Periprostatic fat thickness measured on MRI correlates with lower urinary tract symptoms, erectile function, and benign prostatic hyperplasia progression

Bo Zhang, Xiang Chen, Yu-Hang Liu, Yu Gan, Pei-Hua Liu, Zhi Chen, Wei-Ping Xia, Guo-Yu Dai, Feng Ru, Ze-Xiang Jiang, Yao He

This study investigated the correlation between periprostatic fat thickness (PPFT) measured on magnetic resonance imaging and lower urinary tract symptoms, erectile function, and benign prostatic hyperplasia (BPH) progression. A total of 286 treatment-naive men diagnosed with BPH in our department between March 2017 and February 2019 were included. Patients were divided into two groups according to the median value of PPFT: high (PPFT >4.35 mm) PPFT group and low (PPFT <4.35 mm) PPFT group. After the initial evaluation, all patients received a combination drug treatment of tamsulosin and finasteride for 12 months. Of the 286 enrolled patients, 244 completed the drug treatment course. Patients with high PPFT had larger prostate volume (PV; \( P = 0.013 \)), higher International Prostate Symptom Score (IPSS; \( P = 0.008 \)), and lower five-item version of the International Index of Erectile Function (IIEF-5) score \( P = 0.002 \) than those with low PPFT. Both high and low PPFT groups showed significant improvements in PV, maximum flow rate, IPSS, and quality of life score and a decrease of IIEF-5 score after the combination drug treatment. The decrease of IIEF-5 score was more obvious in the high PPFT group than that in the low PPFT group. In addition, more patients in the high PPFT group underwent prostate surgery than those in the low PPFT group. Moreover, Pearson’s correlation coefficient analysis indicated that PPFT was positively correlated with age, PV, and IPSS and negatively correlated with IIEF-5 score; however, body mass index was only negatively correlated with IIEF-5 score.

Asian Journal of Andrology (2021) 23, 80–84; doi: 10.4103/aja.aja_51_20; published online: 25 August 2020

Keywords: benign prostatic hyperplasia; clinical progression; erectile function; lower urinary tract symptoms; periprostatic fat thickness

INTRODUCTION

Lower urinary tract symptoms (LUTS) and erectile dysfunction (ED) caused by benign prostatic hyperplasia (BPH) are common problems in middle-aged and elderly men that can greatly affect their quality of life. A plethora of evidence has suggested that obesity is associated with the severity of LUTS and ED in patients with BPH.\(^1\)\(^-\)\(^3\) The visceral fat tissue secretes various bioactive substances, which can induce inflammatory responses, and is reportedly associated with various benign and malignant diseases.\(^4\)\(^-\)\(^6\)

Periprostatic fat (PPF), which surrounds the prostate, can produce several cytokines and hormones involved in autocrine, paracrine, and endocrine signaling pathways, such as vascular endothelial growth factor, interleukin-1\(\beta\), interleukin-6, adiponectin, and leptin.\(^7\)\(^-\)\(^9\)

To date, most studies on PPF have focused on uncovering its significance in the context of prostatic cancer (PCa). Several clinical studies have shown that PPF thickness (PPFT) correlates with the tumorigenesis and tumor progression of PCa.\(^6\)\(^-\)\(^8\)\(^-\)\(^13\) Cao et al.\(^12\) demonstrated that PPFT measured on magnetic resonance imaging (MRI) was an independent predictor of the development of PCa and high-grade PCa. Huang et al.\(^13\) reported that PPFT is a readily measurable and independent risk factor for castration-resistant prostate cancer in patients with PCa treated with androgen deprivation therapy. Moreover, Ribeiro et al.\(^8\) performed a global gene expression profiling of PPF tissue and showed that the overexpression of the genes leptin (LEP) and angiopoietin 1 (ANGPT1) could contribute to PCa progression. However, the association between PPF and the severity of LUTS and ED in patients with BPH remains elusive. Therefore, we aimed to elucidate whether PPF was associated with the severity of LUTS and ED and could serve as a predictor of clinical progression in patients with BPH.

We investigated the association between PPF measurements and both the clinical data of patients with BPH and the efficacy of medical therapy. In addition, we compared the effects of PPFT and body mass index (BMI) on prostate volume (PV) and the severity of LUTS and ED in patients with BPH. To the best of our knowledge, no previous studies have performed such evaluations.
PATIENTS AND METHODS
Ethics statement
This study was a single-center, retrospective study of a prospectively collected database and conducted in accordance with the Good Clinical Practice and ethical principles outlined in the Declaration of Helsinki. Before initiating this study, we obtained approval (approval number: 201703545) from the Ethics Committee of the Xiangya Hospital of Central South University, Changsha, China, and written informed consent from the participating patients.

Patient selection
A total of 286 treatment-naive men who were diagnosed with BPH at Xiangya Hospital of Central South University between March 2017 and February 2019 were included in this study. The inclusion criteria were as follows: prostate-specific antigen (PSA) <4 ng ml⁻¹, total International Prostate Symptom Score (IPSS) ≥8, IPSS-quality of life (QoL) score ≥3, PV ≥30 ml, maximum urinary flow rate (Qmax) <15 ml s⁻¹ with voided volume ≥100 ml, and age ≥50 years. The exclusion criteria were as follows: previous medical treatment for LUTS or ED, history of preceding prostate surgery, PCa, bladder cancer, bladder stones, urethral stricture, LUTS due to urinary tract infection, neurogenic bladder dysfunction, severe cardiac disease, renal dysfunction, and hepatic dysfunction.

Data collection
Data on age, BMI, serum PSA level, PV, Qmax, IPSS, QoL score, and International Index of Erectile Function (IIEF-5) score were collected prospectively. PPFT, which is defined as the shortest perpendicular distance between the pubic symphysis and prostate on the midsagittal plane, was measured by MRI in each patient. As no previous studies have characterized the relative PPFT, we divided the patients into two groups according to the median value of PPFT: the high (PPFT >4.35 mm) PPFT group and low (PPFT <4.35 mm) PPFT group. After the initial evaluation, all patients received a combination of the α₁-blocker tamsulosin (0.2 mg per day; Astellas, Shenyang, China) and 5-alpha-reductase inhibitor finasteride (5 mg per day; Merck Sharp and Dohme Limited, Hangzhou, China) for 12 months. Of the 286 enrolled patients, 244 patients completed the drug treatment course, and 42 patients discontinued the drug course due to the following reasons: 27 patients were lost in the follow-up process, 9 patients had adverse drug reactions, and 6 patients underwent prostate surgery.

RESULTS
Patient demographics and baseline characteristics
A total of 286 patients met the inclusion criteria and were enrolled in this study. The age (mean ± s.d.) and BMI (mean ± s.d.) were 60.0 ± 6.3 years and 24.8 ± 3.3 kg m⁻², respectively. Based on the median value of PPFT (4.35 mm), the high (PPFT >4.35 mm) and low (PPFT <4.35 mm) PPFT groups included 143 patients each. The demographic and baseline clinical characteristics of these two groups are summarized in Table 1. No significant difference was detected in age, BMI, total serum PSA, Qmax, and QoL score between these two groups. However, the IPSS (P = 0.008) and PV (P = 0.013) were significantly higher in the high PPFT group than those in the low PPFT group. Conversely, the IIEF-5 score (P = 0.002) was lower in the high PPFT group than that in the low PPFT group.

Table 1: Patients’ baseline characteristics

| Variable | Low PPFT group | High PPFT group | P     |
|----------|----------------|-----------------|-------|
| Patient (n) | 143 | 143 | 0.105 |
| Age (year) | 59.4±6.5 | 60.6±6.0 | 0.105 |
| BMI (kg m⁻²) | 24.6±3.4 | 24.9±3.3 | 0.448 |
| Total PSA (ng ml⁻¹) | 2.2±1.0 | 2.3±0.9 | 0.424 |
| PV (ml) | 47.9±19.8 | 54.2±22.7 | 0.013 |
| Qmax (ml s⁻¹) | 9.2±2.9 | 9.0±2.9 | 0.413 |
| IPSS | 17.2±6.0 | 19.1±5.9 | 0.008 |
| QoL score | 4.4±0.9 | 4.6±0.9 | 0.125 |
| IIEF-5 score | 18.1±4.0 | 16.5±4.8 | 0.002 |

PPFT: periprostatic fat thickness; BMI: body mass index; PSA: prostate specific antigen; PV: prostate volume; Qmax: maximum flow rate; IPSS: International Prostate Symptom Score; QoL: quality of life; IIEF-5: International Index of Erectile Function-5.
Changes in the clinical data for patients of two groups after combination therapy

Of the total 286 patients, 244 patients (126 included in the low and 118 included in the high PPFT group) completed the combination therapy of tamsulosin and finasteride for 12 months (Figure 2). In total, 42 patients discontinued the trial. Of the 25 patients in the high PPFT group who discontinued the trial, 15 patients were lost to follow-up; 4 patients discontinued due to adverse drug reactions, and 6 patients underwent prostate surgery. Similarly, of the 17 patients in the low PPFT group who discontinued the trial, 12 patients were lost to follow-up, and 5 patients discontinued due to adverse drug reactions. No patients in the low PPFT group required surgical intervention. Of the 6 patients who underwent prostate surgery in the high PPFT group, 5 patients developed acute urinary retention, and 1 patient developed secondary bladder stones during the 12-month follow-up. The Chi-square test demonstrated that significantly greater number of patients within the high PPFT group underwent prostate surgery than those patients in the low PPFT group (4.2% vs 0.0%, P = 0.013). However, there was no significant difference in the incidence of adverse drug reactions between these two groups (2.8% vs 3.5%, P = 0.735; Figure 2).

Changes in PV, Qmax, IPSS, QoL score, and IIEF-5 score in low and high PPFT group patients postcombination therapy (12 months) are summarized in Table 2. Both groups showed significant improvements in PV, Qmax, IPSS, and QoL score and a decrease in IIEF-5 score after 12 months of combination therapy. No significant difference was detected in Qmax, IPSS, and QoL score between the two groups after 12 months of combination therapy. PV (P = 0.049) was significantly higher in the high PPFT group than that in the low PPFT group. IIEF-5 score (P < 0.001) was significantly lower in the high PPFT group than that in the low PPFT group. Changes in PV, Qmax, IPSS, and QoL scores before and after treatment were not significantly different between the two groups, although the baseline PV and IPSS were significantly higher in the high PPFT patients than those in the low PPFT patients. Interestingly, worsening of IIEF-5 score (P < 0.001) was significantly more severe in the patients with high PPFT than that in the patients with low PPFT.

Correlations between PPFT and other study parameters

The correlations between PPFT, BMI, and other clinical data were evaluated by calculating the Pearson’s correlation coefficient. The results indicated that PPFT had a positive correlation with age (r = 0.185, P = 0.002), PV (r = 0.157, P = 0.008), and IPSS (r = 0.351, P < 0.001) and a negative correlation with the IIEF-5 score (r = −0.294, P < 0.001; Table 3). Unlike PPFT, the BMI was significantly negatively correlated only with the IIEF-5 score (r = −0.169, P = 0.004; Table 4). Interestingly, there was no significant correlation between PPFT and BMI (r = 0.040, P = 0.506).

DISCUSSION

LUTS and ED caused by BPH are very common health problems in aging men that require the use of expensive medical resources and have attracted worldwide attention. Moreover, overweight and
Obesity has become a serious public health concern in most developing countries, especially in China. Many cohort and observational studies have reported that obesity is associated with LUTS and ED.\textsuperscript{1-3} BMI is the most used surrogate marker of obesity; however, BMI does not accurately reflect metabolically active visceral fat distribution. In the peri-prostatic and pelvic cavity fat distribution. Therefore, the relationship between BMI and the clinical progression of BPH remains elusive.\textsuperscript{16-19}

Visceral fat tissue, which is a metabolically active endocrine organ, is reportedly associated with various benign and malignant diseases. Huang et al.\textsuperscript{20} found that high perirenal fat thickness was an independent predictor of tumor progression in localized clear-cell renal cell carcinoma. In a retrospective review of 250 patients with LUTS associated with BPH, Motoya et al.\textsuperscript{21} found that the visceral fat localization significantly and positively correlated with storage symptoms. The relationships between PPF and prostate size and an increased risk of BPH. The authors suggested that the adiponectin signaling could act as a negative regulator of BPH development via inhibition of extracellular signal-regulated kinase (ERK)-mediated cell proliferation. Similarly, Nandeesha et al.\textsuperscript{22} demonstrated that adiponectin could act as a protective regulator of BPH development and progression through its multifunctional effects including antiproliferation, apoptosis induction, and blocking of G1/S-phase progression. Thus, PPF could theoretically act as a promising surrogate for BMI in evaluating the association between obesity and BPH development and progression.

Our study is the first to evaluate the association between PPF measured on MRI and the severity of LUTS and ED and the efficacy of medical therapy in patients with BPH. In this study, the high PPF patients had larger PV, higher IPSS, and lower IIEF-5 score than the low PPF patients. Both high and low PPF groups showed significant improvements in PV, Qmax, IPSS, and QoL score and a decrease in the IIEF-5 score from the baseline after 12 months of combination therapy with finasteride and tamsulosin. These results are in line with data published previously.\textsuperscript{23} As noted elsewhere, the undesirable sexual side effects of finasteride can negatively impact the IIEF-5 score.\textsuperscript{24} Interestingly, we found that patients with BPH and high PPF may have more severe ED and undesirable sexual side effects than patients with BPH and low PPF. As a result, the combination therapy for patients with BPH and high PPF should include tadalafil, which is often used to treat ED in men.\textsuperscript{25} In addition, the data suggest that the clinical progression was more rapid in patients with BPH with high PPF than in patients with BPH with low PPF.

PPF positively correlated with age, PV, and IPSS and negatively correlated with the IIEF-5 score. Unlike PPF, BMI was negatively correlated only with the IIEF-5 score. Thus, PPF may be superior to BMI at identifying patients with BPH who are at high risk of LUTS and ED. PPFA, periprostatic fat area (PPFA), and periprostatic fat volume (PPFV), which are measured on MRI, are frequently used to measure PPF. Although 3.0-T MRI has been widely used at the second and third level medical centers in China, the special radiologic imaging tool required to calculate PPFA and PPFV from MRI data is costly and thus, available in only a few institutions. Huang et al.\textsuperscript{26} reported significant positive correlations between PPF and PPFA (correlation coefficient = 0.939) and PPFV and IPSS (correlation coefficient = 0.825). Therefore, PPF is a readily measurable and reliable surrogate for PPFA or PPFV in predicting BPH development and progression.

An intriguing hypothesis is that adipokines secreted by PPF are involved in the inflammatory response and promote BPH progression. Powell\textsuperscript{27} reported that there are two kinds of adipocytes, "fat" and "thin." The activated "fat" adipocytes produce more adipokines than thin adipocytes, which are involved in the inflammatory response, in obese populations. Fu et al.\textsuperscript{28} analyzed data from 98 Chinese men (48 patients with BPH and 50 healthy individuals) and found that lower serum adiponectin levels were associated with a larger prostate size and an increased risk of BPH. The authors suggested that the adiponectin signaling could act as a negative regulator of BPH development via inhibition of extracellular signal-regulated kinase (ERK)-mediated cell proliferation. Similarly, Nandeesha et al.\textsuperscript{21} reported that adiponectin is reduced in patients with BPH, and there was a negative correlation between adiponectin and prostate volume. Zhang et al.\textsuperscript{29} mentioned that adipokines of PPF are the major secretors of interleukin-6 (IL-6) and leptin, and PPF was associated with IL-6 and leptin. Our study provides a platform for future studies to investigate the association between PPF and BPH development at the molecular level. Understanding such molecular mechanisms could lead to the development of novel diagnostic procedures and therapeutic interventions for patients with BPH.

Although our research has yielded some important findings, this study has several limitations. First, this study was a single-center cohort study with a limited sample size. Second, although PPF positively correlated with age, PV, and IPSS and negatively correlated with the IIEF-5 score, some correlations were weak. Therefore,

---

### Table 3: Correlation between the periprostatic fat thickness (PPFT) and clinical data

| Variable          | Pearson’s correlation coefficient | P     |
|-------------------|----------------------------------|-------|
| Age (year)        | 0.185                            | 0.002 |
| BMI (kg m\(^{-2}\)) | 0.040                            | 0.506 |
| Total PSA (ng ml\(^{-1}\)) | 0.108                           | 0.069 |
| PV (ml)           | 0.157                            | 0.008 |
| Qmax (ml s\(^{-1}\)) | −0.089                          | 0.133 |
| IPSS              | 0.351                            | <0.001|
| QoL score         | 0.103                            | 0.082 |
| IIEF-5 score      | −0.294                           | <0.001|

BMI: body mass index; PSA: prostate-specific antigen; Qmax: maximum flow rate; PV: prostate volume; IPSS: International Prostate Symptom Score; QoL: quality of life; IIEF-5: International Index of Erectile Function-5

### Table 4: Correlation between the body mass index and clinical data

| Variable          | Pearson’s correlation coefficient | P     |
|-------------------|----------------------------------|-------|
| Age (year)        | 0.051                            | 0.387 |
| PPFT (kg m\(^{-2}\)) | 0.040                           | 0.506 |
| Total PSA (ng ml\(^{-1}\)) | 0.078                           | 0.186 |
| PV (ml)           | 0.051                            | 0.391 |
| Qmax (ml s\(^{-1}\)) | −0.085                          | 0.153 |
| IPSS              | 0.093                            | 0.116 |
| QoL score         | 0.021                            | 0.730 |
| IIEF-5 score      | −0.169                           | 0.004 |

PPFT: periprostatic fat thickness; PSA: prostate-specific antigen; Qmax: maximum flow rate; PV: prostate volume; IPSS: International Prostate Symptom Score; QoL: quality of life; IIEF-5: International Index of Erectile Function-5
a multicenter study with a larger cohort should be performed to validate our present findings. Third, all participants were ethnically of Chinese origin with relatively low BMI. Thus, our results should be validated in other ethnic groups. Fourth, the follow-up period of our study was only 12 months. Pharmacotherapy for LUTS and ED should generally be continued for a much longer period. Finally, we did not adjust our data for additional parameters, such as lifestyle (active vs sedentary), habits (e.g., smoking and drinking), and comorbidities (e.g., hypertension and diabetes), which may influence the progression of LUTS and ED or alter efficacy outcomes following medical therapy.

CONCLUSION

Patients with high PPFT have larger PV, higher IPSS, lower IIEF-5 score, more frequent adverse sexual side effects, and a higher incidence of metabolic syndrome. PPFT significantly correlates with PV, LUTS, and ED; however, BMI only significantly correlates with the IIEF-5 score. Collectively, our data suggest that PPFT measured on MRI correlates with LUTS and erectile function, and PPFT could be a convenient indicator for predicting the progression of BPH.

AUTHOR CONTRIBUTIONS

YH, BZ, and XC contributed to the clinical trial design, data acquisition, and data interpretation. YH, BZ, YG, PHL, WPX, FR, and ZXJ performed the study. YHL, ZC, and GYD contributed to data acquisition. YH and BZ drafted the manuscript and contributed to the critical revision of the manuscript. All authors read and approved the final manuscript.

COMPETING INTERESTS

All authors declared no competing interests.

ACKNOWLEDGMENTS

This work was supported by grant from the National Natural Science Foundation of China (No. 81700663) to YH, and the Fundamental Research Funds for the Central Universities of Central South University to BZ.

REFERENCES

1. Gacci M, Sebastiani A, Salvi M, De Nunzio C, Tubaro A, et al. Central obesity is predictive of persistent storage lower urinary tract symptoms (LUTS) after surgery for benign prostatic enlargement: results of a multicentre prospective study. BJU Int 2015; 116: 271–7.
2. Corona G, Vignozzi L, Rastrelli G, Lotti F, Cipriani S, et al. Benign prostatic hyperplasia: a new metabolic disease of the aging male and its correlation with sexual dysfunctions. Int J Endocrinol 2014; 2014: 329456.
3. Calogero AE, Burgio G, Condorelli RA, Cannarella R, La Vignera S. Epidemiology and risk factors of lower urinary tract symptoms/benign prostatic hyperplasia and erectile dysfunction. Aging Male 2019; 22: 12–9.
4. Gacci M, Vignozzi L, Sebastiani A, Salvi M, Giannessi C, et al. Metabolic syndrome and lower urinary tract symptoms: the role of inflammation. Prostate Cancer Prostatic Dis 2013; 16: 101–6.
5. Russo Gi, Cimino S, Castelli T, Favilla V, Gacci M, et al. Benign prostatic hyperplasia, metabolic syndrome and non-alcoholic fatty liver disease: is metaflammation the link? Prostate 2016; 76: 1528–35.
6. Silva A, Faria G, Araujo A, Monteiro MP. Impact of adiposity on staging and prognosis of colorectal cancer. Crit Rev Oncol Hematol 2020; 145: 102957.
7. Laurent V, Guerard A, Mazeroles C, Le Gonidec S, Toulet A, et al. Periprostatic adipocytes act as a driving force for prostate cancer progression in obesity. Nat Commun 2016; 7: 10230.
8. Ribeiro R, Monteiro C, Catalan V, Hu P, Cunha V, et al. Obesity and prostate cancer: gene expression signature of human prostatic adipose tissue. BMC Med 2012; 10: 108.
9. Dahran N, Szewczyk-Bieda M, Vinnicombe S, Fleming S, Nabi G. Peri-prostatic fat adipokine expression is correlated with prostate cancer aggressiveness in men undergoing radical prostatectomy for clinically localized disease. BJU Int 2019; 123: 985–93.
10. Woo S, Cho JY, Kim SY, Kim SH. Peri-prostatic fat thickness on MRI: correlation with Gleason score in prostate cancer. AJR Am J Roentgenol 2015; 204: W43–7.
11. van Roemund JG, Hinnen KA, Tolman CJ, Bol GH, Witjes JA, et al. Periprostatic fat correlates with tumour aggressiveness in prostate cancer patients. BJU Int 2011; 107: 1775–9.
12. Cao Y, Cao M, Chen Y, Yu W, Fan Y, et al. The combination of prostate imaging reporting and data system version 2 (PI-RADS v2) and periprostatic fat thickness on multi-parametric MRI to predict the presence of prostate cancer. Oncotarget 2017; 8: 44040–9.
13. Huang H, Chen S, Li W, Bai P, Wu X, et al. Peri-prostatic fat thickness on MRI is an independent predictor of time to castration-resistant prostate cancer in Chinese patients with newly diagnosed prostate cancer treated with androgen deprivation therapy. Clin Genitourin Cancer 2019; 17: e1036–47.
14. Patel PM, Sweigert SE, Nelson M, Gupta G, Baker M, et al. Disparities in BPH progression: predictors of presentation to the emergency department in urinary retention. J Urol 2020; 204: 332–6.
15. Liu ZM, Wong CK, Chan D, Tse LA, Yip B, et al. Fruit and vegetable intake in relation to lower urinary tract symptoms and erectile dysfunction among Southern Chinese elderly men: a 4-year prospective study of Mr OS Hong Kong. Medicine (Baltimore) 2016; 95: e2597.
16. Bhindi B, Marget D, Trotter G, Hamilton RJ, Kuikanss GI, et al. Obesity is associated with larger prostate volume but with worse urinary symptoms: analysis of a large multiethnic cohort. Urology 2014; 83: 81–7.
17. Chen Y, Wu Y, Zhou L, Wu S, Yang Y, et al. Relationship among diet habit and lower urinary tract symptoms and sexual function in outpatient-based males with LUTS/BPH: a multinational and cross-sectional study in China. BMJ Open 2016; 6: e010863.
18. Yee CH, So WY, Yip SK, Wu E, Yau P, et al. Effect of weight reduction on the severity of lower urinary tract symptoms in obese male patients with benign prostatic hyperplasia: a randomized controlled trial. Korean J Urol 2015; 56: 240–7.
19. Plata M, Caicedo JJ, Trujillo GG, Marino-Alvarez AM, Fernandez N, et al. Prevalence of metabolic syndrome and its association with lower urinary tract symptoms and sexual function. Actas Urol Esp 2017; 41: 522–8.
20. Huang H, Chen S, Li W, Wu X, Xing J. High perinatal fat thickness predicts a poor progression-free survival in patients with localized core needle biopsy. Urol Oncol 2018; 36: 157.e1-6.
21. Motoya T, Matsumoto S, Yamaguchi S, Wada N, Numata A, et al. The impact of abdominal aortic calcification and visceral fat obesity on lower urinary tract symptoms in patients with benign prostatic hyperplasia. Int Urol Nephrol 2014; 46: 1877–81.
22. Zhang B, Chen X, Xie C, Chen Z, Liu Y, et al. Leptin promotes epithelial-mesenchymal transition in benign prostatic hyperplasia through downregulation of BAMB1. Exp Cell Res 2020; 387: 111754.
23. Fu S, Xu H, Gu M, Liu C, Wang G, et al. Adiponectin deficiency contributes to the development and progression of benign prostatic hyperplasia in obesity. Sci Rep 2017; 7: 43771.
24. Shahk S, Pearce G, Mann RD. Finasteride and tamsulosin used in benign prostatic hypertrophy: a review of the prescription-event monitoring data. BJU Int 2001; 87: 789-96.
25. Rigatti P, Brausi M, Scarpa RM, Porr D, Schumacher H, et al. A comparison of the efficacy and tolerability of tamsulosin and finasteride in patients with lower urinary tract symptoms suggestive of benign prostatic hyperplasia. Prostate Cancer Prostatic Dis 2003; 6: 315–23.
26. Ryu YW, Lim SW, Kim JH, Ahn SH, Choi JD. Comparison of tamsulosin plus serenoa repens with tamsulosin in the treatment of benign prostatic hyperplasia in Korean men: 1-year randomized open label study. Urol Int 2015; 94: 187–93.
27. Traish AM, Haider KS, Doros G, Haider A. Finasteride, not tamsulosin, increases severity of erectile dysfunction and decreases testosterone levels in men with benign prostatic hyperplasia. Horm Mol Biol Clin Invest 2015; 23: 85–96.
28. Gacci M, Andersson KE, Chapllo C, Magni M, Mirone V, et al. Latest evidence on the use of phosphodiesterase type 5 inhibitors for the treatment of lower urinary tract symptoms secondary to benign prostatic hyperplasia. Eur Urol 2016; 70: 124–33.
29. Powell K. Obesity: the two faces of fat. Nature 2007; 447: 525–7.
30. Fu S, Xu H, Gu M, Liu C, Wan X, et al. Lack of adiponectin and adiponectin receptor 1 contributes to benign prostatic hyperplasia. Oncotarget 2017; 8: 88537–51.
31. Nandeesha H, Eldhose A, Dorairajan LN, Anandhi B. Hypoadiponectinemia, elevated iron and high-sensitivity C-reactive protein levels and their relation with prostate size in benign prostatic hyperplasia. Andrology 2017; 49: e12715.
32. Zhang Q, Sun LJ, Qi J, Yang ZG, Huang T. Influence of adipocytokines and peri-prostatic adiposity measurement parameters on prostate cancer aggressiveness. Asian Pac J Cancer Prev 2014; 15: 1879–83.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

©The Author(s) (2020)