

Predicted body fat percentage, fat mass and lean body mass in relation to risk of prostate cancer Results from the NHANES 1999 to 2010

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Abstract

The purpose of this study is to examine the relationship between fat mass (FM), body fat percentage (BF%), lean body mass (LM), and prostate cancer (PCa), and evaluate their potential impact on the risk of PCa. Data from the National Health and Nutrition Examination Survey (NHANES) of the United States were utilized. Adult male participants from 6 survey cycles between 1999 and 2010 were selected as the study sample. Multivariable logistic regression analysis was conducted to explore the association between BF%, LM, and PCa, while controlling for potential confounding variables. Among the 8440 participants, 359 cases of PCa were diagnosed. The relationship between BF%, LM, and PCa was nonlinear. In the multivariable logistic regression analysis, there was an independent association between BF% and PCa risk (OR: 1.04, 95% CI: 1.02–1.06), suggesting that higher BF% levels are associated with an increased risk of PCa. Conversely, higher LM levels were associated with a decreased risk of PCa (OR: 0.96, 95% CI: 0.95–0.98). The findings of this study demonstrate a correlation between BF% and LM with PCa, but do not provide direct evidence of a causal relationship. Higher BF% levels are associated with an increased risk. These results provide valuable insights for understanding and potentially preventing PCa, although further research is needed to fully comprehend the underlying mechanisms.

Abbreviations: BF% = body fat percentage, BMI = body mass index, CVD = cardiovascular disease, FFM = fat-free mass, FM = fat mass, LM = lean mass, NHANES = National Health and Nutrition Examination Survey, PCa = prostate cancer.

Keywords: body fat percentage, fat mass, lean mass, NHANES, prostate cancer

1. Introduction

Prostate cancer (PCa) is the second most common cancer in males globally, accounting for 13.5% of new cancer cases. It is important to identify factors that can be modified to prevent PCa.^[1] Previous studies have investigated body size indicators, like body mass index (BMI), as potential risk factors for PCa but have yielded inconsistent results. Some studies suggest a negative association between BMI and PCa risk, while others find a positive association or varying effects.^[1-4]

BMI is commonly used to predict health risks due to its accessibility and affordability. However, it has limitations. One major drawback is that it does not differentiate between different body compartments, specifically fat mass (FM) and fat-free mass (FFM).^[5] Body composition, which includes the distribution of FM and FFM, varies across the BMI spectrum. Body composition can independently predict outcomes after a cancer diagnosis, highlighting the importance of considering not just overall body weight, but also the distribution of fat and lean mass.^[6,7]

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The metabolic activities of FM and FFM differ substantially. FFM can be seen as representing the body metabolic capacity, while FM can be viewed as a metabolic load. This difference in metabolic functioning has significant implications for various physiological processes within the body.^[6,7] Furthermore, the associations between FM and systemic inflammation, insulin, and insulin-like growth factors are important factors to consider.^[8] These biological mechanisms may contribute to tumor growth and impact the overall risk of developing cancer. Considering the specific distribution of FM and FFM, as well as their metabolic activities, may provide a more comprehensive understanding of individual health status and cancer risks.

While there is less research on the association between body composition and PCa risk compared to BMI studies, it is worth noting that body composition factors like FM, body fat percentage (BF%), and lean mass (LM) may independently influence cancer development. The hypothesis of this study is that there is a association between FM, BF%, LM, and PCa. Therefore, the objective of this study is to investigate the relationship between

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The datasets generated during and/or analyzed during the current study are publicly available.

The studies involving human participants were reviewed and approved by the Research Ethics Review Board of the NCHS. The patients/participants provided their written informed consent to participate in this study.

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body composition and the risk of PCa. Data from the NHANES database will be analyzed to address the gaps in epidemiological understanding of PCa, current research findings, and remaining challenges in this field.

2. Method

2.1. Study population

The National Health and Nutrition Examination Survey (NHANES) is a nationwide study conducted in the United States to gather information on the health status of the civilian population. This survey follows a complex and structured design using a stratified and multistage probability approach. It has been conducted every 2 years since 1999. The research ethics board of the National Center for Health Statistics, Centers for Disease Control and Prevention, authorizes NHANES, and all adult participants provide written informed consent.

This study analyzed data from 6 NHANES cycles conducted between 1999 and 2010, focusing on adult males aged 30 and above. Initially, it had a sample size of 62,160 participants. To ensure data quality, this study excluded individuals with missing information on variables such as prostate cancer, BF%, fat mass, LM, marital status, household income, education, smoking, alcohol consumption, hypertension, diabetes, cardiovascular disease (CVD), and hyperlipidemia. As a result of these exclusions, the final analysis included a total of 8440 participants (Fig. 1).

For a more detailed understanding of the statistical data and additional information about NHANES, please visit the official website at https://www.cdc.gov/nchs/nhanes/.

2.2. Assessment of BF%, LM and FM

Whole-body DXA scans were conducted at the Mobile Examination Center using the advanced Hologic QDR 4500 A fan beam X-ray bone densitometer manufactured by Hologic Inc. Strict criteria were followed for participant inclusion or exclusion to ensure accuracy and reliability of the scans. Individuals who had undergone radiographic contrast material tests or nuclear medicine tests within the past 72 hours or 3 days, respectively, were excluded from the study. Additionally, participants who reported a weight exceeding 300 pounds or a height over 6'.5' were also excluded. The DXA scans underwent a rigorous quality control process, and the data collected were analyzed using the Hologic Discovery software, version 12.1. This software provided precise measurements of total and regional body composition, including LM, FM, and percent fat. To maintain data integrity, any invalid DXA scans were identified and marked as missing in the data files. To account for missing DXA values, a multiple imputation method was used. This study focused on analyzing data related to total LM (excluding bone density mineral), total FM, and total BF%.

2.3. Diagnose of prostate cancer

The medical history of prostate cancer was characterized as "ever been told by a doctor or health professional that you had prostate cancer?" Participants who reported this issue indicated a prior diagnosis of prostate cancer.

2.4. Covariates

This study included additional factors that may influence the relationship between BF%, FM, LM, and PCa. These factors



Figure 1. Overview of participants screening. CVD = cardiovascular diseases, NHANES = National Health and Nutrition Examination Survey.

consisted of population characteristics such as age, race/ethnicity, marital status, educational level, smoking and alcohol consumption patterns, BMI, and household income. Moreover, the researchers also considered health risks such as diabetes, hypertension, CVD, and high cholesterol levels. More detailed categorical information can be found in Table 1, providing a comprehensive overview.

2.5. Statistical analysis

A weighted analysis was conducted in accordance with NHANES guidelines to address the complexities of sampling in this study. Weighted Student t tests were employed for the comparison of continuous variables, while weighted chi-square tests were used for categorical variables. These statistical tests allowed us to compare the baseline characteristics of the normal group and the PCa group, while taking into account the sampling design. To examine the relationship between BF%, FM, LM, and PCa, multivariable logistic regression analysis was carried out. Three models were developed for this purpose. Model 1 was unadjusted and did not consider potential confounding variables. Model 2 was adjusted for age and ethnicity, which are established risk factors for PCa. Model 3 further adjusted for additional factors such as education, smoking, alcohol consumption, BMI, diabetes, hypertension, CVD, and hyperlipidemia. To extract and analyze the data obtained from NHANES, the "nhanesR" package was utilized. A significance level of <0.05 (two-sided) was considered to indicate a significant difference. By conducting this weighted analysis and adjusting for potential confounding factors, this study aimed to provide a comprehensive understanding of the relationship between body composition and PCa.

3. Results

Among the 8440 participants in the study, 359 individuals were diagnosed with PCa (Table 1). After applying weighting, the calculated incidence rate of PCa was found to be 2.71%. Participants with PCa had higher BF% and lower LM compared to those without prostate involvement (all P < .05). However, there was no significant difference in the distribution of FM between the 2 groups. Furthermore, individuals with PCa showed statistically significant differences in ethnicity, marital status, annual household income, smoking habits, alcohol consumption, DM, hypertension, and CVD compared to those without PCa (all P < .05).

To assess the individual impact of BF%, FM, and LM on PCa while accounting for other potential factors, multiple models were developed. Univariate analysis revealed significant associations between the incidence of PCa and various factors including age, BF%, LM, ethnicity, marital status, annual household income, smoking, alcohol consumption, DM, hypertension, and CVD (P < .05, Supplementary material 1, http://links.lww.com/MD/M761).

Overall, a nonlinear relationship (nonlinear P < .001) was observed between BF%, LM, and PCa (Fig. 2). The risk of PCa initially increased rapidly with increasing BF%, but gradually decreased as BF% further increased (change point BF% = 32.95). Conversely, the incidence of PCa decreased gradually with increasing LM levels. Univariate Logit model analysis demonstrated that higher levels of BF% were independently associated with a higher risk of PCa (OR: 1.04, 95% CI: 1.02–1.06, P < .001), while higher levels of LM were associated with a decreased risk of PCa (OR: 0.96, 95% CI: 0.95–0.98). High levels of BF% were identified as a risk factor for PCa (Q4 vs Q1, OR = 2.63, 95% CI: 1.66–4.16, P < .0001, *Ptrend* < 0.001). Conversely, high levels of LM were associated with a lower risk of PCa (Q4 vs Q1, OR: 0.40, 95% CI: 0.28–0.57, P < .001, *Ptrend* = 0.542). There was no significant correlation found between FM levels and the occurrence of PCa (OR: 1.00, 95% CI: 0.99-1.01, P = .85, *Ptrend* = 0.609).

After adjusting for age and ethnicity, the model still showed a significant association between higher BF% levels and an increased incidence of PCa (Q4 vs Q1, OR: 1.71, 95% CI: 1.06-2.77, P = .03, *Ptrend* = 0.017) (Table 2). Additionally, higher LM levels were found to be associated with a lower risk of PCa (Q4 vs Q1, OR = 0.49, 95% CI: 0.34–0.72, P < .001, *Ptrend* < 0.001). Finally, after adjusting for age, ethnicity, education, smoking, alcohol consumption, body mass index (BMI), DM, hypertension, CVD, and hyperlipidemia, the multivariable logistic regression model showed that BF% and LM remained significantly associated with PCa.

4. Discussion

In this research study, NHANES data from the United States was utilized to explore the link between BF%, LM, FM, and PCa. The findings of the study revealed a nonlinear relationship between BF% and the occurrence of PCa, indicating a positive association between an increase in BF% and a heightened risk of PCa. On the other hand, an increase in LM levels showed a negative correlation with the risk of PCa. However, no significant connection was found between FM levels and the incidence of PCa. To summarize, the study demonstrated that BF% and LM may be related to the risk of PCa, while FM does not appear to play a significant role.

A review of the literature reveals conflicting findings regarding the link between obesity and prostate cancer. Some articles suggest that obesity may either promote or protect against prostate cancer, while others indicate no significant effect.^[9] While some articles suggest that obesity either promotes or protects against PCa, others indicate no effect. However, a closer examination of the incidence, progression, and mortality rates provides a clearer understanding of this relationship. Recent data suggests that obesity does influence all 3 aspects of prostate cancer. There is particular concern about the association between obesity and worse post-treatment outcomes, as well as an increased risk of prostate cancer-related mortality. BMI is commonly used as a surrogate measure for obesity, with overweight defined as a BMI of 25 kg/m² or higher, and obesity as a BMI of 30 kg/m² or higher. Studies investigating the relationship between adult BMI and the risk of developing prostate cancer have produced mixed results. Some large cohort studies have identified a higher risk of prostate cancer with increased BMI, although the associations were sometimes weak.[10-12] On the other hand, other prospective cohort studies found no significant association between BMI and prostate cancer risk.^[13,14] Compared to BMI, BF% can more accurately reflect the body fat levels. While the calculation method of BMI is simple, it has significant inaccuracies and cannot precisely reflect body fat content, changes in fat composition across different life stages, and the impact of gender differences on fat distribution within the body. In contrast, BF% offers more advantages.^[15,16] In this study, a significant association was discovered between high BF% and an increased incidence of prostate cancer. Furthermore, a marker of body composition, LM, demonstrated a negative correlation with prostate cancer. These findings provide further evidence of the inverse relationship between BF% levels and the occurrence of PCa. It is important to note that this study did not find a significant association between BMI and the incidence of prostate cancer, which is consistent with previous studies with similar results. This could be attributed to the fact that BF% and BMI are 2 distinct measurement methods. While BMI is based on height and weight, BF% measures the proportion of body fat.^[17,18] BF%, being a more accurate reflection of fat distribution in the body, could potentially play a role in the development of prostate cancer.

Table 1

Characteristics of the study population.									
Wandaha	Total	Non-PCa	PCa	Queles					
	(N = 8440)	(N = 8081)	(N = 359)	<i>P</i> value					
Age	54.75 (0.22)	54.28 (0.22)	71.68 (0.66)	<.0001					
Body fat percentage	29.92 (0.12)	29.87 (0.13)	31.52 (0.33)	<.0001					
Q1[8.96–26.11]	27.58%	28.00%	12.82%	<.0001					
Q2(26.11-29.93]	24.52%	24.46%	26.74%						
Q3(29.93–33.88]	23.52%	23.31%	31.21%						
Q4(33.88–55.91]	24.37%	24.24%	29.22%						
Lean mass	60.52 (0.17)	60.61 (0.18)	57.31 (0.58)	<.0001					
Q1[32.48,51.66]	17.14%	16.87%	26.86%	<.0001					
Q2(51.66,57.43]	23.85%	23.73%	28.28%						
Q3(57.43,63.85]	27.63%	27.71%	24.59%						
Q4(63.85,140.29)	31.38%	31.69%	20.27%						
Fat mass	26.93 (0.17)	26.92 (0.18)	27.03 (0.52)	.85					
01[1.29.20.15]	22.67%	22.73%	20.45%	.55					
02(20.15.25.25]	24.31%	24.30%	24.36%						
03(25 25 31 20)	25.60%	25 49%	29.34%						
04(31 20 77 44)	27 43%	27 47%	25.85%						
Fthnic	2111070	21.11/0	20.00 /	< 001					
Black	8 92%	8 84%	11 62%	<.001					
Other	14 01%	14 20%	7 25%						
White	77 07%	76.06%	01 120/						
Marital status	11.01/0	70.90%	01.1370	< 001					
Married	70,600/	70 440/	70.010/	<.001					
SDW	16.06%	16 200/	10.9170						
Jamariad	11.100/	11.20%	0.00%						
	11.12%	11.35%	2.93%	000					
	15 500/	15 500/	10.40%	.002					
0-19.999\$	15.58%	15.50%	18.46%						
20.000-54.999\$	35.44%	35.16%	45.52%						
55.000-74.999\$	14.34%	14.52%	7.98%						
75.000+\$	34.63%	34.81%	28.04%						
Education				.75					
High school graduate	44.57%	44.54%	45.68%						
or									
under									
Some college or above	55.43%	55.46%	54.32%						
Smoke				<.0001					
Former	36.78%	36.23%	56.34%						
Never	40.72%	40.80%	38.01%						
Now	22.50%	22.97%	5.65%						
Alcohol consumption				<.001					
No	29.02%	28.74%	38.76%						
Yes	70.98%	71.26%	61.24%						
BMI (kg/m ²)				.85					
<25	23.35	23.35	23.25	100					
25-29.9	42.59	42.54	44 29						
>30	34 07	34 11	32.45						
DM	01.01	01111	02.10	02					
DM	16 53%	16 33%	23 58%	.02					
IEG	5.01%	5.84%	8.64%						
ICT	0.31/0	2 2 4 %	2.06%						
No	2.30 /0	2.3470	0.30 /0						
Hypertension	13.1070	13.4370	03.0270	~ 0001					
No	E2 00%	E4 E10/	25 280/	<.0001					
NU Vee	23.99%	04.01%	30.38%						
TES	40.01%	40.49%	04.02%	. 0004					
		00.00%	70 1 00/	<.0001					
NO Vec	85.92%	80.30%	72.18%						
res	14.08%	13.70%	27.82%	<i></i>					
Hyperlipidemia	00.000	04.000	17.0.10	.21					
NO	20.99%	21.08%	17.64%						
Yes	79.01%	78.92%	82.36%						

CVD = cardiovascular diseases, DM = diabetes mellitus, IFG = impaired fasting glucose, IGT = impaired glucose tolerance, Q = quartile, SDW = separated, divorced, widowed.

The possible explanations for the potential causes of the association between high levels of fat and prostate cancer are complex and challenge. Firstly, obesity is related to changes in hormone levels. In obese individuals, testosterone (male hormone) levels may decrease, while estrogen (female hormone) levels may increase.^[19] This hormonal imbalance may

play a role in the development of prostate cancer. Secondly, obesity is associated with dietary intake.^[20] Obese individuals often consume more high-fat and high-sugar foods, which can lead to an excess of energy and nutritional imbalance. Such unhealthy dietary habits may increase the risk of prostate cancer. Additionally, obesity is associated with an increase



Figure 2. Relation of body fat percentage, fat mass, and lean mass with risk of prostate cancer.

in inflammatory mediators.^[21] Fat cells release inflammatory mediators such as cytokines and leukotrienes, which may promote the growth and metastasis of prostate cancer cells. Obesity patients often experience a chronic inflammatory state, thereby increasing the risk of developing prostate cancer. Moreover, obesity is highly correlated with metabolic syndrome. Metabolic syndrome is a disorder characterized by a

series of metabolic abnormalities, including high blood pressure, high blood sugar, high blood lipids, and abdominal obesity.^[3,22] These abnormal states may promote the development of prostate cancer through multiple pathways.

The main strengths of this study include the utilization of MR analysis and an observational study design within the NHANES dataset. The large sample size provided by NHANES allowed



Figure 2. Continued

Table 2

Multivariate logistic regression models of body fat percentage, fat mass, and lean mass on PCa.

Body fat percentage	Model 1		Model 2		Model 3	
	OR (95%CI)	P value	OR (95%CI)	P value	OR (95%CI)	P value
Continuous	1.04 (1.02,1.06)	<.0001	1.02 (1.00,1.04)	.04	1.07 (1.02,1.12)	.004
Q1	ref	ref	ref		ref	
Q2	2.39 (1.47,3.88)	<.0001	1.67 (1.01,2.78)	.049	3.00 (1.74, 5.16)	<.001
Q3	2.92 (1.97,4.35)	<.0001	1.89 (1.26,2.82)	.002	5.27 (2.84, 9.79)	<.0001
Q4	2.63 (1.66.4.16)	<.0001	1.71 (1.06.2.77)	.03	9.56 (3.25,28.07)	<.0001
Ptrend	0.006		0.017		< 0.0001	
Fat mass						
Continuous	1.00 (0.99,1.01)	.85	1.00 (0.98,1.01)	.62	0.99 (0.97,1.02)	.63
Q1	ref	ref	ref		ref	
Q2	1.11 (0.77,1.61)	.56	1.08 (0.73,1.61)	.70	1.15 (0.72,1.85)	.55
Q3	1.28 (0.90, 1.82)	.17	1.11 (0.78, 1.58)	.54	1.17 (0.68,2.02)	.57
Q4	1.05 (0.69, 1.58)	.83	0.92 (0.59,1.41)	.69	0.91 (0.45,1.86)	.80
Ptrend	0.609		0.658		0.814	
Lean mass						
Continuous	0.96 (0.95,0.98)	<.0001	0.97 (0.96,0.99)	<.0001	0.95 (0.93,0.97)	<.0001
Q1	ref	ref	ref		ref	
Q2	0.75 (0.52,1.08)	.12	0.80 (0.56,1.16)	.24	0.73 (0.51,1.05)	.09
Q3	0.56 (0.38,0.82)	.004	0.66 (0.44,0.97)	.04	0.52 (0.33,0.82)	.01
Q4	0.40 (0.28,0.57)	<.0001	0.49 (0.34,0.72)	<.001	0.32 (0.20,0.50)	<.0001
Ptrend	0.542		<0.001		0.449	

Model 1: adjust for non.

Model 2: adjust for age, ethnic.

Model 3: adjust for Age, ethnic, education, smoke, alcohol consumption, BMI, Diabetes, Hypertension, CVD, Hyperlipidemia.

CI = confidence interval, OR = odd ratio, PCa = prostate cancer.

us to include numerous potential confounding variables as covariates in multivariable-adjusted logistic regression analysis, enhancing the reliability and efficacy of the findings.

However, it is important to acknowledge several limitations. Firstly, this study relied on predicted body composition, which serves as a surrogate for body composition measured using gold standard methods like DEXA. This approach does not incorporate any unique parameters for each participant; rather, it is based on commonly used anthropometric variables such as waist circumference, which is closely associated with visceral fat deposition and considered a potential risk factor for prostate cancer. Secondly, the predicted fat and lean body weight were determined through a one-time assessment without repeated measurements. Individuals may undergo significant changes over time, making it challenging to fully understand the correlation between these early indicators and subsequent prostate cancer incidence. Thirdly, while we have considered most potential confounders, it is possible that other unmeasured or unknown residual confounding factors may be present. Lastly, the limited number of prostate cancer cases in this study hindered the ability to perform subgroup analyses and conduct sensitivity analyses to validate the results.

5. Conclusion

In conclusion, this study reveals that high levels of BF% were independently associated with an increased risk of PCa, while high levels of LM were associated with a decreased risk of PCa among adult males. These results highlight the relevance of body composition, specifically BF% and LM, in assessing the risk of PCa. By considering these factors, healthcare professionals can better evaluate an individual susceptibility to PCa and tailor appropriate prevention and treatment strategies. Additionally, the nonlinear relationship observed suggests that targeting body composition, particularly reducing excess body fat and increasing LM, may have a potential protective effect against PCa.

Author contributions

Conceptualization: Shuai Wu, Huibing Huang, Zhiyong Xiong. Data curation: Shuai Wu, Huibing Huang, Zhiyong Xiong. Formal analysis: Qi Zou. Investigation: Qi Zou, Qiang Li.

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References

- [1] Bray F, Ferlay J, Soerjomataram I, et al. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2018;68:394–424.
- [2] Amin HA, Kaewsri P, Yiorkas AM, et al. Mendelian randomisation analyses of UK Biobank and published data suggest that increased adiposity lowers risk of breast and prostate cancer. Sci Rep. 2022;12:909.
- [3] Bhaskaran K, Douglas I, Forbes H, et al. Body-mass index and risk of 22 specific cancers: a population-based cohort study of 5.24 million UK adults. Lancet. 2014;384:755–65.
- [4] MacInnis RJ, English DR. Body size and composition and prostate cancer risk: systematic review and meta-regression analysis. Cancer Causes Control. 2006;17:989–1003.

- [5] Prado CM, Siervo M, Mire E, et al. A population-based approach to define body-composition phenotypes. Am J Clin Nutr. 2014;99:1369–77.
- [6] Prado CM, Wells JC, Smith SR, et al. Sarcopenic obesity: a critical appraisal of the current evidence. Clin Nutr. 2012;31:583–601.
- [7] Wolfe RR. The underappreciated role of muscle in health and disease. Am J Clin Nutr. 2006;84:475–82.
- [8] Hardy OT, Czech MP, Corvera S. What causes the insulin resistance underlying obesity? Curr Opin Endocrinol Diabetes Obes. 2012;19:81-7.
- [9] Buschemeyer WC 3rd, Freedland SJ. Obesity and prostate cancer: epidemiology and clinical implications. Eur Urol. 2007;52:331–43.
- [10] Veierød MB, Laake P, Thelle DS. Dietary fat intake and risk of prostate cancer: a prospective study of 25,708 Norwegian men. Int J Cancer. 1997;73:634–8.
- [11] Zhu D, Toker M, Shyr W, et al. Association of obesity and diabetes with prostate cancer risk groups in a multiethnic population. Clin Genitourin Cancer. 2022;20:299–299.e10.
- [12] Lavalette C, Cordina Duverger E, Artaud F, et al. Body mass index trajectories and prostate cancer risk: results from the EPICAP study. Cancer Med. 2020;9:6421–9.
- [13] Putnam SD, Cerhan JR, Parker AS, et al. Lifestyle and anthropometric risk factors for prostate cancer in a cohort of Iowa men. Ann Epidemiol. 2000;10:361–9.
- [14] Schuurman AG, Goldbohm RA, Dorant E, et al. Anthropometry in relation to prostate cancer risk in the Netherlands Cohort Study. Am J Epidemiol. 2000;151:541–9.
- [15] Gómez-Ambrosi J, Silva C, Catalán V, et al. Clinical usefulness of a new equation for estimating body fat. Diabetes Care. 2012;35:383–8.
- [16] Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous and how do we measure them? Int J Epidemiol. 2006;35:83–92.
- [17] Lee DH, Keum N, Hu FB, et al. Development and validation of anthropometric prediction equations for lean body mass, fat mass and percent fat in adults using the National Health and Nutrition Examination Survey (NHANES) 1999-2006. Br J Nutr. 2017;118:858–66.
- [18] Wallström P, Bjartell A, Gullberg B, et al. A prospective Swedish study on body size, body composition, diabetes, and prostate cancer risk. Br J Cancer. 2009;100:1799–805.
- [19] Bandini M, Gandaglia G, Briganti A. Obesity and prostate cancer. Curr Opin Urol. 2017;27:415–21.
- [20] Satia-Abouta J, Patterson RE, Schiller RN, et al. Energy from fat is associated with obesity in U.S. men: results from the prostate cancer prevention trial. Prev Med. 2002;34:493–501.
- [21] Wilson RL, Taaffe DR, Newton RU, et al. Obesity and prostate cancer: a narrative review. Crit Rev Oncol Hematol. 2022;169:103543.
- [22] Purcell SA, Oliveira CLP, Mackenzie M, et al. Body composition and prostate cancer risk: a systematic review of observational studies. Adv Nutr. 2022;13:1118–30.