BMC Public Health

RESEARCH

Open Access

Association between physical activity and visceral adiposity index (VAI) in U.S. population with overweight or obesity: a cross-sectional study

XiaoLiang Tao[1†], Xiang Xu[2†], YaoXin Xu[1†], QianKun Yang[1], TaoTao Yang[3], Xiang Zhou[4], Hao Xue[1], XiaoQin Ren[1*] and Fei Luo[1*]

Abstract

Background Previous studies have revealed the effects of different physical activity (PA) types on visceral adipose tissue (VAT) accumulation in individuals with overweight/obesity. However, the independent association (especially the dose–response relationship) between PA and VAT in individuals with and without overweight/obesity remains less explored. Visceral adiposity index (VAI), calculated from waist circumference, body mass index (BMI), triglyceride and high-density lipoprotein cholesterol, is a novel indicator of VAT. This study aims to elucidate the association between PA and VAI in participants with and without overweight/obesity.

Methods Participants who are overweight or obese and with complete data on VAI, PA, and other essential covariates from the National Health and Nutrition Examination Survey (NHANES) database (2015–2018) were included in this study. PA was evaluated by the PA questionnaire and converted into metabolic equivalent task (MET) hours per week (MET-h/wk) based on the suggested MET scores. Multivariate linear regression models were used to identify the association between PA and VAI. Subgroup analyses, combined with interaction tests and restricted cubic spline (RCS) regression analyses, were utilized to explore the stability and nonlinearity of PA-VAI association, respectively.

Results A total of 4, 312 participants with complete data on PA and VAI was included in this study, with 3, 441 of them being overweight or obese. After adjusting for all potential covariates, increased PA was found to be significantly associated with remarkable lower level of VAI in all participants (β = -0.0004, $P = 0.003$), participants with (β = -0.0013, $P = 0.012$) and without (β = -0.0004, $P = 0.003$) overweight/obesity. Subgroup analyses and interaction tests revealed that the PA-VAI association was not modified by other covariates in individuals with overweight/obesity. Furthermore, RCS analyses revealed that PA was significantly, linearly and negatively associated with VAI in all participants, participants with and without overweight/obesity (all $P < 0.05$, all P for nonlinearity > 0.05). Noteworthily,

[†]XiaoLiang Tao, Xiang Xu and YaoXin Xu contributed equally to this work and shared the co-authorship.

*Correspondence:
XiaoQin Ren
92024328@tmmu.edu.cn
Fei Luo
luofly1009@hotmail.com; luofei@tmmu.edu.cn
Full list of author information is available at the end of the article

as opposed to individuals without overweight/obesity, PA was significantly associated with lower VAI in participants with overweight/obesity after exceeding the threshold of 150 MET-h/wk.

Conclusion Increased PA was significantly associated with lower level of VAI, but a higher level of PA (> 150 MET-h/wk) was needed to obtain significantly lower level of VAI in individuals with overweight/obesity.

Keywords Physical activity, Visceral adiposity index, Obesity, Overweight

Introduction

Overweight or obesity is a multifactorial disease featured by excess fat (especially visceral fat) accumulation [1], which is responsible for various adverse consequences, such as cardiovascular disease, type 2 diabetes, arthritis, several cancers [2, 3], and so on. According to the World Health Organization (WHO), a body mass index (BMI) $\geq 25 kg/m^2$ is considered overweight, and a BMI $\geq 30 kg/m^2$ is defined as obesity [4]. Despite the numerous public health initiatives adopted to tackle this issue, the global prevalence of overweight or obesity is still continuing to escalate rapidly, making it a health issue that need to be addressed urgently. Therefore, studies targeted at elucidating the pathogenesis, identifying monitoring indicators, and developing management strategies for overweight or obesity are urgently needed currently.

For the management of overweight or obesity, finding an accurate and stable monitoring indicators which can effectively and reliably reflect the extent and status of overweight or obesity is one of the vital prerequisites for developing treatments and assess effectiveness. Previous studies have revealed that individuals with overweight or obesity are characterized by two outstanding features, including alterations in body anthropometrics and occurrence of metabolic abnormalities [1, 5]. Therefore, obesity indicators which incorporate metabolic and anthropometric parameters may be more reflective for assessing the onset and progression of overweight or obesity.

Visceral adiposity index (VAI), a sex-specific indicator calculated from waist circumference, BMI, triglyceride and high-density lipoprotein cholesterol (HDL-C), serves as a reliable indicator in various disease states, especially obesity. Compared with traditional body assessment parameters, such as BMI, waist circumference (WC), and waist-to-height ratio (WHtR), VAI is a novel indicator which can reflect the status of visceral adipose tissue accumulation and dysfunction with favorable performance [6, 7]. Previous studies have revealed that higher level of VAI was not only associated with severe fat accumulation [8], but also significantly associated with numerous metabolic diseases, including heart failure [9], abdominal aortic calcification [7], bone loss [10], chronic kidney disease (CKD) [11], etc. Briefly, as a novel obesity-related indicator, VAI can serve as a key parameter to evaluate the cardiometabolic risks and therapeutic effectiveness during obesity management.

In parallel with dietary, pharmaceutical, and behavioral interventions, physical activity (PA) is deemed as one of the "pillars" in controlling overweight and obesity [12]. Abundant evidences have demonstrated that PA or exercise training (ET) can trigger multiple beneficial health effects, including enhancing body weight and fat loss, maintaining body weight and fat reduction, reducing cardiovascular risks, improving metabolic fitness [13], etc. Based on previous studies, two key points can be summarized regarding the role of PA in maintaining health in individuals with overweight or obesity. One aspect is that PA can ameliorate the adverse outcomes of obesity even in the absence of weight loss, especially eliciting some beneficial metabolic alterations, such as enhancing vascular adaptations [14], alleviating insulin and leptin resistance [15, 16], increasing fat mobilization [17], etc. Previous studies have suggested that the increased risks of cardiometabolic diseases of overweight/obesity is mainly derived from its metabolic disturbance, such as insulin resistance, dyslipidemia, glucose intolerance [18], and so on. In other words, increased PA can help an overweight/obese individuals with metabolic abnormalities become an metabolically healthy obese individuals, and thus prevent the reap of additional cardiometabolic risks [19]. Except for the above-mentioned aspect, another aspect of the role PA in overweight/obese individuals is that increased PA interventions alone may only elicit modest or non-significant weight loss, a heartbreaking fact contrary to our expectation. The primary cause for such situation is that increased levels of PA may elicit diminishing returns in energy expenditure for the compensatory responses in non-PA energy expenditures [20], a phenomenon called energy compensation. It implies that increased PA can not directly and fully translate into an increase of total energy expenditure (TEE), because the TEE from other components may reduce accordingly, which mainly refers to the basal energy expenditure for the normal function of body systems and organs, including immune system, cardiovascular system, kidney, brain, liver, skeletal muscle, digestive and respiratory system, etc. Unfortunately, the degree of energy compensation may become more stronger as we get fatter, making the progressive difficulty in losing fat

[20]. These facts indicated that the dose–response relationship between PA and obesity-related indicators may be quite different among individuals with and without overweight/obesity. Additionally, although abundant previous studies have explored the relationship between different PA types (such as aerobic exercise, resistance training, high-intensity interval training, and so on) and VAT accumulation [21], the independent dose–response relationship between PA and VAT-reflected indicators remained unexplored.

Given this, in this study, we aimed to explore the association (especially dose–response relationship) between physical activity and VAI in individuals with overweight/obesity. Our findings may provide useful scientific references and evidences for individuals with overweight or obesity who endeavor to reduce visceral adipose tissue accumulation by PA.

Methods

Data accession and study population

This study was performed based on the data from the National Health and Nutrition Examination Survey (NHANES) conducted between 2015 and 2018 [22]. The NHANES is carried out in the United States by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC). The survey is comprised of cross-sectional interviews, examinations, and laboratory data collected from a complex multistage, stratified, clustered probability sample representative of the US population. The survey protocol has been granted approval by the Institutional Review Board of the CDC, and all the participants have willingly given their informed consent [23–25].

A total of 19, 225 participants from two Sects. (2015–2016, 2017–2018) were preliminarily selected for analyses. After exclusion of those who with incomplete data on VAI, PA, and other essential covariates, a total of 4, 312 participants were finally included for final analyses, and 3, 441 of them with overweight/obesity and 871 of them without overweight/obesity. The flowchart of participant selection was presented in Fig. 1.

Visceral adiposity index and physical activity assessment

VAI is a sex-specific indicator derived from waist circumference (WC), BMI, triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C). VAI can be calculated by the following formula [7]: with $VAI = [WC/(39.68 + 1.88 \times BMI)] \times (TG/1.03) \times (1.31/HDL\text{-}C)$ for males, and with $VAI = [WC/(36.58 + 1.89 \times BMI)] \times (TG/0.81) \times (1.52/HDL\text{-}C)$ for females, respectively.

As previously described [26], physical activity was evaluated by the weekly metabolic equivalent task (MET)-minute aggregated scores, which represented the energy expenditure used for PA. MET, served as a metabolic equivalent, reflected the ratio of metabolic rate during activity and at rest [27]. According to the NHANES recommendations [26], the weekly PA included five parts (including vigorous work-related activity, moderate work-related activity, walking or bicycling for transportation, vigorous leisure-time physical activity, and moderate leisure-time physical activity), and can be calculated by the formula presented in Fig. 2.

Covariates

According to previous studies [1, 5], some essential covariates were included in this study, including demographic characteristics, behavioral risk factors, and

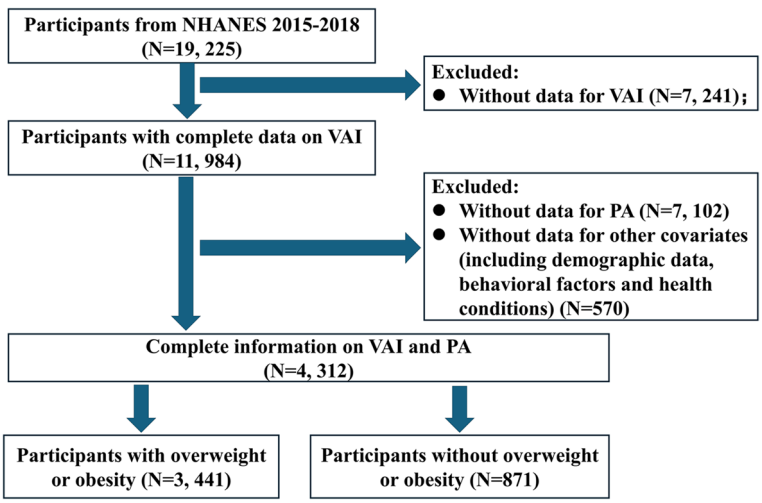


Fig. 1 The flowchart of participant selection

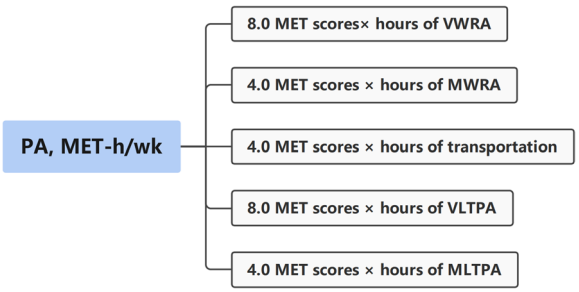


Fig. 2 The assessment of weekly PA. PA is composed of five parts, including the vigorous work-related activity (VWRA), moderate work-related activity (MWRA), walking or bicycling for transportation, vigorous leisure-time physical activity (VLTPA), and moderate leisure-time physical activity (MLTPA)

health conditions. The demographic covariates included age, gender, race, education level, marital status, poverty income ratio (PIR) and BMI. Race was stratified into Mexican American, Non-Hispanic Black, Non-Hispanic White and other races. PIR was divided into three levels as previously described [28], including < 1.30, 1.30–3.50, and ≥ 3.50. Marital status included three categories, including married, unmarried and others (widowed, divorced, and separated). Education was stratified into five levels, which were less than 9th grade, 9th–11th grade, high school, some college and college or above, respectively. Additionally, some other essential covariates were also included in this study, including behavioral risk factors (including smoking and drinking) and some chronic diseases, such as chronic kidney disease (CKD), hypertension, hyperlipidemia, and diabetes. Smoking status, as previously described [5], was divided into three categories, including never smoker, former smoker and current smoker. Similarly, the alcohol use status was categorized into three groups [5], including never drinkers, former drinkers, current drinkers. The Chronic Kidney Disease Epidemiology Collaboration (CKD—EPI) equation to assess CKD [29]. The diagnosis of CKD was based on the estimated glomerular filtration rate (eGFR) < 60 ml/min per 1.73 m^2 or urine albumin-to creatinine ratio (ACR) ≥ 30 mg/g [30]. The definition of hypertension was based on one or more of the following three conditions: (1) Participants responded "Yes" to the blood pressure questionnaire "Has a doctor or any other health professional ever told you that you had hypertension?"; (2) Participants were regarded as hypertensive if their average systolic blood pressure (SBP) was ≥ 130 mmHg or the average diastolic blood pressure (DBP) ≥ 80 mmHg; (3) Individuals were taking any antihypertensive drug were also considered hypertension. The diagnosis of diabetes was based on any of the following criteria: (1) a physician told you that you had diabetes; (2) the level of glycosylated hemoglobin (HbA1c) > 6.5%; (3) the level of fasting glucose ≥ 7.00 mmol/L; (4) the level of random blood glucose ≥ 11.10 mmol/L; (5) the level of two-hour oral glucose tolerance test (OGTT) ≥ 11.10mmo/L; (6) current use of any diabetes medication or insulin.

Statistical analyses

All statistical analyses were conducted by using R software (version 4.2.3) in this study. All analyses were conducted with the incorporation of sample weights, stratification, and cluster to account for the complex survey design. Some essential R packages were utilized to complete the data extraction, processing and analyses, including nhanesR, survery, etc.. Specifically, the baseline characteristics of all participants were presented based on the tertile grouping of PA. Continuous variables, including VAI, BMI, age, and PIR were presented as median (Q1-Q3, interquartile) for their non-normal distribution characteristics. Categorical variables were presented as numbers and percentages. The differences of baseline characteristics among different groups were compared using Rao-Scott chi-squared test or Kruskal–Wallis test. Besides, multivariate linear regression models were utilized to explore the association between PA and VAI. Additionally, subgroup analyses, interaction tests and restricted cubic spline (RCS) regression analysis were also conducted to investigate the robustness and nonlinearity of PA-VAI association, respectively. *P* values less than 0.05 (two sided) were considered as statistically significant.

Results

Baseline characteristics of the study population

As depicted in Fig. 1, after exclusion of participants with incomplete information on VAI, PA, and other essential covariates, including demographic characteristics (age, gender, race, education, marital status, PIR), behavioral factors (smoking and drinking), and health conditions (CKD, hypertension, hyperlipidemia and diabetes), a total of 4, 312 participants were selected with complete information on PA and VAI, and a total of 3, 441 participants with overweight and obesity were selected for final analyses.

The baseline characteristics of all enrolled participants were presented in Table 1. Variables of VAI, gender, education, marital status, PIR, alcohol use, CKD, hypertension, hyperlipidemia, and diabetes were all found to be significantly associated with PA groups ($P < 0.05$). Specifically, participants in the third tertile group were more likely to get lower VAI level, with age < 60 years, be male, be Non-Hispanic White, with higher education level, be

Table 1 Baseline characteristics of the study participants

Variables		PA, MET-h/wk			
	Overall	**Tertile 1**	**Tertile 2**	**Tertile 3**	***P* value**
N	3441	1121	1173	1147	
VAI	1.47 (0.90–2.45)	1.68 (1.02–2.61)	1.51 (0.92–2.40)	1.28 (0.80–2.17)	0.006
Age, years	50 (34–64)	59 (42–69)	51 (35–64)	42 (28–57)	< 0.001
Age categorical					< 0.001
< 60	2330 (67.71%)	615 (54.86%)	791 (67.43%)	924 (80.56%)	
> = 60	1111 (32.29%)	506 (45.14%)	382 (32.57%)	223 (19.44%)	
BMI, kg/m^2	30.70 (27.70–35.20)	30.70 (27.70–35.50)	30.70 (27.60–35.20)	30.60 (27.70–34.80)	0.566
BMI categorical					0.469
< = 30	1567 (45.54%)	512 (45.67%)	530 (45.18%)	525 (45.77%)	
30–35	989 (28.74%)	304 (27.12%)	339 (28.90%)	346 (30.17%)	
35–40	502 (14.59%)	176 (15.70%)	164 (13.98%)	162 (14.12%)	
> 40	383 (11.13%)	129 (11.51%)	140 (11.94%)	114 (9.94%)	
Gender, n(%)					< 0.001
Female	1765 (51.29%)	679 (60.57%)	653 (55.67%)	433 (37.75%)	
Male	1676 (48.71%)	442 (39.43%)	520 (44.33%)	714 (62.25%)	
Race, n(%)					< 0.001
Mexican American	638 (18.54%)	233 (20.79%)	172 (14.66%)	233 (20.31%)	
Non-Hispanic Black	772 (22.44%)	256 (22.84%)	256 (21.82%)	260 (22.67%)	
Non-Hispanic White	1153 (33.51%)	324 (28.90%)	436 (37.17%)	393 (34.26%)	
Other races	878 (25.52%)	308 (27.48%)	309 (26.34%)	261 (22.76%)	
Education, n(%)					< 0.001
Less than 9th grade	348 (10.11%)	188 (16.77%)	82 (6.99%)	78 (6.80%)	
9th-11th grade	380 (11.04%)	169 (15.08%)	91 (7.76%)	120 (10.46%)	
High school	741 (21.53%)	235 (20.96%)	241 (20.55%)	265 (23.10%)	
Some college	1033 (30.02%)	312 (27.83%)	360 (30.69%)	361 (31.47%)	
College or above	939 (27.29%)	217 (19.36%)	399 (34.02%)	323 (28.16%)	
Marital Status, n(%)					< 0.001
Unmarried	511 (14.85%)	121 (10.79%)	183 (15.60%)	207 (18.05%)	
Married	1687 (49.03%)	564 (50.31%)	593 (50.55%)	530 (46.21%)	
Others	1243 (36.12%)	436 (38.89%)	397 (33.84%)	410 (35.75%)	
PIR	2.03 (1.10–3.77)	1.73 (0.98–3.09)	2.29 (1.22–4.54)	2.09 (1.13–3.86)	< 0.001
PIR, n(%)					< 0.001
< 1.30	945 (30.95%)	350 (35.53%)	298 (28.44%)	297 (29.12%)	
1.30–3.50	1267 (41.50%)	425 (43.15%)	401 (38.26%)	441 (43.24%)	
≥ 3.50	841 (27.55%)	210 (21.32%)	349 (33.30%)	282 (27.65%)	
Smoke, n(%)					0.064
Never Smoker	1874 (54.46%)	648 (57.81%)	627 (53.45%)	599 (52.22%)	
Former Smoker	994 (28.89%)	309 (27.56%)	343 (29.24%)	342 (29.82%)	
Current Smoker	573 (16.65%)	164 (14.63%)	203 (17.31%)	206 (17.96%)	
Alcohol Use, n(%)					< 0.001
Never drinker	405 (11.77%)	183 (16.32%)	121 (10.32%)	101 (8.81%)	
Former drinker	930 (27.03%)	352 (31.40%)	313 (26.68%)	265 (23.10%)	
Current drinker	2106 (61.20%)	586 (52.27%)	739 (63.00%)	781 (68.09%)	
CKD, n(%)					< 0.001
Yes	615 (17.87%)	276 (24.62%)	196 (16.71%)	143 (12.47%)	
No	2826 (82.13%)	845 (75.38%)	977 (83.29%)	1004 (87.53%)	
Hypertension, n(%)					< 0.001
Yes	2014 (58.53%)	725 (64.67%)	700 (59.68%)	589 (51.35%)	

Table 1 (continued)

Variables		PA, MET-h/wk			
	Overall	Tertile 1	Tertile 2	Tertile 3	*P* value
No	1427 (41.47%)	396 (35.33%)	473 (40.32%)	558 (48.65%)	
Hyperlipidemia, n(%)					0.045
Yes	2049 (59.55%)	701 (62.53%)	684 (58.31%)	664 (57.89%)	
No	1392 (40.45%)	420 (37.47%)	489 (41.69%)	483 (42.11%)	
Diabetes, n(%)					< 0.001
Yes	855 (24.85%)	377 (33.63%)	281 (23.96%)	197 (17.18%)	
No	2586 (75.15%)	744 (66.37%)	892 (76.04%)	950 (82.82%)	

Abbreviations: *VAI* Visceral adiposity index, *BMI* Body mass index, *PIR* Ratio of family income to poverty, *PA* Physical activity, *MET* Metabolic equivalent. Variables of VAI, age, PIR, and BMI were presented as Median (Q1-Q3)

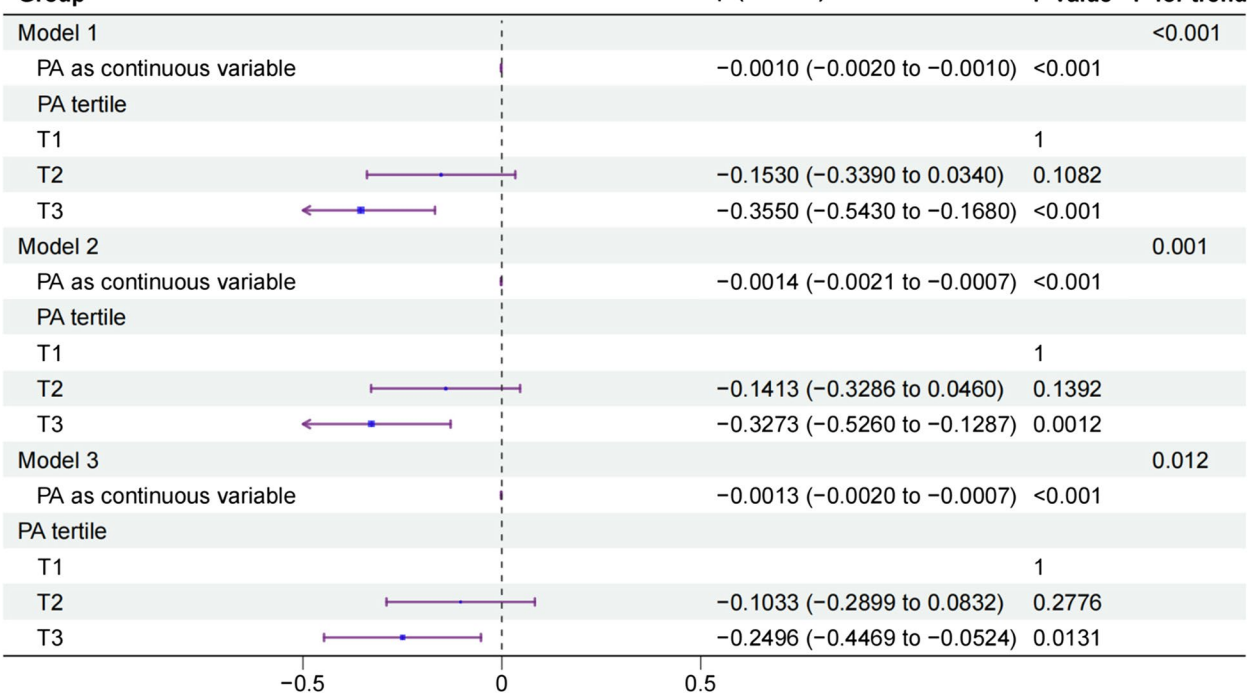
married, with PIR level of 1.30–3.50, be current drinkers, without CKD and diabetes.

Association between PA and VAI

Multivariate linear regression models were utilized to explore the association of PA and VAI in all participants ($N=4312$, regardless of BMI), participants with ($N=3441$, BMI > 25 kg/m^2) and without ($N=871$, BMI ≤ 25 kg/m^2) overweight or obesity. After adjusting for all potential covariates, increased PA (as continuous variable) was found to be significantly associated with remarkably lower VAI in all participants (Model 3; β = -0.0004, 95%CI:-0.0007 to -0.0001, $P=0.003$; STable 1), participants with overweight or obesity (Model 3; β = -0.0013, 95%CI:-0.0020 to -0.0007, $P=0.012$; Table 2), and participants without overweight or obesity (Model 3; β = -0.0004, 95%CI:-0.0007 to -0.0001, $P=0.003$; STable 2). Similarly, when PA was analyzed

Table 2 The Association between physical activity and visceral adiposity index in participants with overweight or obesity

Group	β (95% CI)	P value	P for trend
Model 1			<0.001
PA as continuous variable	−0.0010 (−0.0020 to −0.0010)	<0.001	
PA tertile			
T1		1	
T2	−0.1530 (−0.3390 to 0.0340)	0.1082	
T3	−0.3550 (−0.5430 to −0.1680)	<0.001	
Model 2			0.001
PA as continuous variable	−0.0014 (−0.0021 to −0.0007)	<0.001	
PA tertile			
T1		1	
T2	−0.1413 (−0.3286 to 0.0460)	0.1392	
T3	−0.3273 (−0.5260 to −0.1287)	0.0012	
Model 3			0.012
PA as continuous variable	−0.0013 (−0.0020 to −0.0007)	<0.001	
PA tertile			
T1		1	
T2	−0.1033 (−0.2899 to 0.0832)	0.2776	
T3	−0.2496 (−0.4469 to −0.0524)	0.0131	



Model 1, no covariates were adjusted; Model 2, Adjusted for age, gender and race; Model 3, Adjusted for age, gender, race, education level, marital status, PIR, BMI, smoke, alcohol use, CKD, hypertension, hyperlipidemia, and diabetes

Abbreviations: 95% CI 95% Confidence Interval, *RCS* Restricted cubic spline, *VAI* Visceral adiposity index, *PIR* Ratio of family income to poverty, *BMI* Body mass index, *CKD* Chronic kidney disease

as three categorical variables, increased PA levels were also associated with significantly lower VAI in all participants (Model 3; β = -0.1183, 95%CI:-0.1972 to -0.0394, $P = 0.003$; STable 1), participants with (Model 3; β = -0.2496, 95%CI:-0.4469 to -0.0524, $P = 0.0131$; Table 2) and without (Model 3; β = -0.1361, 95%CI:-0.2402 to -0.0320, $P = 0.010$; STable 2) overweight or obesity.

Subgroup analysis

In order to further confirm the robustness of PA-VAI association across different subgroups, subgroup analyses and interaction tests based on essential covariates were performed. As is shown in Table 3, the associations between PA and VAI remained consistent in different subgroups for covariates of age, gender, race, BMI, education, marital status, PIR, smoking, alcohol use, CKD, hypertension, hyperlipidemia, and diabetes. Additionally, interactions tests revealed that the PA-VAI association were not modified by these variables ($P > 0.05$).

Exploration of the non-linear relationship between total PA and LAP

RCS analysis was employed to explore the non-linear relationship between PA and VAI in all participants, participants with and without overweight or obesity. The results, revealed a significant, linear, and negative association between PA and VAI in all participants (regardless of BMI; $P = 0.001$, P for nonlinearity = 0.156; SFig.1), participants with overweight or obesity (BMI > 25 kg/m^2; $P = 0.031$, P for nonlinearity = 0.259; Fig. 3), and participants without overweight or obesity (BMI ≤ 25 kg/m^2; $P = 0.023$, P for nonlinearity = 0.169; SFig.2). Although a similar linear PA-VAI association was found in all participants, participants with and without overweight or obesity, it should be noted that a threshold of PA level (150 MET-h/wk) existed for PA to get significantly lower VAI in participants with overweight or obesity (Fig. 3). As presented in STable 3, once the weekly PA level exceed 150 MET-h/wk, higher PA levels were obviously associated with significantly lower VAI (β = -0.540, -0.501, -0.463 for model 1, model 2 and model 3, respectively). Additionally, in contrast to PA, a significant, positive and linear relationship between sedentary time and VAI was determined by RCS analysis (Fig. 4; $P = 0.027$, P for nonlinearity = 0.357).

Discussion

In this study, we mainly concentrated on exploring the association between PA and VAI in participants with overweight/obesity. Our results firstly demonstrated that increased PA levels were significantly associated with remarkable VAI reduction in individuals with and without overweight/obesity. Furthermore, subgroup analyses and interaction tests revealed that the PA-VAI association was not modified by other covariates. Additionally, although a similarly linear and negative association between PA and VAI was identified by RCS analysis in participants with and without overweight/obesity, it seems to be more difficult for individuals with overweight/obesity to trigger VAI reduction as opposed to individuals without overweight/obesity. Noteworthily, only PA reaching the threshold of 150 MET-h/wk can significantly elicit VAI reduction in participants with overweight/obesity.

In our study, a higher level of PA was identified by RCS analysis for individuals with overweight/obesity to induce the beneficial effect of VAI reduction as opposed to individuals without overweight/obesity, suggesting that it may be more difficult for overweight/obese individuals to consume fat by increased PA. The primary cause for this was energy compensation [20], a phenomenon that elevated exercise or other physical activities induced by long-term lifestyle changes didn't contribute to commensurate increases in energy expenditure, which is widely occurred both in humans and other animals. Moreover, there existed evidence revealed that the degree of energy compensation may be more progressively aggravated as individuals become fatter [20], thus forming the huge impediment for individuals with overweight/obesity to combat obesity with increased PA. Fortunately, some interventions can be taken to alleviate energy compensation, such as changing the patterns of PA (e.g., non-structured physical activity), alter dietary composition or patterns, and so on. Previous studies have revealed that structured aerobic exercise at the dose of 20 kcal per kilogram of weight per week contributed to less weight loss than expected for the reason of energy compensation induced by behavioral adaptations [31]. Therefore, choosing the pattern of non-structured physical activity may successfully circumvent the adaptations triggered by structured PA and thus alleviate energy compensation. Moreover, abundant evidences has indicated the crucial role of dietary composition or patterns in energy expenditure and metabolism [32]. The study performed by Nina Vujović, et al. [32]. indicated that late eating was significantly associated with various adverse effects, including increased hunger, altered appetite-regulating hormones, elevated waketime and 24-h ghrelin-to-leptin ratio, and altered adipose tissue gene expression responsible for lipid metabolism (a shift of pathways from lipolysis towards adipogenesis), etc. Briefly, energy compensation hindered the beneficial effects elicited by increased PA in individuals with overweight/obesity, and thus other interventions should also be taken to optimize these effects.

Table 3 The association between physical activity and visceral adiposity index in different subgroups

Subgroup	Number	β (95% CI)	P value	P interaction
Age				0.7738
<60	2362	−0.0011 (−0.0018 to −0.0004)	0.0016	
>=60	1195	−0.0008 (−0.0025 to 0.0009)	0.3370	
Gender				0.8861
Female	1765	−0.0010 (−0.0022 to 0.0002)	0.0897	
Male	1676	−0.0011 (−0.0018 to −0.0004)	0.0036	
Race				0.5990
Mexican American	638	−0.0006 (−0.0022 to 0.0010)	0.4435	
Non−Hispanic Black	772	−0.0007 (−0.0024 to 0.0009)	0.3725	
Non−Hispanic White	1153	−0.0007 (−0.0016 to 0.0001)	0.1020	
Other race	878	−0.0019 (−0.0035 to −0.0003)	0.0200	
Education				0.1730
Less than 9th grade	348	−0.0021 (−0.0044 to 0.0003)	0.0847	
9th−11th grade	380	−0.0015 (−0.0036 to 0.0005)	0.1449	
High school	741	−0.0007 (−0.0019 to 0.0004)	0.2296	
Some college	1033	−0.0005 (−0.0015 to 0.0006)	0.3941	
College or above	939	−0.0029 (−0.0045 to −0.0012)	<0.001	
Marital Status				0.4586
Unmarried	511	−0.0019 (−0.0035 to −0.0004)	0.0133	
Married	1687	−0.0011 (−0.0020 to −0.0001)	0.0228	
Others	1243	−0.0005 (−0.0016 to 0.0005)	0.3309	
PIR				0.8224
<=1.30	945	−0.0008 (−0.0020 to 0.0005)	0.2216	
1.30−3.49	1267	−0.0010 (−0.0020 to 0.0000)	0.0437	
>=3.50	841	−0.0015 (−0.0028 to −0.0002)	0.0277	
Smoke				0.3158
Never smoker	1874	−0.0009 (−0.0018 to 0.0000)	0.0425	
Former smoker	994	0.0001 (−0.0011 to 0.0013)	0.9165	
Current smoker	573	−0.0016 (−0.0029 to −0.0002)	0.0204	
Alcohol Use				0.9815
Never drinker	405	−0.0010 (−0.0032 to 0.0012)	0.3577	
Former drinker	930	−0.0013 (−0.0034 to 0.0008)	0.222	
Current drinker	2106	−0.0009 (−0.0016 to −0.0002)	0.0129	
CKD				0.6003
Yes	615	−0.0004 (−0.0025 to 0.0017)	0.6854	
No	2826	−0.0010 (−0.0017 to −0.0004)	0.0022	
Hypertension				0.5936
Yes	2014	−0.0012 (−0.0021 to −0.0003)	0.0126	
No	1427	−0.0008 (−0.0017 to 0.0000)	0.0532	
Hyperlipidemia				0.3959
Yes	2049	−0.0012 (−0.0020 to −0.0003)	0.0062	
No	1392	−0.0006 (−0.0016 to 0.0003)	0.1697	
Diabetes				0.5275
Yes	855	−0.0005 (−0.0022 to 0.0012)	0.5517	
No	2586	−0.0011 (−0.0018 to −0.0004)	0.0011	

The variables adjusted for subgroup analyses were consistent with Model 3 in Table 2 except the stratifying variable

Abbreviations: 95% CI 95% Confidence Interval, *RCS* Restricted cubic spline, *VAI* Visceral adiposity index, *PIR* Ratio of family income to poverty, *BMI* Body mass index, *CKD* Chronic kidney disease

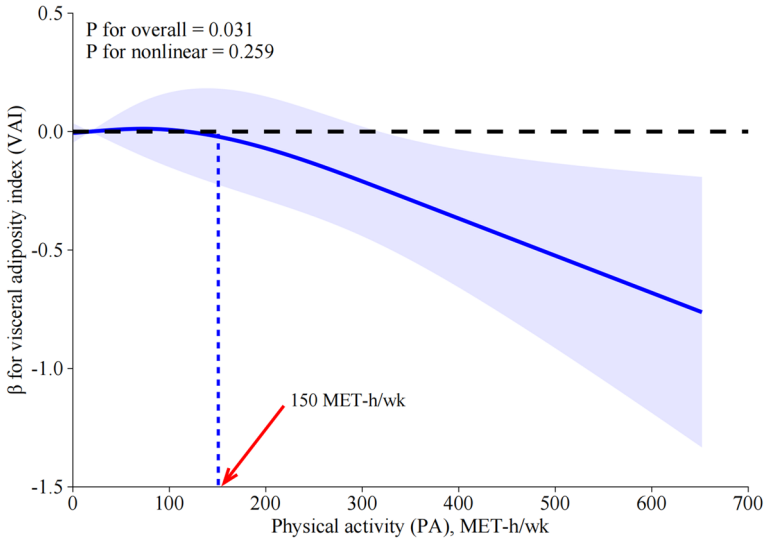


Fig. 3 RCS analysis to determine the association between PA and VAI in participants with overweight or obesity. Variables of age, gender, race, education level, marital status, PIR, BMI, smoke, alcohol use, CKD, hypertension, hyperlipidemia, and diabetes were adjusted during RCS analyses. Abbreviations: RCS, restricted cubic spline; VAI, visceral adiposity index; PIR, ratio of family income to poverty; BMI, body mass index; CKD, chronic kidney disease

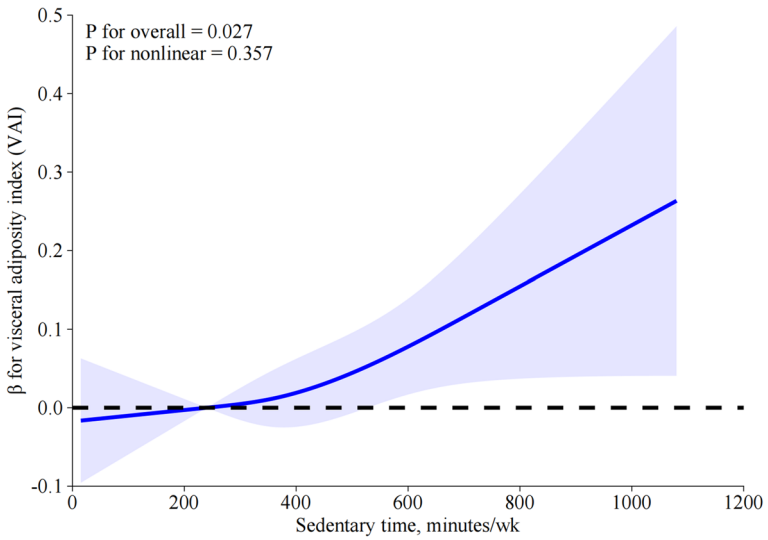


Fig. 4 RCS analysis to determine the association between weekly sedentary time and VAI in participants with overweight or obesity. Variables of age, gender, race, education level, marital status, PIR, BMI, smoke, alcohol use, CKD, hypertension, hyperlipidemia, and diabetes were adjusted during RCS analyses. Abbreviations: RCS, restricted cubic spline; VAI, visceral adiposity index; PIR, ratio of family income to poverty; BMI, body mass index; CKD, chronic kidney disease

Although increased PA may not contribute to successful weight loss for individuals with overweight/obesity, it indeed improved fitness via modulating metabolic health. Previous studies have indicated that metabolic health (especially in overweight/obesity) is being increasingly utilized to evaluate the risks for cardiovascular diseases (CVD), diabetes and diabetes-related complications [33]. Existing evidences have demonstrated that significant metabolic heterogeneity existed among individuals with overweight/obesity [34], and those who are metabolically healthy are defined as metabolically healthy overweight/obesity, whereas others are classified as metabolically unhealthy overweight/obesity [35, 36]. Actually, such metabolic heterogeneity also existed among individuals

with normal weight, and similarly those with and without healthy metabolic conditions were classified as metabolically healthy normal weight and metabolically unhealthy normal weight [37], respectively. A series of studies have revealed that being metabolically unhealthy (even with normal weight), or the transition from metabolically healthy to metabolically unhealthy, was significantly associated with increased risk for various adverse outcomes, including elevated obesity-induced cancer risk [38], enhanced CVD incidence [39, 40], deterioration of cognitive function [41], reduced fat utilization and enhanced insulin resistance [42], etc. Given this, individuals with overweight/obesity are encouraged to complete more PA to reap more metabolic benefits instead of just concentrating on weight loss. Accumulating evidences have uncovered that PA can reshape or reborn fitness via multiple ways, such as improving insulin/leptin resistance [43, 44], activating hepatic autophagy [45], stimulating the secretion of cytokines (e.g., interleukin-13) to enhance exercise endurance [46], and so on. Briefly, the above-mentioned facts all highlighted the critical role of PA in improving metabolic status and thus lowering the risks of obesity-related diseases or complications.

According to the 2020 guidelines from the World Health Organization, all adults are recommended to complete at least 150–300 min of moderate-intensity PA (MPA), or 75–150 min of vigorous-intensity PA (VPA), or equivalent combination of MPA and VPA per week to maintain fitness [47]. This level of PA represented the minimal amount of PA which can effectively reduce cardiometabolic risks, instead of the level to arouse significant weight loss without other interventions. Since about two-thirds of overweight and obese individuals were metabolically unhealthy based on previous epidemiological surveys [36], a higher level of PA was thus needed to elicit favorable health benefits. According to the results from our study, a minimum of 150 MET-h/wk was needed to arouse significant VAI reduction in individuals with overweight/obesity.

Several limitations in the current should be acknowledged. Firstly, the study only included samples from specific regions or populations, which may limit the generalizability of the results to other populations. Secondly, the sample size might be relatively small, which could affect the reliability and generalizability of the study findings. Additionally, the cross-sectional design employed in this study does not allow for establishing causality between PA and visceral fat accumulation. Finally, the evaluation of the PA was based on the self-reported Global Physical Activity Questionnaire (GPAQ), instead of accelerometer, which may introduce some bias to our results. Therefore, future researches based on accelerometer-measured physical activity are needed to further confirm the conclusions of the current study. Moreover, integrated analyses based on high-throughput omics (such as metabolomics, microbiomics, proteomics, and so on) can provide a deeper insight about the association between PA and overweight/obesity.

In summary, our findings from the current study revealed the essential role of PA in controlling overweight/obesity and eliciting VAI reduction. Increased levels of PA were significantly associated with remarkable VAI reduction, but a higher level of PA (>150 MET-h/wk) was needed to trigger remarkable VAI reduction in individuals with overweight/obesity.

Abbreviations

BMI	Body mass index
CKD	Chronic kidney disease
CVD	Cardiovascular diseases
DBP	Diastolic blood pressure
eGFR	Estimated glomerular filtration rate
HbA1c	Glycosylated hemoglobin
HDL-C	High-density lipoprotein cholesterol
MET	Metabolic equivalent task
MPA	Moderate-intensity physical activity
OGTT	Oral glucose tolerance test
PA	Physical activity
PIR	Poverty income ratio
RCS	Restricted cubic spline
SBP	Systolic blood pressure
TEE	Total energy expenditure
TG	Triglyceride
VAI	Visceral adiposity index
VPA	Vigorous-intensity physical activity
WC	Waist circumference
WHtR	Waist-to-height ratio

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-024-19810-1.

Supplementary Material 1.

Acknowledgements

The authors thank all the volunteers for the participation and personnel for their contribution to the NHANES study. In addition, we also thank Zijiao Li, Yichong Niu and Jie Zhang for their kind help in completing this study.

Authors' contributions

Fei Luo and XiaoQin Ren guided the whole research, from research design to data analysis and article writing. XiaoLiang Tao, Xiang Xu and YaoXin Xu wrote the main manuscript text for this research article and conducted the main statistical analyses. QianKun Yang and TaoTao Yang helped to complete data analyses. Xiang Zhou and Hao Xue revised the manuscript and were responsible for writing the response letter and making the additional statistical analyses. All authors gave final approval of the version to be published and agreed to be accountable for all aspects of the work.

Funding

This study was supported by the outstanding talent pool key support object projects of Army Medical University (Number: XZ-2019–505-021), and the project of Military key clinical specialty (Number: 41561Z23711).

Availability of data and materials
Publicly available datasets were analyzed in this study. This data can be accessible at: https://www.cdc.gov/nchs/nhanes/index.htm.

Declarations

Ethics approval and consent to participate
The program was approved by the National Center for Health Statistics Ethics Review Board. All of the participants provided written informed consent. No additional ethical review board approval was required to analyze the anonymized NHANES data.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Orthopaedics, National & Regional United Engineering Lab of Tissue Engineering, Southwest Hospital, Army Medical University, 30 Gaotanyan Street, Shapingba District, Chongqing 400038, China. [2]Department of Cardiology, Southwest Hospital, Army Medical University, Chongqing 400038, China. [3]Department of Radiology, Southwest Hospital, Army Medical University, Chongqing 400038, China. [4]College of Basic Medicine, Army Medical University, Cadet Brigade 4, Chongqing 400038, China.

Received: 13 September 2023 Accepted: 16 August 2024
Published online: 26 August 2024

References

1. Dai W, Zhang D, Wei Z, Liu P, Yang Q, Zhang L, et al. Whether weekend warriors (WWs) achieve equivalent benefits in lipid accumulation products (LAP) reduction as other leisure-time physical activity patterns? -Results from a population-based analysis of NHANES 2007–2018. BMC Public Health. 2024;24:1550.
2. Hainer V, Toplak H, Mitrakou A. Treatment modalities of obesity: what fits whom? Diabetes Care. 2008;31(Suppl 2):S269-277.
3. Kessler C. Pathophysiology of Obesity. Nurs Clin North Am. 2021;56:465–78.
4. Lassale C, Péneau S, Touvier M, Julia C, Galan P, Hercberg S, et al. Validity of web-based self-reported weight and height: results of the Nutrinet-Santé study. J Med Internet Res. 2013;15: e152.
5. Xue H, Zou Y, Yang Q, Zhang Z, Zhang J, Wei X, et al. The association between different physical activity (PA) patterns and cardiometabolic index (CMI) in US adult population from NHANES (2007–2016). Heliyon. 2024;10: e28792.
6. Amato MC, Giordano C, Galia M, Criscimanna A, Vitabile S, Midiri M, et al. Visceral Adiposity Index: a reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care. 2010;33:920–2.
7. Qin Z, Jiang L, Sun J, Geng J, Chen S, Yang Q, et al. Higher visceral adiposity index is associated with increased likelihood of abdominal aortic calcification. Clin Sao Paulo Braz. 2022;77: 100114.
8. Jabłonowska-Lietz B, Wrzosek M, Włodarczyk M, Nowicka G. New indexes of body fat distribution, visceral adiposity index, body adiposity index, waist-to-height ratio, and metabolic disturbances in the obese. Kardiol Pol. 2017;75:1185–91.
9. Xu C, Guo Y, Zhang S, Lai Y, Huang M, Zhan R, et al. Visceral adiposity index and the risk of heart failure, late-life cardiac structure, and function in ARIC study. Eur J Prev Cardiol. 2023;30:1182–92.
10. Sun A, Hu J, Wang S, Yin F, Liu Z. Association of the visceral adiposity index with femur bone mineral density and osteoporosis among the U.S. older adults from NHANES 2005-2020: a cross-sectional study. Front Endocrinol (Lausanne). 2023;14:1231527.
11. Fang T, Zhang Q, Wang Y, Zha H. Diagnostic value of visceral adiposity index in chronic kidney disease: a meta-analysis. Acta Diabetol. 2023;60:739–48.
12. Mehta M, Istfan NW, Apovian CM. Obesity: Overview of Weight Management. Endocr Pract. 2021;27:626–35.
13. Swift DL, McGee JE, Earnest CP, Carlisle E, Nygard M, Johannsen NM. The Effects of Exercise and Physical Activity on Weight Loss and Maintenance. Prog Cardiovasc Dis. 2018;61:206–13.
14. Kojda G, Hambrecht R. Molecular mechanisms of vascular adaptations to exercise. Physical activity as an effective antioxidant therapy? Cardiovasc Res. 2005;67:187–97.
15. Platat C, Wagner A, Klumpp T, Schweitzer B, Simon C. Relationships of physical activity with metabolic syndrome features and low-grade inflammation in adolescents. Diabetologia. 2006;49:2078–85.
16. Saeidi A, Haghighi MM, Kolahdouzi S, Daraei A, Abderrahmane AB, Essop MF, et al. The effects of physical activity on adipokines in individuals with overweight/obesity across the lifespan: A narrative review. Obes Rev Off J Int Assoc Study Obes. 2021;22: e13090.
17. Thompson D, Karpe F, Lafontan M, Frayn K. Physical activity and exercise in the regulation of human adipose tissue physiology. Physiol Rev. 2012;92:157–91.
18. Stefan N, Schulze MB. Metabolic health and cardiometabolic risk clusters: implications for prediction, prevention, and treatment. Lancet Diabetes Endocrinol. 2023;11:426–40.
19. Karelis A, Brochu M, Rabasa-Lhoret R. Can we identify metabolically healthy but obese individuals (MHO)? Diabetes Metab. 2004;30:569–72.
20. Careau V, Halsey LG, Pontzer H, Ainslie PN, Andersen LF, Anderson LJ, et al. Energy compensation and adiposity in humans. Curr Biol CB. 2021;31:4659-4666.e2.
21. Chen X, He H, Xie K, Zhang L, Cao C. Effects of various exercise types on visceral adipose tissue in individuals with overweight and obesity: A systematic review and network meta-analysis of 84 randomized controlled trials. Obes Rev Off J Int Assoc Study Obes. 2024;25: e13666.
22. Song Y, Guo W, Li Z, Guo D, Li Z, Li Y. Systemic immune-inflammation index is associated with hepatic steatosis: Evidence from NHANES 2015–2018. Front Immunol. 2022;13:1058779.
23. Liu B, Wang J, Li Y-Y, Li K-P, Zhang Q. The association between systemic immune-inflammation index and rheumatoid arthritis: evidence from NHANES 1999–2018. Arthritis Res Ther. 2023;25:34.
24. Jayanama K, Theou O, Godin J, Mayo A, Cahill L, Rockwood K. Relationship of body mass index with frailty and all-cause mortality among middle-aged and older adults. BMC Med. 2022;20:404.
25. Shi L, Zhang L, Zhang D, Chen Z. Association between systemic immune-inflammation index and low muscle mass in US adults: a cross-sectional study. BMC Public Health. 2023;23:1416.
26. Afarideh M, Sartori-Valinotti JC, Tollefson MM. Association of Sun-Protective Behaviors With Bone Mineral Density and Osteoporotic Bone Fractures in US Adults. JAMA Dermatol. 2021;157:1437–46.
27. Tucker LA. Physical activity and telomere length in U.S. men and women: An NHANES investigation. Prev Med. 2017;100:145–51.
28. Zhou X, Tao X-L, Zhang L, Yang Q-K, Li Z-J, Dai L, et al. Association between cardiometabolic index and depression: National Health and Nutrition Examination Survey (NHANES) 2011–2014. J Affect Disord. 2024;351:939–47.
29. Wang Y, Katzmarzyk PT, Horswell R, Zhao W, Johnson J, Hu G. Comparison of the heart failure risk stratification performance of the CKD-EPI equation and the MDRD equation for estimated glomerular filtration rate in patients with Type 2 diabetes. Diabet Med J Br Diabet Assoc. 2016;33:609–20.
30. Wang Y-J, Du Y, Chen G-Q, Cheng Z-Q, Liu X-M, Lian Y. Dose-response relationship between dietary inflammatory index and diabetic kidney disease in US adults. Public Health Nutr. 2022;26:1–9.
31. Broskey NT, Martin CK, Burton JH, Church TS, Ravussin E, Redman LM. Effect of Aerobic Exercise-induced Weight Loss on the Components of Daily Energy Expenditure. Med Sci Sports Exerc. 2021;53:2164–72.
32. Vujović N, Piron MJ, Qian J, Chellappa SL, Nedeltcheva A, Barr D, et al. Late isocaloric eating increases hunger, decreases energy expenditure, and modifies metabolic pathways in adults with overweight and obesity. Cell Metab. 2022;34:1486-1498.e7.
33. Schulze MB, Stefan N. Metabolically healthy obesity: from epidemiology and mechanisms to clinical implications. Nat Rev Endocrinol. 2024. https://doi.org/10.1038/s41574-024-01008-5.

34. Neeland IJ, Poirier P, Després J-P. Cardiovascular and Metabolic Heterogeneity of Obesity: Clinical Challenges and Implications for Management. Circulation. 2018;137:1391–406.
35. Zheng R, Yang M, Bao Y, Li H, Shan Z, Zhang B, et al. Prevalence and Determinants of Metabolic Health in Subjects with Obesity in Chinese Population. Int J Environ Res Public Health. 2015;12:13662–77.
36. He D, Qiu Y, Yan M, Zhou T, Cheng Z, Li J, et al. Associations of metabolic heterogeneity of obesity with frailty progression: Results from two prospective cohorts. J Cachexia Sarcopenia Muscle. 2023;14:632–41.
37. Zheng Q, Lin W, Liu C, Zhou Y, Chen T, Zhang L, et al. Prevalence and epidemiological determinants of metabolically obese but normal-weight in Chinese population. BMC Public Health. 2020;20:487.
38. Sun M, Fritz J, Häggström C, Bjørge T, Nagel G, Manjer J, et al. Metabolically (un)healthy obesity and risk of obesity-related cancers: a pooled study. J Natl Cancer Inst. 2023;115:456–67.
39. Abiri B, Koohi F, Ebadinejad A, Valizadeh M, Hosseinpanah F. Transition from metabolically healthy to unhealthy overweight/obesity and risk of cardiovascular disease incidence: A systematic review and meta-analysis. Nutr Metab Cardiovasc Dis NMCD. 2022;32:2041–51.
40. Cordola Hsu AR, Xie B, Peterson DV, LaMonte MJ, Garcia L, Eaton CB, et al. Metabolically Healthy/Unhealthy Overweight/Obesity Associations With Incident Heart Failure in Postmenopausal Women: The Women's Health Initiative. Circ Heart Fail. 2021;14: e007297.
41. Kouvari M, M D'Cunha N, Tsiampalis T, et al. Metabolically Healthy Overweight and Obesity, Transition to Metabolically Unhealthy Status and Cognitive Function: Results from the Framingham Offspring Study. Nutrients. 2023;15(5):1289.
42. Pujia A, Gazzaruso C, Ferro Y, Mazza E, Maurotti S, Russo C, et al. Individuals with Metabolically Healthy Overweight/Obesity Have Higher Fat Utilization than Metabolically Unhealthy Individuals. Nutrients. 2016;8:2.
43. Marson EC, Delevatti RS, Prado AKG, Netto N, Kruel LFM. Effects of aerobic, resistance, and combined exercise training on insulin resistance markers in overweight or obese children and adolescents: A systematic review and meta-analysis. Prev Med. 2016;93:211–8.
44. Peng J, Yin L, Wang X. Central and peripheral leptin resistance in obesity and improvements of exercise. Horm Behav. 2021;133: 105006.
45. Kuramoto K, Liang H, Hong J-H, He C. Exercise-activated hepatic autophagy via the FN1-α5β1 integrin pathway drives metabolic benefits of exercise. Cell Metab. 2023;35:620-632.e5.
46. Knudsen NH, Stanya KJ, Hyde AL, Chalom MM, Alexander RK, Liou Y-H, et al. Interleukin-13 drives metabolic conditioning of muscle to endurance exercise. Science. 2020;368:eaat3987.
47. Bull FC, Al-Ansari SS, Biddle S, Borodulin K, Buman MP, Cardon G, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. Br J Sports Med. 2020;54:1451–62.

Publisher's Note