




Association Between Body Composition and Risk of Type 2 Diabetes Mellitus: A Systematic Review and Meta-Analysis

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Background: Although being overweight or obese is a significant risk factor for type 2 diabetes mellitus (T2DM), conventional measures like body weight and body mass index have limitations in distinguishing between muscle and fat. Body composition metrics have superior value in explaining metabolic heterogeneity.

Objective: This study conducted the systematic review and meta-analysis to investigate the relationship between body composition (fat/muscle-related indicators) and T2DM.

Methods: We conducted a systematic search of eight databases to identify observational studies reporting the relationship between body composition and T2DM up to July 31, 2025. To pool effect sizes, random/fixed-effects models were employed. Subgroup and sensitivity analyses explored heterogeneity sources. Study quality was assessed via the Agency for Healthcare Research and Quality and Newcastle-Ottawa Scale tools.

Results: A total of 36 studies with 2,614,625 participants were included. Fat-related analyses demonstrated that leg fat mass reduces T2DM risk (OR = 0.54 [0.46, 0.63], $P < 0.0001$), while body fat mass (OR = 2.36 [1.39, 4.02], $P < 0.0001$), trunk fat mass (OR = 1.35 [1.12, 1.62], $P < 0.0001$), visceral fat area (OR = 3.18 [1.28, 7.89], $P < 0.0001$), body fat percentage (OR = 1.67 [1.31, 2.14], $P < 0.0001$), and fat mass index (OR = 1.17 [1.08, 1.27], $P < 0.0001$) increase the risk. Muscle-related analyses indicated that lower skeletal muscle mass index is a risk element for T2DM (OR = 1.46 [1.17, 1.82], $P = 0.0007$), while lean mass changes showed no significant association.

Conclusion: The results concluded that T2DM is associated with both abnormal fat/muscle mass and imbalanced fat distribution. Our findings highlighted that based on the results of body composition assessment, we can identify high-risk populations and develop personalized interventions to optimize T2DM prevention strategies.

Keywords: type 2 diabetes mellitus, body composition, observational study, meta-analysis

Introduction

Overweight and obesity are characterized by excessive body fat accumulation.¹ The number of people with overweight and obesity globally has increased rapidly in recent years, and it is projected to exceed 50% by 2025.² The rising trend has been paralleled by a proportional increase in the incidence of type 2 diabetes mellitus (T2DM).³ In 2024, the 11th edition of the IDF Diabetes Atlas reported that about 589 million adults worldwide were living with T2DM. By 2050, projections indicate that this figure will rise to 853 million, with T2DM accounting for over 90% of all cases of diabetes.⁴ Diabetes-related healthcare expenditures have surpassed one trillion dollars for the first time and continue to rise.⁵ Overweight and obesity, together with genetic predisposition, unhealthy lifestyle behaviors (eg, physical inactivity and poor diet), and socioeconomic factors, are well-established risk factors for T2DM, collectively contributing to the escalating global burden.^{6,7}

Weight management is essential to prevent and treat T2DM. A significant dose-response relationship has been observed, with each 1% body weight reduction associated with a 2.74% increase in partial diabetic remission probability and a 2.17% increase in complete diabetic remission probability.⁸ Although body mass index (BMI) and body weight are

simple measures commonly used for obesity screening, they cannot distinguish between muscle and fat tissue.⁹ Meanwhile, BMI can lead to the misclassification of obesity across populations with varying body compositions and metabolic risks, and its application can give rise to the “obesity paradox” observed when assessing disease risk.¹⁰

This review focuses on body composition as a modifiable factor that can be directly targeted in clinical and public health interventions, thereby offering actionable guidance for weight management and metabolic risk reduction. Specifically, body composition indicators can be assessed through multiple technologies such as dual-energy X-ray absorptiometry (DXA), bioelectrical impedance analysis (BIA), magnetic resonance imaging (MRI), computed tomography (CT), predictive equations, and the hydrodensitometry weighing method to allow for the capture of individual variations in metabolic heterogeneity. This approach precisely identifies specific conditions like sarcopenic obesity, avoiding biases associated with an excessive focus on weight loss alone. Multiple body composition metrics can reflect body fat quality, fat percentage, fat distribution, and muscle composition in detail.¹¹

Recently, a growing number of studies have noted the significance of body composition changes in the onset and progression of T2DM. However, due to variations in indicators, measurement methods, and research conclusions, the relationship between specific body composition indicators and T2DM remains unclear. To provide precise evidence for weight management strategies for patients with T2DM, we intend to conduct a systematic review and meta-analysis to thoroughly assess the influence of fat and muscle-related indicators measured by various methods on the risk of T2DM.

Materials and Methods

This systematic review and meta-analysis investigated the impact of body composition on the risk of T2DM in accordance with the Cochrane Handbook for Systematic Reviews. Additionally, we followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and the Meta-analysis of Observational Studies in Epidemiology report guideline (MOOSE) while reporting this review,^{12,13} ([Supplementary Tables S1](#) and [S2](#)). We registered it in PROSPERO (CRD420251080770).

Literature Search

We systematically searched the literature, including PubMed, Embase, Cochrane Library, Web of Science, Scopus, China National Knowledge Infrastructure (CNKI), Wanfang Data Knowledge Service Platform, and China Science and Technology Journal Database (VIP), from the establishment of the database to July 31, 2025. Search strategies for each database combined subject headings with free-text terms: (“Diabetes Mellitus, Type 2”[Mesh] OR “Diabetes Mellitus, Noninsulin Dependent”[Title/Abstract] OR “Diabetes Mellitus, Adult-Onset”[Title/Abstract]) AND (“Body Composition”[Mesh] OR “body fat mass”[Title/Abstract] OR “trunk fat mass”[Title/Abstract] OR “leg fat mass”[Title/Abstract] OR “body fat percentage”[Title/Abstract] OR “visceral fat mass”[Title/Abstract] OR “skeletal muscle mass”[Title/Abstract]). [Supplementary Table S3](#) details the specific search strategy. To ensure comprehensiveness, we manually identified potentially eligible studies by reviewing review articles in relevant fields and checking the references lists of included literature.

Study Selection

After duplicates were removed, the literature screening process was conducted in two stages. First, two researchers (CXR and SGL) individually scanned the title and abstract of each study against the inclusion criteria. Then, they read the entire article in the remaining studies to judge whether they satisfied the criteria. The senior author (ST) was consulted to settle any possible disputes. All of the above steps were performed in EndNote X9 software.

Inclusion and Exclusion Criteria

The inclusion criteria were based on the PICOS (Participants, Intervention, Comparability, Outcomes, Study Design) framework recommended by the PRISMA guidelines: (1) Participants: patients with T2DM (age \geq 18 years). (2) Exposures: body composition indicators related to body fat and muscle mass measured using methods such as BIA, DXA, CT, MRI, and predictive equations. (3) Outcomes: the prevalence or occurrence of T2DM. Additionally, we included research that revealed hazard ratios (HR), relative risks (RR), or odds ratios (OR) adjusted for relevant

confounding factors, along with 95% confidence intervals (CI). (4) Study design: observational studies, including cohort studies, case-control studies, and cross-sectional studies.

The exclusion criteria were as follows: (1) We did not include gestational diabetes patients and type 1 diabetes mellitus individuals due to significant differences in the pathological mechanisms, and prediabetes was not included, as it represented a reversible state of metabolic instability rather than a definitive disease endpoint. (2) publications in languages other than English or Chinese; (3) literature of the following types: conference abstracts, clinical trials, reviews, meta-analyses, and animal studies; (4) duplicated publications; (5) studies with incomplete data where multiple attempts to contact the study authors were unsuccessful.

When multiple articles involved the same cohort population, we selected the one with the most complete information. For our study, we only used the most recently published data.

Data Extraction

Authors, publication year, location, design, sample size, number of T2DM cases, body composition measurement tools, body composition indicators, adjustment factors, and OR/RR/HR values with 95% CI were extracted from each study by two researchers (CXR and SGL) independently. Different opinions were handled by consulting the senior researcher (ST). Additionally, we prioritized the effect estimates from the most fully adjusted model reported in each study.

Quality Assessment

The quality of the included cross-sectional studies was assessed based on the Agency for Healthcare Research and Quality (AHRQ) criteria, consisting of 11 items.¹⁴ For each of the 11 items, a study received 1 point if the criterion was met, and 0 points otherwise. The studies were categorized into low level (0–3 points), medium level (4–7 points), and high level (8–11 points) according to their scores. The quality of cohort studies was evaluated using the Newcastle-Ottawa Scale (NOS).¹⁵ This evaluation was based on the selection of the exposed and unexposed cohorts, comparability, and outcome. Ultimately, studies were categorized into three levels: low quality (1–3 stars), medium quality (4–6 stars), and high quality (7–9 stars). Two researchers (CXR and SGL) conducted the quality assessments independently and resolved any discrepancies by consulting the senior author (ST).

Statistical Analysis

A quantitative synthesis was performed when two or more independent studies reported the effect size of the same body composition indicator. Given the generally low incidence of T2DM in the cohort studies included in this research, HRs and RRs were treated as ORs during data merging.

In this meta-analysis, we processed the effect sizes based on the variable types of the body composition indicators. For continuous variables, we first extracted the effect size representing the risk per unit change. If a study reported the effect size per standard deviation (SD) change, we converted this estimate to the effect size per unit change. For categorical variables, we directly included the effect size if the indicator was dichotomized. When studies provided effect sizes across three or more categories, we combined them into a single estimate. Subsequently, all effect sizes were first converted to their logarithmic forms and standard errors, then weighted and pooled using the generic inverse variance method. Finally, the effects of each study and the comprehensive results were visualized using a forest plot.

Research heterogeneity was analyzed by the Cochrane Q test and the I^2 index. The fixed-effects model was applied when $I^2 \leq 50\%$ and $P \geq 0.05$, whereas the random-effects model was applied when $I^2 > 50\%$ and $P < 0.05$. For studies providing subgroup data, we conducted the subgroup analysis (study type, body composition measurement method, gender, and region) to identify possible heterogeneity. The sensitivity analysis was conducted by successively eliminating individual studies to examine the impact of each study on the aggregate estimate.

We evaluated publication bias by plotting a funnel plot when the meta-analysis included ≥ 10 studies. Egger's test and Begg's test were employed to verify the funnel plot's symmetry. Additionally, to address the potential impact of publication bias on the overall effect size, the trim-and-fill technique was used.

We used R 4.4.3 software (meta and metafor packages) for all statistical analyses. Statistical significance was defined as $P < 0.05$. If the conditions for quantitative synthesis were not met, we proceeded with a descriptive analysis of the research results.

Results

Study Selection

Figure 1 displays the literature selection process and outcomes. This flow diagram shows the standard procedure from database retrieval to the final inclusion of studies in the research. A total of 19,044 records were retrieved. There were 4,675 duplicate records among them. An initial screening was conducted by looking at the titles and abstracts and finding 684 appropriate records. After reviewing the full text, we removed 157 articles that failed to address body composition; 229 studies that did not include participants who met the criteria for T2DM; 141 articles that lacked the appropriate statistical analysis and extracted data; 106 articles that were not observational studies; and 17 articles that were not available in full text. Additionally, we conducted a manual search using the snowball method and identified four relevant references, two of which did not examine the impact of body composition on T2DM. Ultimately, we included 36 articles in the review.

Information on Selected Studies

Supplementary Table S4 presents comprehensive information on the included studies. Our review included 20 cohort studies^{16–35} and 16 cross-sectional studies,^{36–51} involving 2,614,625 participants. All studies were original research published from 2003 to 2025. 21 publications were carried out in Asia (9 in Korea,^{17,18,23,24,29,30,32,43,47} 7 in China,^{26,31,36,39,40,46,51} 4 in Japan,^{33,35,37,50} and 1 in Malaysia⁴⁸), 11 in North America (8 in the United States,^{16,20,22,25,28,34,41,42} 2 in Mexico,^{45,49} and 1 in Canada¹⁹), 3 in Europe (2 in the United Kingdom^{27,44} and 1 in Germany³⁸), and 1 in Peru in South America.²¹ All studies adjusted for covariates during regression analysis, with most

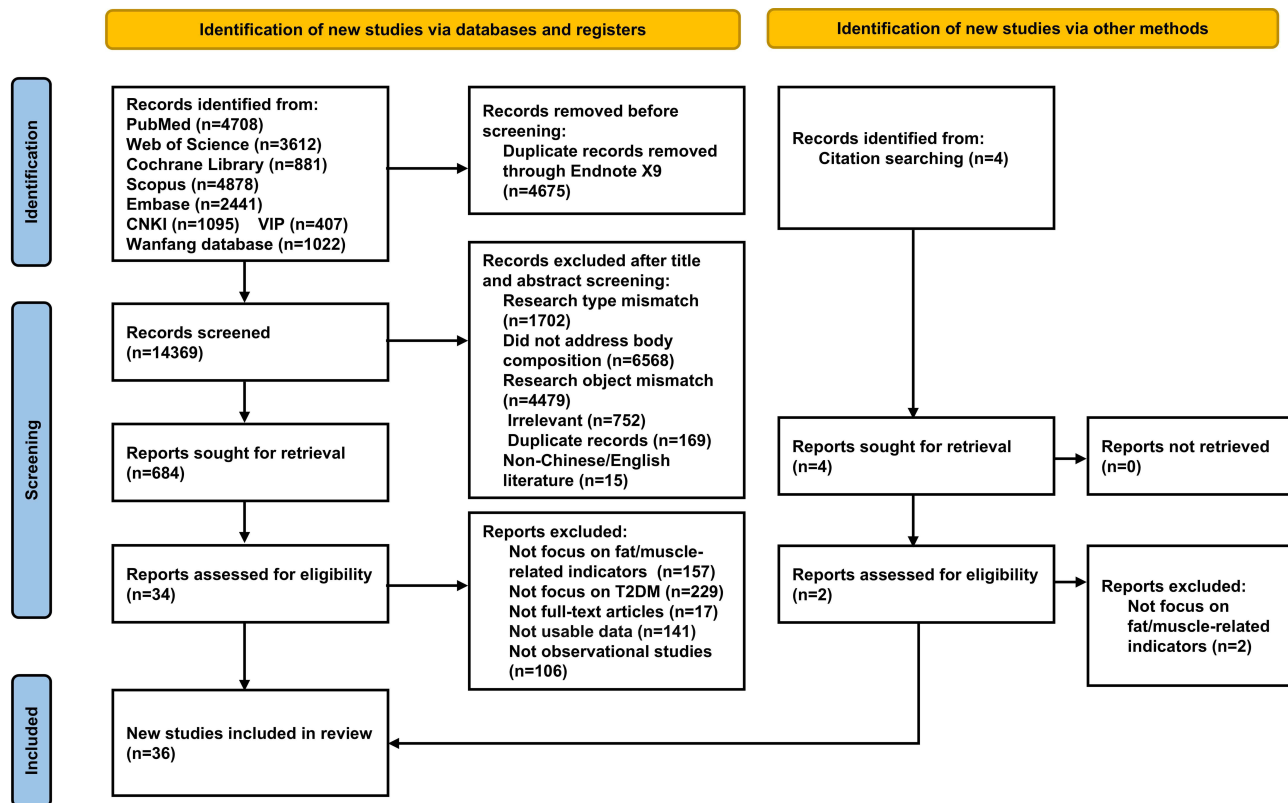


Figure 1 PRISMA flow diagram for the selection of included studies.

adjusting for baseline factors, lifestyle habits, blood biochemical indicators, and other chronic diseases. 19 studies used BIA,^{16,17,20,21,23,24,26,27,29,30,32,38,40,45,46,48–51} 6 studies used DXA,^{28,33,34,42,43,47} 2 studies were based on CT scans,^{22,37} 1 study was based on MRI,³⁹ 7 studies were based on validated predictive equations,^{18,25,31,35,36,41,44} and 1 study used the hydrodensitometry weighing method for body composition measurement.¹⁹ The diagnostic criteria for T2DM primarily included random blood glucose, two-hour postprandial blood glucose, or two-hour post-oral glucose tolerance test results ≥ 11.1 mmol/L, fasting blood glucose ≥ 7.0 mmol/L, hemoglobin A1c (HbA1c) $\geq 6.5\%$, physician-diagnosed diabetes, self-reported diagnosis of diabetes, and use of insulin or anti-diabetic medications.

Study Quality Evaluation

[Supplementary Tables S5](#) and [S6](#) display the evaluation outcomes of the selected publications. The quality of all publications ranged from moderate (12/36)^{31–34,44–51} to high (24/36).^{16–30,35–43} No significant risk of bias was associated with any of the studies. Consequently, the meta-analysis incorporated all 36 investigations.

Results of Meta-Analysis

Body Fat Percentage (BF%)

BF% is defined as the proportion of body fat mass relative to total body mass. There were nine observational studies (three cross-sectional studies and six cohort studies) with 308,815 participants.^{19,24,26,29,31,35,44–46} According to the random-effects model analysis, we found that the higher BF% was linked to higher odds of T2DM prevalence (OR = 1.67 [1.31, 2.14], $P < 0.0001$) ([Figure 2A](#)). Subgroup results from cohort studies further validated that high BF% constituted a risk factor for T2DM (OR = 1.57 [1.20, 2.05], $P < 0.0001$) ([Supplementary Table S7](#)). Other subgroup analysis results were revealed in [Supplementary Tables S8–S10](#), but these subgroups did not differ significantly from one another. Sensitivity analysis validated the robustness of the outcomes (pooled OR range: 1.53–1.81) ([Supplementary Figure S1a](#)). After exclusion of Hong et al 2017, heterogeneity decreased to $I^2 = 67.2\%$, and the pooled OR was 1.75 (95% CI: 1.42–2.14, $P = 0.0004$), identifying it as a major source of heterogeneity ([Supplementary Figure S1b](#)). Funnel plot symmetry tests along with Egger's test ($P = 0.0056$) and Begg's test ($P = 0.0204$) suggested possible publication bias ([Supplementary Figure S1c](#)). After adjusting for six additional studies using the trim-and-fill method, the effect size weakened and lost statistical significance (OR = 1.26 [0.92, 1.71]), indicating that the influence of small-sample research may have led to the initial results being exaggerated.

According to random-effects model analysis, we found a dose-response relationship between BF% and T2DM (OR = 1.10 [1.08, 1.12], $P < 0.0001$) ([Figure 2B](#)).^{16,20,21,25,31,35,38} The subgroup results from cohort studies indicated that T2DM risk increased by 10% for each unit rise in BF% (OR = 1.10 [1.07, 1.13], $P < 0.0001$) ([Supplementary Table S7](#)). Subgroup analysis also demonstrated that body composition measurement methods were the primary source of heterogeneity ($P = 0.0007$) ([Supplementary Table S8](#)). Studies using the BIA method showed lower heterogeneity (OR = 1.07 [1.06, 1.09], $I^2 = 41.6\%$, $P_{\text{heterogeneity}} = 0.1278$), while the predictive equation method showed significant heterogeneity (OR = 1.13 [1.10, 1.16], $I^2 = 80.3\%$, $P_{\text{heterogeneity}} = 0.0001$). Regional differences also represented a potential source of heterogeneity: Europe subgroup results were most consistent ($I^2 = 30.4\%$), while North America exhibited extremely high heterogeneity ($I^2 = 95.3\%$) ([Supplementary Table S10](#)). There were no significant differences between the gender subgroups ([Supplementary Table S9](#)). Sensitivity analysis validated the robustness of the results (pooled OR range: 1.09–1.10) ([Supplementary Figure S2a](#)). The funnel plot symmetry tests and the Egger's test ($P = 0.0039$) suggested possible publication bias ([Supplementary Figure S2b](#)). After imputing six additional studies using the trim-and-fill method, the adjusted effect size increased to OR = 1.14 (95% CI: 1.11–1.17), indicating that the original analysis may have slightly underestimated the effect due to the excluded studies with partially positive results.

Body Fat Mass (BFM)

BFM is defined as the absolute weight of adipose tissue in the human body. There were three cohort studies with 113,251 participants.^{25,31,35} Random-effects model analysis indicated that the higher the BFM, the higher the incidence of T2DM (OR = 2.36 [1.39, 4.02], $P < 0.0001$) ([Figure 3A](#)). There was no significant difference between the gender subgroups ($P > 0.05$) ([Supplementary Table S9](#)). Sensitivity analysis found Tang et al 2024 (M) as the key source of heterogeneity

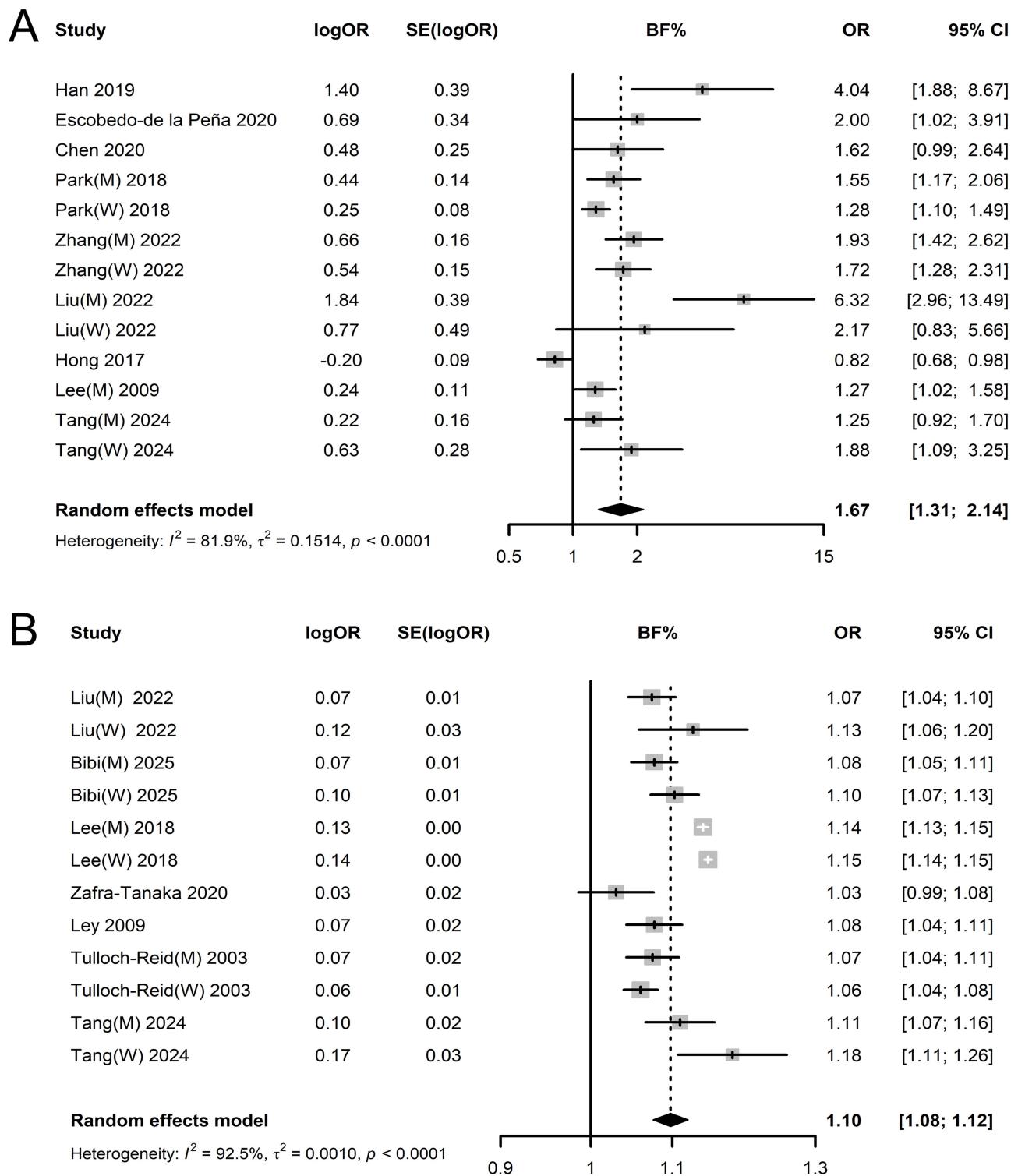


Figure 2 The association between body fat percentage (BF%) and T2DM. **(A)** Q_{high} vs Q_{low} . **(B)** For every increase of 1 unit. The pooled effect estimates, represented by the diamond, are displayed in bold.

([Supplementary Figure S3a](#)). After exclusion, the results showed OR = 2.90 (95% CI: 1.84–4.56, $I^2 = 57.1\%$, and $P_{heterogeneity} = 0.0534$) ([Supplementary Figure S3b](#)).

Meta-analysis results indicated that each kilogram (kg) increment in BFM was positively correlated with the risk of T2DM (OR = 1.04 [1.02, 1.07], $P < 0.0001$) ([Figure 3B](#)).^{25,31,35,38,43,49} The subgroup results from cohort studies

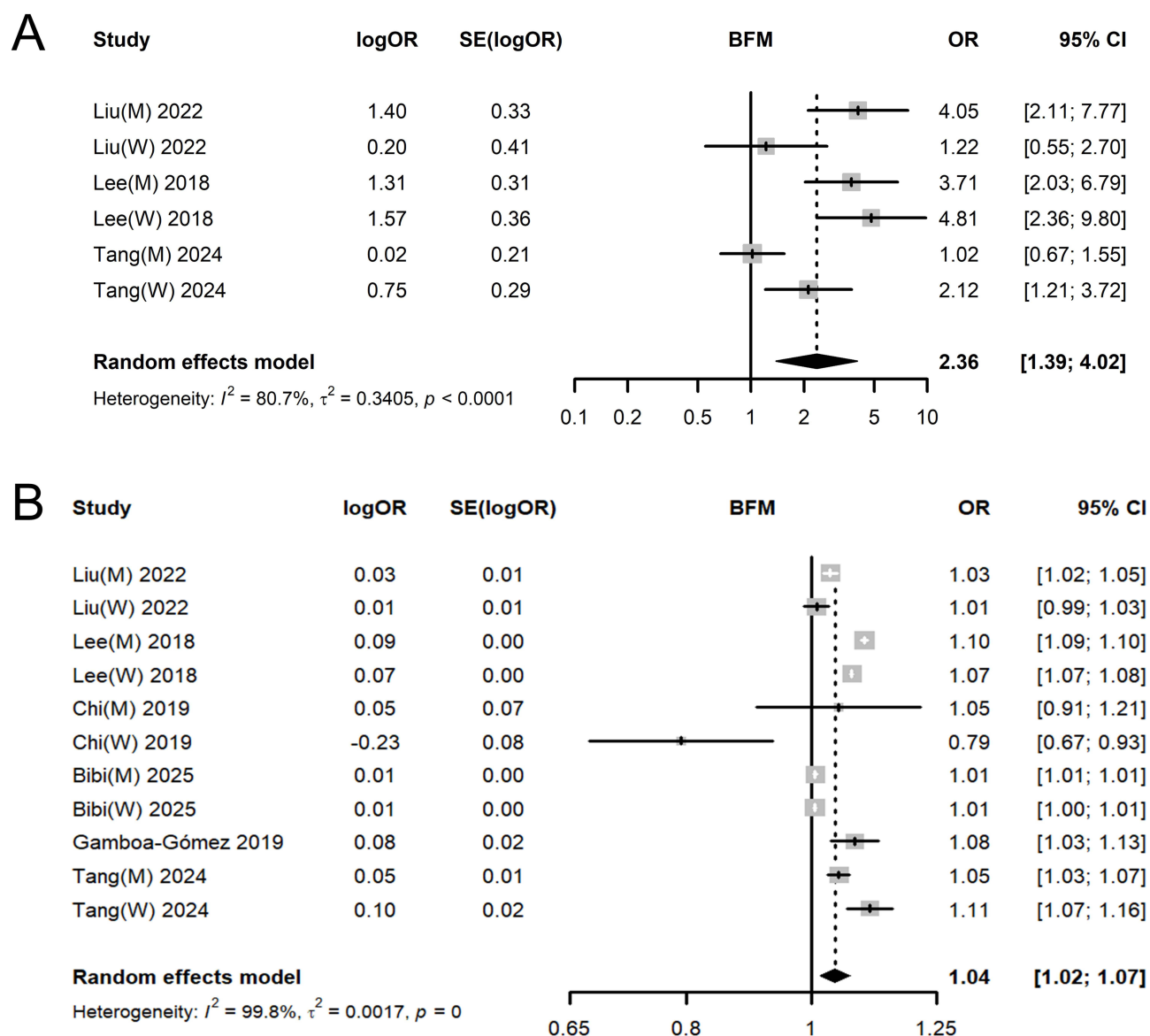


Figure 3 The association between body fat mass (BFM) and T2DM. **(A)** Q_{high} vs Q_{low} . **(B)** For every increase of 1 unit. The pooled effect estimates, represented by the diamond, are displayed in bold.

indicated that the probability of T2DM rose by 6% for an additional 1 kg in BFM (OR = 1.06 [1.03, 1.09], $P < 0.0001$) ([Supplementary Table S7](#)). Subgroup analysis revealed that body composition measurement method and region were the primary sources of heterogeneity ($P < 0.0001$) ([Supplementary Tables S8](#) and [S10](#)). Subgroup analysis by gender failed to explain heterogeneity ([Supplementary Table S9](#)). Sensitivity analysis results validated the robustness of findings (pooled OR range: 1.04–1.05) ([Supplementary Figure S4a](#)).

Trunk Fat Mass (TFM)

TFM refers to the sum of subcutaneous fat and visceral fat within the trunk region, including the chest, abdomen, and back. There were two cross-sectional studies with 14,510 participants.^{43,47} The random-effects model showed that an additional 1 kg in trunk fat mass correlated with higher probabilities of T2DM prevalence (OR = 1.35 [1.12, 1.62], $P < 0.0001$) ([Figure 4](#)). Subgroup analysis by gender indicated no significant difference in effect size ([Supplementary Table S9](#)). Excluding Chi et al 2019 (W) reduced heterogeneity to 69.4%, and the pooled OR = 1.23 (95% CI: 1.10–1.39, $P = 0.0383$), suggesting it may be the primary source of heterogeneity ([Supplementary Figure S5a](#) and [b](#)).

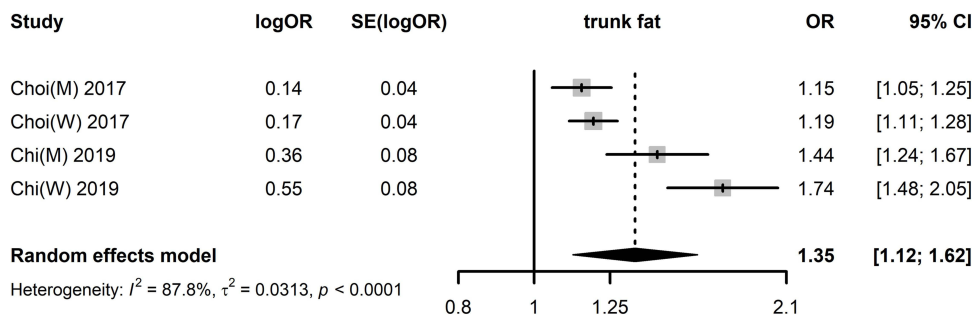


Figure 4 The association between trunk fat mass (TFM) (for every increase of 1 unit) and T2DM. The pooled effect estimates, represented by the diamond, are displayed in bold.

Leg Fat Mass (LFM)

LFM refers to the absolute fat weight in both lower limbs. There were two cross-sectional studies with 14,510 participants.^{43,47} The random-effects model showed that an additional 1 kg in leg fat mass correlated with significantly lower probabilities of T2DM prevalence (OR = 0.54 [0.46, 0.63], $P < 0.0001$) (Figure 5). Sensitivity analysis and subgroup analysis results were presented in [Supplementary Figure S6a](#) and [Table S9](#). Excluding Chi et al 2019 (M) reduced heterogeneity to 73.2%, with the pooled OR = 0.50 (95% CI: 0.44–0.57, $P = 0.0241$) ([Supplementary Figure S6b](#)). However, heterogeneity remained high and needed further study.

Fat Mass Index (FMI)

FMI is defined as the ratio of fat mass to the square of height, which eliminates the influence of height on fat quantity. There were four observational studies (three cohort studies and one cross-sectional study) with 1,731,984 participants.^{18,25,32,41} According to the random-effects model, the pooled OR was 1.17 (95% CI: 1.08–1.27, $P < 0.0001$) (Figure 6). The results from cohort studies further validated that each unit increment of FMI was linked to an 18% increase in the risk of T2DM (OR = 1.18 [1.08, 1.30], $P < 0.0001$), and subgroup analysis results were presented in [Supplementary Tables S7–S10](#). Sensitivity analysis validated the stability of the results (pooled OR range: 1.13–1.19) ([Supplementary Figure S7a](#)), but the heterogeneity of its effect size should be interpreted with caution.

Visceral Fat Area (VFA)

VFA is defined as the cross-sectional area of visceral fat tissue measured in a specific abdominal cross-sectional image. It specifically refers to fat located around the visceral organs within the abdominal cavity. There were two observational studies (one cross-sectional study and one cohort study) involving 12,789 participants.^{17,37} The random-effects model analysis revealed that higher VFA was linked to increased odds of T2DM (OR = 3.18 [1.28, 7.89], $P < 0.0001$) (Figure 7). Subgroup analysis by gender failed to resolve heterogeneity ([Supplementary Table S9](#)). Sensitivity analysis found Kim et al 2022 (W) as the primary source of heterogeneity ([Supplementary Figure S8a](#)). After removing this

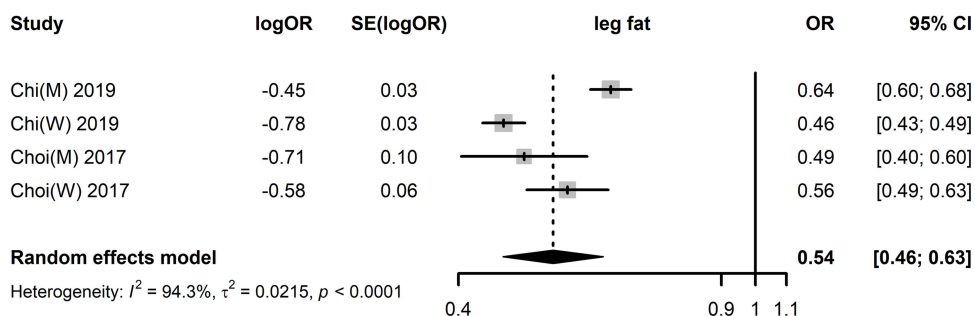


Figure 5 The association between leg fat mass (LFM) (for every increase of 1 unit) and T2DM. The pooled effect estimates, represented by the diamond, are displayed in bold.

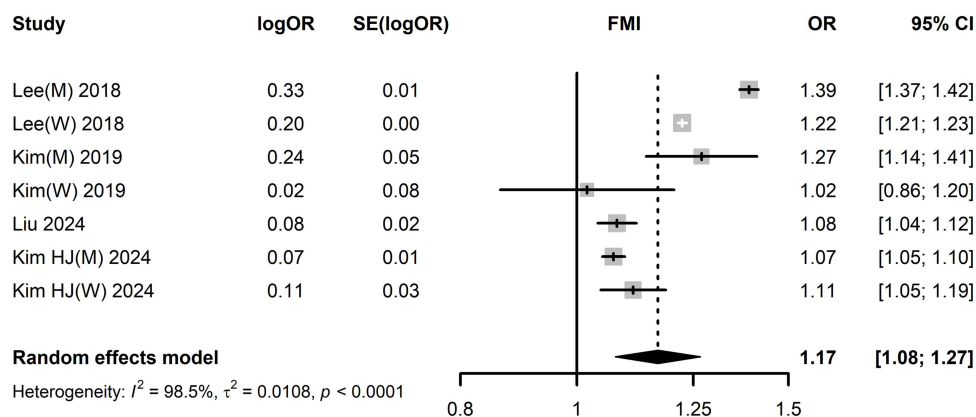


Figure 6 The association between fat mass index (FMI) (for every increase of 1 unit) and T2DM. The pooled effect estimates, represented by the diamond, are displayed in bold.

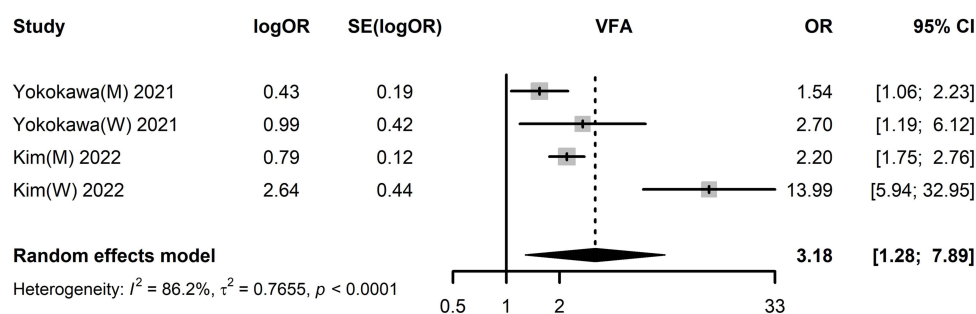


Figure 7 The association between visceral fat area (VFA) (Q_{high} vs Q_{low}) and T2DM. The pooled effect estimates, represented by the diamond, are displayed in bold.

outlier, heterogeneity significantly decreased ($I^2 = 35.3\%$), with the pooled OR = 1.99 (95% CI: 1.49–2.64, $P = 0.2132$) ([Supplementary Figure S8b](#)).

The meta-analysis results suggested that the dose-response association between VFA and T2DM was not significant and required further investigation ([Supplementary Figures S9a–c](#) and [Table S9](#)).^{39,40,46,50}

Lean Mass (LM)

LM refers to the weight remaining after subtracting fat mass from total body weight. We also analyzed the impact of LM on T2DM incidence. The outcomes indicated that statistical significance remained unclear. The dose-response relationship between LM and T2DM required additional research to confirm ([Supplementary Table S7](#), [S9](#) and [S10](#), and [Figure S10a](#) and [b](#)).^{31,34,35,49}

Skeletal Muscle Mass Index (SMI)

SMI is defined as the percentage of skeletal muscle mass relative to total body mass. This systematic review examined the association between SMI and T2DM, incorporating two cohort studies and one cross-sectional study with 248,751 participants.^{29,30,36} According to the random-effects model, the pooled OR was 1.46 (95% CI: 1.17–1.82, $P = 0.0007$) ([Figure 8A](#)). The subgroup results from cohort studies further validated that low SMI was a significant predictor of T2DM (OR = 1.56 [1.18, 2.06], $P = 0.0005$) ([Supplementary Table S7](#)). Sensitivity analysis revealed that heterogeneity significantly decreased to an acceptable level after excluding the Hong et al 2017 (W) study ($I^2 = 48.1\%$, $P_{heterogeneity} = 0.1456$), and the effect size remained robust (OR = 1.26 [1.17, 1.36], $P = 0.1456$) ([Supplementary Figure S11a](#) and [b](#)).

According to two cohort studies, the random-effects model showed that the probability of T2DM rose by 8% for each unit decrease in SMI (OR = 1.08 [1.04, 1.11], $P < 0.0001$) ([Figure 8B](#)).^{29,30} Sensitivity analysis revealed that excluding Hong et al 2017 (M) significantly reduced heterogeneity ($I^2 = 65.1\%$, $P_{heterogeneity} = 0.0903$), while the effect size remained stable (OR = 1.06 [1.04, 1.08], $P = 0.0903$) ([Supplementary Figure S12a](#) and [b](#)).

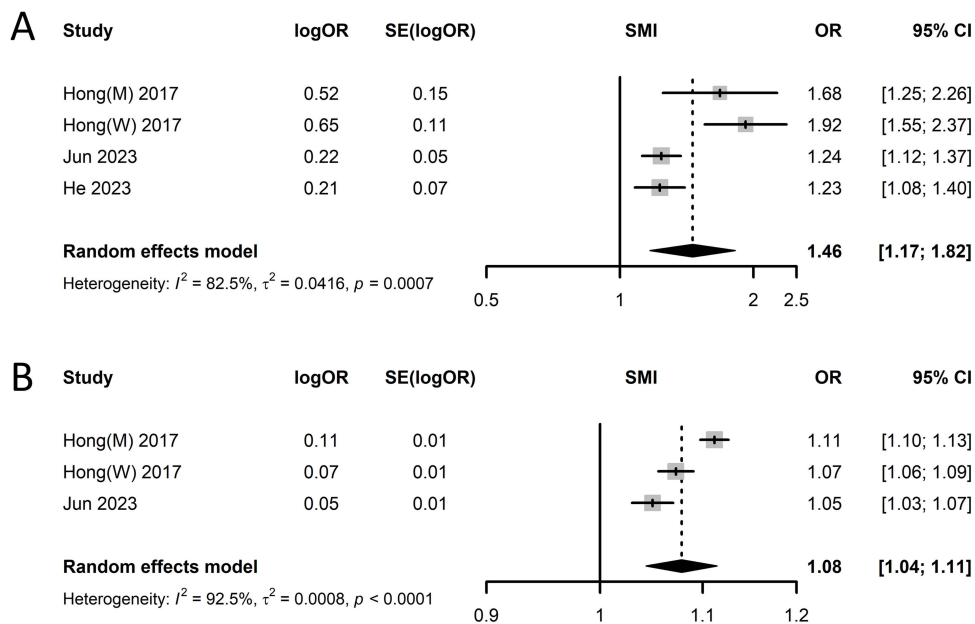


Figure 8 The association between skeletal muscle mass index (SMI) and T2DM. **(A)** Q_{low} vs Q_{high} . **(B)** For every decrease of 1 unit. The pooled effect estimates, represented by the diamond, are displayed in bold.

Descriptive Systematic Review

Eight studies have also shown the correlation between body composition and T2DM. However, due to heterogeneity in indicator measurement methods, unit conversions, and statistical models, meta-analysis was not feasible. Kim et al confirmed that appendicular skeletal muscle mass index (ASMMI) and lean body mass index (LBMI) had a protective effect against T2DM, while body fat mass index (BFMI) increased T2DM risk.¹⁸ Higher fat-to-muscle mass ratio increased the incidence of T2DM.²⁷ Increased trunk fat percentage was linked to higher T2DM risk, while increased leg fat percentage showed a protective effect.³³ Son et al indicated that for each unit decrease in muscle mass index, T2DM risk increased by 35%.²³ Phase angle (PhA) exhibited a significant dose-response protective effect in men, but the relationship was not statistically significant for women.⁴⁸ Both studies indicated that higher visceral adipose tissue (VAT) was associated with increased T2DM risk, but since Granados et al did not provide standard deviation data, standardized effect size conversion could not be performed.^{22,38} Overall, the evidence supported that muscle mass, regional fat distribution, and fat-muscle interaction indicators might have important clinical predictive value.

Discussion

This study focused on a comprehensive assessment of regional fat distribution (such as trunk fat, leg fat, and visceral fat), aiming to address gaps in the existing evidence. Fat-related indicators (BF%, BFM, TFM, FMI, and VFA) were all positively correlated with T2DM risk, but increased leg fat mass significantly decreased the risk. Previous large-scale studies also confirmed that higher BF% or VFA increased T2DM risk, consistent with our findings.^{52,53} Muscle-related analyses showed that low SMI is an independent predictor of T2DM, while LM has no significant association. Tang et al discovered a U-shaped relationship between LM and T2DM.³⁵ The heterogeneity in findings stems mainly from differences in sample characteristics and analytical approaches. Moreover, many studies overlooked the critical confounding effect of the interaction between adiposity and lean mass, thereby compromising the validity of their results.

Anatomical differences in fat distribution sites can produce opposing metabolic effects. Visceral fat directly interferes with insulin signaling and reduces adiponectin secretion by releasing pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6), ultimately inducing insulin resistance.^{40,54,55} In contrast, subcutaneous fat in the legs, due to its reduced lipolysis rate and strong free fatty acid storage capacity, can reduce ectopic lipid deposition and maintain lower inflammation levels, thereby improving metabolic homeostasis.^{56,57} As the primary insulin-dependent

organ for glucose uptake (accounting for 80–90% of postprandial glucose disposal), skeletal muscle directly weakens glucose storage capacity when its mass decreases.⁵⁵ Additionally, dysregulation of myokines (such as irisin and IL-6) may further exacerbate insulin resistance by regulating lipolysis and inflammatory responses.^{58–60}

The NOS/AHRQ criteria were used to evaluate the quality of selected studies, and the results indicated moderate to high quality. Notably, to clarify the relationship between T2DM and body composition, we performed the study-type subgroup analysis to distinguish between cross-sectional and cohort studies, with a particular focus on the results obtained from the cohort study subgroup. However, this study observed significant heterogeneity across most body composition metrics. There are many ways to measure body composition; therefore, caution is warranted when comparing results obtained from different measurement methods. Imaging methods (DXA, MRI, and CT) provide precise tissue distribution data with minimal error, leading to stable estimates and narrow confidence intervals.⁶¹ Although BIA is convenient and widely used, its results are susceptible to hydration and device-specific factors, which introduce variability.⁶² Prediction equations based on variables like age and BMI are population-specific; applying them to other cohorts may reduce their validity.¹¹ This methodological variability likely accounts for the higher heterogeneity, inflated effect sizes, and wider confidence intervals associated with prediction equations.

Additionally, differences in statistical models may constitute a source of internal heterogeneity. All included studies controlled for age, and most studies also adjusted for consistent core confounders, such as BMI, sex, and key clinical biomarkers (including lipid profile and blood pressure). Notably, most studies additionally adjusted for genetic susceptibility (family history of diabetes), socioeconomic factors (educational attainment, income level), and lifestyle factors (smoking, alcohol intake, dietary habits, and physical activity patterns). Subgroup analysis results further suggested that demographic factors such as gender and region contributed to inconsistencies in baseline body composition characteristics and threshold definitions. The observed heterogeneity across different populations can be partially attributed to interactions between environmental exposures and genetic background. For example, dietary patterns characterized by high salt and carbohydrate intake, as well as substantial meat consumption, are prevalent in many Asian populations. Consequently, compared to Africans and Europeans with the same BMI levels, Asians typically exhibit lower muscle mass and a more pronounced central obesity pattern, making them more susceptible to T2DM.⁶³ Moreover, genetic variations, including differences in adipokine regulation or muscle fiber composition, may further modulate these associations. From a clinical perspective, although genetic susceptibility and environmental determinants cannot be directly altered at the individual level, body composition provides evidence for directly implementable interventions to reduce the risk of T2DM.

The findings of our study suggest that healthcare professionals should routinely measure body composition in clinical practice to assess metabolic status. In terms of nutritional intervention, different body composition outcomes require various energy intakes and dietary patterns.^{64,65} For exercise plans, aerobic training aids in fat loss, while resistance training promotes muscle gain.^{66,67} Furthermore, for individuals at high risk of diabetes (such as those with a family history of the disease or unhealthy lifestyle habits), implementing precise assessments and personalized interventions aimed at reducing fat and increasing muscle mass can help reduce the future burden of T2DM.^{10,68} Future studies should conduct multicenter, prospective, large-scale cohort studies incorporating longitudinal body composition tracking data and fully accounting for the interactions between muscle and fat to further elucidate the influence of body composition on T2DM development. Furthermore, standardized methodologies for assessing body composition should be instituted, cross-technology measurement protocols should be formulated, and a consensus on threshold values for these indicators must be achieved.

Nonetheless, this study has specific limitations. First, 16 cross-sectional surveys were included, which cannot establish a causal relationship between body composition and T2DM. Second, due to resource constraints, this study only included English and Chinese literature, potentially overlooking regional evidence and introducing a risk of selection bias. Third, the majority of the included studies were conducted in Asia. The generalizability of these conclusions to other ethnic populations requires further validation. Additionally, the results of the publication bias test suggested the potential publication bias. Finally, all studies were observational and thus susceptible to confounding.

Conclusions

Our study demonstrated a suggestive correlation between abnormal body composition and T2DM risk. While leg fat mass had a protective effect, the increased visceral fat area and trunk fat mass greatly raised T2DM risk. Additionally, the loss of muscle mass was an independent risk factor. Although genetic and environmental factors also influence T2DM development, body composition represents a modifiable and clinically actionable determinant. Focusing on fat and muscle distribution allows for targeted interventions that can be implemented directly to reduce risk, providing practical guidance beyond non-modifiable or long-term environmental influences. By monitoring and analyzing body composition, patients can be provided with personalized weight management intervention plans to reduce the incidence of T2DM.

Data Sharing Statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

Author Contributions

Xinrui Cao: Conceptualization, Formal analysis, Data curation, Investigation, Visualization, Writing-original draft, Writing-review & editing. Guoli Shen: Investigation, Data curation, Validation, Writing-review & editing. Ting Sun: Conceptualization, Funding acquisition, Project administration, Supervision, Writing-review & editing. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declared no conflict of interest.

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