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*Original Communication*

# The Association Between a Body Shape Index and Testosterone Among U.S. Adult Males: National Health and Nutrition Examination Survey (2011–2016)

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**Abstract**

**Background**: Obesity, a prevalent global health issue, is associated with testosterone deficiency (TD). A body shape index (ABSI) provides a more precise assessment of obesity and visceral fat, but its relationship with testosterone remains unclear. This study aimed to explore the association between ABSI and testosterone levels leading to TD. **Methods**: Data from 5256 adult males participating in the National Health and Nutrition Examination Survey (NHANES) from 2011 to 2016 were collected to analyze of the association between ABSI and TD. The data underwent analysis using multivariate linear regression, logistic regression, restricted cubic spline (RCS) analysis, subgroup analysis, and interaction testing. The predictive ability of ABSI based on weight, height, and waist circumference, as well as body mass index (BMI) based on weight and height, alongside a multiplicative combination of both metrics, BMI $\times$ ABSI, and optimal proportional combination $O_{BMI+ABSI}$ for assessing TD risk, was valuated using receiver operating characteristic (ROC) curves. **Results**: Following adjustment for all confounding factors, ABSI exhibited a negative linear correlation with testosterone ($\beta = -6.99$, 95% confidence interval (CI): –8.25 to –5.73; $p < 0.001$) and a positive association with TD risk (odds ratio (OR) = 1.06, 95% CI: 1.04–1.08; $p < 0.001$). Notably, these associations remained consistent in the subgroup analysis. Additionally, age and hypertension demonstrated significant interactions between ABSI and TD ($p < 0.05$). Moreover, combining metrics, such as BMI $\times$ ABSI and $O_{BMI+ABSI}$, proved to be more reliable predictors of TD compared to BMI or ABSI alone. **Conclusions**: This study identified a negative linear correlation between ABSI and total testosterone levels in adult American males, as well as a positive linear correlation with TD prevalence. ABSI represents a valuable addition to BMI for assessing obesity and the association between obesity and TD.

**Keywords:** obesity; ABSI; testosterone; testosterone deficiency

## 1. Introduction

Testosterone, a hormone associated with male sex characteristics, is produced by the Leydig cells within the testes. It not only helps sustain male secondary sexual traits and boost sperm generation but also increases muscle mass and bone strength, while being crucial for the normal functioning of the reproductive system and multiple organs in the body [1,2]. Although testosterone level in male serum is highest during adolescence, they gradually decline with age and significantly decrease in old age. When testosterone level in male serum drops to 300 ng/dL, it can be diagnosed as testosterone deficiency (TD) [3]. This condition may lead to various age-related issues such as sexual dysfunction, reproductive disorders, cardiovascular diseases, and metabolic disturbances, severely affecting men's quality of life and health [4,5]. Reports indicate that up to 40% of men over the age of 45 in the United States experience TD, a figure anticipated to rise in the future as life expectancy continues to grow [4,6]. Therefore, TD has become an increasingly concerning global health issue.

Obesity greatly increases the likelihood of various heart and metabolic disorders, such as coronary artery disease, high blood pressure, stroke, type 2 diabetes, high cholesterol, and gout [7]. Moreover, research indicates that obesity is linked to reduced testosterone level [8]. Additionally, the distribution of fat in the body can affect the level of testosterone. Excessive accumulation of subcutaneous fat tissue typically does not affect the body's metabolism or the synthesis and metabolism of serum testosterone [9]. However, if obesity leads to substantial fat deposition around internal organs in the abdominal region, these adipose tissues can impede skeletal muscle function and disrupt liver metabolism. Furthermore, excessive abdominal fat accumulation interferes with organ metabolism leading to insulin resistance, inflammation, and dyslipidemia. This often results in metabolic disturbances such as high waist circumference (WC) or abdominal obesity which may con-

tribute to decreased serum testosterone concentration [10, 11]. Obesity is closely associated with reduced testosterone level, and it is imperative to comprehend the obesity index that can forecast the risk of testosterone decline and TD.

Out of numerous indexes for obesity, body mass index (BMI) stands out as a widely accepted and commonly utilized tool to evaluate obesity [12–14]. The World Health Organization defines obesity as having a BMI $\geq$30 kg/m$^2$ primarily relevant in Western societies [15]. Some research revealed an inverse relationship between BMI and testosterone, suggesting that obese individuals are more prone to experiencing reduced testosterone level in comparison to those with a normal BMI. Nevertheless, if obese individuals undergo testosterone replacement therapy and achieve weight loss, their testosterone level will rise [16,17]. These outcomes underscore a significant interplay linking both aspects together. However, as research progressed, it became evident that BMI is limited in its ability to directly measure the degree of obesity and distinguish between lean body mass and fat. Consequently, there is a growing consensus within the scientific community that BMI alone cannot reliably predict obesity in clinical practice, prompting the need for a new indicator to either replace or complement BMI for assessing obesity [18]. As investigations into conditions linked with obesity persistently evolve; emphasis has shifted towards abdominal visceral fat assessment offering superior insights into overall adiposity distribution patterns [19]. Due to the side effects and cost considerations associated with computed tomography (CT), magnetic resonance imaging (MRI), and dual X-ray absorptiometry (DXA), these imaging modalities are not widely applicable in clinical practice. Therefore, simple clinical anthropometric indices such as WC, waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) should be utilized as alternative or supplementary indicators to BMI for identifying metabolic diseases [19]. Even though there is an association between these alternative markers and low testosterone, the correlation of these waist-based obesity indicators with BMI may confound the observation of the link between obesity and diseases, rendering them an unreliable indicator of visceral obesity [20–23]. Hence underscoring the continued necessity for novel precise markers effectively appraising visceral adipose accumulations thereby comprehensively unraveling their intricate ties with TD.

Krakauer and Krakauer [24] devised a body shape index (ABSI) as a more accurate measure for assessing abdominal visceral obesity. Unlike BMI, which primarily reflects overall body mass and does not distinguish between fat and lean mass, ABSI is specifically designed to capture information about body shape, particularly the distribution of abdominal fat, independent of overall adiposity. In contrast to BMI, which reflects overall body fat, ABSI utilizes WC for adjusting the impact of height and weight largely independent of these variables as well as from BMI itself [25,26]. This is crucial because visceral adipose tissue, which ABSI is thought to better reflect, is metabolically more active and strongly linked to adverse health outcomes compared to subcutaneous fat. ABSI provides a better explanation for the degree and distribution of central abdominal obesity. Clinically, ABSI has shown promise in predicting various health risks beyond what BMI or WC alone can offer. Prior research has shown a link between ABSI and ailments like high blood pressure, diabetes, and heart disease [27]. Importantly, it suggests ABSI may be a stronger predictor of mortality and certain metabolic diseases than BMI or WC, particularly because it is less confounded by factors like muscle mass and height. Furthermore, it exhibits a stronger correlation with death rates than BMI or WC individually. Moreover, the ABSI can adjust the proportion index based on the population of other groups, exhibiting only marginal discrepancies. In contrast to BMI, it is applicable across diverse populations for investigating obesity and associated conditions [28]. Therefore, investigating ABSI in the context of TD is clinically relevant because it could offer a more refined and potentially more predictive tool for identifying men at risk, compared to relying solely on BMI or WC. Nevertheless, there remains a lack of comprehensive observational or laboratory investigations exploring the link between ABSI and TD.

This research sought to investigate the link between the novel obesity indicator ABSI and testosterone level in adult American men aged 20 and above, with the objective of raising awareness about the detrimental impact of obesity on adult testosterone level and TD. This provides a fresh perspective on male reproductive health and obesity issues.

## 2. Materials and Methods

### *2.1 Research Methodology and Demographic Investigation*

This study made use of data from the National Health and Nutrition Examination Survey (NHANES) carried out between 2011 and 2016. For further details about the survey, please refer to https://wwwn.cdc.gov/Nchs/Nhanes (accessed on 14 August 2024). Nationally representative sample survey NHANES is conducted by the Centers for Disease Control and Prevention (CDC). Notably, NHANES integrates population demographic, socioeconomic, dietary, health-related interview questions, physiological and medical measurements, as well as medical-related laboratory tests. The outcomes of this comprehensive survey will be employed to ascertain disease prevalence, identify disease risk factors, evaluate nutritional status in relation to health promotion and disease prevention efforts, conduct epidemiological studies, and facilitate health research endeavors. The NHANES research protocol has been granted approval by the U.S. institutional review board, and all participants have provided written informed consent prior to survey participation, thereby addressing any ethical concern.

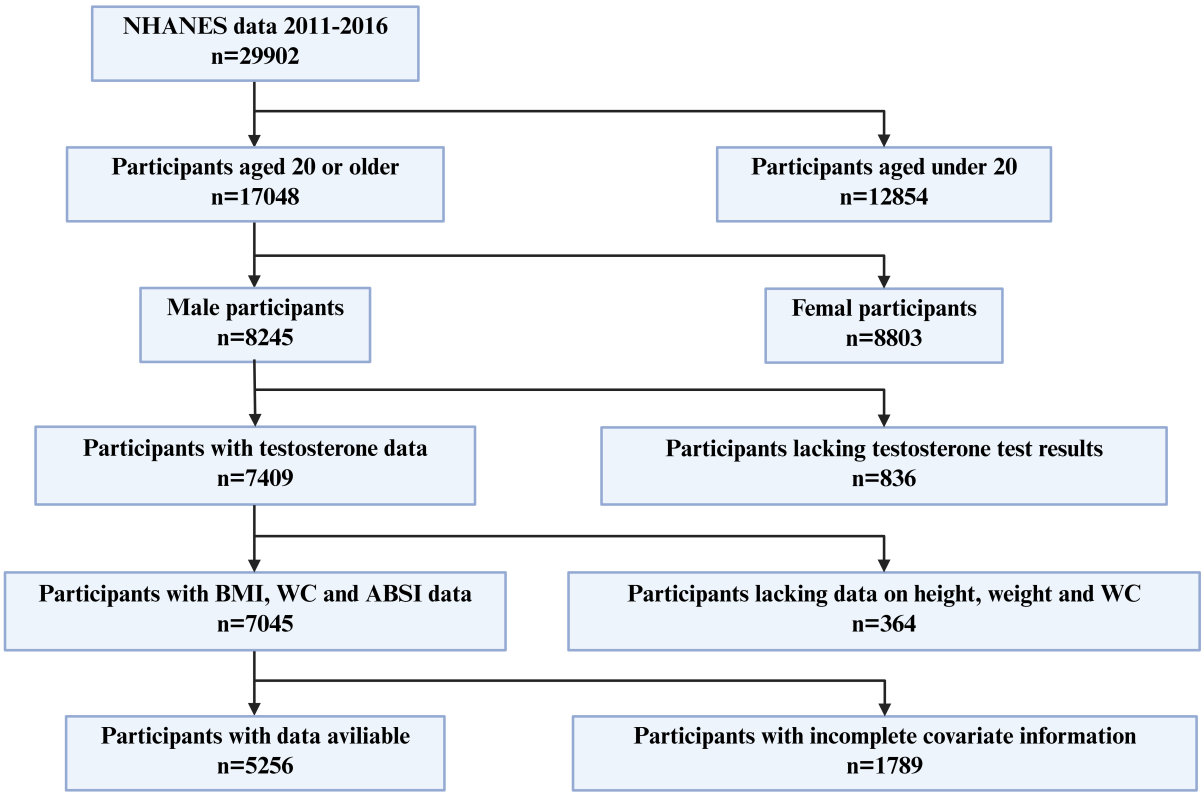


**Fig. 1. Flowchart or the selection and recruitment of study participants.**

This study encompassed data from 29,902 individuals meeting specific inclusion criteria: (1) aged $\geq$20 years; (2) male gender; (3) availability of testosterone level along with height/weight/WC measurements and a two-day 24-hour dietary report; (4) race, education level, marital status, poverty-income ratio (PIR), smoking, diabetes, hypercholesterolemia and hypertension as covariates. Individuals failing to meet these criteria or lacking essential survey questionnaires pertaining to demographics smoking diabetes hyperlipidemia or hypertension variables were excluded from this analysis. Ultimately our study included a cohort of 5256 participants (Fig. 1).

### *2.2 Testosterone and TD*

The concentration of testosterone was assessed utilizing the isotope dilution liquid chromatography-tandem mass spectrometry instrument developed by the CDC. If the measured testosterone value falls below the predetermined minimum, the detection value is calculated by dividing this minimum by the square root of 2. Detailed test methods for testosterone can be accessed from NHANES's official website. TD is diagnosed when serum testosterone level is below 300 ng/dL [3].

### *2.3 Body Measurement Indicators and Calculation Method*

Body measurement data were collected by health technicians commissioned by NHANES. The technicians utilized precise measuring instruments to obtain height (m), weight (kg), and WC (m). Specifically, During the standing position, the examiner measures WC at the midpoint between the lowest rib and the highest point of the iliac crest after exhalation. BMI is determined by dividing weight by height squared. Additionally, ABSI was calculated by 1000 $\times$ WC (m) $\times$ [weight (kg)]$^{-2/3}$ $\times$ [height (m)]$^{5/6}$ [29].

The study evaluated the predictive capacity for testosterone occurrence risk using ABSI, BMI, and their multiplicative combination ABSI $\times$ BMI and optimal proportional combination $O_{BMI+ABSI}$ [29]. Here, $O_{BMI+ABSI} = n_{ABSI} \times ABSI + n_{BMI} \times BMI$. The optimal proportion coefficients ABSI ($n_{ABSI}$) and BMI ($n_{BMI}$) for ABSI and BMI were determined as $n_{ABSI} = \beta_{ABSI}/(\beta_{BMI} + \beta_{ABSI})$ and $n_{BMI} = \beta_{BMI}/(\beta_{BMI} + \beta_{ABSI})$, respectively; $\beta_{ABSI}$ and $\beta_{BMI}$ are the regression coefficients assigned to ABSI and BMI, respectively, within a multivariable logistic regression model. To estimate the regression coefficients for ABSI and BMI, we included these variables in a multivariable-adjusted logistic regression model, with TD occurrence as the primary outcome. Furthermore, adjustments were made for age, race, marital status, education level, PIR, smoking, diabetes, hyperlipidemia, hypertension, and alcohol intake as covariates. To derive these coefficients for ABSI and BMI variables, they were included in a multivariable-adjusted logistic regression model with TD occurrence as an outcome of interest while adjusting for age, race, marital status, education level, PIR, smoking, diabetes, hypertension, hyperlipidemia and alcohol intake.

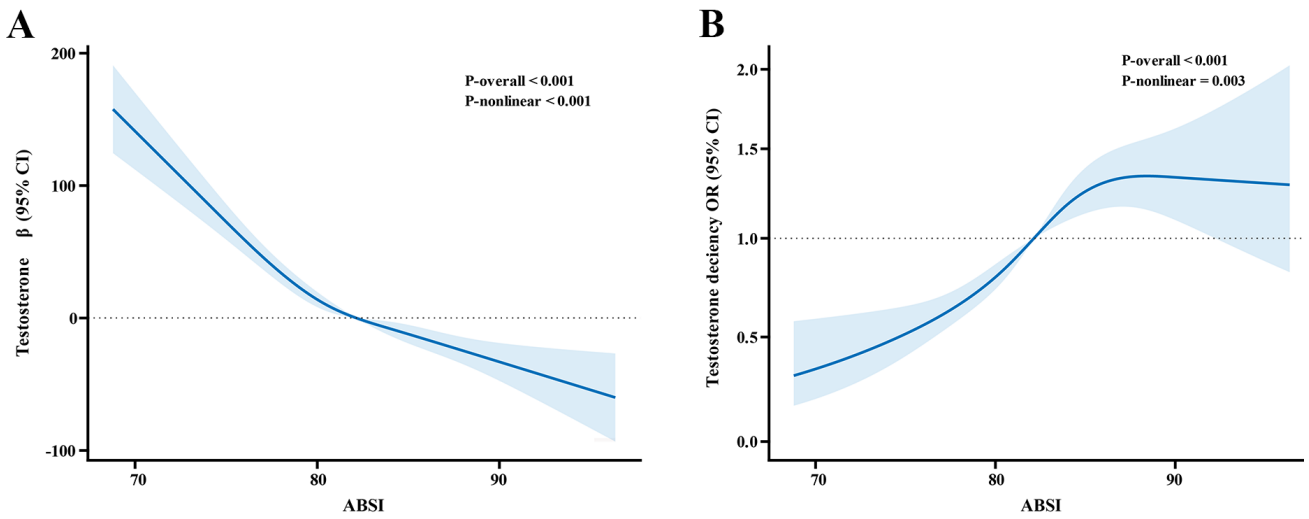


**Fig. 2. Restricted cubic spline (RCS) curve illustrates the relationship between a body shape index (ABSI) and testosterone and testosterone deficiency (TD) in males aged over 20 years in U.S., as well as the risk of TD, with the blue band representing the 95% CI.** (A) RCS analysis of the association between ABSI and testosterone. (B) RCS analysis of the association between ABSI and TD. The RCS curves were both adjusted according to age, race, marital status, education level, PIR, BMI, diabetes, hypercholesterolemia, hypertension, smoking and alcohol consumption. Significant overall association was observed ($p$-overall $< 0.001$), with no clear evidence of non-linearity ($p$-nonlinear $< 0.01$).

### *2.4 Confounding Variables*

In order to improve the accuracy of the analysis, we incorporated a comprehensive set of confounding variables associated with testosterone in this study. Demographic factors encompassed age (<40, 41–60, ≥60 years), race (Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black and other race), education level (Less than 9th grade, 9–11th grade, high school, some college or AA degree, college graduate or above), marital status (married, separated, divorced, widowed, never married, living with a partner) and PIR (<1.7, 1.7–3.3, ≥3.3). Alcohol consumption was determined as the mean from the first and second 24-hour dietary survey questionnaires and categorized as non-drinkers (0 g/day), moderate drinkers (0–30 g/day) and heavy drinkers (≥30 g/day). Additionally obtained from the questionnaire were data on smoking history and hypercholesterolemia. Diabetes was identified by satisfying any of the following conditions: fasting blood sugar of 126 mg/dL or higher; hemoglobin A1c of 6.5% or above; plasma glucose level of at least 200 mg/dL during an oral glucose tolerance test (OGTT); self-reported diagnosis by a doctor; or self-reported use of blood sugar-lowering medications or insulin. A diagnosis of high blood pressure was confirmed when the average systolic readings were 140 mmHg or higher, or the average diastolic readings were 90 mmHg or above, based on three measurements, combined with either self-reported hypertension or the use of blood pressure medication.

### *2.5 Statistical Analysis*

This research employed normality tests to assess the data distribution and utilized suitable descriptive statistical techniques for both normally and non-normally distributed variables. Wilcoxon rank-sum test was used to analyze continuous variables, with results presented as the mean ± standard deviation (M ± SD). Categorical variables may be numerically represented and in percentages, and assessed via Pearson's Chi-squared test. Multivariate linear regression analysis was used to compute the $\beta$ values and 95% confidence intervals (CI) for evaluating the correlation between ABSI and testosterone. Additionally, logistic regression modeling was applied to ascertain the odds ratio (OR) and 95% CI in order to explore the link between ASBI and TD. We employed three different models for this purpose: Model 1 without adjustment for confounding factors; Model 2 adjusted for age and race; Model 3 adjusted for age, race, marital status, education level, PIR, BMI, diabetes, hypercholesterolemia, hypertension, smoking and alcohol consumption among other confounding variables. Potential nonlinear relationships between ASBI and testosterone or TD were explored using restricted cubic spline (RCS) regression. For the RCS analysis, we placed knots at the recommended default quantiles of the ABSI distribution, specifically at the 5th, 25th, 50th, 75th, and 95th percentiles. This approach ensures a reasonable distribution of flexibility across the range of ABSI values observed in our data. Subgroup analyses based on Model 3 investigated the associations between ASBI and testosterone or TD across various stratifying factors including age, race,

marital status, education level, PIR, smoking, diabetes, hypertension, hyperlipidemia, alcohol consumption and BMI. Interaction tests were conducted to examine consistency in these relationships across different subgroups. Specifically, we employed Likelihood Ratio Test to assess the statistical significance of interaction terms. For each subgroup analysis, we incorporated interaction terms into our fully adjusted regression models (Model 3). For example, when examining interaction by age, we included an interaction term between ABSI (continuous) and age category (as a categorical variable: $<$40, 40–60, $\geq$60) in the linear regression model for testosterone, and similarly in the logistic regression model for TD risk. Pearson correlation analysis examined the correlation between ABSI, BIM, and WC. We utilized receiver operating characteristic (ROC) curves to assess the efficacy of continuous obesity metrics such as BMI, ABSI, $O_{BMI+ABSI}$ and BMI $\times$ ABSI in identifying TD. The study utilized the area under the curve (AUC) and Delong's test to assess variations in the efficacy of each obesity indicator in recognizing TD risk. Additionally, the optimal diagnostic threshold for each indicator was determined using the Youden index. Youden Index was chosen primarily for its simplicity and its intuitive appeal in balancing sensitivity and specificity, aiming to maximize the overall accuracy of the diagnostic test.

## 3. Results

### *3.1 Baseline Characteristics of TD Patients and Individuals With Normal Testosterone Level*

This research involved 5256 individuals in total, among whom 1466 (27.89%) were diagnosed with TD. Comparative findings between men with TD and those exhibiting normal testosterone level are presented in Table 1. Men with TD demonstrated elevated BMI, WC and ASBI, and exhibited a higher prevalence of being over 40 years old, married, having a history of smoking, diabetes, hypertension, and hyperlipidemia compared to men with normal testosterone level. However, individuals with TD reported lower alcohol consumption and had a decreased likelihood of being single compared to those with normal testosterone level.

### *3.2 The Association Between ABSI and TD*

In all statistical models, especially the fully adjusted one, the results suggest an inverse relationship between ABSI and testosterone (Model 3: $\beta$ = –6.99, 95% CI: –8.25 to –5.73, $p < 0.001$). Upon stratifying ABSI into quartiles, this adverse correlation continued to be statistically significant in all of our models (all $p$-values $< 0.001$). Subsequently, a logistic regression study was performed to explore the relationship between ABSI and TD. The findings indicated a positive association between ABSI and TD (Model 3: OR = 1.06, 95% CI: 1.04–1.08, $p < 0.001$). This positive correlation persisted consistently across quartiles and trends (all $p$-values $< 0.001$). Furthermore, individuals in quartile Q4 exhibited significantly higher risk of TD compared to those in quartile Q1 for ABSI (all $p$-values $< 0.001$). Detailed results are presented in Table 2. To assess whether the link between ABSI and testosterone or TD was non-linear, additional RCS analysis was conducted. The outcomes confirmed that the negative correlation between ABSI and testosterone followed a non-linear pattern; notably, this negative association weakened considerably when ABSI surpassed 80.43 ($\beta$ = –0.25, 95% CI: –0.3 to –0.21, $p < 0.001$) (Fig. 2A, Table 3). The positive correlation between ABSI and TD was also non-linear, but there was no correlation between ABSI and TD after ABSI exceeded 84.31, indicating a U-shaped curve relationship (Fig. 2B, Table 3).

### *3.3 Subgroup Analysis Results*

Subsequently, a subgroup analysis was conducted to pinpoint individuals potentially sensitive to the link between ABSI and testosterone and TD and to confirm the strength of this connection across various groups. The findings indicated that, with the exception of the widowed males, the correlation between ASBI and testosterone remained consistent across all subgroups, with age, smoking, hypertension, and BMI demonstrating an interaction effect on ASBI and testosterone. However, apart from participants categorized as other race, widowed, divorced or separated individuals, participants with 9–11th grade education level or those diagnosed with diabetes, the correlation between ASBI and TD exhibited stability across all subgroups except for these specific cases. Additionally, age along with diabetes and hypertension were found to have an interactive influence on ASBI and TD (Table 4).

### *3.4 Evaluation of ABSI and Its Combination With BMI in Detecting TD*

The correlation matrix reveals a statistically significant weak association (r = 0.13, $p < 0.001$) between ABSI and BMI, while a moderate correlation is observed between ABSI and WC (r = 0.46, $p < 0.001$). The relationship between WC and BMI demonstrates high significance (r = 0.92, $p < 0.001$) (Fig. 3). Consequently, we have discontinued the utilization of ABSI and WC for joint prediction of TD risk. Instead, we have proposed two composite indicators, $O_{BMI+ABSI}$ and BMI $\times$ ABSI, and assessed the TD risk in conjunction with the use of ABSI and BMI. ROC curves were utilized to assess the predictive ability of BMI, ABSI, BMI $\times$ ABSI and $O_{BMI+ABSI}$ for TD (Fig. 4). Table 5 presents a summary of the AUC and optimal diagnostic threshold for ABSI, BMI, and their combination. The AUCs for predicting TD were as follows: BMI 0.695 (95% CI: 0.679–0.711), ABSI 0.617 (95% CI: 0.600–0.633), BMI $\times$ ABSI 0.712 (95% CI: 0.697–0.728), and $O_{BMI+ABSI}$ 0.715 (95% CI: 0.700–0.731). Compared to individual predictors like BMI and ABSI, $O_{BMI+ABSI}$ demonstrated the highest AUC in detecting TD among American adult males, with

**Table 1. Patient demographics and baseline characteristics in males aged over 20 years in U.S.**

| Characteristic | Testosterone | | *p*-value |
|---|---|---|---|
| | <300, N = 1466 | ≥300, N = 3790 | |
| Age (year, %) | | | <0.001[b] |
| <40 | 385 (26.3%) | 1432 (37.8%) | |
| 40–60 | 514 (35.1%) | 1231 (32.5%) | |
| ≥60 | 567 (38.7%) | 1127 (29.7%) | |
| Ethnicity (%) | | | 0.074[b] |
| Mexican American | 195 (13.3%) | 481 (12.7%) | |
| Other Hispanic | 134 (9.1%) | 356 (9.4%) | |
| Non-Hispanic white | 662 (45.2%) | 1575 (41.6%) | |
| Non-Hispanic black | 275 (18.8%) | 816 (21.5%) | |
| Other ethnicity | 200 (13.6%) | 562 (14.8%) | |
| Education level (%) | | | 0.427[b] |
| Less than 9th grade | 118 (8.0%) | 283 (7.5%) | |
| 9–11th grade | 189 (12.9%) | 468 (12.3%) | |
| High school | 352 (24.0%) | 883 (23.3%) | |
| Some college or AA degree | 425 (29.0%) | 1068 (28.2%) | |
| College graduate or above | 382 (26.1%) | 1088 (28.7%) | |
| Marital status (%) | | | <0.001[b] |
| Married | 950 (64.8%) | 2030 (53.6%) | |
| Widowed | 63 (4.3%) | 115 (3.0%) | |
| Divorced | 117 (8.0%) | 344 (9.1%) | |
| Separated | 31 (2.1%) | 109 (2.9%) | |
| Never married | 196 (13.4%) | 841 (22.2%) | |
| Living with partner | 109 (7.4%) | 351 (9.3%) | |
| Poverty-income ratio (PIR, %) | | | 0.357[b] |
| <1.7 | 581 (39.6%) | 1465 (38.7%) | |
| 1.7–3.3 | 360 (24.6%) | 1004 (26.5%) | |
| ≥3.3 | 525 (35.8%) | 1321 (34.9%) | |
| Smoking (%) | | | <0.001[b] |
| Yes | 225 (15.3%) | 851 (22.5%) | |
| Former | 552 (37.7%) | 1106 (29.2%) | |
| No | 689 (47.0%) | 1833 (48.4%) | |
| Diabetes (%) | | | <0.001[b] |
| Yes | 438 (29.9%) | 588 (15.5%) | |
| No | 1028 (70.1%) | 3202 (84.5%) | |
| Hypertension (%) | | | <0.001[b] |
| Yes | 752 (51.3%) | 1455 (38.4%) | |
| No | 714 (48.7%) | 2335 (61.6%) | |
| Hypercholesterolemia (%) | | | <0.001[b] |
| Yes | 740 (50.5%) | 1333 (35.2%) | |
| No | 726 (49.5%) | 2457 (64.8%) | |
| Alcohol consumption (gm) (%) | | | 0.002[b] |
| Non-drinkers | 999 (68.1%) | 2384 (62.9%) | |
| Moderate drinkers | 289 (19.7%) | 878 (23.2%) | |
| Heavy drinkers | 178 (12.1%) | 528 (13.9%) | |
| Body mass index (BMI) (kg/m$^2$), M ± SD | 31.8 ± 6.8 | 27.6 ± 5.3 | <0.001[a] |
| Waist circumference (WC) (m), M ± SD | 1.1 ± 0.17 | 0.98 ± 0.14 | <0.001[a] |
| A body shape index (ABSI), M ± SD | 83.5 ± 4.2 | 81.7 ± 4.8 | <0.001[a] |

Abbreviations: ABSI, a body shape index; BMI, body mass index; PIR, poverty-income ratio; M ± SD, mean ± standard deviation; WC, waist circumference.

[a]Continuous variables: mean ± standard deviation, tested by Wilcoxon rank sum test.

[b]Categorical variables: %, tested by Pearson's Chi-squared test.

**Table 2. Association between ABSI and testosterone as well as testosterone deficiency (TD) in males aged over 20 years in U.S.**

| Characteristic | Model $1_a$ | | | Model $2_b$ | | | Model $3_c$ | | |
|---|---|---|---|---|---|---|---|---|---|
| | OR/$\beta$ | 95% CI | $p$-value | OR/$\beta$ | 95% CI | $p$-value | OR/$\beta$ | 95% CI | $p$-value |
| Testosterone (ng/dL) | | | | | | | | | |
| ABSI (continuous) | –8.75 | –9.77, –7.72 | <0.001 | –9.24 | –10.55, –7.92 | <0.001 | –6.99 | –8.25, –5.73 | <0.001 |
| ABSI | | | | | | | | | |
| Quartile 1 | — | — | | — | — | | — | — | |
| Quartile 2 | –72.19 | –86.00, –58.38 | <0.001 | –69.30 | –83.77, –54.84 | <0.001 | –46.97 | –60.56, –33.38 | <0.001 |
| Quartile 3 | –88.68 | –102.49, –74.87 | <0.001 | –86.32 | –101.67, –70.97 | <0.001 | –56.86 | –71.38, –42.34 | <0.001 |
| Quartile 4 | –107.86 | –121.67, –94.05 | <0.001 | –106.77 | –123.76, –89.78 | <0.001 | –78.41 | –94.64, –62.18 | <0.001 |
| $p$ for trend | | | <0.001 | | | <0.001 | | | <0.001 |
| TD | | | | | | | | | |
| ABSI (continuous) | 1.09 | 1.07, 1.10 | <0.001 | 1.08 | 1.07, 1.10 | <0.001 | 1.06 | 1.04, 1.08 | <0.001 |
| ABSI | | | | | | | | | |
| Quartile 1 | — | — | | — | — | | — | — | |
| Quartile 2 | 1.75 | 1.44, 2.12 | <0.001 | 1.67 | 1.37, 2.04 | <0.001 | 1.35 | 1.10, 1.66 | 0.005 |
| Quartile 3 | 2.44 | 2.02, 2.93 | <0.001 | 2.28 | 1.86, 2.80 | <0.001 | 1.73 | 1.39, 2.14 | <0.001 |
| Quartile 4 | 3.06 | 2.55, 3.68 | <0.001 | 2.83 | 2.27, 3.53 | <0.001 | 2.15 | 1.70, 2.71 | <0.001 |
| $p$ for trend | | | <0.001 | | | <0.001 | | | <0.001 |

Abbreviations: TD, testosterone deficiency; OR, odds ratio; CI, confidence interval.

$_a$Model 1: no covariates were adjusted.

$_b$Model 2: adjusted for age and ethnicity.

$_c$Model 3: adjusted for age, ethnicity, marital status, education level, PIR, BMI, diabetes, hypercholesterolemia, hypertension, smoking and alcohol consumption.

**Table 3. Effect of standardized ABSI on testosterone and TD: adjusted coefficients/OR from segmented regression analysis.**

| Association | Characteristic | $\beta$/OR per SD | 95% CI | $p$-value |
|---|---|---|---|---|
| Testosterone | ABSI (<80.43) | –0.25 | –0.30, –0.21 | <0.001 |
| Testosterone | ABSI (≥80.43) | –0.07 | –0.10, –0.03 | <0.001 |
| TD | ABSI (<84.31) | 1.56 | 1.43, 1.70 | <0.001 |
| TD | ABSI (≥84.31) | 0.98 | 0.89, 1.08 | 0.690 |

an optimal diagnostic threshold of 48.808 along with sensitivity and specificity values at 64.3% and 67.1% respectively. However, the AUC, sensitivity, and specificity of BMI × ABSI are slightly lower but comparable to those of $O_{BMI+ABSI}$ (AUC, sensitivity and specificity: 0.712, 64.1% and 66.3%, respectively). Furthermore, it exhibits superior predictive capability for TD in American adult men compared to BMI and ABSI.

## 4. Discussion

This research employed NHANES data to investigate the link between the new obesity measure ABSI and testosterone. Our findings revealed a robust negative correlation between ABSI and testosterone, as well as a positive correlation between ABSI and TD. Even after controlling for demographic characteristics, comorbidities, and dietary factors, the positive correlation remained consistent. Subgroup analysis and interaction test results demonstrated that the association between ABSI and testosterone, as well as the risk of TD, persisted across different subgroups. To our knowledge, this is the first comprehensive study investigat-

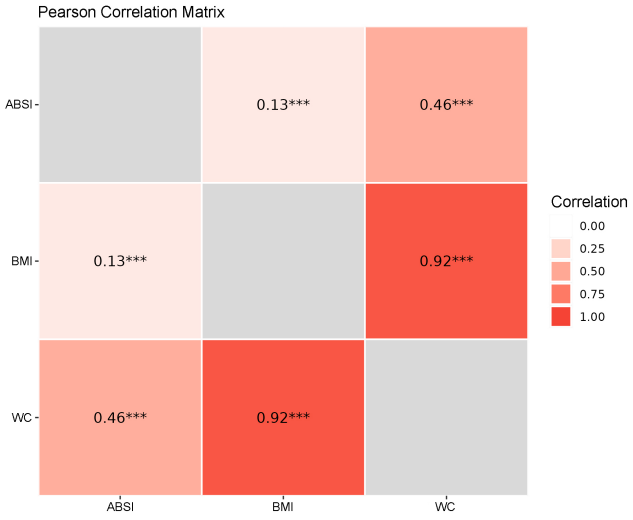


**Fig. 3. The Pearson correlation matrix between ABSI, body mass index (BMI), and waist circumference (WC).** *** is $p$-value < 0.001.

**Table 4. Subgroup analysis of the association between ABSI and testosterone and TD in males aged over 20 years in U.S.**

| Subgroup[a] (N) | Testosterone (ng/dL) | *p*-value | *p* for interaction | TD | *p*-value | *p* for interaction |
|---|---|---|---|---|---|---|
| | $\beta$ (95% CI) | | | OR (95% CI) | | |
| Age | | | <0.001 | | | 0.001 |
| <40 (1850) | –10.89 (–12.98, –8.81) | <0.001 | | 1.12 (1.08–1.17) | <0.001 | |
| 40–60 (1900) | –4.89 (–7.09, –2.69) | <0.001 | | 1.04 (1.01–1.07) | 0.018 | |
| ≥60 (1506) | –5.56 (–7.91, –3.22) | <0.001 | | 1.05 (1.02–1.08) | 0.002 | |
| Ethnicity | | | 0.080 | | | 0.236 |
| Mexican American (680) | –5.78 (–9.07, –2.49) | 0.001 | | 1.08 (1.03–1.14) | 0.003 | |
| Other Hispanic (450) | –7.89 (–12.44, –3.34) | 0.001 | | 1.10 (1.02–1.18) | 0.010 | |
| Non-Hispanic white (2200) | –6.68 (–8.64, –4.73) | <0.001 | | 1.06 (1.03–1.09) | <0.001 | |
| Non-Hispanic black (900) | –10.32 (–13.21, –7.42) | <0.001 | | 1.12 (1.07–1.16) | <0.001 | |
| Other ethnicity (626) | –4.18 (–7.45, –0.90) | 0.013 | | 1.02 (0.97–1.07) | 0.498 | |
| Education level | | | 0.481 | | | 0.432 |
| Less than 9th grade (350) | –10.29 (–15.42, –5.17) | <0.001 | | 1.15 (1.07–1.23) | <0.001 | |
| 9–11th grade (400) | –6.77 (–10.56, –2.99) | <0.001 | | 1.04 (0.98–1.09) | 0.201 | |
| High school (1100) | –7.51 (–10.18, –4.84) | <0.001 | | 1.06 (1.02–1.10) | 0.002 | |
| Some college or AA degree (1000) | –6.69 (–8.93, –4.45) | <0.001 | | 1.07 (1.04–1.11) | <0.001 | |
| College graduate or above (2406) | –6.19 (–8.58, –3.80) | <0.001 | | 1.05 (1.01–1.09) | 0.007 | |
| Marital status | | | 0.781 | | | 0.613 |
| Married (3000) | –6.46 (–8.09, –4.83) | <0.001 | | 1.07 (1.04–1.09) | <0.001 | |
| Widowed (200) | –6.14 (–15.06, 2.78) | 0.179 | | 1.03 (0.94–1.13) | 0.565 | |
| Divorced (350) | –5.24 (–10.01, –0.48) | 0.031 | | 1.04 (0.98–1.11) | 0.206 | |
| Separated (100) | –9.24 (–17.97, –0.52) | 0.040 | | 1.13 (0.98–1.31) | 0.086 | |
| Never married (700) | –7.41 (–10.32, –4.50) | <0.001 | | 1.07 (1.02–1.12) | 0.003 | |
| Living with partner (506) | –10.67 (–15.01, –6.33) | <0.001 | | 1.11 (1.03–1.19) | 0.007 | |
| PIR | | | 0.457 | | | 0.953 |
| <1.7 (1800) | –7.33 (–9.46, –5.21) | <0.001 | | 1.07 (1.04–1.10) | <0.001 | |
| 1.7–3.3 (1200) | –6.71 (–9.06, –4.36) | <0.001 | | 1.06 (1.02–1.10) | 0.004 | |
| ≥3.3 (2256) | –6.39 (–8.49, –4.28) | <0.001 | | 1.06 (1.03–1.10) | <0.001 | |
| BMI | | | 0.002 | | | 0.182 |
| <25 (1500) | –8.28 (–10.91, –5.66) | <0.001 | | 1.07 (1.02–1.11) | 0.002 | |
| 25–30 (1800) | –6.30 (–8.33, –4.28) | <0.001 | | 1.06 (1.03–1.10) | <0.001 | |
| ≥30 (1956) | –5.67 (–7.74, –3.60) | <0.001 | | 1.06 (1.03–1.09) | <0.001 | |
| Diabetes | | | 0.066 | | | 0.014 |
| No (3700) | –7.79 (–9.20, –6.38) | <0.001 | | 1.08 (1.06–1.10) | <0.001 | |
| Yes (1556) | –4.26 (–7.07, –1.44) | 0.003 | | 1.02 (0.98–1.06) | 0.334 | |
| Hypercholesterolemia | | | 0.095 | | | 0.159 |
| No (3500) | –8.20 (–9.84, –6.57) | <0.001 | | 1.08 (1.05–1.11) | <0.001 | |
| Yes (1756) | –4.73 (–6.73, –2.73) | <0.001 | | 1.04 (1.02–1.07) | 0.002 | |
| Hypertension | | | 0.003 | | | 0.004 |
| No (3200) | –8.84 (–10.47, –7.21) | <0.001 | | 1.10 (1.07–1.12) | <0.001 | |
| Yes (2056) | –4.46 (–6.46, –2.46) | <0.001 | | 1.03 (1.00–1.06) | 0.034 | |
| Smoking | | | 0.041 | | | 0.419 |
| No (2300) | –6.41 (–8.12, –4.71) | <0.001 | | 1.06 (1.04–1.09) | <0.001 | |
| Former (1800) | –7.87 (–10.19, –5.55) | <0.001 | | 1.08 (1.04–1.11) | <0.001 | |
| Yes (1156) | –7.30 (–10.37, –4.22) | <0.001 | | 1.05 (1.01–1.10) | 0.017 | |
| Alcohol consumption | | | 0.621 | | | 0.324 |
| ≤0 (2500) | –6.54 (–8.09, –5.00) | <0.001 | | 1.06 (1.03–1.08) | <0.001 | |
| 0–30 (1500) | –6.59 (–9.47, –3.70) | <0.001 | | 1.05 (1.01–1.10) | 0.011 | |
| >30 (1256) | –9.41 (–12.78, –6.04) | <0.001 | | 1.11 (1.05–1.17) | <0.001 | |

[a]All subgroup analyses were adjusted for age, ethnicity, marital status, education level, PIR, BMI, diabetes, hypercholesterolemia, hypertension, smoking and alcohol consumption.

**Table 5. The cutpoint, sensitivities, specificities, Youden's index, and area under curve (AUC) of $O_{BMI+ABSI}$, BMI × ABSI, ABSI and BMI.**

| Predictor | AUC | 95% CI | Sensitivity | Specificity | Youden's Index | Cutpoint |
|---|---|---|---|---|---|---|
| $O_{BMI+ABSI}$ | 0.715 | 0.700–0.731 | 64.3% | 67.1% | 0.314 | 48.808 |
| ABSI × BMI | 0.712 | 0.697–0.728 | 64.1% | 66.3% | 0.304 | 2389.261 |
| ABSI | 0.617 | 0.600–0.633 | 69.6% | 48.8% | 0.184 | 81.428 |
| BMI | 0.695 | 0.679–0.711 | 66.3% | 62.1% | 0.284 | 28.400 |

Abbreviations: AUC, area under curve.

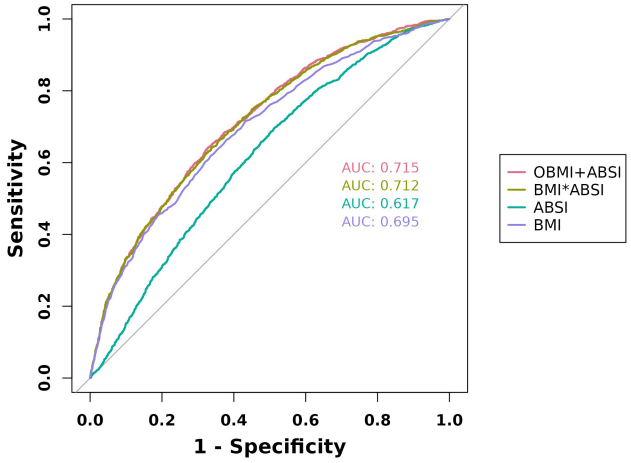


**Fig. 4. Receiver operating characteristic (ROC) curve analysis of optimal proportion coefficient ABSI, BMI, and their multiplicative combination ABSI × BMI and optimal proportional combination $O_{BMI+ABSI}$ in males aged over 20 years in U.S.**

ing the link between ABSI and TD risk using nationally representative data.

ABSI was found to have a significant negative correlation with serum total testosterone levels and a positive correlation with the risk of TD. Our findings not only support the established association between traditional adiposity measures BMI and WC and TD, but more importantly, they reveal that ABSI, which is based on body size distribution, may serve as a more precise clinical predictor [30–36]. While BMI and WC primarily reflect global or regional fat accumulation, ABSI offers a more sensitive measure of the ectopic distribution of abdominal fat by integrating height, weight, and WC data. This characteristic may elucidate the stronger association observed between ABSI and testosterone compared to traditional measures in the present study. The preferential accumulation of abdominal fat may contribute to TD risk through a dual mechanism: firstly, visceral adipose tissue exhibits higher aromatase activity, potentially accelerating the conversion of testosterone to estrogen, which inhibits the function of the hypothalamic-pituitary-gonadal (HPG) axis via a negative feedback mechanism [37–39]. Secondly, central obesity, as characterized by ABSI, may be spatially linked to an imbalance in adipocytokine secretion (e.g., leptin resistance, proinflammatory factor release) [40,41]. ABSI maintained its independent association with TD risk, suggesting that the pattern of abdominal fat distribution itself may directly influence the steroidogenic function of Leydig cells through oxidative stress and a localized inflammatory microenvironment [42]. The correlation between ABSI, testosterone, and the TD risk suggests that interventions aimed at reducing abdominal fat—such as high-intensity interval training and medications targeting visceral fat—may be more effective than traditional weight-loss strategies in enhancing gonadal function [35,40,43].

Common risk factors contributing to reduced or insufficient testosterone include not only obesity but also advanced age, smoking habits, hypertension as well as obesity-related conditions. Subgroup analysis within ABSI reveals that age along with hypertension serves as a significant regulatory element for testosterone insufficiency across all subgroups. Consistent with previous research, smoking and alcohol consumption have been associated with testosterone decreases and the risk of TD [44,45]. Among non-drinkers, each additional unit of ABSI is linked to a lower decrease in testosterone levels and reduced risk of hypogonadism compared to heavy drinkers. Furthermore, non-smokers and former smokers exhibit a lesser degree of testosterone decline and decreased risk of hypogonadism when compared to smokers. The mechanisms underlying these associations involve heightened concentrations from prolonged alcohol intake stimulating hypothalamus-pituitary-adrenal axis activity thereby inducing inflammation alongside oxidative stress while concurrently inhibiting HPG axis function resulting in decreased production rates within hepatic sex hormone-binding globulin (SHBG) ultimately culminating in lowered serum concentrations [46,47]. Furthermore, tobacco exposure emerges as another pertinent contributor towards diminished Testosterone; it has been shown capable even among rodent models [48]. Smoking elevates hepatic testosterone metabolism, leading to reduced testosterone level [49]. Additionally, the cigarette metabolite cotinine disrupts the equilibrium between total antioxidant capacity and ROS production, resulting in heightened levels of active oxygen, oxidative stress, increased lipid peroxidation, diminished antioxidant activity, and amplified toxic effects on Leydig cells with consequent impairment of testosterone synthesis [50,51]. In summary, chronic smoking coupled with excessive alcohol usage fosters accruals pertaining to ROS elevation

alongside augmented lipid peroxidation thus interfering with pathways governing biosynthesis consequently depleting circulating hormone quantities whilst elevating risks associated with TD. Conversely, individuals afflicted by comorbidities such as hypertension, diabetes and hypercholesterolemia have a lesser decrease in their testosterone level per unit of ABSI and a diminished likelihood of developing TD. Furthermore, obesity can also contribute to metabolic disturbances that precipitate the onset of diabetes and high cholesterol. Patients with diabetes frequently encounter insulin resistance, hyperlipidemia and elevated cholesterol levels—all of which are notable risk factors for TD [1,9]. Despite these factors increasing the likelihood of TD, individuals with comorbid conditions such as hypertension, diabetes and hypercholesterolemia may prioritize lifestyle and dietary management, enhanced physical activity and weight control [52]. Meanwhile, our data collection on subjects with hypertension, diabetes and hypercholesterolemia includes individuals who are undergoing medication for reducing their blood pressure, glucose level and cholesterol level. These medication variables may attenuate the observed correlation between ABSI and TD. Naturally, future cohort studies are imperative for further elucidating the interplay between these factors in individuals with hypertension diabetes and elevated cholesterol level.

Currently, researchers have identified numerous indicators related to obesity, such as BMI, WC, WHR and WHtR. These indicators are utilized for assessing the degree of obesity and investigating the correlation between obesity and disease risk [23]. However, these indicators are not comprehensive in detecting the degree of obesity and phenotypes. Their associations and predictive strengths vary across different diseases [23,53,54]. For instance, BMI fails to differentiate between fat and muscle mass or reflect body fat distribution. Therefore, in the assessment of testosterone deficiency risk among obese patients, a supplementary index is essential alongside BMI or WC. ABSI is a recently introduced parameter that incorporates WC, body weight, and height to characterize body shape [24]. From a statistical perspective, ABSI differs from height, BMI and WC by better reflecting visceral fat accumulation degree and fat distribution associated with endocrine and metabolic disorders compared to traditional body measurement indices. Our study demonstrates a negative correlation between ABSI and TD. Moreover, regardless of BMI strata within each subpopulation group ABSI consistently correlates with TD risk. Interestingly enough previous research has shown an inverse relationship between ABSI and lean mass [55]. Modified ABSI also exhibits a positive association with fat mass index but a negative association with appendicular skeletal mass index [56]. Additionally, several studies indicate high sensitivity of ABSI in predicting metabolic disease risk but low specificity/accuracy suggesting it may not be suitable as a standalone indicator for detecting metabolic disease risk [57]. Nevertheless, recent research from Japan evaluated the combination of ABSI, BMI and non-alcoholic fatty liver disease (NAFLD) risk, finding that combining BMI with ABSI significantly outperforms individual predictive abilities of BMI or ABSI alone and further improves NAFLD risk prediction [29].

Furthermore, the researchers conducted a study involving 11,872 Taiwanese participants aged 30 and above who have diabetes. They utilized various anthropometric indexes, including ABSI, BMI, WC, WHR, WHtR and Body Roundness Index (BRI), either individually or in combination, to predict mortality risk among the diabetic population. The findings revealed that the combined effect of BMI and ABSI on mortality risk was greater than their individual effects and surpassed the combined effects of other anthropometric measures [58]. Consequently, ABSI is likely to serve as a complementary predictor of TD risk in addition to BMI. Our study has validated these findings, demonstrating that the combined predictive ability of ABSI and BMI for TD in American adult men surpasses their individual predictive abilities. Furthermore, our results indicate that $O_{BMI+ABSI}$ outperforms BMI $\times$ ABSI in predicting TD, suggesting the need for further research across a broader spectrum of metabolic diseases to confirm the superiority of combined indicators.

Our research has multiple advantages, being the first and most extensive examination of the relationship between ABSI, testosterone and TD risk. Moreover, the extensive and dependable NHANES dataset empowers us to explore potential variables that could impact the relationship between ABSI and testosterone. Additionally, we conducted subgroup analyses across diverse populations to assess the strength of the relationship between ABSI, testosterone, and TD. Our research also indicates that ABSI serves as a valuable complement to BMI in assessing obesity and its relation to TD, with their combined predictive capacity for TD risk surpassing that of either measure alone. The Youden Index maximizes the sum of sensitivity and specificity, effectively identifying a threshold that optimizes the ability of the test to correctly classify both those with and without the condition (TD in this case). This approach is particularly useful when equal weight is given to the importance of correctly identifying both true positives (those with TD) and true negatives (those without TD). Consequently, our study results hold significant public health implications for obesity management and TD prevention.

Nevertheless, this study is subject to certain constraints. Firstly, it is a cross-sectional study, precluding the establishment of a causal relationship between ABSI and TD. As the original data for this study was sourced from the NHANES database, the findings derived from the analysis are only generalizable to the US population and may not be extrapolated to other global regions. Secondly, there exist numerous potential confounding factors influencing BMI, weight-adjusted waist index (WWI), ABSI, and TD; some of these warrant further investigation by re-

searchers. Given our inability to account for all potential confounders using the NHANES database, caution should be exercised in interpreting our results and subsequent research should validate the conclusions drawn from this study. The collection of a substantial number of questionnaires in this study encompassed dietary factors, comorbidities, and demographic information reliant on self-reporting by subjects. Consequently, introducing bias due to missing data. Furthermore, as NHANES' medical questionnaire lacked comprehensive information on testosterone and TD symptoms/signs/comorbidities/laboratory data related to TD were not considered. TD is multifaceted beyond being merely a biochemical marker and necessitates consideration alongside other clinical conditions. However, owing to constraints within the NHANES database we could only define TD as total testosterone below 300 ng/dL; additionally lacking access to testicular examination/imaging data which could have been included as covariates in our study. Last but not least, it is important to acknowledge that the Youden Index has limitations and may not be the most appropriate threshold selection criterion in all contexts. A key limitation is that the Youden Index gives equal weight to sensitivity and specificity and does not consider the relative clinical consequences of false positives versus false negatives, or the prevalence of the condition in the population.

Another important consideration is the generalizability of our findings, which are primarily based on data from the NHANES survey, a nationally representative sample of the U.S. adult population. While NHANES provides a robust snapshot of the U.S., it is essential to acknowledge that the observed associations may not directly translate to other populations globally due to several factors.

Despite our efforts to adjust for a comprehensive set of known confounders, it is essential to acknowledge the inherent limitation of observational studies in fully accounting for all potential confounding variables. Unmeasured confounders—factors that are associated with both ABSI and testosterone levels/TD risk but were not directly assessed or adequately controlled for in the NHANES dataset—could potentially influence our findings and introduce bias. For example, physical activity levels, while partially captured through general survey questions in NHANES, may not have been assessed with sufficient detail or granularity to fully account for its complex influence. Individuals with higher levels of physical activity might tend to have both lower ABSI (due to better body composition) and healthier testosterone profiles, independent of a direct causal link between ABSI and testosterone [59]. Similarly, genetic factors influencing both body fat distribution patterns (and thus ABSI) and hormonal regulation could act as unmeasured confounders. While we adjusted for race/ethnicity as a proxy for some genetic background, this is a crude measure and does not capture the full spectrum of genetic variability that might predispose individuals to certain body shapes and hormonal profiles [60]. For instance, certain genetic variants might predispose individuals to both higher visceral adiposity (potentially leading to higher ABSI) and a tendency towards lower testosterone levels, again creating an association that is not directly causal. Considering the calculation formula for ABSI, WC is positioned in the numerator and its error directly and linearly affects ABSI. Despite the large-scale nature of NHANES measurements, staff techniques, cooperation and physiological conditions of the participants, measurement timing, and equipment precision give rise to some inevitable errors in the measurements of WC, height, and weight. Underestimation of height concurrently increases BMI and decreases height terms, ultimately resulting in an underestimation of ABSI. Therefore, ABSI is more sensitive to WC error than BMI or WC alone as its calculation involves multivariable interactions. In the future, sensitivity analyses can be carried out to simulate the robustness of the association between ABSI and outcomes under different error margins. Another way to reduce the error is to verify the reproducibility of ABSI in a multicenter study and clarify its clinical application threshold.

## 5. Conclusions

This study has unveiled a negative linear correlation between ABSI and testosterone in U.S. male adults aged 20 and above, while also identifying a positive linear correlation between ABSI and the incidence of TD. We acknowledge that, like all observational studies, our findings are susceptible to the influence of unmeasured confounding factors, such as physical activity levels and genetic predispositions, which could partially explain the observed associations. It is important to note that, due to the cross-sectional design of our study, we have established associations, but cannot infer causal relationships between ABSI and testosterone or TD. In future applications for the prevention and management of TD, particular attention should be given to individuals aged 20–40 without hypertension. Additionally, consideration should be directed towards individuals aged 60 and older, non-Hispanic blacks, those with less than a 9th-grade education, individuals with PIR $<1.7$, smokers, drinkers, as well as those diagnosed with diabetes, hypertension, and hypercholesterolemia. ABSI serves as a valuable complement to BMI in assessing obesity and its association with TD. The combined use of ABSI and BMI demonstrates stronger predictive capabilities for the risk of TD compared to their individual use alone. These findings enhance our understanding of low testosterone levels related to obesity and offer fresh insights into the prevention and management of TD alongside male health. Future longitudinal studies are needed to elucidate potential causal pathways and confirm the directionality of the observed associations.

## Availability of Data and Materials

The study includes the original contributions, which are detailed in the article. For additional information, inquiries may be directed to the corresponding author.

## Author Contributions

FG and CD designed this study. FG and PL analyzed the data and wrote the original draft. All authors have contributed to the editorial changes made to the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

## Ethics Approval and Consent to Participate

The data for this study are all from the NHANES database and do not require ethical approval. The NHANES database has been approved by the National Health Statistics Ethics Review Board in the U.S., and all participants have provided written informed consent prior to data collection.


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## Conflict of Interest

The authors declare no conflict of interest.

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