

Association of sarcopenic obesity with cognitive dysfunction: A systematic review and meta-analysis

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ABSTRACT

الأهداف: تقييم العلاقة بين السمنة المفرطة وضعف الإدراك. قد ترتبط التغيرات في تكوين جسم الإنسان بتطور ضعف الإدراك. تُعتبر السمنة المفرطة، التي تتميز بتراكم مفرط للدهون وانخفاض كتلة العضلات، لها العديد من النتائج الصحية السلبية.

المنهجية: أجرينا مراجعة منهجية وتحليلًا تلويًا. بحثنا في قواعد بيانات PubMed، ومكتبة كوكرين، وشبكة العلوم، وEmbase، وCINAHL، وCNKI، وSinomed، وWanfang، وVIP عن دراسات تبحث في العلاقة بين السمنة المفرطة وضعف الإدراك. التزمت العملية بإرشادات «بنود التقارير المفضلة للمراجعات المنهجية والتحليلات التلوية» (PRISMA).

النتائج: شملت ثمانين دراسة، شملت 87,520 مشاركًا (5 دراسات جماعية و3 دراسات مقطعية). تناول التحليل التلوي باستخدام نموذج التأثيرات العشوائية التباين العالي ($p=0.020$)، وأظهر ارتباطًا ذا دلالة إحصائية بين السمنة المفرطة والخلل الإدراكي (نسبة الأرجحية=1.77، فاصل ثقة 95%: 1.48-2.12، $p<0.001$). أكد تحليل الحساسية صحة هذه النتائج، على الرغم من أن مخططات القمع أشارت إلى بعض التحيز في التشتت. كشفت تحليلات المجموعات الفرعية، المستندة إلى معايير تشخيصية مختلفة للسمنة المفرطة والخلل الإدراكي، عن ارتباطات متسقة.

الخلاصة: ترتبط السمنة المفرطة بالخلل الإدراكي. ومع ذلك، يُعد إجراء المزيد من البحوث باستخدام معايير ومنهجيات تشخيصية موحدة أمرًا ضروريًا لتأكيد هذه النتائج.

Objectives: To evaluate the association between sarcopenic obesity and cognitive dysfunction. Changes in human body composition may be linked to the development of cognitive dysfunction. Sarcopenic obesity, characterized by excessive fat accumulation and reduced muscle mass, is implicated in various adverse health outcomes.

Methods: We conducted a systematic review and meta-analysis. PubMed, Cochrane Library, Web of Science, Embase, CINAHL, CNKI, Sinomed, Wanfang, and VIP databases were searched for studies

examining the link between sarcopenic obesity and cognitive dysfunction. The process adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Results: Eight studies, comprising 87,520 participants (5 cohort and 3 cross-sectional studies) were included. Meta-analysis using a random effects model addressed high heterogeneity ($p=0.020$, $I^2=50.1\%$) and demonstrated a statistically significant association between sarcopenic obesity and cognitive dysfunction (odds ratio=1.77, 95% confidence interval 1.48-2.12, $p<0.001$). Sensitivity analysis confirmed the robustness of these findings, although funnel plots indicated some dispersion bias. Subgroup analyses based on varying diagnostic criteria for sarcopenic obesity and cognitive dysfunction revealed consistent associations.

Conclusion: Sarcopenic obesity is associated with cognitive dysfunction. However, further research utilizing standardized diagnostic criteria and methodologies is essential to corroborate these findings.

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Cognitive dysfunction refers to impairments in mental processes that affect memory, attention, reasoning, and problem-solving abilities.¹ This term

encompasses a range of conditions, including dementia, milder cognitive impairments, and mild cognitive decline.² Cognitive dysfunction diminishes individual quality of life and significantly impacts families and society.^{3,4} Early diagnosis and treatment may delay the progression of mental status decline and the onset of dementia. Recent studies have suggested that changes in body composition might be associated with cognitive dysfunction, potentially offering a novel approach to dementia prevention through the optimization of body fat and muscle mass.^{5,6}

Changes in body composition have been shown to contribute to various diseases, including cardiovascular diseases, metabolic disorders, decreased bone density, cognitive impairment, and an increased risk of mortality.⁷⁻¹⁰ Human body composition can be measured using various methods. Obesity is defined as a condition of excessive body fat. Sarcopenia is characterized by low muscle mass and function. Sarcopenic obesity (SO) is a condition that combines excessive obesity with low muscle mass or function.^{11,12,13} Compared to obesity and sarcopenia alone, SO often poses greater health and functional risks due to the concurrent presence of excess body fat and reduced muscle mass.¹⁴ While the associations between obesity or sarcopenia and cognitive dysfunction have been extensively studied,¹⁵⁻¹⁷ the understanding of the relationship between SO and cognitive dysfunction remains insufficient.

Here, we evaluated the relationship between SO and cognitive dysfunction through systematic evaluation and meta-analysis to explore the relationship between SO and the risk of cognitive dysfunction, which could provide evidence for SO management to prevent cognitive dysfunction in future clinical practice.

Methods. *Study design and search strategy.* We performed this systematic review and meta-analysis following the Preferred Reporting Items for Systematic

Reviews and Meta-Analyses (PRISMA) statement for observational studies.¹⁸ We applied the Newcastle-Ottawa Quality Scale (NOS) and the Joanna Briggs Institute (JBI) methodological guidance on systematic reviews of observational epidemiological studies. The study protocol was registered at PROSPERO (CRD42024544920).

We performed a literature search in English and Chinese language databases, including PubMed, Cochrane Library, Embase, Web of Science, Cumulative Index to Nursing and Allied Health Literature (CINAHL), China National Knowledge Infrastructure (CNKI), Sinomed, Wanfang, Vip Database, OpenGrey, ClinicalTrials.gov, and World Health Organization (WHO) International Clinical Trials Registry Platform (ICTRP) with a time frame from inception to May 1st, 2024. A combination of subject terms and free terms was used. Specifically, the search terms were “sarcopenia obesity/sarcopenic obesity/SO” “cognitive dysfunction/cognitive impairment/cognitive disorder /cognitive function/neurocognitive disorder/mild cognitive impairment/Alzheimer’s disease/dementia” (Table 1).

Literature selection criteria. We selected studies using the Condition, Context, and Population (CoCoPop) framework.

Condition. Observational studies examining the association between SO and cognitive dysfunction were included in our analysis. The diagnostic criteria for SO were adopted from the European Association for the Study of Obesity (EASO) and the European Society for Clinical Nutrition and Metabolism (ESPEN), along with additional criteria.¹⁹ Obesity was diagnosed using several metrics: body mass index (BMI), percent body fat (PBF), visceral fat area (VFA), waist circumference (WC), and fat mass index (FMI). Sarcopenia was identified based on the criteria from the European Working Group on Sarcopenia in Older People (EWGSOP; EWGSOP2), the Asian Working Group for Sarcopenia (AWGS; AWGS 2019), and the Foundation for the National Institutes of Health Sarcopenia project (FNIH).²⁰ Included cognitive impairments ranged from dementia and mild cognitive impairment to global cognition impairment and Alzheimer’s disease.

Context. There was no restriction on the context or setting of the studies included for analysis. We also included studies in hospital outpatient clinics, inpatient services, and community-based facilities.

Population. We included studies performed in patients with SO.

Exclusion criteria. The following criteria were used to exclude the studies: 1) no prevalence reported

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at baseline, 2) sarcopenia was measured by indirect instruments, 3) no diagnosis criteria reported, 4) animal studies, 5) conference abstracts, 6) theses, and 7) correspondences or letters.

Study selection. Two researchers independently screened the titles, abstracts, and full-text manuscripts based on the predetermined eligibility criteria in Endnote X9. Any disagreements between these two researchers were resolved after discussing with a third researcher. In addition, we also hand-screened the reference lists in the included articles to avoid missing potentially relevant studies.

Data extraction. A researcher performed the data extraction of eligible articles into a standardized spreadsheet. A second researcher double-checked the data accuracy independently.

Methodological quality assessment. The quality of the included article was assessed using the NOS for cohort studies and the JBI Evidence-based Health Care Quality Assessment tool for cross-sectional research.

Data analysis. Stata 18.0 (StataCorp, USA) was selected for statistical analysis. We reported the odds ratio (OR) and 95% confidence interval (CI) to assess the association between SO and cognitive dysfunction. We applied Cochran's Q and Higgins' I² tests to evaluate inter-study heterogeneity. Significant heterogeneity was indicated by a *p*-value less than 0.05 or an I² greater than 50%, necessitating the use of a random effects model. Subgroup analyses were conducted to explore the sources of heterogeneity. A fixed-effects model was employed in the absence of significant heterogeneity. These subgroup analyses differentiated between various categories of SO and cognitive dysfunction. Sensitivity analyses were performed to test the robustness of the results by systematically excluding studies. Funnel plots were utilized to detect potential publication bias, with statistical significance set at a *p* value less than 0.05.

GRADE evidence quality rating. The quality of evidence in each included study was assessed and graded using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework.²¹

Results. Literature search results. The electronic search retrieved 671 articles, with 8 literatures (87520 patients) included in the final review (Figure 1).

Narrative synthesis of study characteristics. There were 5 cohorts and 3 cross-sectional studies. The characteristics of the included studies are listed in Tables 2 & 3. Most of these studies were conducted in China (*n*=4). Among these studies, Zhang et al²² categorized the participants into male and female. Fu et al²³ categorized

the data into AWGS+WC, AWGS+VFA, AWGS+BMI, and AWGS+PBF groups according to inclusion criteria. Someya et al²⁴ analyzed the data on both mild cognitive impairment and dementia separately.

Methodological quality. Two studies scored 8 after evaluating the quality on the NOS scale.^{22,25} Six studies rated the entries as "yes" after evaluating the quality of the literature on JBI.^{23,24,26,27,28,29} The methodological quality assessments of the included literature are shown in Tables 4 & 5.

Sarcopenic obesity and cognitive impairment. There was a high heterogeneity among these 8 studies (*p*=0.020, I²=50.1%).²²⁻²⁹ The meta-analysis was performed using a random effects model, which showed the association between SO and an increased risk of cognitive dysfunction (OR=1.77, 95% CI 1.48-2.12, *p*<0.001) (Figure 2).

Subgroup analysis. Subgroup analyses were performed using different diagnostic criteria for SO or cognitive dysfunction (Table 6).

Different diagnostic criteria for sarcopenic obesity. Subgroup analyses were conducted based on different diagnostic criteria for SO (Figure 3). Three studies employed the simultaneous criteria of PBF for obesity and the AWGS criteria for sarcopenia to define SO.^{23,27,29} Therefore, these studies exhibited low heterogeneity (I²=1.7%, *p*=0.361) and were analyzed using a fixed-effects model. The results indicated that using PBF combined with AWGS as diagnostic criteria for SO was associated with an increased risk of cognitive dysfunction (OR=2.18, 95% CI 1.53-3.09, *p*<0.001).

Two studies used the simultaneous fulfillment of the diagnostic criteria of obesity with BMI and of sarcopenia with the diagnostic criterion of sarcopenia with AWGS as the diagnostic criterion of SO,^{23,24} with a high heterogeneity among the studies (I²=66.9%, *p*=0.049), which was analyzed using a random effects model. The results showed that SO with BMI+AWGS as diagnostic criteria was associated with an increased risk of cognitive dysfunction development (OR=2.53, 95% CI 1.22-5.26, *p*=0.013). Other studies with BMI+EWGSOP, FAT+AWGS, VFA+AWGS, WC+AWGS, BMI+FNIH, and FMI+AWGS as the diagnostic criteria for sarcopenia all had only one article, with OR (95% CI) of 1.68 (1.21-2.33), 1.54 (1.14-2.09), 1.75 (1.14-2.68), 1.96 (1.29-2.98), 1.20 (1.03-1.40), and 1.62 (0.58-4.50), respectively.²²⁻²⁸

Different diagnostic criteria for cognitive dysfunction. Subgroup analyses were further performed for different diagnostic criteria on cognitive functions (Figure 4). Two studies had a diagnosis of dementia,^{22,24}

Table 1 - Literature search strategy.

Data database	Search strategy
Cochrane Library	#1 [sarcopenia obesity] explode all trees #2 [cognitive dysfunction] explode all trees #3 (sarcopenia obesity OR sarcopenic obesity OR SO): ti, ab, kw #4 (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia): ti, ab, kw #5 (#1 or #3) and (#2 or #4)
Web of Science	(TS=(sarcopenia obesity OR sarcopenic obesity OR SO) AND TS=(cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia))
PubMed	#1 sarcopenia obesity [Title/Abstract] OR sarcopenic obesity [Title/Abstract] OR SO[Title/Abstract] #2 cognitive impairment [Title/Abstract] OR dementia [MeSH Terms] OR alzheimer's disease [MeSH Terms] OR cognitive dysfunction [MeSH Terms] OR cognitive function [Title/Abstract] OR cognitive disorder [Title/Abstract] OR neurocognitive disorder [Title /Abstract] OR mild cognitive impairment [Title/Abstract] #3 #1 and #2
Embase	#1 (sarcopenia obesity OR sarcopenic obesity OR SO).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword heading word, floating subheading word, candidate term word] #2 (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia).mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword heading word, floating subheading word, candidate term word] #3 #1 and #2
CHINAL	MH (sarcopenia obesity OR sarcopenic obesity OR SO) AND (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia)
CNKI	#1 TS = sarcopenia obesity OR sarcopenic obesity OR SO #2 TS = cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia #3 #1 and #2
Wanfang	#1 TS = sarcopenia obesity OR sarcopenic obesity OR SO #2 TS = cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia #3 #1 and #2
Sinomed	#1 sarcopenia obesity OR sarcopenic obesity OR SO #2 cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia #3 #1 and #2
Vip	#1 Title or keywords = sarcopenia obesity OR sarcopenic obesity OR SO #2 Title or keywords = cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia #3 #1 and #2
OpenGrey	(sarcopenia obesity OR sarcopenic obesity OR SO) AND (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia)
ClinicalTrials.gov	(sarcopenia obesity OR sarcopenic obesity OR SO) AND (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia)
WHO ICTRP	(sarcopenia obesity OR sarcopenic obesity OR SO) AND (cognitive dysfunction OR cognitive impairment OR cognitive disorder OR cognitive function OR neurocognitive disorder OR mild cognitive impairment OR Alzheimer's disease OR dementia)

with a high heterogeneity ($I^2=88.3\%$, $p<0.001$), which was analyzed using random effects model, showing an association between SO and dementia development (OR=2.00, 95% CI 1.35-2.97, $p=0.001$). Three studies had a diagnosis of cognitive impairment,^{26,28,29} with a low heterogeneity ($I^2=0.0\%$, $p=0.480$), which was analyzed using a fixed-effects model, showing an association between SO cognitive impairment development (OR=1.65, 95% CI 1.26-2.17, $p<0.001$). Two studies

had a diagnosis of mild cognitive impairment,^{23,24} with a low heterogeneity ($I^2=0.0\%$, $p=0.941$), which was analyzed using a fixed-effects model, showing an association between SO and MCI (OR=1.88, 95% CI 1.52-2.32, $p<0.001$). Other studies had diagnoses of AD and overall cognitive impairment ($p=0.025$, $p=0.020$, respectively).^{25,27}

Sensitivity analysis and publication bias. Sensitivity analyses were conducted to assess the reliability of the

Table 2 - Article characteristics, study design, confounders, and diagnostic criteria of included literature.

Author	Year	Country	Study design	Confounder
Zhang et al ²²	2024	United Kingdom	Cohort	Sociodemographic factors (age, TDI, race, education), lifestyle (physical activity, smoking status, alcohol status), and dietary intake (vegetables, fruits, red meat, processed meats, oily fish, coffee, and dairy products)
Zhou et al ²⁶	2023	China	Cross-sectional	Age, gender, educational status, current smoking, current alcohol consumption, history of hypertension, stroke and diabetes, and year on dialysis
Weng et al ²⁷	2023	China	Cross-sectional	Age, sex, height, weight, smoking, alcohol consumption, hypertension, diabetes, education, low physical activity, nutritional status, and depressive state
Fu et al ²³	2023	China	Cross-sectional	Age, sex, employment status, living alone status, income, education level, marital status
Someya et al ²⁴	2022	Japan	Cross-sectional	Smoking status, alcohol intake, physical activity, hypertension, diabetes and cardiovascular disease
Batsis et al ²⁵	2021	USA	Cohort	Age, gender, years of education, physical activity, hypertension, diabetes mellitus, dyslipidemia, and depressive state
Tou et al ²⁸	2021	Singapore	Cross-sectional	Age category, gender, smoking status, education, comorbidities (heart disease, high blood pressure, diabetes, lung disease, stroke, cancer, ever walked)
Wang et al ²⁹	2019	China	Cross-sectional	Age, gender, education level, smoking history and physical activity level, diabetes, hypertension, cardiovascular disease, high cholesterol, stroke, depression
				Age, sex, smoking status, alcohol use, physical activity, burden of chronic comorbidities, nutritional status, and depressive state

ALMI - appendicular lean mass relative to fat mass index; ASMI - appendicular skeletal muscle mass index; BMI - body mass index; FMI - fat mass index; PBF - percent body fat; MCI - mild cognitive insufficiency; MMSE - mini mental status examination; MoCA - Montreal Cognitive Assessment; SMI - skeletal muscle mass index; SO - sarcopenic obesity; TDI - Townsend Deprivation Index

Table 2 - Article characteristics, study design, confounders, and diagnostic criteria of included literature.

Obesity	Diagnostic criteria		
	Sarcopenia	Sarcopenic obesity	Cognitive dysfunction
BMI \geq 30 kg/m ²	Grip strength<27 kg (male), <16 kg (female); SMI<7.0 kg/m ² (male), <5.5 kg/m ² (female)	Reach diagnosis for obesity and sarcopenia	Dementia is defined as hospitalization or death from the 10th Revision of the International Classification of Diseases (ICD-10) or ICD-9 code: ICD-10 codes A81.0, F00-F03, G30, G31.0, G31.1, and G31.8, and ICD-9 codes 290.2-290.4, 291.2, 294.1, 331.0-331.2, and 331.5.
PBF \geq 35% (female), >30% (male)	Grip strength<26 kg (male), <18 kg (female); SMI<7.0 kg/m ² (male), <5.7 kg/m ² (female)	Reach diagnosis for obesity and sarcopenia	MMSE< 27: cognitive impairment
PBF \geq 35% (female), \geq 25% (male)	ASMI<7.0 kg/m ² (male), <5.7 kg/m ² (female); grip strength<28 kg (male), <18 kg (female)	Reach diagnosis for obesity and sarcopenia	21 \leq MMSE \leq 26 mild AD; 10 \leq MMSE \leq 20 severe AD
BMI \geq 28 kg/m ² ; PBF \geq 35% (female), PBF \geq 25% (male); VFA>100 cm ² ; WC>80 cm (female), WC>90 cm (male)	ASMI<7.0 kg/m ² (male), <5.7 kg/m ² (female); grip strength<28 kg (male), <18 kg (female); SPPB \leq 9	Reach diagnosis for obesity and sarcopenia	Dementia was assessed using CDR; MCI was determined using Petersen criteria
BMI \geq 25 kg/m ²	Grip strength<28 kg (male), <18 kg (female); SMI<7.0 kg/m ² (male), <5.4 kg/m ² (female)	Reach diagnosis for obesity and sarcopenia	MoCA \leq 22 MCI, MMSE \leq 23 dementia
BMI \geq 30 kg/m ²	Grip strength<35.5 kg (male), <20 kg (female)	Reach diagnosis for obesity and sarcopenia	Impaired overall cognitive functioning, abnormal scores on the AD8 scale or any of the 3 domains (memory, orientation, executive functioning)
FMI \geq 7.63 kg/m ² (male), FMI \geq 9.93 kg/m ² (female)	ALMI<7.0 kg/m ² (male), ALMI<5.4 kg/m ² (female); grip strength<28 kg (male), grip strength<18 kg (female)	Reach diagnosis for obesity and sarcopenia	Cognitive functioning was measured using RBANS Total Scale Index and Domain-Specific Index scores for all subjects according to the RBANS manual, where each score is expressed as a standardized score with a mean of 100 and a standard deviation of 15. Cognitive impairment was defined using a critical index score <80, which corresponds to the 9th percentile
PBF \geq 31.61% (male), \geq 40.68% (female)	ASMI<7.0 kg/m ² (male); <5.7 kg/m ² (female)	Reach diagnosis for obesity and sarcopenia	MMSE \leq 17 severe cognitive impairment, 18 \leq MMSE \leq 23 MCI, and subjects with MCI and severe cognitive impairment were included in the cognitive impairment

ALMI - appendicular lean mass relative to fat mass index; ASMI - appendicular skeletal muscle mass index; BMI - body mass index; FMI - fat mass index; PBF - percent body fat; MCI - mild cognitive insufficiency; MMSE - mini mental status examination; MoCA - Montreal Cognitive Assessment; SMI - skeletal muscle mass index; SO - sarcopenic obesity; TDI - Townsend Deprivation Index

combined results concerning the association between SO and cognitive function. The analyses indicated that when individual studies were sequentially omitted, the overall effect sizes for the association between SO and cognitive function remained statistically significant, suggesting reliable findings (Figure 2B). However, funnel plots revealed some dispersion bias in the included literature (Figure 2C).

GRADE evidence quality rating. The primary outcomes of the included studies were assessed using the GRADE framework. SO diagnosed by the PBF+AWGS criteria and cognitive dysfunction diagnosed by cognitive impairment criteria were rated as low-quality evidence, while all other outcomes were rated as very low-quality evidence (Table 7).

Discussion. Our meta-analysis indicated that SO is associated with a 1.77-fold increased risk of cognitive dysfunction. Specifically, SO diagnosed using PBF+AWGS criteria was linked to a 2.18-fold increase in risk, while SO diagnosed using BMI+AWGS criteria showed a 2.53-fold increase. Subgroup analyses, based on different diagnostic criteria for cognitive dysfunction—such as dementia, cognitive impairment, and mild cognitive impairment—revealed an increased risk of dementia, cognitive impairment, and mild cognitive impairment by 2.00-, 1.65-, and 1.88-fold, respectively.

Zhou et al²⁵ found that, among patients requiring maintenance hemodialysis, the incidence of cognitive impairment in patients with SO was approximately 34.6%. Moreover, the risk of developing cognitive impairment in these patients was significantly higher than that in patients with either sarcopenia or obesity. After adjusting for confounding factors such as age, gender, and educational status, the associations between either sarcopenia or obesity and cognitive impairment disappeared, whereas a significant correlation between SO and cognitive impairment remained (OR=1.47). This result indicated that SO might be an independent predictor of cognitive dysfunction. It also suggested that the demographic characteristics of the study participants could have a specific impact on the research findings. Future research could further explore the independent and interactive effects of sarcopenia, obesity, and others on cognitive dysfunction to achieve a deeper understanding of the underlying mechanisms.

The association between SO and the risk of cognitive dysfunction may vary depending on the diagnostic criteria for SO and cognitive dysfunction. Currently, different diagnostic criteria for SO and

cognitive dysfunction have been adopted in various studies, making it difficult to compare the results of these studies. Fu Y et al²² evaluated the prevalence of SO and its correlation with MCI by combining different obesity diagnostic indicators with the criteria of AWGS. The study found that, according to different diagnostic criteria, the prevalence of SO fluctuated between 1.7% and 8.0%. Although there was a consistent association between SO and MCI under different diagnostic criteria, compared with other obesity indicators, the prevalence of SO defined based on BMI was lower, and its consistency with MCI was also relatively weak. This was different from the results of our study. It might be related to the heterogeneity of the data and the insufficient sample size included in this study. There are various types of cognitive dysfunction. Currently, research on SO and cognitive dysfunction primarily focuses on dementia, mild cognitive impairment, and Alzheimer's disease. The specific impacts of SO on these three conditions are still unclear and require further investigation.

The global prevalence of SO is approximately 1% in the general population but increases to 17% among those aged 80 to 89 years.⁷ Previous research has established a strong link between obesity and sarcopenia with cognitive dysfunction. Our findings further demonstrate that SO, a condition characterized by both sarcopenia and obesity—is also associated with cognitive dysfunction. This association may stem from the synergistic effects of obesity and sarcopenia on the development of abnormal mental processes. Semenova et al³⁰ identified numerous risk alleles common to both sarcopenia and obesity. Dowling et al²⁴ discovered differentially expressed miRNAs in cases of sarcopenia or obesity, which may play crucial roles in the pathogenesis of SO.³¹ Moreover, Livshits et al³² suggested that obesity, sarcopenia, and SO are inflammation-driven disorders that may share common pathogenic mechanisms. The pathogenesis of SO may be linked to factors such as a sedentary lifestyle, adipose tissue disorders, comorbidities, and metabolic changes associated with aging. The interrelated and synergistic nature of obesity and sarcopenia contributes to a vicious cycle of fat gain and muscle loss.¹³

Currently, the most effective treatments for SO include aerobic or resistance exercise and nutritional interventions.^{33,34} Hsu et al³³ showed that aerobic exercise reduced fat mass and body weight, resistance exercise reduced fat mass and improved muscle strength, and the combination of aerobic and resistance exercises reduced fat mass and improved walking speed, while

Table 3 - Study characteristics and group assignments of included literature.

Author	Characteristics	Non-sarcopenia and obesity	Sarcopenic obesity
Zhang et al ²²	Sample size (female), n	71532	4837
	Age (female), years, M±SD	63.89±2.78	64.34±2.82
	Dementia (female), HR	1.00	1.424(1.227-1.653)
	Sample size (male), n	66948	2676
	Age (male),	64.14±2.80	64.66±2.81
	Dementia HR (male)	1.00	1.989(1.702-2.323)
Zhou et al ²⁶	Sample size (male/female)	875(547/328)	592(351/241)
	Age, years, M±SD	47.0(37.0-57.0)	65.0(56.0-73.0)
	Incidence of cognitive impairment, %	15.5	34.9
	Cognitive impairment, OR	1.00	1.54(1.13-2.08)
Weng et al ²⁷	Sample size (male/female), n	36(10/16)	25(13/12)
	Age, years, M±SD	70.1±7.8	75.2±3.5
	Incidence of AD, %	/	/
	AD, OR	1.00	5.84 (1.23–27.11)
Fu et al ²³	Sample size (male/female), n	1038(125/913)	181(26/155)
	Age, years, M±SD	61.33±6.1	64.82±6.0
	Incidence of MCI, %	/	/
	MCI OR - AWGS + VFA	1.00	1.75(1.14, 2.68)
	MCI OR - AWGS+WC	1.00	1.96(1.29, 2.99)
	MCI OR - AWGS+PBF	1.00	1.94(1.29, 2.93)
	MCI OR - AWGS + BMI	1.00	1.45(0.67,3.12)
Someya et al ²⁴	Sample size (male/female), n	960(349/611)	76(35/41)
	Age, years, M±SD	72(68-76)	79(74-80)
	Incidence of MCI, %	14.5	40.8
	Incidence of dementia, %	1.6	14.5
	MCI, OR	1.00	2.11(1.23-3.62)
	Dementia, OR	1.00	6.17(2.50-15.27)
Batsis et al. ²⁵	Sample size (male/female), n	5822(2578/3244)	750(318/432)
	Age, years, M±SD	/	/
	Incidence of overall cognitive function, %	16.8	22.6
	Overall cognitive function, OR	1.00	1.20(1.03-1.40)
Tou et al ²⁸	Sample size (male/female), n	221(90/131)	39(14/25)
	Age, years, M±SD	/	/
	Incidence of cognitive impairment, %	/	/
Wang et al ²⁹	Sample size (male/female), n	479(248/231)	57(34/23)
	Age, years, M±SD	67.54±5.83	71.56±7.49
	Incidence of cognitive impairment, %	9.6	22.8
	Cognitive impairment, OR	1.00	2.550(1.196-5.435)

AD - Alzheimer dementia, AWGS - Asian Working Group for Sarcopenia, BMI - body mass index, HR - hazard ratio, M±SD - mean±standard deviation, PBF - percent body fat, OR - odds ratio, VFA - visceral fat area, WC - waist circumference.

nutritional interventions, especially the low-calorie, high-protein diet, reduced fat mass without affecting muscle mass and grip strength. Alizadeh et al³⁴ showed that low-intensity blood flow restriction training improved muscle mass and strength and effectively prevented the worsening of obesity in sarcopenia. Prokopidis et al³⁵ showed that a high abundance

of the specific gut microbiome was associated with better protein synthesis and overall metabolic health, which might be driven by fiber and fat depletion to counteract the progression of sarcopenia and obesity. Therefore, they recommended dietary modifications based on the gut microbiome profile to manage obesity and sarcopenia in the elderly population. Whether

Table 4 - Evaluation of included cohort literature using the Newcastle-Ottawa Quality Scale.

Author	Selection			Demonstration that the outcome of interest was not present at baseline	Comparability	Outcome			Total score
	Representativeness of exposed	Representativeness of unexposed	Ascertainment of exposure		Control for important confounding factors	Blinded independent evaluation	Adequate follow-up period	Adequacy of follow-ups	
Zhang et al ²²	1	1	1	1	2	0	1	1	8
Batsis et al ²⁵	1	1	1	1	2	0	1	1	8

Table 5 - Evaluation of included cross-sectional literature by Joanna Briggs Institute Evidence-Based Healthcare quality assessment.

Author	A	B	C	D	E	F	G	H
Zhou et al ²⁶	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weng et al ²⁷	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Fu et al ²³	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Someya et al ²⁴	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Tou et al ²⁸	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Wang et al ²⁹	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

A - Determination of whether the inclusion criteria for the research subjects were clearly defined, B - Determination of whether the research subjects and research sites were described in detail, C - Determination of whether standard, effective, and credible methods were used to measure the exposure factors, D - Determination of whether objective and standard methods were used to measure health issues, E - Determination of whether the confounding factors were clarified, F - Determination of whether measures were taken to control the confounding factors, G - Determination of whether effective and credible methods were used to evaluate the outcome indicators, H - Determination of whether the data analysis method was appropriate.

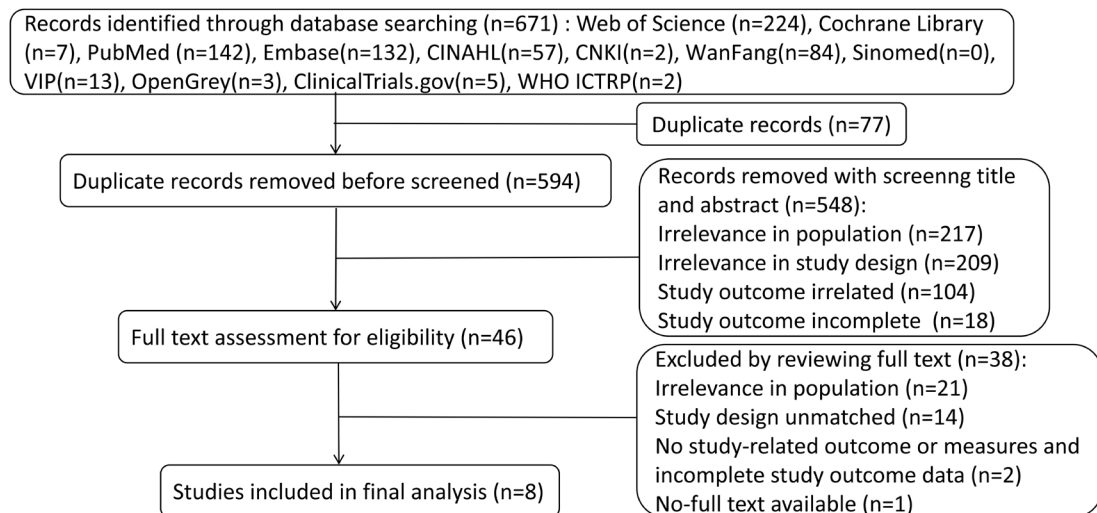
**Figure 1 -** Flowchart for the literature selection.

Table 6 - Subgroup analysis on associations between sarcopenic obesity and cognitive dysfunction.

Subgroups	Number of studies	Heterogeneity		Effect model	OR (95% CI)	P-value
		I ²	P			
Different diagnostic criteria for SO						
PBF+AWGS	3	1.7%	0.361	fixed effect model	2.18 (1.53, 3.09)	<0.001
BMI+AWGS	2	66.9%	0.049	random effect model	2.53 (1.22, 5.26)	0.013
Different diagnostic criteria for cognitive dysfunction						
SO and dementia	2	88.3%	<0.001	random effect model	2.00 (1.35, 2.97)	0.001
SO and cognitive impairment	3	0.0%	0.480	fixed effect model	1.65 (1.26, 2.17)	<0.001
SO and MCI	2	0.0%	0.941	fixed effect model	1.88 (1.52, 2.32)	<0.001

AWGS, Asian Working Group for Sarcopenia; CI, confidence interval; MCI, mild cognitive impairment; OR, odds ratio; PBF, percent body fat; SO, sarcopenic obesity.

Table 7 - Quality of evidence evaluated by GRADE framework.

Outcome measures	Quality of evidence assessment					OR (95% CI)	Quality of evidence
	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication bias		
SO and cognitive dysfunction	Serious ^a	Serious ^b	Not Serious	Not Serious	Serious ^d	1.77(1.48-2.12)	Very low
<i>Different diagnostic criteria for SO</i>							
PBF+AWGS	Serious ^a	Not Serious	Not Serious	Not Serious	Serious ^d	2.18 (1.53-3.09)	Low
BMI+AWGS	Serious ^a	Serious ^b	Not Serious	Serious ^c	Serious ^d	2.53 (1.22- 5.26)	Very low
<i>Different diagnostic criteria for cognitive dysfunction</i>							
SO and dementia	Serious ^a	Serious ^b	Not Serious	Not Serious	Serious ^d	2.00 (1.35-2.97)	Very low
SO and cognitive impairment	Serious ^a	Not Serious	Not Serious	Not Serious	Serious ^d	1.65 (1.26-2.17)	Low
SO and MCI	Serious ^a	Not Serious	Not Serious	Serious ^c	Serious ^d	1.88 (1.52-2.32)	Very low

Risk of bias exists in random sequence generation, allocation concealment, and blinding; ^aSignificant heterogeneity is present; ^cSmall sample size; ^dFunnel plot suggests potential publication bias

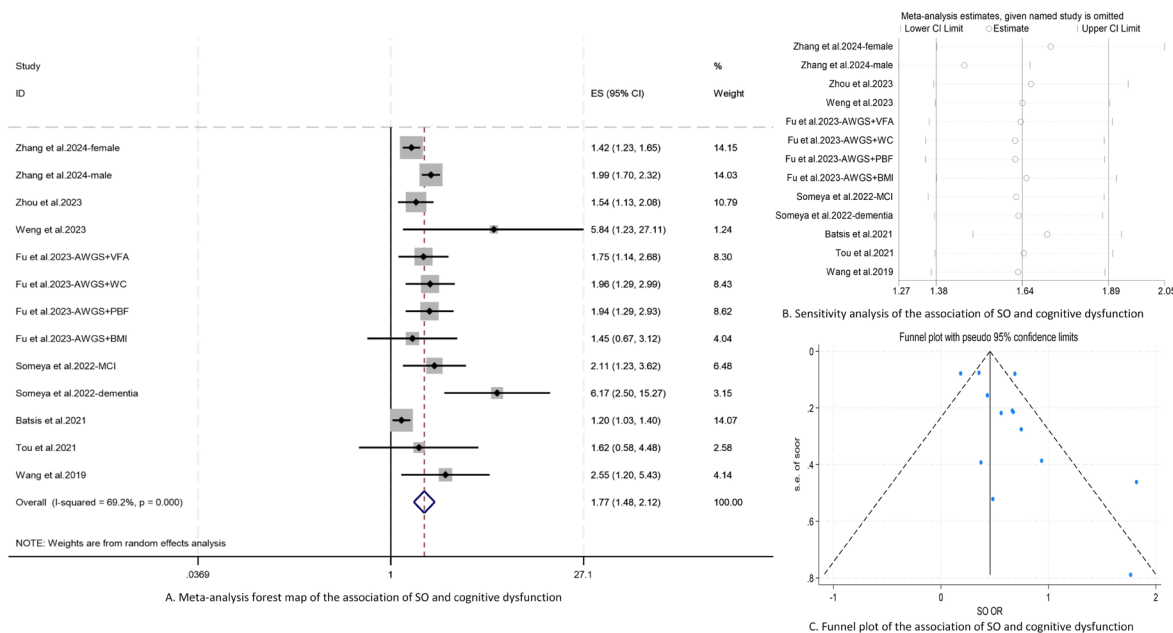


Figure 2 - Association of sarcopenic obesity and cognitive dysfunction, A) meta-analysis forest map of the association between sarcopenic obesity and cognitive dysfunction; B) sensitivity analysis of the association between sarcopenic obesity and cognitive dysfunction; C) funnel plot of the association between sarcopenic obesity and cognitive dysfunction.

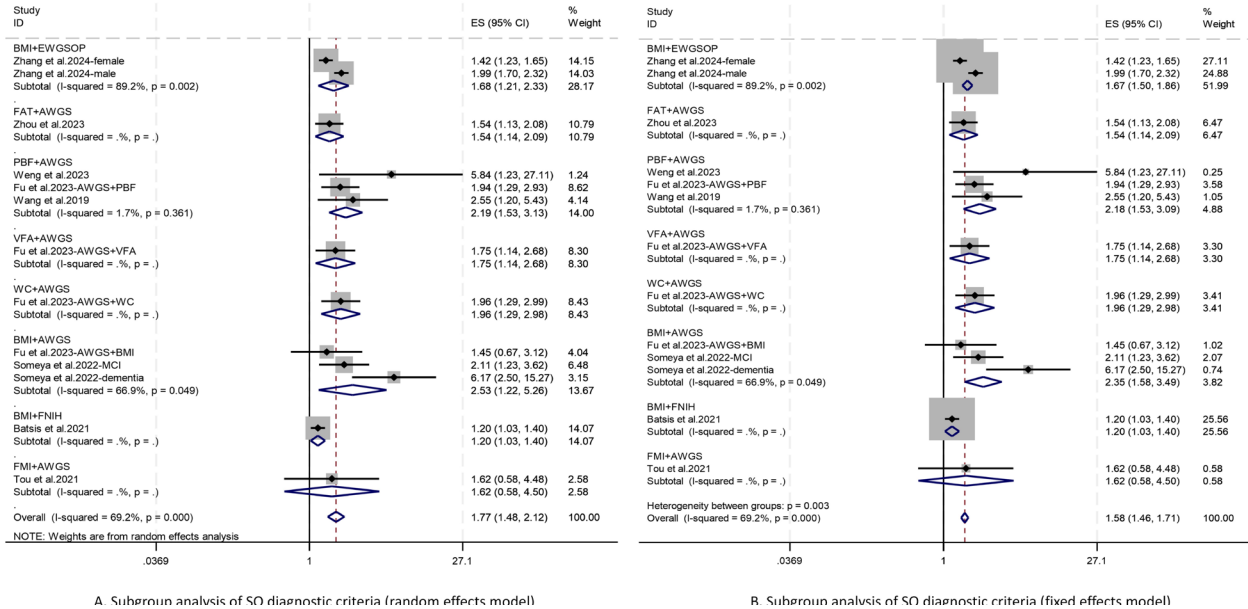


Figure 3 - Subgroup analysis based on different diagnostic criteria on sarcopenic obesity, A) random effects model; B) fixed effects model.

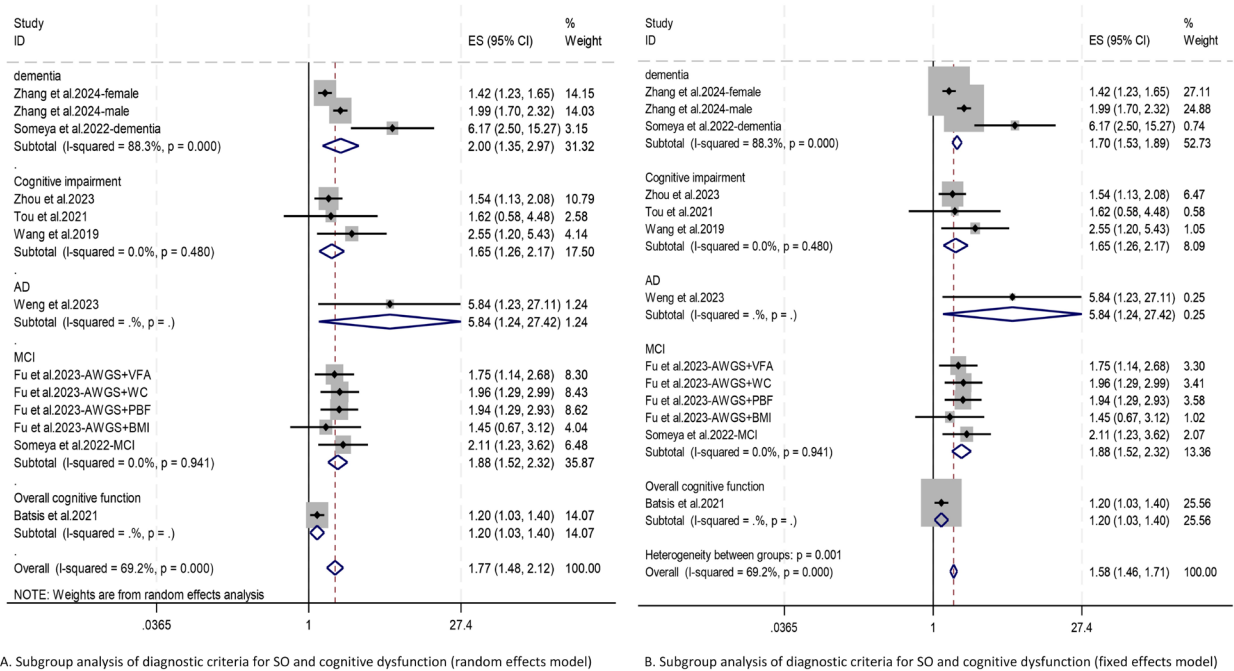


Figure 4 - Subgroup analysis based on different diagnostic criteria on cognitive dysfunction A) subgroup analysis of diagnostic criteria for sarcopenic obesity and cognitive dysfunction with random effects model; B) subgroup analysis of diagnostic criteria for sarcopenic obesity and cognitive dysfunction with fixed effects model.

combined exercise and nutritional interventions could increase muscle mass and promote fat metabolism requires further studies. Whether treatments on SO could delay or stop cognitive decline also requires further investigations.

Study limitations. Our study carried several limitations. Of the eight studies included in our meta-analysis, 6 were cross-sectional research that could not allow us to determine a causal relationship between SO and cognitive decline. To address this limitation, future research should incorporate prospective longitudinal studies to better understand the potential causal relationship between sarcopenic obesity and cognitive impairment. Additionally, the small number of studies and potential publication bias might limit the robustness of our conclusions. Efforts should be made to include several high-quality studies in future meta-analyses to enhance the credibility of the findings. Moreover, most studies were conducted on elderly participants in China. To overcome this limitation, subsequent studies should include more diverse demographic groups, such as different age ranges, ethnicities, and geographic regions, to improve the generalizability of the findings. Future research should also focus on elucidating the underlying biological mechanisms linking SO and cognitive impairment, including the roles of inflammation, insulin resistance, and hormonal changes. Randomized controlled trials should be conducted to evaluate the effectiveness of interventions in mitigating the cognitive decline associated with SO. Finally, efforts should be made to establish standardized diagnostic criteria for SO and cognitive impairment to facilitate more consistent and comparable research outcomes.

Conclusion. Our findings indicate an association between SO and cognitive dysfunction. However, more rigorous research is needed to expand the study population, standardize research methodologies, and refine the focus of studies. Such efforts are essential to validate the relationship between SO and cognitive dysfunction under varied diagnostic criteria.

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