Association between weight-adjusted waist index and thoracic cancer: evidence from two national population surveys

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Abstract

Introduction: Obesity is closely associated with the risk of cancers. This study analyzed national population surveys from the United States and China to investigate the association between weight-adjusted waist index (WWI) and thoracic cancer.

Material and methods: Data from two national population surveys were used in the study. Logistic regression analysis was used to analyze the association between WWI and thoracic cancer. Restricted cubic spline (RCS) analysis was used to investigate a potential nonlinear relationship. The stability of this relationship across different subgroups was assessed through subgroup analysis. ROC curve analysis facilitated the evaluation of different obesity indicators in terms of their efficacy in predicting thoracic cancer.

Results: A relationship between thoracic cancer and WWI was identified. RCS analysis confirmed the nonlinear relationship between thoracic cancer and WWI (p for nonlinear < 0.001). The analysis of subgroups indicated that the association between thoracic cancer and WWI was broadly applicable across different populations, further confirming the robustness of the study findings. ROC analysis showed that WWI possessed satisfactory predictive capability for thoracic cancer.

Conclusions: The study showed that WWI was independently associated with thoracic cancer prevalence in both the United States and Chinese populations. Furthermore, the accuracy of WWI in predicting thoracic cancer risk surpasses that of conventional obesity indicators.

Key words: thoracic cancer, weight-adjusted waist index, lung cancer, obesity.

Introduction

Cancer is among the most prevalent diseases on a global scale, representing a grave threat to public health. According to global cancer statistics [1], each year, approximately 20 million new cancer cases are diagnosed. Lung cancer and esophageal cancer are among the common types of thoracic cancers. Annually, more than 2 million new diagnoses of lung cancer are made, resulting in approximately 1.8 million mortalities. Globally, 604,000 new esophageal cancer cases are diagnosed, resulting in 544,000 deaths [2]. The majority of cases are identified in the later stages [3], resulting in a five-year survival rate that is generally under

20%. The development of cancer is related to a variety of lifestyle factors. Extensive research has indicated a direct correlation between smoking and lung cancer [4]. Furthermore, it has been identified that excessive alcohol consumption is associated with elevated risk of various cancers [5–7]. Moreover, research shows an association between unhealthy diets and colorectal cancer [8]. A sedentary lifestyle has been proven to correlate with a greater likelihood of developing various cancers [9–11]. The early detection of tumor-related risk factors is of particular importance.

The weight-adjusted waist index (WWI) is a recently developed indicator that integrates weight and waist circumference data to more accurately reflect visceral fat distribution and central obesity than traditional indicators [12]. Obesity is widely regarded as a significant risk factor for developing cancer, and conventional obesity indicators have been demonstrated to correlate with diverse cancer types [13–17]. Research findings have demonstrated a correlation between elevated body mass index (BMI) and increased risk of lung cancer [18]. Research has indicated that obesity, as measured by BMI, is associated with increased overall survival in individuals with lung cancer who have undergone surgical resection [19]. Furthermore, research has demonstrated that higher BMI is associated with an increased risk of esophageal adenocarcinoma. For instance, individuals with a BMI ≥ 35 kg/m^2 exhibit a 2.5-fold elevated risk of developing esophageal adenocarcinoma in comparison to those within the normal BMI range [20]. WWI may also be applicable to the risk assessment of thoracic cancer.

The relationship between WWI and thoracic cancer has yet to be fully clarified. This study utilizes data from the China Health and Retirement Longitudinal Study (CHARLS) and the National Health and Nutrition Examination Survey (NHANES) to further analyze the association between WWI and thoracic cancer.

Material and methods

Study design and participants

Data from CHARLS and NHANES surveys were used in the study. These two surveys evaluate nutrition, health and socioeconomic conditions of adults in China and the United States, respectively. CHARLS is a national representative longitudinal study that is currently ongoing. It received approval from the Peking University Ethics Review Committee and is administered by the China Center for Economic Research. NHANES is a program conducted by the Centers for Disease Control and Prevention (CDC) and administered by the National Center for Health Statistics (NCHS). In 2011, baseline data were collected from CHARLS, with follow-up data collected in 2013, 2015, and 2018, involving 17,708 participants in total. In addition, data from NHANES from 1999 to 2018 were collected, and a total of 101,316 participants were screened. Prior to enrollment, every participant provided written informed consent. Figure 1 shows the specific criteria for inclusion and exclusion.

Measurement of WWI

The formula for WWI involves dividing the waist circumference in cm by the square root of the weight in kg. Professional recorders and trained medical staff meticulously document human measurements to maintain data accuracy.

Diagnosis of cancer

The study collected information on cancer prevalence from self-administered question-

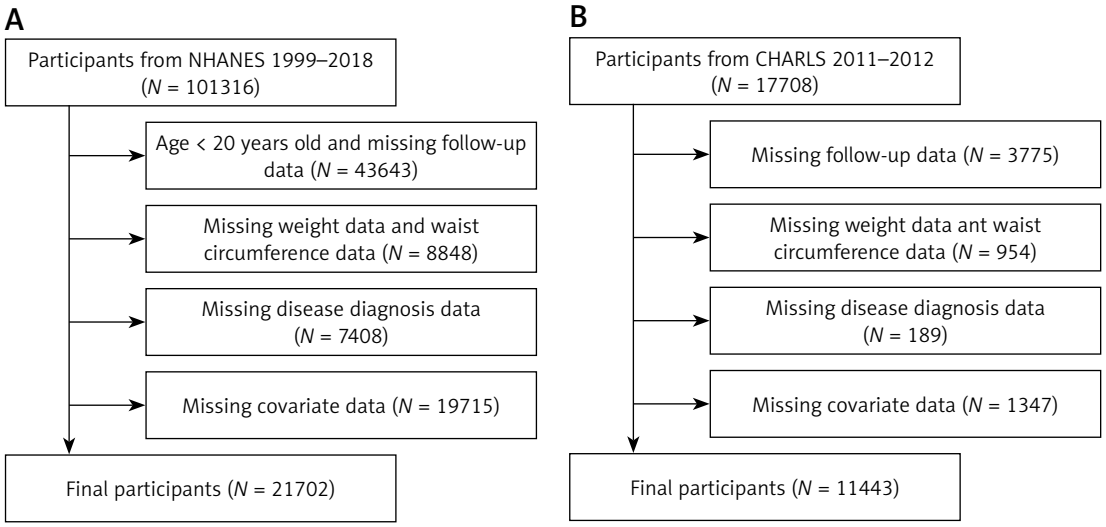

Figure 1. Flowchart of participant selection from NHANES 1999–2018 (**A**) and CHARLS 2011–2012 (**B**)

naires obtained from CHARLS and NHANES. In both studies, participants were queried with inquiries such as, "Have you been informed by a medical professional about a diagnosis of cancer?" and "Have you been informed by a medical professional about a diagnosis of lung cancer, esophageal cancer, or any other type of thoracic cancer?" Participants who responded in the affirmative were subsequently classified as having thoracic cancer.

Definitions of covariates

This study also considered the potential for confounding factors that could influence the association between thoracic cancer and WWI. These covariates include demographic data (including age, sex, and economic status), health status data (including BMI, smoking, drinking, history of hypertension, and history of diabetes), and laboratory-related data (including triglycerides, high-density lipoprotein, creatinine, and uric acid).

Statistical analysis

In this study, continuous variables were analyzed using *t*-tests, whereas categorical variables were examined with χ^2 tests. Weight adjustments were made in accordance with the relevant regulations for both database studies. According to WWI, three-digit groups were identified, designated T1, T2, and T3. Logistic regression analysis was used to evaluate the association between thoracic cancer and WWI. Four models were constructed in the NHANES and CHARLS studies, respectively. No adjustments for covariates were made in Model 1. Model 2 was adjusted for age, sex, and income. With these adjustments being based on Model 2, Model 3 made further adjustments to medical history indicators, including alcohol use, smoking status, hypertension, and diabetes. Model 4 made further adjustments to laboratory indicators such as triglycerides, uric acid, creatinine, and high-density lipoprotein, as based on Model 3. In Model 4, we used restricted cubic splines (RCS) to examine the nonlinear connection between WWI and tho-

Table I. Characteristics of the NHANES study population

Characteristics	Normal (*N* = 21,012)	Thoracic cancer (*N* = 690)	*P*-value
Age [years]	50.18 ±10.66	53.77 ±10.16	< 0.001
Sex, *n* (%)			< 0.001
Female	10,236 (48.72)	274 (39.71)	
Male	10,776 (51.28)	416 (60.29)	
Poverty income ratio (%)			< 0.001
< 1.3	6,516 (31.01)	197 (28.55)	
1.3–3.5	8,826 (42.01)	324 (46.96)	
≥ 3.5	5,670 (26.98)	169 (24.49)	
Alcohol use (%)			< 0.001
No	11,031 (52.50)	374 (54.20)	
Yes	9,981 (48.50)	316 (45.80)	
Smoking status (%)			< 0.001
No	12,544 (59.70)	283 (41.01)	
Yes	8,468 (40.30)	407 (58.99)	
BMI [kg/m^2]	30.22 ±6.67	31.06 ±7.07	0.008
Diabetes (%)			< 0.001
No	18,428 (87.70)	535 (77.54)	
Yes	2,584 (12.30)	155 (22.46)	
Hypertension (%)			< 0.001
No	17,438 (82.99)	559 (81.02)	
Yes	3,574 (17.01)	131 (19.98)	
Triglycerides [mg/dl]	157.07 ±46.34	149.77 ±48.86	< 0.001
HDL-C [mg/dl]	47.06 ±8.08	48.33 ±8.57	< 0.001
Creatinine [mg/dl]	1.01 ±0.48	1.07 ±0.39	0.005
Uric acid [mg/dl]	5.97 ±1.12	5.66 ±1.03	< 0.001

Percentages represent categorical variables, and continuous variables are shown as mean ± standard deviation. HDL-C – high-density lipoprotein cholesterol.

racic cancer. Subgroup analyses were performed for age, sex, alcohol use, smoking status, hypertension, and diabetes. Furthermore, ROC curve analysis was employed to evaluate the predictive capacity of WWI in the context of thoracic cancer. R 4.4.1 software was used for data analysis, with $p < 0.05$ regarded as statistically significant.

Results

Baseline characteristics

Data on 21,702 NHANES participants and 11,443 CHARLS participants were utilized. Tables I and II display their specific characteristics. In the baseline table, "normal population" refers to individuals who have not received a thoracic cancer diagnosis. A comparison of the basic information of the populations in the two databases revealed a degree of similarity. For instance, patients diagnosed with thoracic cancer are typically older, predominantly male, and have a history of smoking.

The relationship between WWI and thoracic cancer

The results of the multifactor logistic regression analysis are detailed in Table III. In both the NHANES and CHARLS studies, a significant association was observed between WWI and thoracic cancer. Thoracic cancer risk rises with an increase in WWI. For further analysis, we converted WWI into categorical variables (T1, T2, and T3). The NHANES study revealed that, in Model 4A, thoracic cancer risk increased by 36% with each one-unit increment in WWI (OR = 1.36 95% CI: 1.08–1.83, $p < 0.05$). A comparison of the T1A group with the T2A and T3A groups revealed an elevated risk of thoracic cancer, with an increase of 25% and 62%, respectively (p for trend = 0.002). The CHARLS study indicated that in Model 4B, thoracic cancer risk increased by 8% with each one-unit increment in WWI (OR = 1.08 95% CI: 1.02–1.13, $p < 0.05$). A comparison of the T1B group with the T2B and T3B groups revealed an elevated risk of thoracic cancer, with an increase of 19% and 31%, respectively (p for trend < 0.001).

Table II. Characteristics of the CHARLS study population

Characteristics	Normal (*N* = 11,331)	Thoracic cancer (*N* = 112)	*P*-value
Age [years]	58.71 ±9.56	55.36 ±8.87	0.006
Sex, *n* (%)			< 0.001
Female	5,410 (47.75)	43 (48.39)	
Male	5,921 (52.25)	69 (61.61)	
Income [yuan]	20,037 ±8869	19,136 ±7796	< 0.001
Alcohol use (%)			< 0.001
No	7,308 (64.50)	73 (65.18)	
Yes	4,023 (35.50)	39 (34.82)	
Smoking status (%)			< 0.001
No	6,538 (57.70)	46 (41.07)	
Yes	4,793 (42.30)	66 (58.93)	
BMI, *n* (%)			< 0.001
Underweight	673 (5.94)	7 (6.25)	
Normal	6,684 (58.99)	69 (61.60)	
Overweight	2,821 (24.89)	30 (26.79)	
Obesity	1,153 (10.18)	6 (5.36)	
Diabetes (%)			< 0.001
No	9,484 (83.70)	88 (78.57)	
Yes	1,847 (16.30)	24 (21.43)	
Hypertension (%)			< 0.001
No	6,708 (59.20)	65 (58.04)	
Yes	4,623 (40.80)	47 (41.96)	
Triglycerides [mg/dl]	106.37 ±37.22	116.67 ±40.05	0.033
HDL-C [mg/dl]	49.16 ±8.32	46.87 ±8.67	0.013
Creatinine [mg/dl]	0.78 ±0.22	0.86 ±0.21	0.002
Uric acid [mg/dl]	4.47 ±0.92	4.83 ±0.87	< 0.001

Percentages represent categorical variables, and continuous variables are shown as mean ± standard deviation. HDL-C – high-density lipoprotein cholesterol.

Table III. The relationship between WWI and thoracic cancer

Characteristic	OR (95% CI), *p*-value			
NHANES	**Model 1A**	**Model 2A**	**Model 3A**	**Model 4A**
WWI (Continuous)	1.74 (1.37, 2.22), < 0.001	1.53 (1.12, 1.97), 0.007	1.49 (1.13, 1.99), 0.005	1.36 (1.08, 1.83), 0.011
Categories				
T1A	Reference	Reference	Reference	Reference
T2A	1.50 (1.11, 2.59), 0.010	1.36 (1.08, 2.42), 0.019	1.32 (1.05, 2.36), 0.021	1.25 (1.06, 2.15), 0.009
T3A	2.44 (1.51, 3.97), < 0.001	1.88 (1.14, 3.22), 0.014	1.74 (1.03, 3.18), 0.039	1.62 (1.01, 2.78), 0.045
P for trend	< 0.001	< 0.001	0.003	0.002
CHARLS	Model 1B	Model 2B	Model 3B	Model 4B
WWI (Continuous)	1.45 (1.39, 1.52), < 0.001	1.24 (1.18, 1.31), < 0.001	1.12 (1.06, 1.19), < 0.001	1.08 (1.02, 1.13), 0.007
Categories				
T1B	Reference	Reference	Reference	Reference
T2B	1.61 (1.42, 1.83), < 0.001	1.40 (1.21, 1.61), < 0.001	1.38 (1.13, 1.51), 0.002	1.19 (1.04, 1.35), 0.012
T3B	2.15 (1.91, 2.45), < 0.001	1.88 (1.70, 2.24), < 0.001	1.52 (1.31, 1.75), < 0.001	1.31 (1.12, 1.51), < 0.001
P for trend	< 0.001	< 0.001	< 0.001	< 0.001

Model 1 did not include any adjustments for covariates; Model 2: adjusted for age, sex, and income; Model 3: Model 2 + adjusted for alcohol use, smoking status, diabetes and hypertension; Model 4: Model 3 + adjusted for triglycerides, creatinine, HDL-C, and uric acid.

Nonlinear association between WWI and thoracic cancer

We applied RCS analysis to assess the nonlinear association between WWI and thoracic cancer. As demonstrated in Figures 2 and 3, a significant nonlinear positive association was found between WWI and thoracic cancer (*p* for nonlinear < 0.001). This suggests that in both populations, the risk of thoracic cancer increases with increasing WWI.

Subgroup analyses

The analyses of subgroups were performed based on age, sex, smoking status, alcohol use, hypertension, and diabetes to further investigate the reliability of the observed associations. As demonstrated in Figures 4 and 5, no substantial interaction was identified between subgroups (*p* for interaction > 0.05). The findings from the analyses of subgroups reveal that the association between thoracic cancer risk and WWI is broadly applicable across different populations, further confirming the robustness of the study findings.

Predictive ability of different obesity indicators

Figure 6 shows the results of the ROC curve analysis. In the NHANES study, WWI AUC (95% CI):

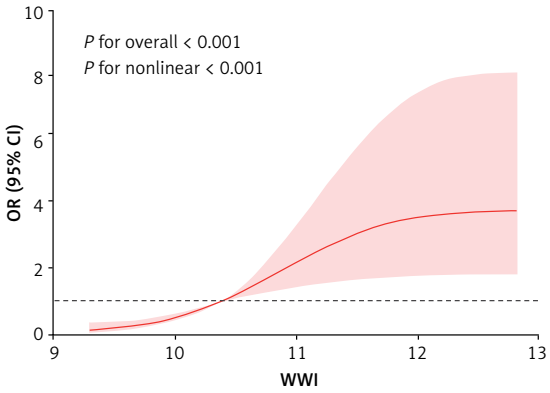

Figure 2. Nonlinear association between WWI and thoracic cancer in NHANES

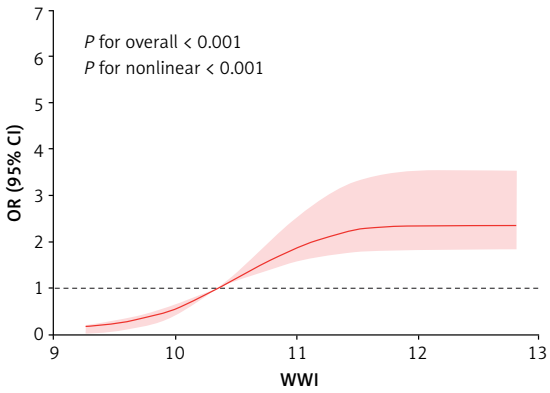

Figure 3. Nonlinear association between WWI and thoracic cancer in CHARLS

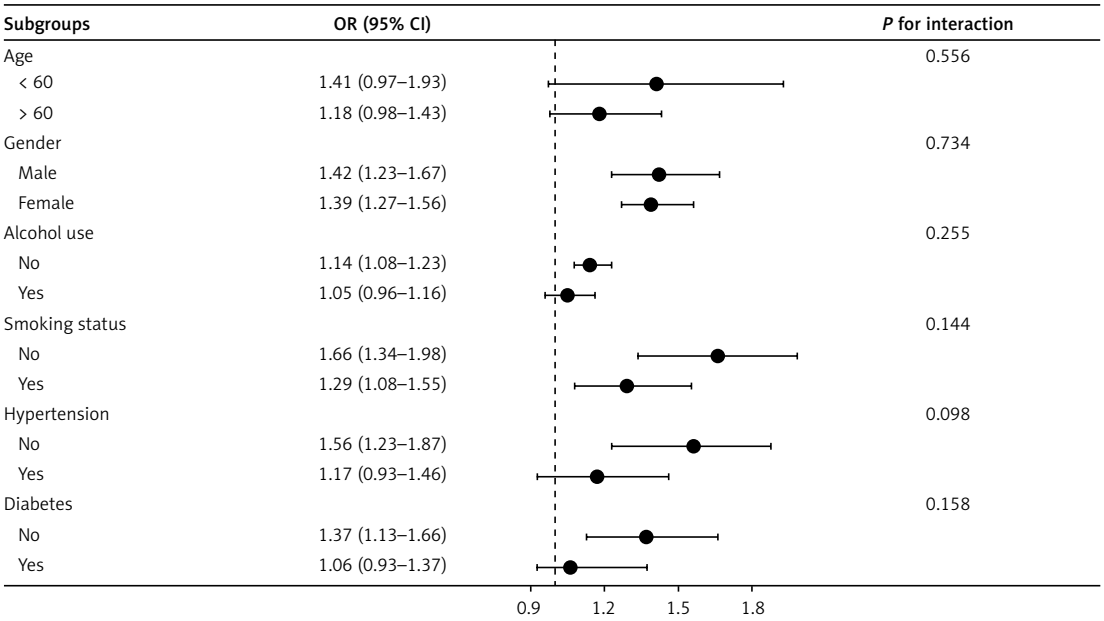

Figure 4. Subgroup analysis of the association between WWI and thoracic cancer in NHANES

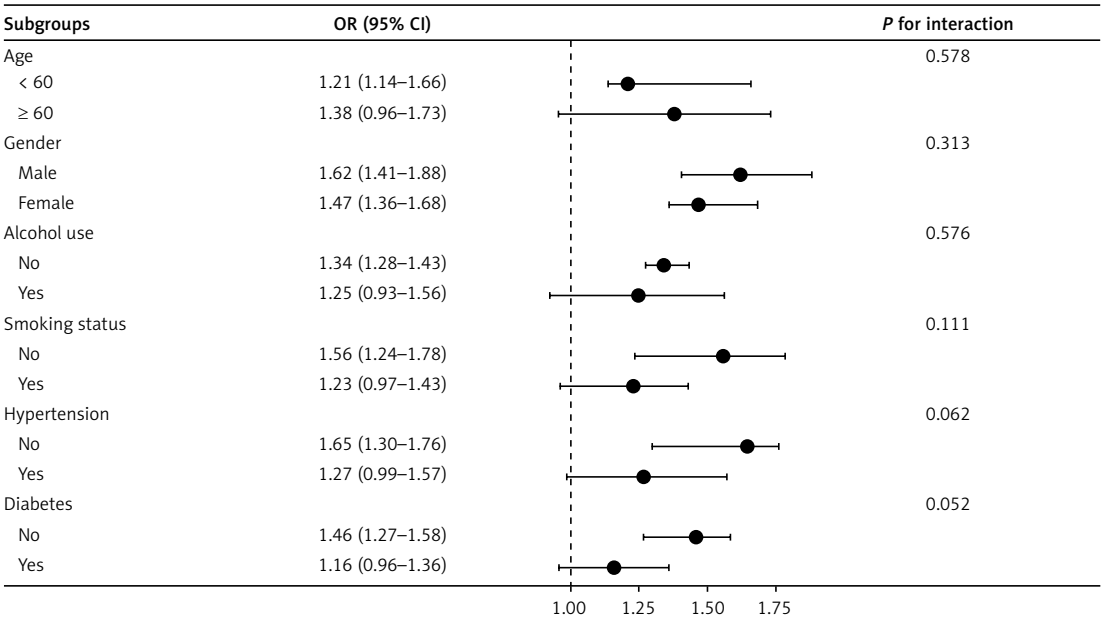

Figure 5. Subgroup analysis of the association between WWI and thoracic cancer in CHARLS

0.725 (0.701–0.750). In the CHARLS study, WWI AUC (95% CI): 0.712 (0.673–0.749). The findings of both studies indicated that WWI exhibited superior predictive capability for thoracic cancer in comparison to conventional obesity indicators, including waist-to-height ratio (WHtR) and BMI (DeLong test, $p < 0.05$).

Discussion

In this study, a substantial amount of patient information was extracted from the CHARLS and NHANES databases. The data were then screened, and multiple models were established. Logistic regression analysis was performed, and RCS curves were plotted. The research suggests a nonlinear association between WWI and thoracic cancer risk. The subgroup analyses demonstrated that the relationship between WWI and thoracic cancer was not affected by confounding variables, including age, sex, alcohol use, smoking status, hypertension, and diabetes. In comparison with conventional obesity indicators, such as WHtR and BMI, WWI exhibits excellent predictive ability for thoracic cancer.

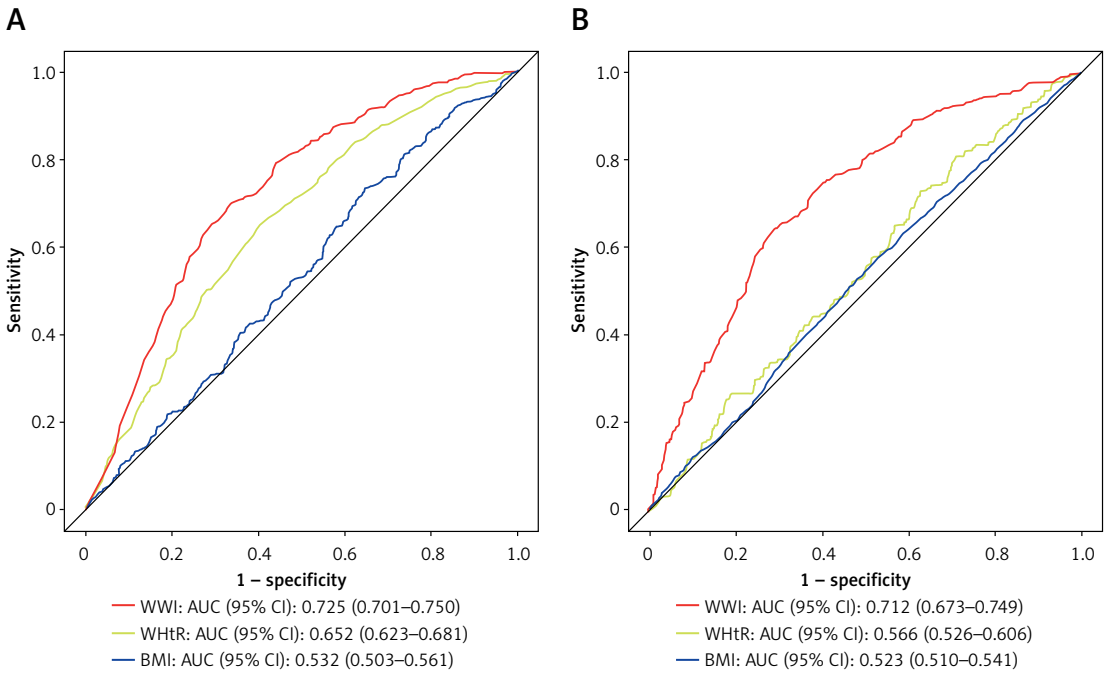

Figure 6. The prediction of thoracic cancer by different obesity indicators in NHANES (**A**) and CHARLS (**B**)

Obesity is a well-recognized risk factor for multiple types of cancer. A significant number of epidemiological and experimental studies have found a robust connection between cancer and obesity [21]. Obesity has been proven to exert a detrimental influence on a minimum of 13 distinct types of cancer [22], including rectal cancer, gallbladder cancer, and ovarian cancer [23]. Concurrently, obesity is increasingly recognized as one of the leading preventable risk factors for cancer [24]. The global increase in obesity prevalence has been associated with a rising burden of obesity-related cancers [25]. Furthermore, obesity may also influence the genomic subtypes of cancers. For instance, a relationship between obesity and specific driver mutations has been observed in endometrial cancer, lung adenocarcinoma, and cancers of unknown primary origin [26]. Studies have shown that presence of excess fat around the abdomen is associated with elevated likelihood of lung cancer [18]. The link is further substantiated by the significant association observed between increased visceral fat tissue and an elevated risk of lung cancer. There is a demonstrated association between central obesity and increased risk of developing esophageal adenocarcinoma. Research has shown that obesity accounts for approximately 27.0% of the attributable risk estimates for the development of esophageal adenocarcinoma [27].

WWI is a newly developed physical measurement indicator that offers a superior reflection of central obesity in comparison to conventional indicators, including BMI and waist circumference (WC) [28]. BMI is a measure of weight in relation to height that is calculated on a per-unit basis. WC is a static measurement that does not take into account fluctuations in weight. Conversely, the WWI approach considers the interrelated changes in weight and WC, thereby offering a more comprehensive reflection of the dynamic evolution of body composition.

Previous studies have demonstrated an association between weight, WWI, and abdominal fat, suggesting that WWI may serve as a marker of visceral fat accumulation [29]. Another study has shown that the combination of body weight and waist circumference offers a more precise measure of visceral fat, which plays a key role in predicting microvascular complications such as retinopathy [30]. Mechanistically, excess visceral adiposity may contribute to lung cancer development through chronic low-grade inflammation, altered adipokine secretion, insulin resistance and IGF pathway activation, oxidative stress, and immune dysregulation [31–33]. In a retrospective study, the authors evaluated whether obesity-induced immunomodulation affected the expression of programmed death ligand-1 (PD-L1) in lung cancer cells, and identified differences in PD-L1 expression between overweight women and women of normal weight [34]. In addition, obesity-associated increases in leptin and decreases in adiponectin may create a pro-tumorigenic pulmonary microenvironment [35], while experimental studies have shown that obesity can promote lung tumorigenesis via leptin-mediated activation of the PI3K/Akt/mTOR/STAT3 pathway [36]. Therefore, WWI may represent a simple anthropometric

marker that captures biologically meaningful central adiposity relevant to thoracic cancer risk. WWI also showed stronger associations with specific health outcomes. A study found a marked association between WWI and mortality in diabetes [37]. Furthermore, some investigations have revealed that WWI performs well in predicting symptoms of depression [38] and anxiety [39].

A separate study found that BMI was a less stable measure than WWI when examining the association between stroke risk and obesity indicators [40]. In related research on thoracic cancer, it was found that an elevated BMI may be associated with increased risk of developing small cell lung cancer [41]. In advanced lung cancer patients, a decrease in BMI combined with weight loss was associated with significantly reduced survival time [42]. A recent study yielded findings showing an association between genetic risk scores for high BMI and an increased risk of esophageal cancer [43]. An investigation of Asian populations indicated a negative association between BMI and esophageal cancer mortality [44]. Moreover, previous studies have identified associations between WWI and both prostate [45] and gynecological cancers [46].

To our knowledge, this study is one of the first to investigate the association between WWI and thoracic cancer. We found that elevated WWI was positively associated with thoracic cancer risk and has superior predictive performance compared to conventional obesity indicators. The present study has several strengths. First, two public databases, NHANES and CHARLS, were used for the study to ensure the objectivity of the information. Second, we implemented a series of adjustments to account for confounding variables and performed subgroup analyses to further substantiate the reliability of the results. Furthermore, there is a notable consistency in the results obtained from these two datasets, which serves to bolster the credibility of our research results. The findings of this study can serve as a starting point for future research, such as studies on the effects on treatment outcomes and toxicity.

However, the study has several limitations. First, the thoracic cancer diagnosis information in both databases is contingent on self-reported diagnoses by doctors, which may be subject to recall bias. Second, the exclusion of certain participants from the analysis resulted from an absence of pertinent tumor data. This potential limitation may restrict the applicability of our findings. Third, although the study considered two different populations in China and the United States, it is unclear if it can be applied to other groups. Finally, reverse causality is possible, as cancer-related weight loss may alter WWI, and longitudinal risk prediction cannot be inferred from cross-sectional data. In the future, it will be necessary to carry out studies targeting multiple regions and populations to further validate the research results.

In conclusion, the study demonstrated that WWI was independently associated with thoracic cancer prevalence in both the United States and Chinese populations. WWI showed better discriminatory ability for thoracic cancer risk than traditional obesity indicators. WWI may serve as an economical and readily available anthropometric indicator for the assessment of thoracic cancer risk.

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Data availability

The datasets used in the study are available on the CHARLS website (https://charls.pku.edu.cn/en) and the NHANES website (https://www.cdc.gov/nchs/nhanes/).

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Ethical approval

Every protocol utilized in NHANES has received approval from NCHS. Every protocol employed in CHARLS has been sanctioned by Peking University's Ethics Review Committee. Written informed consent was obtained from every participant.

Conflict of interest

The authors declare no conflict of interest.

References

1. Bray F, Laversanne M, Sung H, et al. Global Cancer Statistics 2022: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin 2024; 74: 229-63.
2. Thrift AP. Global burden and epidemiology of Barrett oesophagus and oesophageal cancer. Nat Rev Gastroenterol Hepatol 2021; 18: 432-43.
3. Morgan E, Soerjomataram I, Rumgay H, et al. The global landscape of esophageal squamous cell carcinoma and esophageal adenocarcinoma incidence and mortality in 2020 and projections to 2040: new estimates from GLOBOCAN 2020. Gastroenterology 2022; 163: 649-58.
4. Malcomson FC, Parra-Soto S, Ho FK, et al. Adherence to the 2018 World Cancer Research Fund (WCRF)/American Institute for Cancer Research (AICR) Cancer Prevention Recommendations and risk of 14 lifestyle-related cancers in the UK Biobank prospective cohort study. BMC Med 2023; 21: 407.
5. Song C, Lv J, Yu C, et al. Adherence to healthy lifestyle and liver cancer in Chinese: a prospective cohort study of 0.5 million people. Br J Cancer 2022; 126: 815-21.

6. Bharti JL, Wankhade A, Morey P, Ruikar P. The role of obesity in cancer incidence, treatment, and outcomes. Med Pharm J 2023; 2: 181-92.
7. Rumgay H, Shield K, Charvat H, et al. Global burden of cancer in 2020 attributable to alcohol consumption: a population-based study. Lancet Oncol 2021; 22: 1071-80.
8. Aleksandrova K, Reichmann R, Kaaks R, et al. Development and validation of a lifestyle-based model for colorectal cancer risk prediction: the LiFeCRC score. BMC Med 2021; 19: 1.
9. Joffe L, Mirzaei S, Bhatia S, et al. Body mass index, physical activity, and subsequent neoplasm risk among childhood cancer survivors. JAMA Oncol 2025; 11: 835-45.
10. Kerr J, Anderson C, Lippman SM. Physical activity, sedentary behaviour, diet, and cancer: an update and emerging new evidence. Lancet Oncol 2017; 18: e457-71.
11. Lynch BM, Bassett JK, Milne RL, et al. Estimating cancer incidence attributable to physical inactivity in the United States. Cancer 2025; 131: e35725.
12. Stanley A, Schuna J, Yang S, et al. Distinct phenotypic characteristics of normal-weight adults at risk of developing cardiovascular and metabolic diseases. Am J Clin Nutr 2020; 112: 967-78.
13. Recalde M, Davila-Batista V, Díaz Y, et al. Body mass index and waist circumference in relation to the risk of 26 types of cancer: a prospective cohort study of 3.5 million adults in Spain. BMC Med 2021; 19: 10.
14. Rubinstein, MM, Brown KA, Iyengar NM. Targeting obesity-related dysfunction in hormonally driven cancers. Br J Cancer 2021; 125: 495-509.
15. Leitzmann, MF, Stein MJ, Baurecht H, Freisling H. Excess adiposity and cancer: evaluating a preclinical-clinical obesity framework for risk stratification. EClinicalMedicine 2025; 83: 103247.
16. Xu M, Li M, Zhang Y, Li L, Shen Y, Hu G. Dynamic phenotypes of preclinical and clinical obesity in relation to new-onset cancer risk: a longitudinal analysis from the UK biobank. Diabetes Obes Metab 2025; 27: 5291-301.
17. Fontvieille E, Viallon V, Recalde M, et al. Body mass index and cancer risk among adults with and without cardiometabolic diseases: evidence from the EPIC and UK Biobank prospective cohort studies. BMC Med 2023; 21: 418.
18. Liu EE, Suthahar N, Paniagua SM, et al. Association of cardiometabolic disease with cancer in the community. JACC Cardiooncol 2022; 4: 69-81.
19. Petrelli F, Cortellini A, Indini A, et al. Association of obesity with survival outcomes in patients with cancer. JAMA Netw Open 2021; 4: e213520.
20. Sweetland S, Floud S, Gaitskell K, et al. Adiposity and risk of oesophageal cancer subtypes in the Million Women Study. Int J Epidemiol 2023; 52: 1795-804.
21. Thi, YN, Vu TD, Lan T, Chu DT. Epigenetic contribution to the relationship between obesity and cancer. Int Rev Cell Mol Biol 2024; 387: 195-213.
22. Lee H. Obesity-associated cancers: evidence from studies in mouse models. Cells 2022; 11: 1472.
23. Rakib A, Mamun MAA, Mandal M, Sinha P, Singh UP. Obesity–cancer axis crosstalk: molecular insights and therapeutic approaches. Acta Pharm Sinica B 2025; 15: 2930-44.
24. Lam TK, Daschner P, Ishibe N, et al. Metabolic Dysregulation and Cancer Risk Program (MeDOC): a transdisciplinary approach to obesity-associated cancers. J Natl Cancer Inst 2024; 116: 1555-61.
25. Pagano C, di Zazzo E, Avilia G, et al. Advances in 'adiponcosis': Insights in the inner mechanisms at the base of adipose and tumour tissues interplay. Int J Cancer 2022; 152: 2464-73.
26. Tang C, Castillon VJ, Waters M, et al. Obesity-dependent selection of driver mutations in cancer. Nat Genet 2024; 56: 2318-21.
27. Laaksonen MA, Li S, Canfell K, et al. The future burden of oesophageal and stomach cancers attributable to modifiable behaviours in Australia: a pooled cohort study. Br J Cancer 2023; 128: 1052-69.
28. Kunutsor SK, Jae SY, Laukkanen JA. Association between weight-adjusted waist index and cardiometabolic multimorbidity in older adults: findings from the English Longitudinal Study of Ageing. Geroscience 2025; 47: 6429-6438.
29. Kim NH, Park Y, Kim NH, Kim SG. Weight-adjusted waist index reflects fat and muscle mass in the opposite direction in older adults. Age Ageing 2021; 50: 780-6.
30. Li J, Yin Q, Hao J, et al. Association between weight-adjusted-waist index and retinopathy among American adults: a cross-sectional study and mediation analysis. Front Nutr 2025; 12: 1556065.
31. Renehan AG, Zwahlen M, Egger M. Adiposity and cancer risk: new mechanistic insights from epidemiology. Nat Rev Cancer 2015; 15: 484-98.
32. Shen S, Brown KA, Green AK, Iyengar NM. Obesity and cancer: a translational science review. JAMA 2026; 335: 1341-50..
33. Hirano T. IL-6 in inflammation, autoimmunity and cancer. Int Immunol 2020; 33: 127-48.
34. Gałązka JK, Czeczelewski M, Kucharczyk T, Szklener K, Mańdziuk S. Obesity and lung cancer – is programmed death ligand-1 (PD-L1) expression a connection? Arch Med Sci 2024; 20: 313-6.
35. Lim JY, Templeton SP. Regulation of lung inflammation by adiponectin. Front Immunol 2023; 14: 1244586.
36. Shi D, Wu J, Wu Y, Lin X, Xu C, Lian X. High-fat diet-related obesity promotes urethane-induced lung tumorigenesis in C57BL/6J mice. Front Oncol 2021; 11: 620993.
37. Zhang T, Zhang Z, Wang X, et al. Relationship between weight-adjusted-waist index and all-cause and cardiovascular mortality in individuals with type 2 diabetes. Diabetes Obes Metab 2024; 26: 5621-9.
38. Guo Y, Song M, Wang C. Weight-adjusted waist circumference index and chronic diseases as predictors of depression risk in U.S. adults: a cross-sectional study with mediation analysis. Front Nutr 2025; 12: 1568193.
39. Liang X, Chen K, Xia Y, et al. Association between weight-adjusted-waist index and anxiety among adults in the National Health and Nutrition Examination Survey (NHANES), 2007–2012. Front Nutr 2025; 12: 1530028.
40. Pillay P, Lewington S, Taylor H, Lacey B, Carter J. Adiposity, body fat distribution, and risk of major stroke types among adults in the United Kingdom. JAMA Netw Open 2022; 5: e2246613.
41. Zhou W, Liu G, Hung RJ, et al. Causal relationships between body mass index, smoking and lung cancer: univariable and multivariable Mendelian randomization. Int J Cancer 2020; 148: 1077-86.
42. Oswalt C, Liu Y, Pang H, et al. Associations between body mass index, weight loss and overall survival in patients with advanced lung cancer. J Cachexia Sarcopenia Muscle 2022; 13: 2650-60.
43. Carter P, Yuan S, Kar S, et al. Coffee consumption and cancer risk: a Mendelian randomisation study. Clin Nutr 2022; 41: 2113-23.
44. Lee S, Jang J, Abe SK, et al. Association between body mass index and oesophageal cancer mortality: a pooled

analysis of prospective cohort studies with >800 000 individuals in the Asia Cohort Consortium. Int J Epidemiol 2022; 51: 1190-203.
45. Gao Q, Lin Z, Zhang Y, Cai J, Li S. The association between waist-to-height index and prostate cancer risk: evidence from the NHANES study. Front Nutr 2025; 12: 1610288.
46. Fang L, Li X, Fang Y, et al. Association between weight-adjusted-waist index and gynecologic cancers: a population-based study. Front Nutr 2024; 11: 1449643.