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ABOUT COVER

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Association between sarcopenic obesity and higher risk of type 2 diabetes in adults: A systematic review and meta-analysis

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Abstract

BACKGROUND

The coexistence of sarcopenia and obesity is referred to as sarcopenic obesity (SO) and it has been hypothesized that the two components of SO may synergistically increase their negative effects. However, many uncertainties still surround this condition especially with regard to its potential negative effects on health outcomes.

AIM

To conduct a systematic review to determine the prevalence of sarcopenia among adults with overweight and obesity and to investigate whether SO was associated with a higher risk of type 2 diabetes (T2D).

METHODS

This study was conducted in adherence with the Preferred Reporting Items for Systematic Review and Meta-Analyses guidelines. Literature searches, study selection, methodology development and quality appraisal were performed independently by two authors and the data were collated by means of meta-analysis and narrative synthesis.

RESULTS

Of the 606 articles retrieved, 11 studies that comprised a total of 60118 adults with overweight and obesity of both genders met the inclusion criteria and were reviewed, revealing two main findings. First, the overall prevalence of sarcopenia is 43% in females and 42% in males who are with overweight and obesity. Secondly, the presence of SO increases the risk of T2D by 38% with respect to those without SO (OR = 1.38, 95% CI: 1.27-1.50).

CONCLUSION

A high prevalence of sarcopenia has been found among adults with overweight and obesity regardless of their gender and this condition seems to be associated

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with a higher risk of T2D. Clinician should be aware of this scenario in their clinical practice for the better management of both obesity and T2D.

Key words: Obesity; Overweight; Sarcopenia; Type 2 diabetes; Reduced lean body mass

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Core tip: The coexistence of sarcopenia and obesity is referred to a phenotype termed sarcopenic obesity, defined as the increase in body fat deposition, and the reduction in lean mass and muscle strength. Since many uncertainties still surround this condition, especially with regard to its potential negative effects on health outcomes, we conducted this systematic review and found a high prevalence of sarcopenia among adults with obesity. Moreover, this condition seems to be associated with a higher risk of type 2 diabetes (T2D). Clinicians should be aware of this scenario in their clinical practice for better management of obesity and T2D.

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INTRODUCTION

A condition that occurs because of the coexistence of sarcopenia and obesity has been termed sarcopenic obesity (SO)^[1-7]. Many uncertainties still surround this phenomenon with regard to its definition and its potential negative effects on health outcomes, especially those related to obesity, namely the so-called cardio-metabolic diseases^[8,9] such as type 2 diabetes (T2D), cardiovascular diseases, dyslipidaemia and metabolic syndrome^[5,6,10-13]. In fact, it has been hypothesized that the two components of SO may synergistically increase their negative effects on health, however this is still a matter of debate^[14-16].

Several studies have been conducted with a specific focus on determining the association between SO and T2D, however data regarding the contention that individuals with SO are likely to have poorer glycaemic profiles (*i.e.*, hyperglycaemia, high HbA1c, insulin resistance, *etc.*) are still contradictory and require further clarification^[11,17-23]. Moreover, to the best of our knowledge no systematic review posing this issue as a primary outcome has yet been conducted in order to provide an unbiased interpretation of the evidence published to date. In light of these considerations, we set out to systematically review the published literature with the aim of determining the prevalence of sarcopenia among adults with overweight and obesity and to investigate whether SO was associated with higher risk of T2D, in accordance with the PICO process^[24-26] as detailed below: P - population: Individuals in the overweight or obese categories, however they were defined [*i.e.*, body mass index (BMI), body fat percentage, waist circumference, *etc.*]^[27]; I - seeking treatment (*i.e.*, weight-loss or any other treatment if recruited from a clinical setting), otherwise non-treated if subjects were recruited from the general population; C - comparison: Comparison between individuals with sarcopenia and those without SO and with the healthy control group (when available); O - outcome: (i) Prevalence of SO however it was defined in the studies' Methods section (*i.e.*, low muscle mass, low muscle strength, low physical performance, increased visceral adiposity, increased waist circumference *etc.*) and assessed [*i.e.*, bioelectric impedance analysis (BIA), dual-energy X-ray absorptiometry (DXA), handgrip, *etc.*] among the entire obesity groups in the two genders; (ii) The prevalence of T2D however it was defined in the studies' Methods section (*i.e.*, fasting plasma glucose and glycated hemoglobin A1c, oral glucose tolerance test *etc.*) in the SO and non-SO groups.

MATERIALS AND METHODS

The review conformed to the Preferred Reporting Items for Systematic Review and

Meta-Analyses guidelines^[28-30] and was registered in the PROSPERO Registry, No. CRD42018111931^[31].

Inclusion and exclusion criteria

All studies that evaluated SO and T2D in adults were included, provided that they met the following criteria: (i) Studies written in English; (ii) Original research with a cross sectional or longitudinal design; and (iii) Prospective or retrospective observational (analytical or descriptive), experimental or quasi-experimental controlled or non-controlled studies, documenting clearly the prevalence of SO, as well as the association or relationship between SO and T2D. No reviews or non-original articles (*i.e.*, case reports, editorials, "Letters to the Editor" and book chapters) were included.

Information source and search strategy

The literature search was designed and performed independently in duplicate by two authors, namely the principal (DK) and the senior investigator (ME). The PubMed and Science direct databases^[32] were systematically screened using the MeSH terms and a manual search was carried out to retrieve other articles that had not been identified via the initial search strategy. Publication date was not considered an exclusion criterion for the purposes of this review.

Study selection

Two independent authors (DK and ME) screened the articles for their methodology and suitability for inclusion. The quality appraisal was conducted according to the Newcastle-Ottawa Scale (NOS), which relies on a 9-star system whereby scores of 0-3, 4-6 and 7-9 are considered poor, moderate and good quality, respectively^[33]. Consensus discussion was used to resolve disagreements between reviewers.

Data collection process and data items

The title and abstract of each paper were initially assessed by two independent authors (DK and ME) for language suitability and subject matter relevance, the selected studies were then assessed in terms of their suitability for inclusion and the quality of the methodology. The studies that passed both rounds of screening are presented in [Table 1](#).

Data synthesis

The 11 studies that met the inclusion criteria have been presented as a narrative synthesis. In addition, a meta-analysis was conducted on the included studies using Med Calc. software^[34]. The Mantel Haenszel fixed and random effect models were used to estimate the overall effect size and 95%CI. The pooled estimate and 95%CI of the prevalence of SO among males and females in the included studies was estimated similarly.

RESULTS

The initial search retrieved 606 papers. After the first round of screening, 366 papers were excluded for: (i) Languages other than English; (ii) Non-human studies; and (iii) Dealing with obesity without sarcopenia, or the latter without the former. The second round of screening excluded 229 articles due to: (i) Inappropriate paper type, not original research articles (*i.e.*, clinical reviews, Letters to the Editor, chapters in a book and case reports); (ii) Descriptions of SO, but not health-related outcomes; and (iii) An unclear definition of SO or identification of individuals with this condition. Accordingly, following the screening process, 11 articles were included in the systematic review and underwent narrative synthesis and meta-analysis ([Figure 1](#)). The NOS checklist proved that the studies were of a high quality ($n = 11$) (mean score = 7.36 points) ([Table 2](#)).

Narrative synthesis

In 2012, Sénéchal *et al*^[35] conducted a cross-sectional evaluation in which the authors assessed dynapenic obesity, defined as low leg muscle strength combined with abdominal obesity, in 1963 individuals with abdominal obesity. Of these patients, 566 had dynapenic obesity (data per gender is not available). Regardless of gender the mean age and mean BMI in the dynapenic obesity and non-dynapenic obesity groups were 65.4 ± 9.9 years and 29.9 ± 4.6 kg/m² and 65.5 ± 9.6 years and 30.8 ± 4.5 kg/m² respectively. Furthermore, 130 of the 566 individuals with dynapenic obesity had T2D compared to 196 of the 1397 individuals in the non-dynapenic obesity group.

One year later, Lu *et al*^[18] completed a cross sectional study in which they assessed

Table 1 Studies included in the systematic review

| Study | Design | Definition of SO | Body composition | Gender | Sample | Mean age | Mean BMI | Prevalence Sarcopenic Obesity | Prevalence of Diabetes |
|---|--------------------------------|--|--|--------|---------------------------------|--|--|---|--|
| Sénéchal <i>et al</i> ^[35] , 2012 | Cross sectional | Dynapenic obesity, defined as low leg muscle strength, combined with abdominal obesity | Kin- Com dynamometer | M-F | T = 1963 | Non DO: 65.5 ± 9.6; DO: 65.4 ± 9.9 | Non DO: 30.8 ± 4.5; DO: 29.9 ± 4.6 | DO: <i>n</i> = 566/1963 (Did not distinguish in gender) | T2D: Non DO: <i>n</i> = 196; DO: <i>n</i> = 130 |
| Lu <i>et al</i> ^[18] , 2013 | Cross sectional | Defined by combination of total skeletal muscle mass/wt. (100) and BMI ≥ 25 kg/m ² | BIA | M-F | T = 180; M = 60; F = 120 | Non SO: 69.9 ± 7.3; SO: 61.1 ± 9.9 | Non SO: 26.8 ± 1.6; SO: 27.8 ± 2.6 | <i>n</i> = 35/60 in males; <i>n</i> = 80/120 in females | T2D: Non SO: <i>n</i> = 12/65; SO: <i>n</i> = 17/115 |
| Poggiogalle <i>et al</i> ^[36] , 2015 | Cross sectional | Defined by ASMM/h ² or ASMM/wt. < 2SD of sex specific mean combined with assessment of FM and FFM | DXA | M-F | T = 727; M = 141; F = 586 | 46.49 ± 13.73; 46.99 ± 13.76 | 38.85 ± 5.88; 38.84 ± 5.79 | SO: <i>n</i> = 68/141 in males; <i>n</i> = 350/586 in females | Pre-diabetes or T2D: Non SO: <i>n</i> = 69; SO: <i>n</i> = 155 |
| Ma <i>et al</i> ^[37] , 2016 | Retrospective; Cross sectional | SO: BMI > 30 kg/m ² and 24 h- UC < median | Sex-specific 24-h urinary creatinine excretion | M-F | T = 310; M = 144; F = 166 | 71.8 ± 7.6 | 34.1 ± 4.0 | SO: <i>n</i> = 44/144 in males; <i>n</i> = 52/166 in females | T2D: Non SO: <i>n</i> = 51; SO: <i>n</i> = 40 |
| Xiao <i>et al</i> ^[38] , 2017 | Retrospective | FMI/FFMI ratio > 95 percentile of sex, BMI and ethnicity specific population-representative references | BIA | M-F | T = 144; M = 45; F = 99 | Non SO: 56.6 ± 12.7; SO: 54.6 ± 10.1 | Non SO: 44.0 ± 7.6; SO: 49.1 ± 8.3 | SO: 73/144 in total; (Did not distinguish in gender) | T2D: Non SO: <i>n</i> = 36/71; SO: <i>n</i> = 34/71 |
| Kang <i>et al</i> ^[39] , 2017 | Cross sectional | ASM/Wt < 1 SD the mean of the reference group, and BMI ≥ 25 kg/m ² | DXA | F | T = 1555 | Non SO: 61.05 ± 0.44; SO: 62.91 ± 0.44 | Non SO: 26.80 ± 0.07; SO: 27.93 ± 0.11 | SO: <i>n</i> = 855/1555 (All females) | T2D: Non SO: <i>n</i> = 105/700; SO: <i>n</i> = 165/855 |
| Aubertin-Leheudre <i>et al</i> ^[40] , 2017 | Cross sectional | Dynapenic obesity, defined as low handgrip strength (≤ 19.9 in females; ≤ 31.9 in males), combined with BMI ≥ 30 kg/m ² | Jamar Handheld Dynamometer | M-F | T = 670; M = 213; F = 457 | Non SO: 76.3 ± 4.7; SO: 78.0 ± 4.6 | Non SO: 35.6 ± 4.8; SO: 34.9 ± 4.8 | SO: <i>n</i> = 77/213 in males; <i>n</i> = 179/457 in females | T2D: Non SO: <i>n</i> = 133/414; SO: <i>n</i> = 81/256 |
| Park <i>et al</i> ^[41] , 2018 | Cross sectional | SO defined by combination of SMI < 2 SD and WC ≥ 90 cm for men and ≥ 85 cm women | BIA | M-F | T = 53818; M = 38820; F = 14998 | Non SO: 40.5 ± 9.2; SO: 40.0 ± 11.3 | Non SO: 26.9 ± 2.2; SO: 30.7 ± 3.4 | <i>n</i> = 6513; M = 3341; F = 3172 | T2D; Non-SO: <i>n</i> = 2176; SO: <i>n</i> = 391 |
| Kreidieh <i>et al</i> ^[42] , 2018 | Cross sectional | ALM/BMI < 0.512 | BIA | F | T = 154 | 33.26 ± 14.65 | 31.42 ± 4.94 | <i>n</i> = 31 | T2D: Non SO: <i>n</i> = 3/123; SO: <i>n</i> = 4/31 |

| | | | | | | | | | |
|--|--|---|----------------------------------|---|---------|------------------------------------|------------------------------------|-------------|--|
| Khazem <i>et al</i> ^[43] , 2018 | Cross sectional | ALM/BMI < 0.789, (ALM/Wt.) × 100% < 25.72, and (ALM/Wt.) × 100% < 29.60 | BIA | M | T = 72 | 32.79 ± 13.65 | 33.69 ± 5.85 | 23.9%-69.4% | T2D: Non SO: n = 1/22; SO: n = 3/50 |
| Scott <i>et al</i> ^[46] , 2018 | Cross sectional (includes a longitudinal part) | ALM/height < 7.26 kg/m ² combined with handgrip strength < 30 kg and/or low gait speed ≤ 0.8 m/s. Obesity was defined as body fat percentage ≥ 30% | DXA Handgrip strength Gait speed | M | T = 525 | Non SO: 75.9 ± 4.7; SO: 80.3 ± 6.5 | Non SO: 30.7 ± 3.4; SO: 27.2 ± 2.3 | n = 80 | High fasting glucose or diabetes medications: Non SO: n = 177/445; SO: n = 29/80 |

SO: Sarcopenic obesity; DO: Dynapenic obesity; BMI: Body mass index; M: Male; F: Female; BIA: Bioelectric impedance analysis; T2D: Type 2 diabetes; DXA: Dual-energy X-ray absorptiometry.

SO defined as the coexistence of obesity (BMI ≥ 25 kg/m²) and sarcopenia based on the skeletal muscle index estimated by BIA. A sample of 180 individuals with obesity (60 males and 120 females) was recruited. Of the 60 males included in the sample 35 had SO compared to 80 of the 120 females. Regardless of gender the mean age and BMI in the SO group were 61.1 ± 9.9 years and 27.8 ± 2.6 kg/m², and 69.9 ± 7.3 years and 26.8 ± 1.6 kg/m² in the non-SO group. Moreover, 12 of the 65 patients in the SO group had T2D compared to 17 of the 115 patients in the non-SO group.

In early 2016, Poggiogalle *et al*^[36] conducted a cross sectional study in which the authors assessed SO using DXA, with SO defined as the coexistence of obesity (BMI ≥ 30 kg/m²) and sarcopenia (ASMM: height² < 6.54 and < 4.82 kg/m² for males and females respectively) or (ASMM: weight < 0.2827 and < 0.2347 for males and females respectively). This study enrolled a sample of 727 individuals with obesity (141 males and 586 females), with mean ages of 45.63 ± 13.53 and 45.76 ± 13.58 years, and mean BMIs of 37.56 ± 5.99 and 37.80 ± 5.77 kg/m² respectively for each gender. Of the 141 male patients 68 had SO, while 350 of the 586 females had the condition. In addition, 155 of the 418 patients had pre-diabetes or T2D in the SO group compared to 70 of the 309 patients in the non-SO group.

In the same year, Ma *et al*^[37] performed a cross-sectional evaluation on SO defined by BMI and sex-specific 24-h urinary creatinine excretion, in 310 patients (166 females and 144 males) with obesity (BMI ≥ 30 kg/m²). Fifty-four of the 144 males and 52 of the 166 females had SO. The mean BMI and age of the SO group were 34.1 ± 4.0 kg/m² and 71.8 ± 7.6 years, while they were 34.9 ± 4.4 kg/m² and 67.8 ± 6.8 years in the non-SO group, respectively. Furthermore, 40 of the 106 patients had T2D in the SO group in comparison to 51 of the 204 patients in the non-SO group.

In 2017, Xiao *et al*^[38] performed a retrospective study on the prevalence of SO and its association with health outcomes in patients seeking weight loss treatment in a bariatric surgery setting. Body composition analysis was conducted by means of BIA and SO was defined by a fat mass:fat-free mass index (FMI: FFMI) ratio greater than the 95th percentile of sex, BMI and ethnicity-specific population-representative references. A sample of 144 adults with obesity (99 females and 45 males) were enrolled, with a mean age of 55.6 ± 11.5 years and a mean BMI of 46.6 ± 8.4 kg/m². Of the 144 patients included in the sample 73 had SO (data per gender is not available). The mean age and BMI of the individuals with obesity only were 56.6 ± 12.7 years and 44.0 ± 7.6 kg/m², compared to 54.6 ± 10.1 years and 49.1 ± 8.3 kg/m² in those with SO. Furthermore, 34 of the 73 patients had T2D in the SO group in comparison to 36 of the 71 patients in the non-SO group.

In 2017, Kang *et al*^[39] conducted a large cross-sectional study to assess the association between SO and metabolic syndrome in postmenopausal women. SO was defined by the co-existence of sarcopenia (ASM/weight < 1 standard deviation below the mean of the reference group) and a BMI cut-off point for obesity which referred to a score of 25 kg/m² on the basis of the Asia-Pacific obesity criterion. The study included 1555 females with obesity, of whom 855 had SO, with a mean age of 62.91 ± 0.44 years and a mean BMI of 27.93 ± 0.11 kg/m². On the other hand, 700 did not have SO and had a mean age of 61.05 ± 0.44 years and a mean BMI of 26.80 ± 0.07 kg/m². In

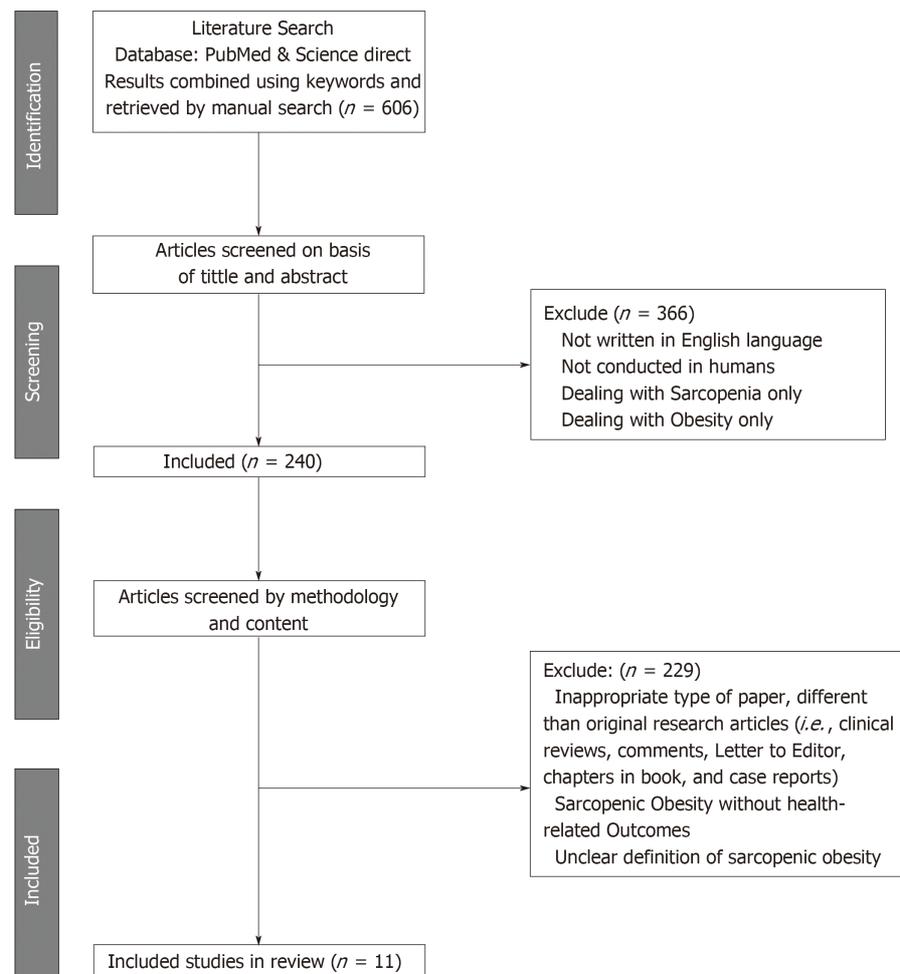


Figure 1 The flowchart summarizing the study selection procedure.

addition, 165 of the 855 patients had T2D in the SO group while 105 of the 700 patients in the non-SO group had T2D.

In the same year, a cross-sectional study by Aubertin-Leheudre *et al*^[40] aimed to examine the association between dynapenic obesity and metabolic risk factors in older adults (age ≥ 70 years). Dynapenic obesity was defined as low handgrip strength (u 19.9 in females; ≤ 31.9 in males) combined with a BMI of ≥ 30 kg/m². The study included 670 participants with obesity (213 males and 457 females), of whom 256 had dynapenic obesity, with a mean age of 78.0 ± 4.6 years and a mean BMI of 34.9 ± 4.8 kg/m², and 414 did not have dynapenic obesity, with a mean age of 76.3 ± 4.7 years and a mean BMI of 35.6 ± 4.8 kg/m². Furthermore, 81 of the 256 individuals in the dynapenic obesity group had T2D while 133 of 414 individuals in the non-dynapenic obesity group had T2D.

In 2018, Park *et al*^[41] conducted a large cross sectional study in two sites, which included a total of 53818 adults with overweight and obesity of both genders (38820 males and 14998 females), of whom 6513 had SO defined as below two standard deviations of the mean of the skeletal muscle mass index for young adults assessed by BIA and a waist circumference of ≥ 90 cm for men and ≥ 85 cm for women. The mean age and BMI of the individuals with obesity only were 40.5 ± 9.2 years and 26.9 ± 2.2 kg/m² compared to those with SO who had a mean age of 40.0 ± 11.3 years and a mean BMI of 30.7 ± 3.4 kg/m². Moreover, 391 of the 6513 patients had T2D in the SO group compared to 2176 of the 47305 patients in the non-SO group.

In 2018, Kreidieh *et al*^[42] conducted a cross sectional controlled study in which body composition measurements were conducted by BIA using a definition that in addition to appendicular lean mass (ALM) also involved BMI, and patients were considered affected by SO if ALM: BMI < 0.512. The study included 154 females with overweight and obesity with a mean age of 33.26 ± 14.65 years and a mean BMI of 31.42 ± 4.94 kg/m². Of the 154 female patients 31 had SO. Moreover, four of the 31 patients had T2D in the SO group compared to three of the 123 patients in the non-SO group.

In 2018, Khazem *et al*^[43] performed a cross-sectional controlled study on 72 adult

Table 2 Quality assessment of the included studies

| Author | Sénéchal <i>et al</i> ^[35] , 2012 | Lu <i>et al</i> ^[18] , 2013 | Poggiogale <i>et al</i> ^[36] , 2016 | Ma <i>et al</i> ^[37] , 2016 | Xiao <i>et al</i> ^[38] , 2017 | Kang <i>et al</i> ^[39] , 2017 | Aubertin-Leheudre <i>et al</i> ^[40] , 2017 | Park <i>et al</i> ^[41] , 2018 | Scott <i>et al</i> ^[46] , 2018 | Kreidieh <i>et al</i> ^[42] , 2018 | Khazem <i>et al</i> ^[43] , 2018 |
|---|--|--|--|--|--|--|---|--|---|--|--|
| Selection | | | | | | | | | | | |
| Represents cases with independent validation | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Cases are consecutive or obviously representative | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Controls from the community | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Controls have no history of sarcopenic obesity | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Comparability | | | | | | | | | | | |
| Controls are comparable for the most important factors | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Control for any additional factor | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| Ascertainment of exposure | | | | | | | | | | | |
| Secured record or structured interview where blind to /control status | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 |
| Same method of ascertainment for cases and controls | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Cases and controls have completed follow up | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Total score | 7 | 7 | 8 | 8 | 6 | 8 | 7 | 8 | 8 | 7 | 7 |

Newcastle-Ottawa Scale (NOS) for longitudinal and cross-sectional studies. Yes = 1, No (not reported, not available) = 0; Studies with scores of 0-3, 4-6, 7-9 were considered as low, moderate and high quality, respectively.

males with overweight and obesity with a mean age of 32.79 ± 13.65 years and a mean BMI of 33.69 ± 5.84 kg/m². In this study the authors used three different definitions proposed by Batsis *et al*^[44], Levine and Crimmins^[21], and Oh *et al*^[45] based on ALM: BMI

and $(\text{ALM: weight}) \times 100\%$ to define SO. Body composition was assessed by BIA. Based on each formula the prevalence of SO varied between 23.9% and 69.4%. However, based on the definition that was revealed to be more useful from the clinical perspective, 50 of the 72 patients had a reduced lean body mass with a prevalence of 69.4%. Moreover, three of the 50 patients had T2D in the SO group in comparison to one of the 22 patients in the non-SO group.

Finally, in 2018 Scott *et al.*^[46] conducted a large sampled study that aimed to investigate the cross-sectional association between SO and components of metabolic syndrome in community-dwelling older men. SO was defined by the co-existence of sarcopenia as $\text{ALM/height} < 7.26 \text{ kg/m}^2$ combined with handgrip strength $< 30 \text{ kg}$ and/or low gait speed $\leq 0.8 \text{ m/s}$, while obesity was defined as a body fat percentage of $> 30\%$. The study included 525 males with obesity, of whom 80 had SO, with a mean age of 80.3 ± 6.5 years and mean BMI of $27.2 \pm 2.3 \text{ kg/m}^2$ and 445 did not have SO, with a mean age of 75.9 ± 4.7 years and mean BMI of $30.7 \pm 3.4 \text{ kg/m}^2$. Furthermore, 29 of the 80 individuals in the SO group had T2D in comparison to 177 of the 445 individuals in the non-SO group.

Meta-analysis

The meta-analysis estimated the overall prevalence of SO among males and females. With high heterogeneity among the included studies, a random effect model was considered for the estimation of the overall prevalence of SO. The forest plots in Figures 2 and 3 show that SO affected 43% (95%CI: 28-59) of females and 42% (95%CI: 31-53) of males. In addition, the overall odds ratios of T2D in patients with SO as compared to those without SO are presented in Figure 4. The fixed effect weighted pooled odds for T2D in patients with SO indicated an increased risk of T2D of approximately 38% compared to those without SO (OR: 1.38, 95%CI: 1.27-1.50). The heterogeneity analysis revealed moderate variability ($I^2 = 60\%$).

DISCUSSION

This systematic review aimed to provide benchmark data on the prevalence of sarcopenia in individuals with overweight and obesity and to assess any potential association between SO and T2D in this population. The major finding is that sarcopenia seems to affect approximately 40%-45% of individuals with overweight and obesity of both genders, and the co-existence of both conditions, namely sarcopenia and excess weight/obesity increases the risk of T2D by nearly 38% when compared with those who had excess weight or obesity alone. The underlying mechanism behind this association is still unclear, however it seems that there is a bi-directional interaction between obesity, chronic inflammation, insulin resistance and sarcopenia^[19]. In fact, the chronic inflammation plays an important role in the pathogenesis of T2D. For this reason we speculate that coexistence of both obesity and sarcopenia under the so-called phenotype "SO", may have a synergistic effect with chronic inflammation being a common "denominator" seen in both conditions, which seems to exacerbate further glucose metabolism impairment (*i.e.*, insulin resistance, pre-diabetes and T2D)^[19].

The clinical implication of this review's findings is the awareness of the high prevalence of sarcopenia in the overweight/obese population that should be raised among clinicians and patients. Secondly, these results reveal the importance of screening for SO in individuals affected by excess weight and obesity, since this condition also seems to be strongly associated with T2D.

This systematic review has certain strengths. To the best of our knowledge this is the first systematic review to assess the overall prevalence of SO in males and females with overweight and obesity. In fact, the studies that have been conducted on this topic reported varying levels of prevalence that ranged between 0 and 100%, depending on the applied definition of SO^[47,48]. Higher prevalence tends to be reported in studies that accounted for body mass (*i.e.*, BMI), whereas a lower prevalence is reported in those that did not^[43,49]. A low prevalence may also be explained by the use of definitions that have primarily been developed from studies in older cohorts and these may not be applicable to younger adults^[47].

However, this systematic review also has certain limitations. Foremost, our results need to be interpreted with caution with regard to the association between SO and the prevalence of T2D, since the cross-sectional design of the studies (*i.e.*, non cohort), included in our systematic review indicates only simple associations between SO and T2D at best and does not provide solid information regarding any causal relationships between the two conditions^[50,51]. In other words, these studies lack evidence to determine if SO may lead to the onset or deterioration of T2D, since very few studies

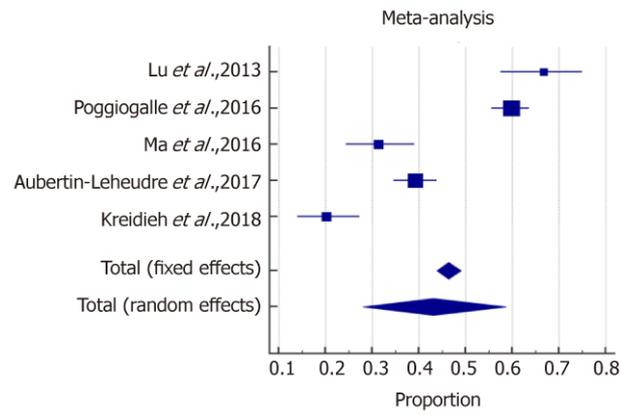


Figure 2 Forest plot for the pooled estimate of proportion of females with sarcopenic obesity.

have longitudinally investigated the “real” effects of SO on health^[52]. These shortcomings in the current research indicate the need to design longitudinal studies to clarify the real effect of SO on the onset and progression of T2D.

In conclusion, a high prevalence of sarcopenia has been found among adults with overweight and obesity regardless of their gender, and this condition seems to be associated with a higher risk of T2D. Clinicians should be aware of this scenario in their clinical practice for better management of both obesity and T2D.

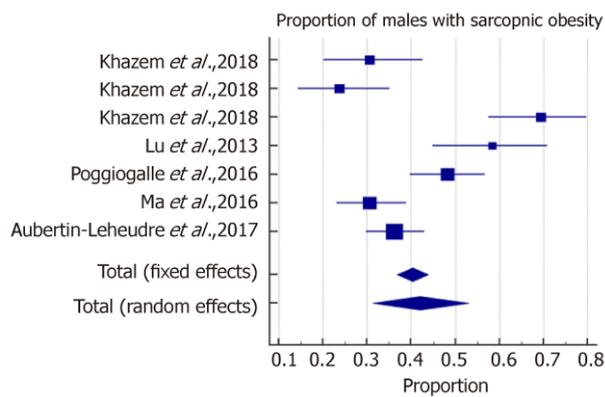


Figure 3 Forest plot for the pooled estimate of proportion of males with sarcopenic obesity.

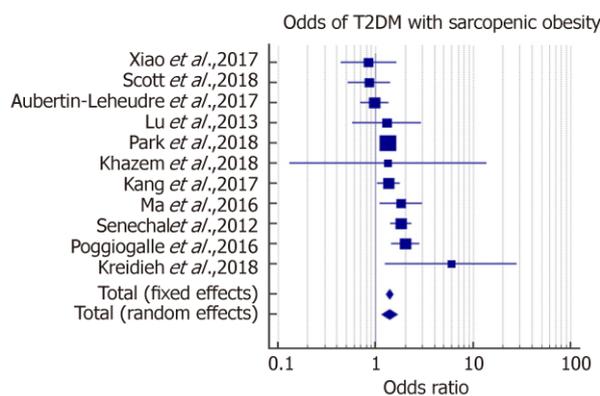


Figure 4 Forest plot for the pooled estimate of the odds of type 2 diabetes with sarcopenic obesity.

ARTICLE HIGHLIGHTS

Research background

The coexistence of sarcopenia and obesity has been termed as sarcopenic obesity (SO). Several studies have been conducted in order to determine any potential association between SO phenotype and type 2 diabetes (T2D). However, the available data are still contradictory and require further clarification.

Research motivation

To our knowledge no systematic review on the primary outcome related to the association between SO and T2D has been conducted yet to provide an unbiased interpretation of the evidence published to date.

Research objectives

We set out to systematically review the published literature with the aim of determining the prevalence of sarcopenia among adults with overweight and obesity and to investigate whether SO was associated with higher risk of T2D.

Research methods

The review conformed to the Preferred Reporting Items for Systematic Review and Meta-Analyses guidelines, and data were collated by means of narrative synthesis and meta-analysis.

Research results

The prevalence of SO in adult with overweight and obesity is 43% in females and 42% in males, and the presence of this condition increases the risk of T2D by 38% with respect to those without SO.

Research conclusions

A high prevalence of sarcopenia has been found among adults with overweight and obesity regardless of their gender, and this condition seems to be associated with a higher risk of T2D. The clinical implication of our findings is to raise awareness of the high prevalence of this phenotype in the overweight/obese population, and the importance of screening for SO in

individuals affected by excess weight, since this condition seems to be strongly associated with T2D. However, our results need to be interpreted with caution with regard to the association between SO and the prevalence of T2D, since the cross-sectional design of the studies included in our systematic review indicates only associations between the two conditions and that does not provide information regard the causal relationships.

Research perspectives

The current research indicates the need to design longitudinal studies to clarify the real effect of SO on the onset and progression of T2D. In other words, the available studies lack in evidence to determine if SO may lead to the onset or deterioration of T2D, since very few studies have longitudinally investigated the “real” effects of SO on health.

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