Menopausal symptom network differences between women with and without waist-to-height ratio-defined abdominal obesity

Diya Wang, MN,[1] Feng Wang, MN,[2] Jiaqi Zheng, MN,[3] Xiaoxun Chen, MN,[1] Yunzhe Lei, MN,[1] and Jing Su, PhD[1]

Abstract

Objectives: To compare the structure of menopausal symptom networks between women with and without abdominal obesity (AO), defined as waist-to-height ratio (WHtR).

Methods: This cross-sectional analysis included 1,150 women from visit 6 of the study of Women's Health Across the Nation (SWAN). AO was defined as a WHtR of ≥ 0.5. We adjusted for demographic and clinical differences by regressing each symptom score on covariates, and subsequently using the residualized symptom scores for network estimation. We used centrality indices and community structure analysis to characterize the networks, and applied the Network Comparison Test (NCT) to evaluate differences in network structure and global strength.

Results: Women with AO reported higher prevalence and greater severity of several symptoms. Network density was marginally higher in the AO group (0.59; 46 of 78 edges) than in the non-AO group (0.55; 43 of 78 edges). The NCT revealed significant differences in network structure, whereas global strength remained comparable. Core symptoms differed by AO status: in the AO network, forgetfulness, irritability, and night sweats exhibited the highest centrality, whereas in the non-AO network, night sweats, palpitations, and depression demonstrated the highest centrality. In addition, symptom clusters also differed between the groups.

Conclusions: Women with AO exhibit both a higher prevalence and greater severity of symptoms, as well as a distinct symptom network structure. These findings highlight distinct symptom network profiles in women with AO, which may reflect different patterns of symptom interaction or underlying biological processes that warrant further investigation. Assessment of AO using WHtR may help stratify women who are likely to benefit from targeted, network-based interventions over isolated symptom management.

Key Words: Abdominal obesity, Menopausal symptoms, Network analysis, Symptoms management.

(*Menopause* 2026;33:000–000)

Menopause is a retrospective diagnosis established 12 months after permanent cessation of menstruation.[1] This cessation stems from declining ovarian hormone production, primarily estrogen, which gives rise to a spectrum of physiological and psychological symptoms collectively known as menopausal symptoms.[2] Estrogen deficiency disrupts lipid metabolism, energy expenditure, and fat distribution, promoting accumulation of abdominal and visceral adipose tissue.[3] Furthermore, abdominal adipose tissue has greater lipogenic potential, leading to lipid deposition and increased release of free fatty acids. These metabolic changes render menopausal women more susceptible to abdominal obesity (AO).[4,5]

AO, characterized by excess abdominal fat, particularly visceral adiposity, carries greater health risks than BMI-defined obesity.[6–8] A cohort study of 458,543 participants demonstrated that AO is associated with a higher incidence of colorectal cancer than BMI-based obesity.[9] Similarly, a cross-sectional study spanning more than 90 countries found that normal-weight individuals with AO face significantly increased risks of hypertension, diabetes, and dyslipidemia.[10] Consequently, global medical guidelines have increasingly incorporated AO into health assessments.[11–13] The International Menopause Society's 2025 white paper further emphasizes the impact of AO on women's health.[14]

AO also exacerbates the menopausal symptom burden.[5] Tang et al,[15] using data from the Peking Union

Received for publication October 16, 2025; accepted February 19, 2026.
From the [1]Department of Nursing, Shantou University Medical College, Shantou, Guangdong Province, China; [2]Guangzhou Women and Children's Medical Center, Guangzhou Medical University, Guangzhou, Guangdong, China; and [3]The Fourth Affiliated Hospital of Guangzhou Medical University, Guangzhou, Guangdong, China.
Funding/Support: None reported.
Financial disclosure/Conflicts of interest: None reported.
Supplemental Digital Content is available for this article. Direct URL citations are provided in the HTML and PDF versions of this article on the journal's website, www.menopause.org.
Address correspondence to: Jing Su, PhD, Department of Nursing, Shantou University Medical College, No. 22 Xinling Road, Shi Paotai Street, Jinping District, Shantou City, Guangdong Province, 515041, China. E-mail: jsu@stu.edu.cn

eISSN: 1530-0374
DOI: 10.1097/GME.0000000000002794

Medical College Hospital Aging Longitudinal Cohort of Women in Midlife (PALM) study, found AO to be associated with vasomotor symptoms (VMS) severity but not prevalence among Chinese women, although only 58 participants (13.8%) met AO criteria. Similarly, Jing et al's[16] study of Chinese women found that normal-weight women with AO experienced greater menopausal symptom severity, particularly insomnia, VMS, and paresthesia. Studies from Mediterranean countries further confirm associations between AO and both VMS and somatic symptoms.[17]

Despite regional variations in both obesity patterns (eg, higher rates in the Americas versus Asia) and menopausal symptom burden (eg, more severe hot flashes in African women), research on the impact of AO on these symptoms remains scarce.[18,19] This paucity underscores the need for region-specific studies to clarify the impact across diverse populations.[18,19] Moreover, existing researches have predominantly examined isolated symptom-AO associations, thereby overlooking complex interrelationships among symptoms (eg, the interconnections among VMS, anxiety, sleep disturbances, and depression).[20,21] Given that AO prevalence among menopausal women exceeds 60%, investigating symptom interaction patterns may inform more targeted interventions.[22]

Network analysis, widely applied in psychopathology, regards symptoms as interconnected nodes and quantifies their relationships as weighted edges.[23,24] This approach has gained traction in menopausal symptom research for identifying central symptoms. For example, Zhao et al[25] used network analysis to demonstrate that anxiety, despite a prevalence of $<60\%$, was the most central symptom in Chinese menopausal women, acting as a key node connecting psychological and somatic symptoms. Zhang et al[26] replicated this finding in a separate Chinese database. Among US women, Min et al[27] identified psychological symptoms as central but observed no significant network differences between perimenopausal and postmenopausal stages. Notably, no studies have examined how AO status influences symptom network structures, despite evidence linking AO to increased symptom burden.

Accurate assessment of AO is critical for such comparisons. Although waist circumference and waist-to-hip ratio have historically been used, waist-to-height ratio (WHtR) provides superior predictive accuracy with a consistent cutoff (≥ 0.5) across sex and ethnicity.[12,28,29] We therefore used WHtR ≥ 0.5 to classify AO in this study.[30]

On the basis of these considerations, we applied network analysis to compare symptom network structures between women with and without AO. We hypothesized that women with AO would exhibit both more severe symptoms and more densely interconnected symptom networks compared with women without AO.

METHODS

The Study of Women's Health Across the Nation (SWAN, www.swanstudy.org) is a multi-site, longitudinal cohort study initiated in 1994 to evaluate midlife women's health. Data collection was conducted at the following research centers: Ypsilanti and Inkster, MI (University of Michigan); Boston, MA (Massachusetts General Hospital); Chicago, IL (Rush Presbyterian-St. Luke's Medical Center); Alameda and Contra Costa County, CA (University of California-Davis and Kaiser Permanente); Los Angeles, CA (University of California-Los Angeles); Hackensack, NJ (Hackensack University Medical Center); and Pittsburgh, PA (University of Pittsburgh). At baseline, 3,302 participants met the eligibility criteria, and subsequent follow-up assessments included standardized physical examinations, fasting morning blood draws, interviewer-administered questionnaires, and self-administered forms completed either in person or by telephone.

Participants

Participants were drawn from SWAN Visit 6 (2002-2004).[31] Of the 2,448 women who completed this assessment wave, we restricted eligibility to women who (a) were aged 40-55 years, (b) had not used estrogen or progestin in the preceding 12 months, (c) were not pregnant, and (d) had not undergone hysterectomy or bilateral salpingo-oophorectomy. Given evidence that menopausal symptoms can occur both before and after perimenopause, we included women in all three stages: premenopause, perimenopause, and postmenopause.[1,32,33] WHtR was calculated as waist circumference divided by height (both in cm) and dichotomized at the 0.5 cutoff to define AO.[30] Ultimately, 1,150 women met all inclusion criteria: 705 with AO and 445 without.

Measures

Demographic and clinical characteristics

All variables were obtained from self-reported questionnaire, including age, race/ethnicity, marital status, annual household income, and chronic diseases. We classified menopausal status according to STRAW+10 criteria: premenopause (minimal menstrual cycle change); perimenopause (persistent $>$ 7 days cycle deviation or amenorrhea of $\geq$ 60 days); and postmenopause (12 consecutive months of amenorrhea).[1]

Physical measures

Height, weight, waist circumference, and hip circumference were obtained through physical examination data. WHtR was calculated from these measurements.

Menopausal symptoms

Thirteen symptoms were selected based on previous research[34] and the modified Kupperman Index (KI),[35] including: hot flashes, night sweats, joint/neck/shoulder stiffness or soreness, sleep disturbance, nocturia, decreased sexual desire, vaginal dryness, depression, dizziness, irritability, forgetfulness, palpitations, and headaches.

Analytic strategy

Descriptive and differential tests

We conducted analyses using SPSS 25.0. Continuous variables were presented as mean ± SD, and categorical variables as frequencies and percentages. We

used χ^2 test, independent sample t test, and Mann-Whitney U test to compare groups.

Covariate selection

Consistent with established methods for symptom network studies, we selected covariates through a two-step procedure.[36] AO was excluded because it was the primary exposure of interest, and waist circumference and height were excluded because they are components of WHtR.

We first screened candidate covariates for associations with the total symptom score (dependent variable). Specifically, we examined continuous variables using Pearson correlation, binary variables using independent-samples t tests, ordinal variables using Spearman correlation, and unordered categorical variables using one-way ANOVA. Variables with $P < 0.05$ were retained for subsequent multivariable modeling.

Subsequently, we coded significant variables as follows: continuous variables were entered directly; binary and ordinal variables were recoded; dummy variables were created for unordered categories. We then entered these variables multivariable regression model using a blockwise approach. This analysis identified six covariates associated with the total symptom score ($P < 0.05$): weight, smoking status, employment status, hypertension, menopausal status, and annual household income.

Network analysis

In network analysis, each menopausal symptom is conceptualized as a node and the associations between them are represented as edges. Each edge reflects the relationship between two symptoms, adjusted for the influence of all other symptoms in the network, thereby providing a clear view of symptom interdependencies. The analytic workflow was guided by the tutorial paper published by Epskamp et al[37] and Borsboom et al.[23]

Covariate adjustment. To account for potential demographic and clinical confounding, symptom networks were estimated based on residuals obtained from regressing each symptom score on the identified covariates.[38,39] These residuals represent symptom variance unexplained by covariates and were used for network estimation, ensuring that the network structure remained independent of demographic and clinical differences. Specifically, residualized symptom scores (ie, residuals from regressing each symptom on identified covariates) were used as input variables, enabling unbiased estimation of symptom associations independent of group-level demographic and clinical differences.

Network structure estimation. Menopausal symptom networks were estimated using the *estimateNetwork* function in R, applying a Gaussian graphical model (GGM), in which edges represent partial correlation coefficients between symptoms. Partial correlations quantify the unique association between two symptoms, while controlling for all remaining symptoms in the network. To minimize spurious edges, overfitting, and instability, the EBICglasso method was applied with a tuning parameter of 0.5.[40] The *averageLayout* function was applied to derive a consensus layout for the two networks, ensuring positional consistency and enabling direct visual comparison.[41]

Network description. Centrality indices (strength, expected influence, closeness, and betweenness) were calculated for each node with the *CentralityPlot* function and reported on a "row" scale to plot the raw coefficients. Centrality indices quantify the relative importance of individual symptoms within the network structure. Strength denotes the sum of absolute edge weights connected to the node, reflecting the direct influence on other nodes. However, in networks containing negative associations, strength can overestimate the influence of nodes by ignoring edge signs. Expected influence (EI) retains the signs of edge weights when summing, allowing positive and negative associations to contribute meaningfully to a node's influence score.[42] Therefore, EI provides a measure of a symptom's overall propensity to activate or inhibit other symptoms in the network, and is particularly appropriate for identifying core symptoms when negative edges are present. Nodes with higher EI were interpreted as more central in terms of their influence on the network structure. Closeness, defined as the inverse of weighted shortest path distances to all other nodes, indicates how rapidly a symptom can affect or be influenced by the entire network. Betweenness, defined as the frequency with which a node lies on the shortest paths between node pairs, captures bridging capacity.

Network comparison test. The network comparison test (NCT) was used to assess two null hypotheses at $\alpha = 0.05$: invariant global strength (H_0: equal sums of absolute edge weights across networks); and invariant network structure (H_0: equal edge weights for every pairwise symptom association).[43]

Community detection. Community detection was performed using the Spinglass algorithm, which accommodates both positive and negative edge weights.[44] Nodes within each cluster were assigned distinct colors.

Network accuracy and stability analysis. Edge weight accuracy was assessed using 1,000 nonparametric bootstrap samples. 95% CIs for each edge weight were calculated to indicate the stability and precision of the associations; narrower CIs reflect greater reliability of connections between symptoms.[37] Centrality stability was assessed through case-dropping bootstrap analysis (1,000 iterations). A centrality stability (CS) coefficient of ≥ 0.25 signifies acceptable stability, whereas a CS of ≥ 0.50 indicates good stability.[37]

Use of AI tools

After the initial draft was completed, grammar and style were reviewed by Kimi 2.5 (www.kimi.com), then verified by the authors and were not utilized in any other aspect of the study.

RESULTS

Participant characteristics

A total of 1,150 women were enrolled. Of these, 445 were classified as non-AO and 705 as AO. Approximately 50% of the sample identified as non-Hispanic White. The overall mean age was 50.91 ± 1.90 years, mean waist circumference was 88.59 ± 16.60 cm, mean hip circumference was 107.58 ± 15.10 cm, mean weight was 76.37 ± 21.26 kg, and mean height was 162.29 ± 6.71 cm. Menopausal status was distributed as follows: 65 participants (5.65%) were premenopausal, 713 (62.00%) were perimenopausal, and 372 (32.35%) were postmenopausal.

The non-AO group had a mean age of 50.75 ± 1.90 years, whereas the AO group had a mean age of 51.01 ± 1.90 years. Employment status, menopausal stage, smoking status, and height did not differ significantly between the two groups ($P > 0.05$). The groups differed significantly in race, marital status, annual household income, chronic diseases (diabetes mellitus, hypertension, and high cholesterol), and weight (Table 1).

TABLE 1. Characteristics of participants by abdominal obesity (AO) status

	Non-AO (n = 445)	AO (n = 705)	*P*
Age (y), mean ± SD[a]	50.75 ± 1.90	51.01 ± 1.90	0.03
Race, n (%)[b]			<0.01
Black/African American	59 (13.26)	273 (38.72)	
Chinese/Chinese American	68 (15.28)	52 (7.38)	
Japanese/Japanese American	94 (21.12)	48 (6.81)	
White Non-Hispanic	222 (49.89)	315 (44.68)	
Hispanic	2 (0.45)	17 (2.41)	
Income, n (%)[c]			<0.01
< $19,999	21 (4.72)	83 (11.77)	
$20,000-$49,999	117 (26.29)	203 (28.79)	
$50,000-$99,999	104 (23.37)	165 (23.40)	
$100,000 or more	203 (45.62)	254 (36.03)	
Employment status, n (%)[b,d]			0.17
No	75 (16.85)	142 (20.14)	
Yes	370 (83.15)	563 (79.86)	
Smoking, n (%)[b]			0.11
No	395	603	
Yes	50	102	
Diabetes mellitus, n (%)[b]			<0.01
No	438 (98.43)	643 (91.21)	
Yes	7 (1.57)	62 (8.79)	
Hypertension, n (%)[b]			<0.01
No	417 (93.71)	503 (71.35)	
Yes	28 (6.29)	202 (28.65)	
Marital status, n (%)[b]			<0.01
Single/never	51 (11.46)	114 (16.17)	
Currently married or living as married	326 (73.26)	412 (58.44)	
Separated	17 (3.82)	26 (3.69)	
Widowed	6 (1.35)	26 (3.69)	
Divorced	45 (10.11)	127 (18.01)	
Menopausal status, n (%)[b]			0.06
Premenopause	34 (7.64)	31 (4.40)	
Perimenopause	274 (61.57)	439 (62.27)	
Postmenopause	137 (30.79)	235 (33.33)	
Height (cm), mean ± SD[a]	162.25 ± 6.92	162.32 ± 6.57	0.13
Weight (kg), mean ± SD[a]	59.9 ± 8.91	86.78 ± 20.18	<0.01
WC (cm), mean ± SD[a]	73.57 ± 5.55	98.08 ± 14.06	<0.01
HC (cm), mean ± SD[a]	96.12 ± 6.68	114.81 ± 14.45	<0.01

[a]Age, height, weight, waist circumference (WC), and hip circumference (HC) were compared between the two groups with independent-samples *t* tests.
[b]Differences in race, employment status, smoking status, diabetes, hypertension, marital status, and menopausal status were examined using χ^2 tests.
[c]Household income was analyzed using the Mann-Whitney *U* test.
[d]Employment status: whether participants were currently employed for pay at follow-up.

Differences in menopausal symptoms

Nocturia (night-time urination), forgetfulness, and stiffness or soreness in joints, neck or shoulders were the three most prevalent symptoms in both groups. The AO group reported higher prevalence of dizziness, hot flashes, night sweats, nocturia, sleep disturbances, and palpitations compared with the non-AO group (Table 2).

Regarding symptom severity, the AO group reported significantly greater severity of dizziness, hot flashes, night sweats, and other somatic symptoms compared with the non-AO group (Table 2).

Covariate selection

We examined associations between the total menopausal symptom score and demographic and clinical characteristics. Age, hip circumference, weight, employment status, smoking, income, diabetes mellitus, hypertension, marital status, and menopausal status showed significant associations with total symptom scores (all $P < 0.05$). Race was not significantly associated with total symptom scores ($P = 0.18$). Variables significantly associated with total symptom scores were entered into the multiple linear regression model.

We coded these variables and included dummy variables in blockwise regression (see SDC1-Table S1, Supplemental Digital Content 1, http://links.lww.com/MENO/B515). Multiple linear regression showed significant multicollinearity between hip circumference and weight (variance inflation factor, VIF > 10). Given that body weight is a widely used indicator of overall adiposity, we retained weight for covariate adjustment and excluded hip circumference from the final covariate set. In the subsequent analysis, marital status, treated as a categorical variable, did not reach statistical significance ($\Delta R^2 = 0.001$, $P = 0.779$); we therefore excluded it from the final model. The final covariate set used for adjustment included weight, smoking status, employment status, hypertension, menopausal status, and annual household income (see SDC1-Table S2, Supplemental Digital Content 1, http://links.lww.com/MENO/B515).

Network analysis

Using covariates identified in the Covariate selection section, we regressed each menopausal symptom score on these covariates and used the resulting residualized scores as input variables for network estimation.

Differences in network structure

Overall, the AO group exhibited higher network connectivity than the non-AO group. Network density

TABLE 2. Symptom prevalence and severity by abdominal obesity (AO) status

	Prevalence rate (%)[a]			Severity, median (IQR)[a]		
	Non-AO (n = 445)	AO (n = 705)	*P*	Non-AO (n = 445)	AO (n = 705)	*P*
Dizziness	15.28	23.68	<0.01	1 (1.1)	1 (1.1)	<0.01
Hot flashes	39.33	54.47	<0.01	1 (1.2)	2 (1.2)	<0.01
Night sweats	33.71	43.26	<0.01	1 (1.2)	1 (1.2)	<0.01
Stiffness or soreness in joints, neck or shoulder	67.64	78.72	0.09	2 (1.3)	2 (2.4)	<0.01
Forgetfulness	68.09	69.93	0.51	2 (1.2)	2 (1.2)	0.20
Vaginal dryness	29.44	28.23	0.66	1 (1.2)	1 (1.2)	0.69
Headaches	54.16	59.86	0.06	2 (1.2)	2 (1.2)	0.02
Get up at night to urinate	82.47	89.79	<0.01	4 (2.5)	4 (2.5)	<0.01
Feeling blue or depressed	54.61	55.18	0.85	2 (1.2)	2 (1.2)	0.32
Desire to engage in any form of sexual activity	53.03	53.62	0.85	2 (2.3)	2 (2.3)	0.50
Trouble falling asleep	38.20	45.82	0.01	1 (1.2)	1 (1.3)	<0.01
Irritability or grouchiness	63.37	67.52	0.15	2 (1.2)	2 (1.2)	0.04
Palpitations	20.67	29.93	<0.01	1 (1.1)	1 (1.2)	<0.01

AO, abdominal obesity.
[a]The χ^2 test and the Mann-Whitney *U* test were used to analyze the prevalence and severity, respectively.

was slightly higher in the AO group (0.59; 46/78 edges) compared with the non-AO group (0.55; 43/78 edges). Average edge weights were comparable across groups (both 0.05). The NCT indicated a significant difference in network structure (M = 0.24, $P = 0.02$), whereas global strength was comparable (S = 0.19, $P = 0.81$). The edge invariance test revealed significant between-group differences for the following edges: forgetfulness – vaginal dryness (E = 0.17, $P = 0.02$), decreased sexual desire – trouble falling asleep (E = 0.12, $P = 0.04$), dizziness – palpitations (E = 0.01, $P = 0.02$), nocturia – palpitations (E = 0.03, $P = 0.02$), and depression – palpitations (E = 0.15, $P = 0.05$).

Network description

The strongest edges were qualitatively similar across groups. In the non-AO group, the strongest edges were hot flashes – night sweats (w = 0.56), irritability – depression (w = 0.38), and palpitations – dizziness (w = 0.35). In the AO group, the strongest edges were hot flashes – night sweats (w = 0.50), irritability – depression (w = 0.32), and stiffness – forgetfulness (w = 0.21) (Fig. 1).

Centrality indices suggested that key symptoms differed by AO status. In the non-AO network, EI and strength centrality were highest for night sweats ($r_{EI} = 0.95$, $r_s = 0.95$), followed by palpitations ($r_{EI} = 0.94$, $r_s = 0.94$) and depression ($r_{EI} = 0.89$, $r_s = 0.89$); closeness centrality peaked for depression and palpitations (both $r_c = 0.007$); and betweenness centrality was greatest for trouble falling asleep ($r_b = 20$), palpitations ($r_b = 18$) and depression ($r_b = 16$). These finding suggest that palpitations were likely the most central symptom despite high EI and strength centrality of night sweats. In the AO network, EI and strength centrality was highest for forgetfulness ($r_{EI} = 0.92$, $r_s = 0.92$) and irritability ($r_{EI} = 0.90$, $r_s = 1.02$), and followed by night sweat ($r_{EI} = 0.83$, $r_s = 0.83$); closeness centrality was maximal for dizziness, forgetfulness, irritability, and headache (all $r_c = 0.006$); betweenness centrality was greatest for dizziness ($r_b = 15$), irritability ($r_b = 13$), and forgetfulness ($r_b = 10$), demonstrating that forgetfulness was the most influential node in this network (Table 3).

We defined any constellation of two or more co-occurring symptoms as a symptom cluster, following Kim et al.[45] The Spinglass algorithm identified four symptom clusters within the non-AO network: hot flashes, night sweats, and vaginal dryness; joint/neck/shoulder stiffness, forgetfulness, irritability, and depression; headache, palpitations, and dizziness; and trouble falling asleep and nocturia (night-time urination). The isolated symptom was decreased sexual desire. The AO group's network contained three symptom clusters: joint/neck/shoulder stiffness, forgetfulness, vaginal dryness, and headache; trouble falling asleep, nocturia (night-time urination), hot flashes, and night sweats; dizziness, palpitations, depression and irritability. Decreased sexual desire was also isolated (Fig. 1).

Network accuracy and stability analysis

In accuracy plots, gray area represents the 95% CIs; narrower intervals reflect higher precision (see SDC2, Supplemental Digital Content 2, http://links.lww.com/MENO/B516). For stability analysis, the CS coefficient for both networks exceeded 0.25, indicating acceptable stability (see SDC3, Supplemental Digital Content 3, http://links.lww.com/MENO/B517).

DISCUSSIONS

To our knowledge, this is the first study to apply symptom network analysis to compare the menopausal symptoms between women with and without AO. Consistent with our hypothesis, women with AO reported higher prevalence and greater severity of several menopausal symptoms, particularly VMS, dizziness, and sleep disturbance. Notably, although the AO group exhibited marginally higher network density than the non-AO group, the NCT revealed significant differences in network structure, whereas global strength was comparable

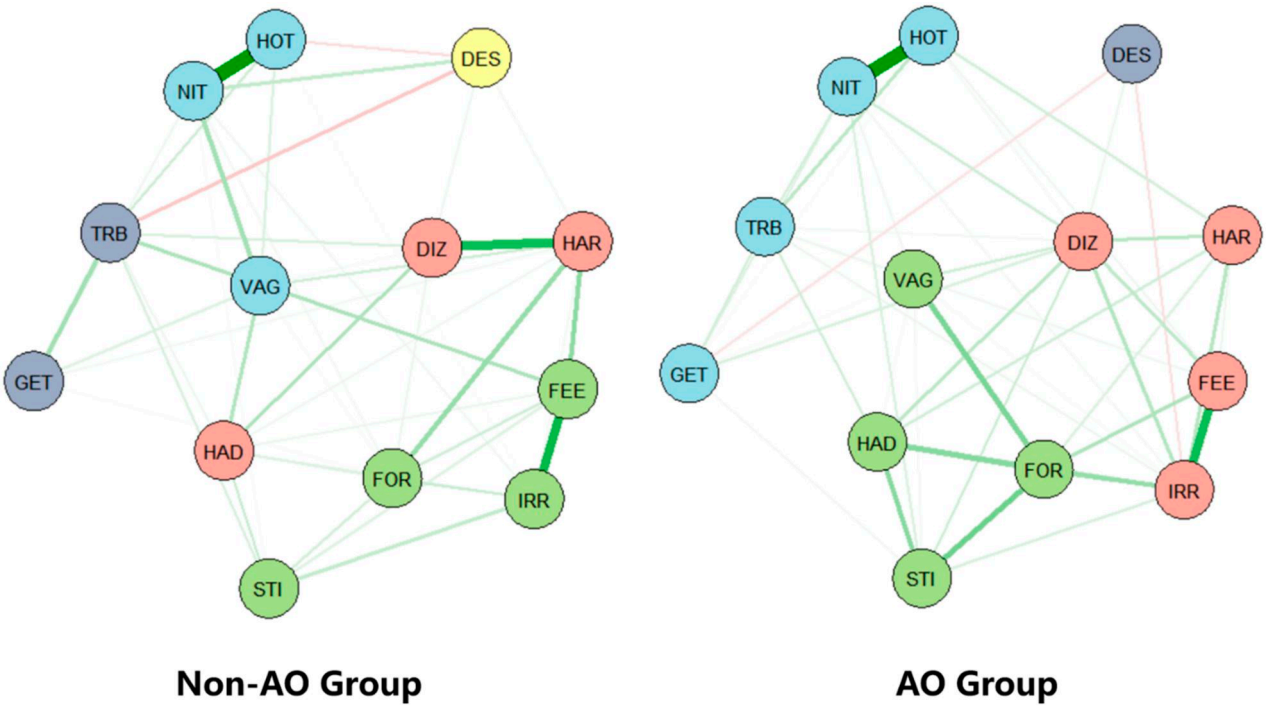

FIG. 1. Symptom networks in women with and without AO (covariate adjusted). Nodes represent symptoms and edges represent regularized partial correlations between symptoms. Both networks are displayed using the same layout to facilitate visual comparison. Thicker edges indicate stronger associations; green edges indicate positive associations and red edges indicate negative associations. Node colors indicate symptom communities (clusters) detected using the Spinglass algorithm; nodes without shared color represent isolated symptoms. AO, abdominal obesity; DES, decreased sexual desire; DIZ, dizziness; FEE, feeling blue/depressed; FOR, forgetfulness; GET, nocturia; HAD, headaches; HAR, palpitations; HOT, hot flashes; IRR, irritability; NIT, night sweats; STI, joint/neck/shoulder stiffness; TRB, trouble falling asleep; VAG, vaginal dryness.

between groups. These findings suggest that AO may be associated less with uniformly stronger symptom networks and more with alterations in specific symptom interrelationships.

Regarding symptom experience, women with AO exhibited higher prevalence and greater severity of VMS, sleep disturbances, and somatic symptoms, findings consistent with previous research.[5] These findings may reflect the complex interplay between AO and declining estrogen levels. Under the influence of ovarian hormones and genetic factors, women accumulate more adipose tissue in the hip region than men.[46] Estrogen plays a significant role in maintaining subcutaneous fat accumulation, particularly in the hips and thighs. This fat distribution contributes to a gynoid (pear-shaped) pattern, which is associated with glucose-insulin homeostasis and lipid metabolic balance.[47] During the menopausal transition, declining ovarian hormone production leads to redistribution of body fat toward the abdominal region, thereby increasing risk of AO and metabolic diseases.[48,49] In addition to hormonal changes, evidence suggests a bidirectional relationship between sleep disturbances and AO. A randomized controlled trial reported that sleep disturbances increased abdominal fat accumulation.[50] Similarly, a cross-sectional study demonstrated an association between sleep disturbances and AO.[51] From a correlation perspective, this association may explain why women with AO experience more severe sleep disturbances, although the underlying physiological mechanisms warrant further investigation.

VMS is one of the most common menopausal symptoms, typically characterized by sudden hot flashes and night sweats.[52] Estrogen decline constitutes an important contributor, as evidenced by the efficacy of estrogen supplementation in reducing hot flashes.[53] However, it is not the sole determinant. VMS also varies by ethnicity and socioeconomic status.[19] Current evidence suggests that VMS pathophysiology involves dysregulation of the thermoregulatory center, mediated by a narrowed thermoneutral zone during the menopausal transition. Specifically, this narrower thermoneutral zone renders menopausal women more sensitive to minor changes in core body temperature, thereby readily activating thermoregulatory pathways that trigger VMS to restore body temperature within the thermoneutral zone.[54] Obese individuals possess greater adipose tissue mass, which confers greater resistance to cold but, conversely, impairs heat dissipation.[55] We hypothesize that the increased abdominal adiposity in women with AO may impair heat dissipation from the viscera organs,

TABLE 3. Centrality indices of symptom networks by abdominal obesity (AO) status[a]

		Expected influence[b]		Strength[c]		Closeness[d]		Betweenness[e]	
label	**Item**	**Non-AO**	**AO**	**Non-AO**	**AO**	**Non-AO**	**AO**	**Non-AO**	**AO**
DIZ	Dizzy	0.67	0.82	0.67	0.82	0.006	0.006	5	15
HOT	Hot flashes	0.65	0.74	0.79	0.74	0.005	0.004	5	3
NIT	Night sweat	0.95	0.83	0.95	0.83	0.005	0.004	9	7
STI	Stiffness or soreness in joints, neck, or shoulder	0.41	0.65	0.41	0.65	0.005	0.005	0	2
FOR	Forgetfulness	0.60	0.92	0.60	0.92	0.005	0.006	0	10
VAG	Vaginal dryness	0.54	0.39	0.54	0.39	0.006	0.005	11	0
HAD	Headaches	0.62	0.72	0.62	0.72	0.005	0.006	3	5
GET	Get up at night to urinate	0.30	0.18	0.30	0.28	0.005	0.003	0	1
FEE	Feeling blue or depressed	0.89	0.66	0.89	0.66	0.007	0.005	16	0
DES	Desire to engage in any form of sexual activity	−0.007	−0.06	0.37	0.15	0.005	0.003	0	0
TRB	Trouble falling asleep	0.46	0.45	0.69	0.45	0.006	0.004	20	1
IRR	Irritability or grouchiness	0.70	0.90	0.70	1.02	0.006	0.006	2	13
HAR	Palpitations	0.94	0.46	0.94	0.46	0.007	0.005	18	0

AO, abdominal obesity; DES, decreased sexual desire; DIZ, dizziness; FEE, feeling blue/depressed; FOR, forgetfulness; GET, nocturia; HAD, headaches; HAR, palpitations; HOT, hot flashes; IRR, irritability; NIT, night sweats; STI, joint/neck/shoulder stiffness; TRB, trouble falling asleep; VAG, vaginal dryness.

[a]Centrality indices were computed from covariate-adjusted (residualized) symptom networks.

[b]Expected Influence: the sum of a node's connected edge weights retaining their signs higher values indicate greater overall influence.

[c]Strength: the sum of the absolute edge weights; higher values indicate stronger direct connectivity.

[d]Closeness: the inverse of weighted shortest path distances; higher values indicate faster influence propagation through the network.

[e]Betweenness: the frequency with which a node lies on shortest paths between other nodes; higher values indicate greater bridging capacity.

which exhibit high resting energy expenditure, potentially exacerbating VMS severity, although this mechanism awaits empirical validation.[56]

Sleep disturbances, AO, and VMS seem to form an interconnected cluster, suggesting complexity beyond simple pairwise relationships. Previous studies have reported an association between sleep disturbances and VMS, potentially attributable to nocturnal VMS episodes.[21] Consistent with the literature, our symptom cluster analysis revealed that VMS (hot flashes and night sweats) co-occurred with sleep disturbances within the same cluster in the AO groups, a clustering pattern notably absent in the non-AO group.

In addition to weight management for women with AO, symptom management strategies should be tailored to AO status. Although the overall edge strength was comparable between networks, the NCT revealed significant structural differences. These findings suggest that intervention strategies should be stratified by AO status. In the AO network, the most central symptoms were forgetfulness, irritability, and VMS. Notably, cognitive and affective symptoms showed higher EI and strength than VMS, suggesting a disproportionate impact on the overall symptom system.[57] These centrality patterns highlight the need for greater attention to cognitive symptoms among women with AO. Emerging evidence indicates that AO is associated with increased risk of cognitive impairment.[58] However, in routine menopausal care, VMS typically receives primary attention due to its recognizable nature, whereas cognitive symptoms are frequently under-recognized. By contrast, in the non-AO group, central symptoms were VMS, palpitations, and depressive symptoms, which exhibited the highest EI and strength across network. Thus, these symptoms warrant priority attention in clinical management of women without AO.

The distinct symptom networks between AO and non-AO women highlight the clinical importance of accurate AO identification. In this context, our findings support WHtR as an effective anthropometric indicator for assessing menopausal symptom burden and health risks. A key advantage of WHtR is its uniform cutoff criterion (≥ 0.5), largely independent of ethnicity and sex, whereas cutoffs for waist circumference or waist-to-hip ratio vary considerably across populations.[59] For example, waist circumference cutoff for AO differ between Chinese women ($\geq$ 80 cm) and Japanese ($\geq$ 90 cm);[60] by contrast, the WHO recommends sex-specific waist-to-hip ratio cutoffs (≥ 0.90 for men and ≥ 0.85 for women).[61] In addition, WHtR exhibits superior predictive validity for disease and mortality risks compared with waist circumference and waist-to-hip ratio.[59,62,63]

This study has several limitations. First, although we included participants from diverse racial/ethnic backgrounds, the predominance of White participants may limit generalizability to other racial/ethnic groups. Secondly, as a secondary analysis employing listwise deletion for missing data, the analytic sample may not fully represent the broader SWAN cohort, and selection bias cannot be ruled out. Third, although SWAN data have been previously validated and demonstrate acceptable reliability, the reliance on self-reports may introduce reporting bias, warranting cautious interpretation. Finally, the cross-sectional design using single-timepoint (visit 6) data precludes establishment of temporal or causal relationships. These findings require validation through longitudinal studies or randomized controlled trials to clarify directional relationships between menopausal symptoms and AO.

CONCLUSIONS

In summary, this network analysis demonstrated that menopausal symptom interrelationships differ between women with and without AO. Network density was slightly higher in the AO group; however, global strength was comparable between groups. Nevertheless, the NCT revealed significant structural differences. Furthermore, symptom clusters differed between groups, with VMS clustering with sleep disturbance in the AO network, a pattern not observed in the non-AO network.

These findings may inform more individualized symptom assessment and management. We recommend screening menopausal women for AO using WHtR and tailoring symptom management strategies to AO status. Longitudinal studies in diverse populations are needed to confirm these findings and clarify temporal and causal relationships.

REFERENCES

1. Harlow SD, Gass M, Hall JE, et al. Executive summary of the stages of reproductive aging workshop + 10: addressing the unfinished agenda of staging reproductive aging. *J Clin Endocrinol Metab* 2012; 97:1159-1168. doi:10.1210/jc.2011-3362
2. Santoro N, Roeca C, Peters BA, Neal-Perry G. The menopause transition: signs, symptoms, and management options. *J Clin Endocrinol Metab* 2021;106:1-15. doi:2021122204215971200
3. Fenton A. Weight, shape, and body composition changes at menopause. *J -Life Health* 2021;12:187-192. doi:10.4103/jmh.jmh_123_21
4. Davis SR, Castelo-Branco C, Chedraui P, et al. Understanding weight gain at menopause. *Climacteric* 2012;15:419-429. doi:10.3109/13697137.2012.707385
5. Opoku AA, Abushama M, Konje JC. Obesity and menopause. *Best Pract Res Clin Obstet Gynaecol* 2023;88:102348. doi:10.1016/j.bpobgyn.2023.102348
6. Dhawan D, Sharma S. Abdominal obesity, adipokines and non-communicable diseases. *J Steroid Biochem Mol Biol* 2020;203: 105737. doi:10.1016/j.jsbmb.2020.105737
7. Ben Ali S, Belfki-Benali H, Ahmed DB, et al. Postmenopausal hypertension, abdominal obesity, apolipoprotein and insulin resistance. *Clin Exp Hypertens* 2016;38:370-374. doi:10.3109/10641963.2015.1131286
8. Sternfeld B, Wang H, Quesenberry CP, et al. Physical activity and changes in weight and waist circumference in midlife women: Findings from the Study of Women's Health Across the Nation. *Am J Epidemiol* 2004;160:912-922. doi:10.1093/aje/kwh299
9. Safizadeh F, Mandic M, Hoffmeister M, Brenner H. Colorectal cancer and central obesity. *JAMA Netw Open* 2025;8:e2454753. doi:10.1001/jamanetworkopen.2024.54753
10. Ahmed KY, Aychiluhm SB, Thapa S, et al. Cardiometabolic outcomes among adults with abdominal obesity and normal body mass index. *JAMA Netw Open* 2025;8:e2537942. doi:10.1001/jamanetworkopen.2025.37942
11. Nadolsky K, Garvey WT, Agarwal M, et al. American association of clinical endocrinology consensus statement: algorithm for the evaluation and treatment of adults with obesity/adiposity-based chronic disease - 2025 update. *Endocr Pract Off J Am Coll Endocrinol Am Assoc Clin Endocrinol* 2025;31:1351-1394. doi:10.1016/j.eprac.2025.07.017
12. Rubino F, Cummings DE, Eckel RH, et al. Definition and diagnostic criteria of clinical obesity. *Lancet Diabetes Endocrinol* 2025;13:221-262. doi:10.1016/S2213-8587(24)00316-4
13. Wharton S, Lau DCW, Vallis M, et al. Obesity in adults: a clinical practice guideline. *CMAJ Can Med Assoc J J Assoc Medicale Can* 2020;192:E875-E891. doi:10.1503/cmaj.191707
14. International Menopause Society. The IMS White Paper: The Role of Lifestyle Medicine in Menopausal Health: A Review of Non-Pharmacologic Interventions. Accessed December 22, 2025 https://www.imsociety.org/education/world-menopause-day-2025/resources/
15. Tang R, Fan Y, Luo M, et al. General and central obesity are associated with increased severity of the VMS and sexual symptoms of menopause among Chinese women: a longitudinal study. *Front Endocrinol* 2022;13:814872. doi:10.3389/fendo.2022.814872
16. Jing T, Guijun Z, Yehua Y, Limei C. The study on the relationship between abdominal obesity and climacteric syndromes in perimenopausal women with normal body weight. *J Chengde Med Univ* 2023;40:381-384. doi:10.15921/icnki.cyxb.2023.05.001
17. El Hajj A, Wardy N, Haidar S, et al. Menopausal symptoms, physical activity level and quality of life of women living in the mediterranean region. *PloS One* 2020;15:e0230515. doi:10.1371/journal.pone.0230515
18. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet Lond Engl* 2024;403:1027-1050. doi:10.1016/S0140-6736(23)02750-2
19. Fang Y, Liu F, Zhang X, et al. Mapping global prevalence of menopausal symptoms among middle-aged women: a systematic review and meta-analysis. *BMC Public Health* 2024;24:1767. doi:10.1186/s12889-024-19280-5
20. Hunter MS, Chilcot J. Testing a cognitive model of menopausal hot flushes and night sweats. *J Psychosom Res* 2013;74:307-312. doi:10.1016/j.jpsychores.2012.12.005
21. Baker FC, Lampio L, Saaresranta T, Polo-Kantola P. Sleep and sleep disorders in the menopausal transition. *Sleep Med Clin* 2018; 13:443-456. doi:10.1016/j.jsmc.2018.04.011
22. Xi X, Pei C, Song N. Prevalence of and trends in obesity and insulin resistance among US perimenopausal women, 2003-2023. *Menopause* 2025;32:1088-1094. doi:10.1097/GME.0000000000002612
23. Borsboom D, Deserno MK, Rhemtulla M, et al. Network analysis of multivariate data in psychological science. *Nat Rev Methods Primer* 2021;1:58. doi:10.1038/s43586-021-00055-w
24. Zhu Z, Xing W, Hu Y, Wu B, So WKW. Paradigm shift: moving from symptom clusters to symptom networks. *Asia-Pac J Oncol Nurs* 2022;9:5-6. doi:10.1016/j.apjon.2021.12.001
25. Zhao D, Lv G, Qi M, et al. The structure of menopausal syndrome: using network analysis to understand unique symptomatic relationships. *Int J Gynecol Obstet* 2023;160:289-296. doi:10.1002/ijgo.14353
26. Zhang T, Wan Y, Geng L. Unraveling the core and bridge menopausal symptoms of perimenopausal women: a network analysis. *Menopause* 2024;31:996-1005. doi:10.1097/GME.0000000000002431
27. Min SH, Yang Q, Docherty SL, Lee C. Comparison of symptoms between midlife women in perimenopause and postmenopause using network comparison test. *West J Nurs Res* 2025;47:630-640. doi:10.1177/01939459251333669
28. Fang H, Berg E, Cheng X, Shen W. How to best assess abdominal obesity. *Curr Opin Clin Nutr Metab Care* 2018;21:360. doi:10.1097/MCO.0000000000000485
29. Gold EB, Crawford SL, Shelton JF, et al. Longitudinal analysis of changes in weight and waist circumference in relation to incident vasomotor symptoms: The study of women's health across the nation (SWAN). *Menopause* 2017;24:9-26. doi:10.1097/GME.0000000000000723
30. Ashwell M, Gibson S. A proposal for a primary screening tool: "keep your waist circumference to less than half your height. *BMC Med* 2014;12:207. doi:10.1186/s12916-014-0207-1
31. Sutton-Tyrrell K, Selzer F, Sowers MFR, et al Study of women's health across the nation (SWAN), 2002-2004: Visit 06 dataset. Published online 2025 doi:10.3886/ICPSR31181.v3
32. Aras SG, Grant AD, Konhilas JP. Clustering of > 145,000 symptom logs reveals distinct pre, peri, and menopausal phenotypes. *Sci Rep* 2025;15:640. doi:10.1038/s41598-024-84208-3
33. Avis NE, Crawford SL, Greendale G, et al. Duration of menopausal vasomotor symptoms over the menopause transition. *JAMA Intern Med* 2015;175:531-539. doi:10.1001/jamainternmed.2014.8063
34. Min SH, Yang Q, Docherty SL, Im EO, Hu X. Symptom clusters and key symptoms among midlife perimenopausal and postmenopausal women with and without metabolic syndrome. *Nurs Res* 2022;71: E28-E38. doi:10.1097/NNR.0000000000000591

35. Teng Y, Tao, Shao, Li. Correlation between the modified Kupperman Index and the Menopause Rating Scale in Chinese women. *Patient Prefer Adherence* 2013;7:223-229. doi:10.2147/PPA.S42852
36. Jing F, Zhu Z, Qiu J, et al. Contemporaneous symptom networks and correlates during endocrine therapy among breast cancer patients: a network analysis. *Front Oncol* 2023;13:1081786. doi:10.3389/fonc.2023.1081786
37. Epskamp S, Borsboom D, Fried EI. Estimating psychological networks and their accuracy: a tutorial paper. *Behav Res Methods* 2018;50:195-212. doi:10.3758/s13428-017-0862-1
38. Yu X, Zhang X, Wu K, et al. Targeting childhood loneliness in China: in silico interventions and moderated network analysis. *Child Adolesc Psychiatry Ment Health* 2025;19:89. doi:10.1186/s13034-025-00947-9
39. Marchetti I. Hopelessness: a network analysis. *Cogn Ther Res* 2019; 43:611-619. doi:10.1007/s10608-018-9981-y
40. Epskamp S, Borsboom D, Fried EI. Estimating psychological networks and their accuracy: a tutorial paper. *Behav Res Methods* 2018;50:195-212. doi:10.3758/s13428-017-0862-1
41. Epskamp S, Cramer AOJ, Waldorp LJ, Schmittmann VD, Borsboom D. qgraph: Network visualizations of relationships in psychometric data. *J Stat Softw* 2012;48:1-18. doi:10.18637/jss.v048.i04
42. Robinaugh DJ, Millner AJ, McNally RJ. Identifying highly influential nodes in the complicated grief network. *J Abnorm Psychol* 2016;125:747-757. doi:10.1037/abn0000181
43. van Borkulo CD, van Bork R, Boschloo L, et al. Comparing network structures on three aspects: a permutation test. *Psychol Methods* 2023;28:1273-1285. doi:10.1037/met0000476
44. Fortunato S, Hric D. Community detection in networks: a user guide. *Phys Rep* 2016;659:1-44. doi:10.1016/j.physrep.2016.09.002
45. Kim HJ, McGuire DB, Tulman L, Barsevick AM. Symptom clusters: concept analysis and clinical implications for cancer nursing. *Cancer Nurs* 2005;28:270-282; quiz 283-284. doi:10.1097/00002820-200507000-00005
46. Fried SK, Lee MJ, Karastergiou K. Shaping fat distribution: new insights into the molecular determinants of depot- and sex-dependent adipose biology. *Obes Silver Spring Md* 2015;23: 1345-1352. doi:10.1002/oby.21133
47. Amati F, Pennant M, Azuma K, et al. Lower thigh subcutaneous and higher visceral abdominal adipose tissue content both contribute to insulin resistance. *Obes Silver Spring Md* 2012;20: 1115-1117. doi:10.1038/oby.2011.401
48. Franklin RM, Ploutz-Snyder L, Kanaley JA. Longitudinal changes in abdominal fat distribution with menopause. *Metabolism* 2009;58: 311-315. doi:10.1016/j.metabol.2008.09.030
49. Leeners B, Geary N, Tobler PN, Asarian L. Ovarian hormones and obesity. *Hum Reprod Update* 2017;23:300-321. doi:10.1093/humupd/dmw045
50. Covassin N, Singh P, McCrady-Spitzer SK, et al. Effects of experimental sleep restriction on energy intake, energy expenditure, and visceral obesity. *J Am Coll Cardiol* 2022;79:1254-1265. doi:10.1016/j.jacc.2022.01.038
51. Ma B, Li Y, Wang X, et al. Association between abdominal adipose tissue distribution and obstructive sleep apnea in Chinese obese patients. *Front Endocrinol* 2022;13:847324. doi:10.3389/fendo.2022.847324
52. Monteleone P, Mascagni G, Giannini A, Genazzani AR, Simoncini T. Symptoms of menopause—global prevalence, physiology and implications. *Nat Rev Endocrinol* 2018;14:199-215. doi:10.1038/nrendo.2017.180
53. The 2022 hormone therapy position statement of The North American Menopause Society. *Menopause* 2022;29:767-794. doi:10.1097/GME.0000000000002028
54. Freedman RR. Menopausal hot flashes: mechanisms, endocrinology, treatment. *J Steroid Biochem Mol Biol* 2014;142: 115-120. doi:10.1016/j.jsbmb.2013.08.010
55. Speakman JR. Obesity and thermoregulation. *Handb Clin Neurol* 2018;156:431-443. doi:10.1016/B978-0-444-63912-7.00026-6
56. Müller MJ, Wang Z, Heymsfield SB, Schautz B, Bosy-Westphal A. Advances in the understanding of specific metabolic rates of major organs and tissues in humans. *Curr Opin Clin Nutr Metab Care* 2013;16:501-508. doi:10.1097/MCO.0b013e328363bdf9
57. Borgatti SP. Centrality and network flow. *Soc Netw* 2005;27:55-71. doi:10.1016/j.socnet.2004.11.008
58. Huang YY, Zhang WS, Wang J, et al. Systematic analysis of associations between obesity and memory decline. *GeroScience* Published online2025. doi:10.1007/s11357-025-01725-3
59. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0·5 could be a suitable global boundary value. *Nutr Res Rev* 2010;23:247-269. doi:10.1017/S0954422410000144
60. Ross R, Neeland IJ, Yamashita S, et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR working group on visceral obesity. *Nat Rev Endocrinol* 2020;16:177-189. doi:10.1038/s41574-019-0310-7
61. Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation World Health Organization; 2008. Accessed September 27, 2025. https://www.who.int/publications/i/item/9789241501491
62. Zhang FL, Ren JX, Zhang P, et al. Strong association of waist circumference (WC), body mass index (BMI), waist-to-height ratio (WHtR), and waist-to-hip ratio (WHR) with diabetes: a population-based cross-sectional study in jilin province, China. *J Diabetes Res* 2021;2021:8812431. doi:10.1155/2021/8812431
63. Jayedi A, Soltani S, Zargar MS, Khan TA, Shab-Bidar S. Central fatness and risk of all cause mortality: systematic review and dose-response meta-analysis of 72 prospective cohort studies. *BMJ* 2020; 370:m3324. doi:10.1136/bmj.m3324