxidative stress. Oxidized LDL particles are known to trigger the activation of pro-inflammatory signaling pathways, leading to the release of cytokines and reactive oxygen species (ROS) that damage tissue. In the prostate, oxidative stress can induce DNA damage, apoptosis, and fibrosis, exacerbating the inflammatory environment within the tissue. Chronic oxidative stress is believed to contribute to the development of benign prostatic hyperplasia (BPH) and prostate cancer, as it can promote cellular mutations and the activation of growth pathways.
3. **Inflammation and Prostate Cell Proliferation:** The inflammatory environment created by dyslipidemia can promote prostate cell proliferation by upregulating growth factors such as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and insulin-like growth factors (IGFs). These factors stimulate angiogenesis, fibrosis, and tissue remodeling, which are critical components of both BPH and prostate cancer progression. The increase in inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukins (IL-6, IL-1) further accelerates cell division, creating a fertile ground for abnormal prostate cell growth.
4. **Role of Growth Factors and Enzymes in Prostate Enlargement:** Dyslipidemia can influence the expression of enzymes and growth factors involved in prostate enlargement. For instance, dysregulated lipid metabolism has been shown to upregulate the expression of 5-alpha-reductase, an enzyme responsible for converting testosterone into its more potent form, dihydrotestosterone (DHT). Elevated DHT levels are closely associated with BPH and prostate enlargement. Furthermore, lipids and their metabolites can influence the activity of matrix metalloproteinases (MMPs), enzymes involved in the degradation of extracellular matrix components, which is a key factor in tissue remodeling and enlargement of the prostate gland.
5. **Impact on Prostate Cancer Development:** In addition to BPH, dyslipidemia plays a significant role in the development and progression of prostate cancer. The chronic inflammation associated with dyslipidemia can promote genomic instability and activate oncogenic signaling pathways, contributing to tumor initiation and progression. For example, increased LDL and oxidized lipids can upregulate pathways involving the nuclear factor kappa B (NF-κB), a key regulator of inflammation and cell survival, which is frequently implicated in cancer cell proliferation and resistance to apoptosis. Moreover, lipid metabolism dysregulation can influence cancer cell metabolism and energy production, enhancing tumor growth and metastatic potential.

Dyslipidemia, by altering lipid metabolism and inducing oxidative stress and inflammation, is a key factor in the pathogenesis of prostate diseases, including BPH and prostate cancer. Its effects on lipid profiles, growth factor expression, and enzyme activity create a conducive environment for prostate cell proliferation, tissue remodeling, and cancer progression. Thus, managing dyslipidemia in patients with MetS could potentially reduce the risk of prostate enlargement and malignancy, highlighting the need for targeted therapeutic interventions aimed at restoring lipid balance and reducing oxidative damage[27].

Inflammation: Chronic low-grade inflammation is increasingly recognized as a central component in the pathophysiology of Metabolic Syndrome (MetS) and its association with benign prostatic hyperplasia (BPH). MetS, a cluster of conditions including obesity, dyslipidemia, insulin resistance, and hypertension, creates an inflammatory environment that contributes to the development and progression of various chronic diseases, including BPH[28].

Inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) are pivotal in this inflammatory cascade. These cytokines are found to be elevated in individuals with MetS and are involved in activating several molecular pathways that influence prostate growth and pathology. TNF-α, a pro-inflammatory cytokine, has been shown to stimulate the androgenic pathway by increasing the expression of androgen receptors in prostate cells. Androgens, particularly dihydrotestosterone (DHT), play a critical role in the normal growth and maintenance of the prostate. When this pathway is dysregulated by inflammatory mediators, it can lead to excessive prostate growth, a hallmark of BPH. IL-6, another key cytokine elevated in MetS, not only exacerbates systemic inflammation but also directly impacts prostate tissue. It is known to activate the Janus kinase-signal transducer and activator of transcription (JAK-STAT) pathway, which leads to the activation of genes that promote cell proliferation and survival. This effect may further contribute to the hyperplasia of prostate tissue, which is characteristic of BPH.

Beyond stimulating cell growth, the chronic inflammatory milieu in the prostate can also lead to fibrosis, a critical feature in advanced stages of BPH. Inflammatory cytokines like TNF-α and IL-6 stimulate the production of collagen and extracellular matrix components through the activation of fibroblasts[29]. This fibrosis can impair normal tissue function, leading to the stiffening and scarring of the prostate. As the prostate

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

undergoes fibrotic remodeling, its normal architecture is disrupted, leading to further enlargement and dysfunction, which can exacerbate urinary symptoms in BPH patients. In addition, the chronic low-grade inflammation associated with MetS can promote oxidative stress, which damages cellular components, leading to further inflammatory responses and tissue remodeling. This chronic cycle of inflammation, tissue damage, and fibrosis creates a vicious cycle that accelerates the progression of BPH, ultimately affecting prostate function and contributing to lower urinary tract symptoms[30].

The interplay between chronic low-grade inflammation in MetS and the development of BPH is complex, involving elevated inflammatory cytokines like TNF-α and IL-6. These cytokines stimulate the androgenic pathway, promote prostate growth, and contribute to fibrosis. The ongoing inflammatory environment may not only lead to the enlargement of the prostate but also result in the progression of BPH, causing long-term health impacts and significant clinical concerns.

Clinical Implications

1. **Diagnosis:** The association between MetS and BPH may have important diagnostic implications. Patients presenting with LUTS and a known history of MetS may require closer monitoring and early intervention. Identifying the metabolic abnormalities that coexist with BPH can help clinicians better assess the severity of the condition and tailor treatment strategies.
2. **Treatment Strategies:** Treatment of BPH often includes medications such as α-blockers, 5α-reductase inhibitors, and, in severe cases, surgery. However, addressing the underlying metabolic dysfunction in patients with MetS may improve outcomes. For example, lifestyle modifications such as weight reduction, dietary changes, and exercise may alleviate symptoms of both MetS and BPH. Pharmacologic interventions targeting insulin resistance, obesity, and inflammation could represent promising therapeutic options for managing BPH in patients with MetS.
3. **Prevention:** Preventive strategies aimed at reducing the risk of MetS may also help prevent the development or progression of BPH. Public health campaigns focused on healthy eating, regular physical activity, and the management of cardiovascular risk factors could play a significant role in reducing the burden of both MetS and BPH.

CONCLUSION

The growing body of evidence suggesting a link between MetS and BPH highlights the need for a holistic approach to managing these conditions in aging men. Addressing metabolic dysfunction, particularly insulin resistance, obesity, and inflammation, may not only help prevent or delay the onset of BPH but also improve overall prostate health. Future studies should further elucidate the complex interactions between MetS components and prostate pathophysiology, paving the way for more effective treatments and preventive strategies for BPH in this high-risk population.

REFERENCES

1. Udensi, U.K., Tchounwou, P.B.: Oxidative stress in prostate hyperplasia and carcinogenesis. J Exp Clin Cancer Res. 35, 139 (2016). https://doi.org/10.1186/s13046-016-0418-8
2. Bortnick, E., Brown, C., Simma-Chiang, V., Kaplan, S.A.: Modern best practice in the management of benign prostatic hyperplasia in the elderly. Therapeutic Advances in Urology. 12, 1756287220929486 (2020). https://doi.org/10.1177/1756287220929486
3. Chen, B., Cao, D., Chen, Z., Huang, Y., Lin, T., Ai, J., Liu, L., Wei, Q.: Estrogen regulates the proliferation and inflammatory expression of primary stromal cell in benign prostatic hyperplasia. Translational Andrology and Urology. 9, 32231–32331 (2020). https://doi.org/10.21037/tau.2020.02.08
4. 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
5. 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., Chinedum, K.E., Agu, P., Nwobodo, V.: In Vivo and in Silico Assessment of Ameliorative Effects of Xylopia aethiopica on Testosterone Propionate-Induced Benign Prostatic Hyperplasia. Pharmaceutical Fronts. 05, e64–e76 (2023). https://doi.org/10.1055/s-0043-1768477
6. Franco, J.V.A., Tesolin, P., Jung, J.H.: Update on the management of benign prostatic hyperplasia and the role of minimally invasive procedures. Prostate International. 11, 1–7 (2023). https://doi.org/10.1016/j.prnil.2023.01.002
7. Djavan, B., Kazzazi, A., Bostanci, Y.: Revival of thermotherapy for benign prostatic hyperplasia. Current Opinion in Urology. 22, 16–21 (2012). https://doi.org/10.1097/MOU.0b013e32834d5469
8. Bawa, I., Uti, D.E., Itodo, M.O., Umoru, G.U., Zakari, S., Obeten, U.N.: Effect of Solvent Extracts of Tephrosia vogelii Leaves and Stem on Lipid Profile of Poloxamer 407-Induced Hyperlipidemic Rats.

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

Ibnosina Journal of Medicine and Biomedical Sciences. 14, 135–144 (2023). https://doi.org/10.1055/s-0042-1760223

9. Umoru, G.U., Atangwho, I.J., David-Oku, E., Uti, D.E., Agwupuye, E.I., Obeten, U.N., Maitra, S., Subramaniyan, V., Wong, L.S., Aljarba, N.H., Kumarasamy, V.: Tetracarpidium conophorum nuts (African walnuts) up-regulated adiponectin and PPAR-γ expressions with reciprocal suppression of TNF-α gene in obesity. Journal of Cellular and Molecular Medicine. 28, e70086 (2024). https://doi.org/10.1111/jcmm.70086
10. Mohamed, S.M., Shalaby, M.A., El-Shiekh, R.A., El-Banna, H.A., Emam, S.R., Bakr, A.F.: Metabolic syndrome: risk factors, diagnosis, pathogenesis, and management with natural approaches. Food Chemistry Advances. 3, 100335 (2023). https://doi.org/10.1016/j.focha.2023.100335
11. Ali, N., Samadder, M., Shourove, J.H., Taher, A., Islam, F.: Prevalence and factors associated with metabolic syndrome in university students and academic staff in Bangladesh. Sci Rep. 13, 19912 (2023). https://doi.org/10.1038/s41598-023-46943-x
12. Kopp, W.: Diet-Induced Hyperinsulinemia as a Key Factor in the Etiology of Both Benign Prostatic Hyperplasia and Essential Hypertension? Nutr Metab Insights. 11, 1178638818773072 (2018). https://doi.org/10.1177/1178638818773072
13. Rył, A., Rotter, I., Miazgowski, T., Słojewski, M., Dołęgowska, B., Lubkowska, A., Laszczyńska, M.: Metabolic syndrome and benign prostatic hyperplasia: association or coincidence? Diabetology & Metabolic Syndrome. 7, 94 (2015). https://doi.org/10.1186/s13098-015-0089-1
14. Rajpathak, S.N., Gunter, M.J., Wylie-Rosett, J., Ho, G.Y.F., Kaplan, R.C., Muzumdar, R., Rohan, T.E., Strickler, H.D.: The role of insulin-like growth factor-I and its binding proteins in glucose homeostasis and type 2 diabetes. Diabetes Metab Res Rev. 25, 3–12 (2009). https://doi.org/10.1002/dmrr.919
15. Al-Samerria, S., Radovick, S.: The Role of Insulin-like Growth Factor-1 (IGF-1) in the Control of Neuroendocrine Regulation of Growth. Cells. 10, 2664 (2021). https://doi.org/10.3390/cells10102664
16. Kojta, I., Chacińska, M., Błachnio-Zabielska, A.: Obesity, Bioactive Lipids, and Adipose Tissue Inflammation in Insulin Resistance. Nutrients. 12, 1305 (2020). https://doi.org/10.3390/nu12051305
17. Hardy, O.T., Czech, M.P., Corvera, S.: What causes the insulin resistance underlying obesity? Curr Opin Endocrinol Diabetes Obes. 19, 81–87 (2012). https://doi.org/10.1097/MED.0b013e3283514e13
18. AlZaim, I., Al-Saidi, A., Hammoud, S.H., Darwiche, N., Al-Dhaheri, Y., Eid, A.H., El-Yazbi, A.F.: Thromboinflammatory Processes at the Nexus of Metabolic Dysfunction and Prostate Cancer: The Emerging Role of Periprostatic Adipose Tissue. Cancers. 14, 1679 (2022). https://doi.org/10.3390/cancers14071679
19. Uti, D.E., Atangwho, I.J., Eyong, E.U., Umoru, G.U., Egbung, G.E., Rotimi, S.O., Nna, V.U.: African Walnuts (Tetracarpidium conophorum) Modulate Hepatic Lipid Accumulation in Obesity via Reciprocal Actions on HMG-CoA Reductase and Paraoxonase. Endocrine, Metabolic & Immune Disorders - Drug Targets(Formerly Current Drug Targets - Immune, Endocrine & Metabolic Disorders). 20, 365–379 (2020). https://doi.org/10.2174/1871530319666190724114729
20. Al-hussaniy, H.A., Alburghaif, A.H., Naji, M.A.: Leptin hormone and its effectiveness in reproduction, metabolism, immunity, diabetes, hopes and ambitions. J Med Life. 14, 600–605 (2021). https://doi.org/10.25122/jml-2021-0153
21. Karnati, H.K., Panigrahi, M.K., Li, Y., Tweedie, D., Greig, N.H.: Adiponectin as a Potential Therapeutic Target for Prostate Cancer. Curr Pharm Des. 23, 4170–4179 (2017). https://doi.org/10.2174/1381612823666170208123553
22. Carlson, N.G., Wieggel, W.A., Chen, J., Bacchi, A., Rogers, S.W., Gahring, L.C.: Inflammatory cytokines IL-1 alpha, IL-1 beta, IL-6, and TNF-alpha impart neuroprotection to an excitotoxin through distinct pathways. J Immunol. 163, 3963–3968 (1999)
23. Gorabi, A.M., Razi, B., Aslani, S., Abbasifard, M., Imani, D., Sathyapalan, T., Sahebkar, A.: Effect of curcumin on proinflammatory cytokines: A meta-analysis of randomized controlled trials. Cytokine. 143, 155541 (2021). https://doi.org/10.1016/j.cyto.2021.155541
24. 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
25. Kopp, W.: Diet-Induced Hyperinsulinemia as a Key Factor in the Etiology of Both Benign Prostatic Hyperplasia and Essential Hypertension? Nutr Metab Insights. 11, 1178638818773072 (2018). https://doi.org/10.1177/1178638818773072
26. Calogero, A.E., Burgio, G., Condorelli, R.A., Cannarella, R., Vignera, S.L.: Epidemiology and risk factors of lower urinary tract symptoms/benign prostatic hyperplasia and erectile dysfunction. The Aging Male. (2019)

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

27. Long, Y., Mao, C., Liu, S., Tao, Y., Xiao, D.: Epigenetic modifications in obesity-associated diseases. MedComm. 5, e496 (2024). https://doi.org/10.1002/mco2.496
28. Zhao, S.-C., Xia, M., Tang, J.-C., Yan, Y.: Associations between metabolic syndrome and clinical benign prostatic hyperplasia in a northern urban Han Chinese population: A prospective cohort study. Sci Rep. 6, 33933 (2016). https://doi.org/10.1038/srep33933
29. Silver, S.V., Tucker, K.J., Vickman, R.E., Lanman, N.A., Semmes, O.J., Alvarez, N.S., Popovics, P.: Characterization of prostate macrophage heterogeneity, foam cell markers, and CXCL17 upregulation in a mouse model of steroid hormone imbalance. Sci Rep. 14, 21029 (2024). https://doi.org/10.1038/s41598-024-71137-4
30. Silver, S.V., Popovics, P.: The Multifaceted Role of Osteopontin in Prostate Pathologies. Biomedicines. 11, 2895 (2023). https://doi.org/10.3390/biomedicines11112895

CITE AS: Omeye Francis I. (2025). Association between Metabolic Syndrome and Benign Prostatic Hyperplasia. NEWPORT INTERNATIONAL JOURNAL OF PUBLIC HEALTH AND PHARMACY, 6(1):134-140. https://doi.org/10.59298/NIJPP/2025/61134140