Prevalence of subclinical hypothyroidism in adults with obesity and its cardiometabolic correlates: a systematic review and meta-analysis

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Abstract

Objective. To estimate the prevalence of subclinical hypothyroidism (SCH) in adults with obesity and to summarise the cardiometabolic differences between adults with overweight or obesity with and without SCH, as a basis for screening and management in military personnel with obesity.

Materials and methods. PubMed was searched on 4 October 2026 without date or language limits. Studies of adults with obesity reporting the number with SCH were pooled after logit transformation with a DerSimonian–Laird random-effects model; studies comparing cardiometabolic factors between adults with overweight or obesity with and without SCH were synthesised narratively. Study quality was appraised with the JBI checklist for prevalence studies.

Results. Of 323 records, 25 studies were included: 19 with 5,847 adults with obesity in the meta-analysis and eight in the narrative synthesis (two in both). SCH was found in 703 participants; the pooled prevalence was 12.6% (95% CI 10.2–15.4), with a 95% prediction interval of 5.0–28.3% and I2 = 86.8%. The prevalence did not differ between bariatric surgery candidates (13.6%), other clinical samples (12.9%) and community or volunteer samples (10.8%; p = 0.76), and it was not associated with mean body mass index (p = 0.24). Five of eight comparative studies reported a less favourable lipid, glucose or metabolic syndrome profile in SCH, and after bariatric surgery SCH resolved in most patients.

Conclusion. About one in eight adults with obesity has SCH, more than in general-population surveys, and the TSH elevation often resolves after surgically induced weight loss. In servicemen with obesity, TSH testing could be considered at periodic examinations, a raised value should be confirmed by repeat testing, and levothyroxine should be considered only for younger patients with a persistent TSH above 10 mU/L or, as a trial, for younger symptomatic patients.

Keywords: *subclinical hypothyroidism; obesity; thyroid-stimulating hormone; prevalence; meta-analysis; military personnel*

Introduction

Subclinical hypothyroidism (SCH) is defined as an elevated serum thyroid-stimulating hormone (TSH) concentration with free thyroxine (FT4) within the reference range [1]. In the general population, SCH was found in 4.3% of the United States population [2] and undiagnosed hypothyroidism in 4.94% of Europeans [3]. SCH has been associated with a higher risk of cardiovascular disease and all-cause mortality, particularly in people with a high cardiovascular risk [4], and with metabolic syndrome (MetS) in meta-analyses of observational studies [5, 6].

Obesity and thyroid function are closely linked. Serum TSH rises with body mass index (BMI) within the reference range [7, 8], obesity activates the hypothalamic–pituitary–thyroid axis [9], and in a meta-analysis obesity was associated with a higher risk of SCH (relative risk 1.70) and of overt hypothyroidism (relative risk 3.21) [10]. Whether a raised TSH in obesity reflects thyroid failure or an adaptive change is debated, because thyroid autoimmunity is less frequent in obese patients with SCH than in normal-weight patients with SCH [11] and TSH falls after weight loss [12].

The question is practical for military medicine. Obesity among United States active duty personnel increased from 5.0% in 1995 to 12.7% in 2008 [13], primary hypothyroidism is the most common incident thyroid disorder in United States service members [14], and current hypothyroidism is disqualifying for enlistment in the United States armed forces unless the person is asymptomatic and euthyroid on TSH testing [15]. Reliable estimates of SCH in obesity are therefore needed to plan screening and to avoid unnecessary treatment. The aim of this study was to estimate the prevalence of SCH in adults with obesity and to summarise its cardiometabolic correlates.

Materials and methods

Protocol, search and eligibility

The review followed the PRISMA 2020 statement [16], the MOOSE recommendations [17] and the JBI guidance for systematic reviews of prevalence data [18]; the protocol, which defined the search, the eligibility criteria and the data items, was written before screening but was not registered. PubMed (MEDLINE) was searched on 4 October 2026 from inception, without language restrictions, with the query ("subclinical hypothyroidism" OR "subclinical hypothyroid" OR "mild thyroid failure") AND (obesity OR obese OR overweight OR bariatric), all terms in title or abstract. Two questions were prespecified. For the prevalence question, studies of adults with obesity (BMI ≥30 kg/m2, ≥27.5–28 or ≥25 kg/m2 by Asian criteria, or candidates for bariatric surgery) reporting the number of participants with SCH, or a percentage from which it could be calculated, and the denominator were eligible. For the comparative question, studies of adults with overweight or obesity comparing cardiometabolic factors between participants with SCH and euthyroid participants were eligible. Children, pregnant women, populations selected for another disease and studies in which SCH could not be separated from overt or treated hypothyroidism were excluded; when several reports described the same cohort, the most complete one was used.

Selection, data extraction and quality

Titles and abstracts were screened by two reviewers, each assessing half of the records; full texts were assessed when available and abstracts otherwise, and uncertain records were decided by a senior reviewer. Data were extracted with a standard form, and a second reviewer checked every value used in the analyses against the source and supplied missing descriptive data. The extracted items were the country, setting, sample size, sex, mean age and BMI, the definition of SCH and the TSH threshold, the exclusion of known thyroid disease and the number of participants with SCH and, for the comparative studies, the size of the groups and all reported lipid, glucose, insulin resistance, blood pressure and MetS outcomes. SCH was accepted as defined by the authors. When only a percentage was reported, the number of participants with SCH was taken as the nearest integer to the percentage multiplied by the denominator and was accepted if it reproduced the published percentage within 0.1 percentage point.

Quality was appraised by one reviewer with the nine-item JBI checklist for prevalence studies [18, 19], using the following decision rules. The sample frame was rated appropriate for clinical or population samples of adults with obesity and inappropriate for volunteers, for the obese group of a study designed for another condition and for samples restricted by antibody status, smoking or completeness of follow-up. Sampling was rated appropriate when consecutive, complete or random enrolment was stated, and the sample size was rated adequate with at least 384 participants, which allows any prevalence to be estimated with a precision of ±5 percentage points. SCH was rated validly identified when it was explicitly defined as a raised TSH with normal FT4, and reliably measured when the assay was described and FT4 was measured in all participants or in all those with a raised TSH. Items not reported were rated unclear.

Statistical analysis

Proportions were logit-transformed and pooled with the DerSimonian–Laird random-effects model [20], with Wilson score 95% confidence intervals (CI) for individual studies [21]. Heterogeneity was quantified with Cochran's Q and I2 [22, 23], and a 95% prediction interval was calculated [24, 25]. Subgroup analyses compared the setting (bariatric surgery candidates, other clinical samples, community or volunteer samples), WHO region, TSH threshold, exclusion of known thyroid disease, publication period, sex and data source, with a random-effects test of subgroup differences; random-effects meta-regression [26] was used for mean BMI and publication year. Small-study effects were assessed with a funnel plot and Egger's test [27]. Sensitivity analyses used the restricted maximum likelihood estimator [28] and the Freeman–Tukey double arcsine transformation, interpreted with caution because of its back-transformation [29]; the analysis was also restricted to studies with reported counts, full-text studies, studies that excluded known thyroid disease, studies with at least 100 participants and samples other than bariatric surgery candidates, and each study was omitted in turn. Because the comparative studies used different outcomes, groups and statistics, they were synthesised narratively. The analyses were performed in Python 3 (NumPy, SciPy) with code checked against the statsmodels implementation. The protocol was changed after screening in four respects: counts derived from published percentages were accepted, the Asian obesity threshold of ≥25 kg/m2 was accepted in addition to ≥27.5–28 kg/m2, comparative studies without numerical results were included in the narrative synthesis, and the JBI checklist for prevalence studies replaced the checklist for analytical cross-sectional studies; the subgroup, meta-regression and sensitivity analyses were not specified in the protocol.

Results

Study selection and characteristics

The search returned 323 records; 261 were excluded on the title and abstract and 62 reports were assessed for eligibility. Of these, 37 were excluded, most often because the population was not restricted to adults with obesity (n = 13) or the data could not be extracted (n = 12); two smaller reports from a centre whose largest report defined SCH as TSH above 2.5 mU/L were also excluded, because they did not state their definition and probably overlapped with it. Twenty-five studies were included: 19 in the meta-analysis of prevalence and eight in the narrative synthesis, two of them in both (Figure 1).

The 19 studies in the meta-analysis were published between 2005 and 2025 and included 5,847 adults with obesity (range 38–1,486) from China (five studies), Brazil, Egypt and Pakistan (two each) and one study each from Syria, Iran, Tunisia, the United States, the Netherlands, Spain, Italy and Greece (Table 1). Eight studies examined candidates for bariatric surgery, seven other

clinical samples and four community or volunteer samples. The TSH threshold for SCH was stated or derivable from the reference range in six studies, known thyroid disease was excluded in eight, and 11 studies were available only as abstracts.

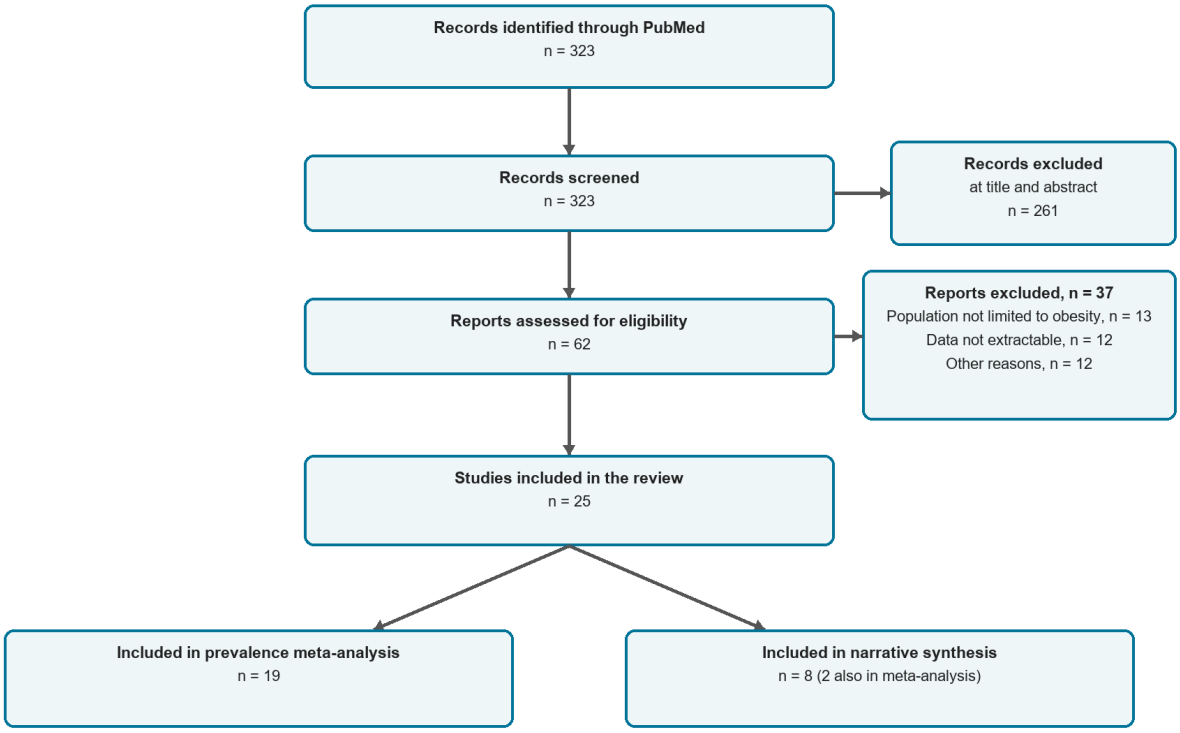


Figure 1. Flow diagram of study selection (PRISMA 2020)

Prevalence of subclinical hypothyroidism

SCH was found in 703 of 5,847 adults with obesity. The prevalence ranged from 4.0% [30] to 25.0% [31]; the pooled prevalence was 12.6% (95% CI 10.2–15.4), with substantial heterogeneity ($I^2 = 86.8\%$, $Q = 136.0$, $p < 0.001$) and a 95% prediction interval of 5.0–28.3% (Figure 2).

Sources of heterogeneity

The prevalence did not differ significantly by setting (13.6% in bariatric surgery candidates, 12.9% in other clinical samples and 10.8% in community or volunteer samples; $p = 0.76$), by region (from 10.5% in the Eastern Mediterranean to 14.9% in China; $p = 0.61$) or by TSH threshold ($p = 0.94$) (Figure 3). It was 11.9% in studies that excluded known thyroid disease, 9.0% in those that did not and 15.3% in those that did not report it ($p = 0.10$). The prevalence was also similar in studies published in 2005–2015 and in 2018–2025 (14.0% and 11.9%; $p = 0.40$), in studies of women only and of both sexes (10.6% and 12.4%; $p = 0.84$) and in full-text and abstract-only studies (11.4% and 13.5%; $p = 0.43$). In meta-regression, mean BMI, reported in 9 studies, was not associated with the prevalence (slope 0.018 per kg/m2 on the logit scale, $p = 0.24$; Figure 4), nor was the publication year ($p = 0.27$).

Table 1. Characteristics of the studies included in the meta-analysis of prevalence

Study	Country	Setting	N	Women, %	Mean BMI, kg/m2	TSH threshold, mIU/L	Thyroid disease excluded	SCH, n	SCH, %
Moulin de Moraes 2005 [31]	Brazil	Bariatric	72	86.1	53.0	—	Yes	18	25.0
Michalaki 2006 [32]	Greece	Clinic	144	76.4	—	—	No	11a	7.6
Chikunguwo 2007 [33]	USA	Bariatric	86	—	49.0	—	Yes	9a	10.5
Rotondi 2009 [11]	Italy	Clinic	350	—	—	—	—	48a	13.7
Ruiz-Tovar 2014 [34]	Spain	Bariatric	60	—	—	—	—	10a	16.7
Janssen 2015 [35]	Netherlands	Bariatric	503	—	—	—	—	71	14.1
Sami 2018 [36]	Pakistan	Clinic	127	53.5	32.1	—	Yes	19	15.0
Wang 2018 [37]	China	Community	397	36.5	30.7	>4.2	No	53	13.4
Zhang 2018 [38]	China	Clinic	534	100	—	—	—	108	20.2
Abdelbaki 2020 [39]	Egypt	Bariatric	554	—	45.0	—	—	71a	12.8
Granzotto 2020 [40]	Brazil	Bariatric	215	75.8	40.2	—	Yes	20	9.3
Najjari 2021 [41]	Iran	Bariatric	1,486	—	—	—	No	102	6.9
Salman 2021 [42]	Egypt	Bariatric	128	—	—	>4.5	—	30	23.4
Yao 2021 [43]	China	Community	280	73.9	—	>4.2	Yes	56	20.0
Yan 2022 [44]	China	Community	289	53.3	30.4	>4.20	Yes	27	9.3
Alourfi 2023 [45]	Syria	Community	292	—	—	>4.6	Yes	13	4.5
Malik 2023 [46]	Pakistan	Clinic	242	86.0	41.6	—	—	31	12.8
Jemai 2025 [30]	Tunisia	Clinic	50	100	41.2	—	—	2	4.0
Xu 2025 [47]	China	Clinicb	38	—	—	>4.20	Yes	4	10.5

Note. a Calculated from the published percentage. b Setting not stated; participants described as patients. Bariatric, bariatric surgery candidates; Clinic, other clinical samples; Community, community or volunteer samples; BMI, body mass index; SCH, subclinical hypothyroidism; TSH, thyroid-stimulating hormone; —, not reported.

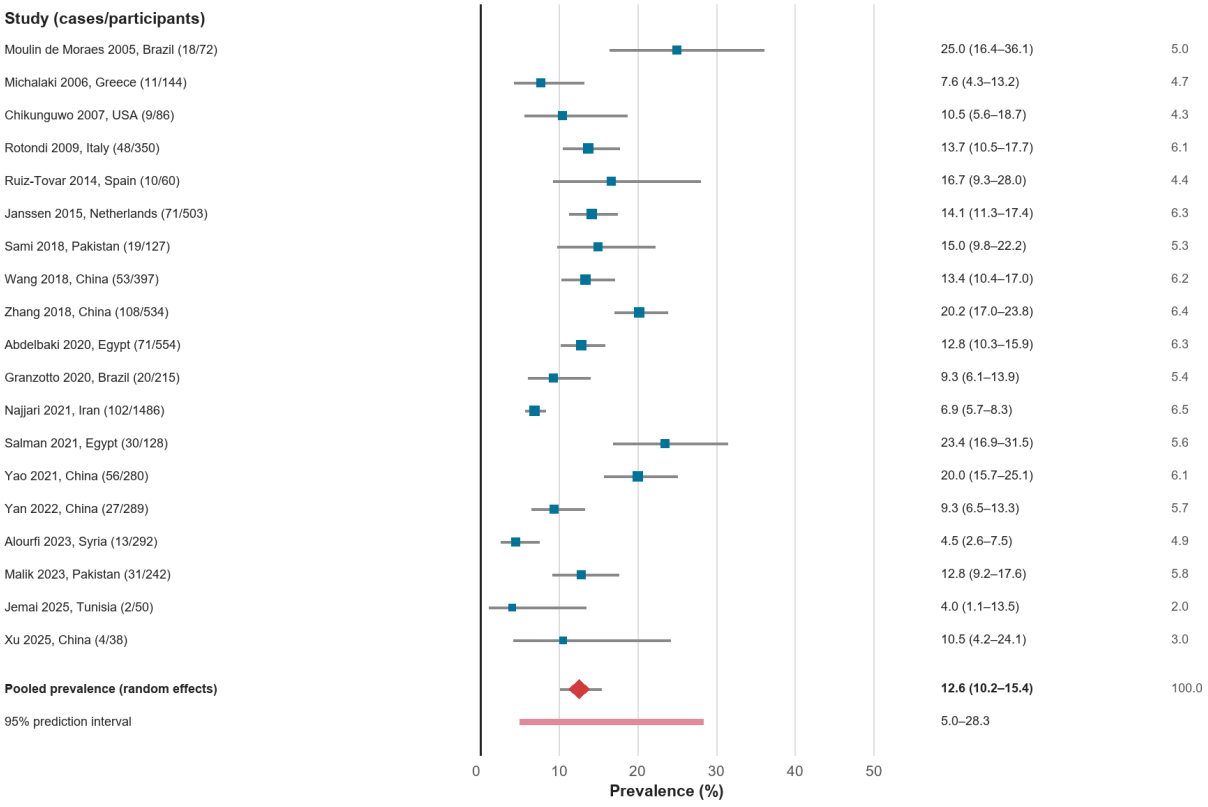


Figure 2. Forest plot of the prevalence of subclinical hypothyroidism in adults with obesity

Dark marks, study estimates (mark width proportional to the weight); light bars, 95% CI (Wilson); red, pooled random-effects estimate (DerSimonian–Laird, logit transformation) with the dashed reference line; pink, 95% prediction interval. SCH, subclinical hypothyroidism.

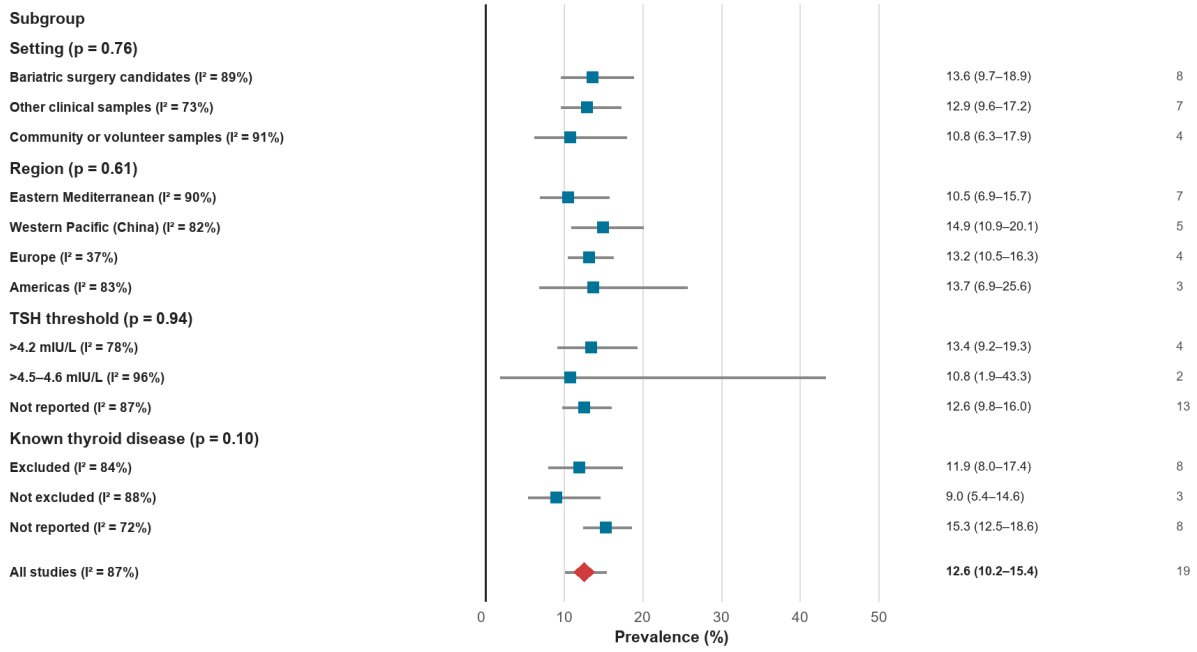


Figure 3. Pooled prevalence of subclinical hypothyroidism in subgroups

Orange, subgroup random-effects estimates with 95% CI; red, all studies. p, random-effects test of subgroup differences. SCH, subclinical hypothyroidism; TSH, thyroid-stimulating hormone.

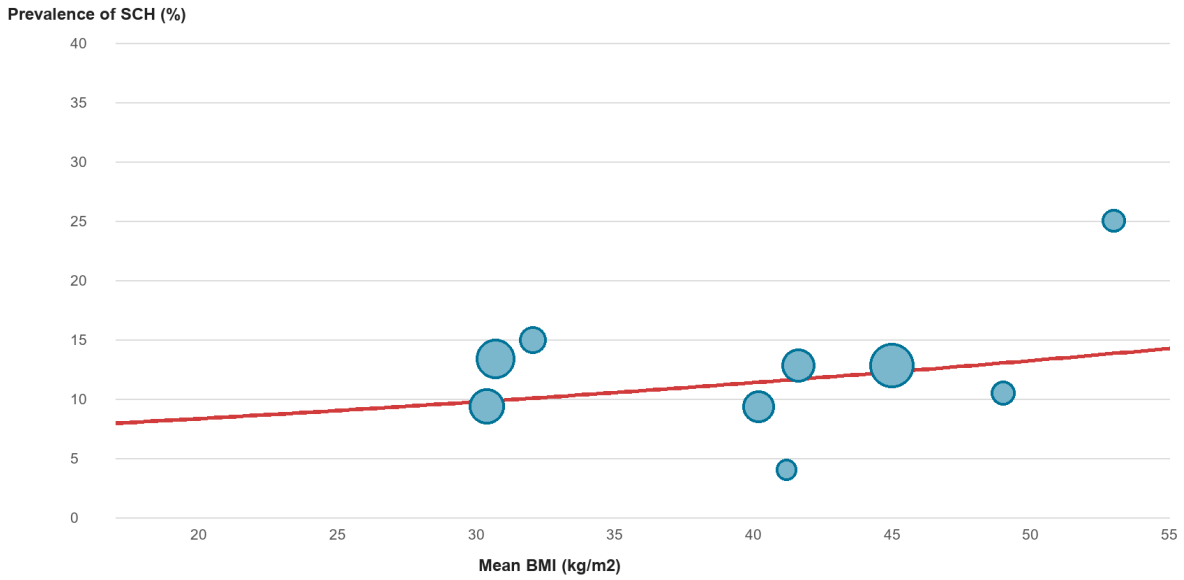


Figure 4. Meta-regression of the prevalence of subclinical hypothyroidism on mean body mass index

Bubble area is proportional to the sample size. Red line, random-effects meta-regression (logit scale, back-transformed). SCH, subclinical hypothyroidism.

Sensitivity analyses, small-study effects and study quality

The pooled estimate was stable with the restricted maximum likelihood estimator (12.6%) and the double arcsine transformation (12.6%), and when the analysis was restricted to studies with reported counts (12.7%), full-text studies (11.4%), studies that excluded known thyroid disease (11.9%), studies with at least 100 participants (12.3%) or samples other than bariatric surgery candidates (11.9%); omitting each study in turn gave estimates from 12.1% (without Salman 2021) to 13.3% (without Najjari 2021) (Table 2). The funnel plot showed no marked asymmetry, and Egger's test showed no evidence of small-study effects (intercept −0.59, p = 0.71; Figure 5).

Table 2. Sensitivity analyses of the pooled prevalence of subclinical hypothyroidism

Analysis	Studies	Participants	Prevalence, %	95% CI	I2, %
Main analysis (DerSimonian–Laird, logit)	19	5,847	12.6	10.2–15.4	87
Restricted maximum likelihood estimator	19	5,847	12.6	10.2–15.4	87
Freeman–Tukey double arcsine transformation	19	5,847	12.6	10.0–15.3	88
Studies with reported counts only	14	4,653	12.7	9.6–16.5	90
Full-text studies only	8	1,880	11.4	8.4–15.3	80
Studies that excluded known thyroid disease	8	1,399	11.9	8.0–17.4	84
Studies with at least 100 participants	14	5,541	12.3	9.8–15.5	89
Without bariatric surgery candidates	11	2,743	11.9	9.2–15.4	83
Leave-one-out analysis (range)	18	—	12.1–13.3	—	79–88

Note. CI, confidence interval; I2, proportion of total variation due to between-study heterogeneity.

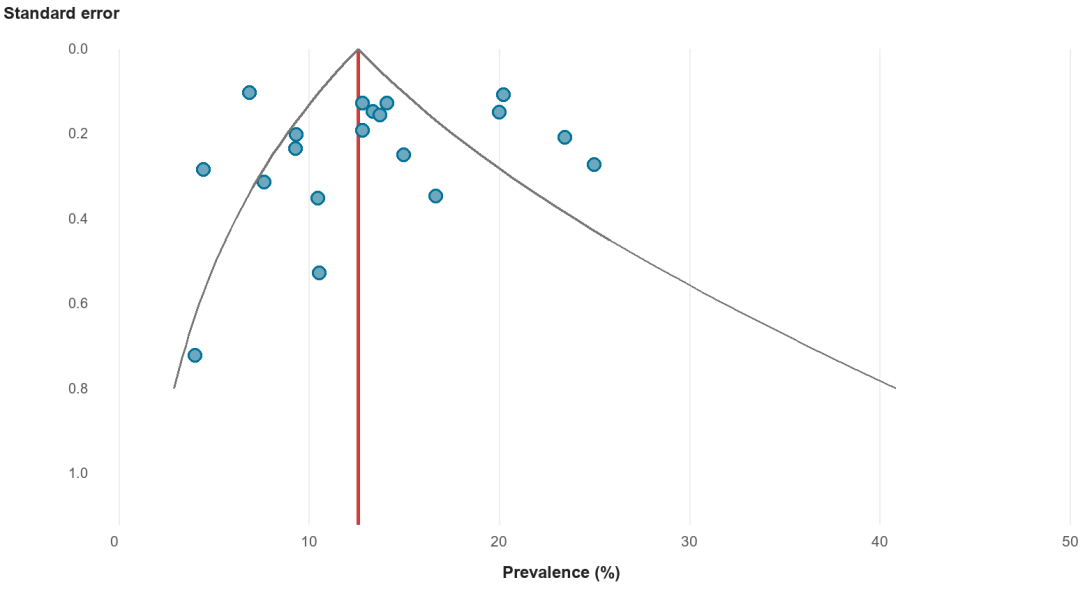


Figure 5. Funnel plot of the prevalence of subclinical hypothyroidism in the included studies

Each circle is a study; dashed lines, pseudo 95% confidence limits around the pooled estimate (logit scale, back-transformed); red line, pooled prevalence. Egger's test: intercept −0.59, p = 0.71. SCH, subclinical hypothyroidism.

On the JBI checklist, the sample frame was appropriate in 14 studies, sampling was consecutive, complete or random in 5 and the sample size was adequate in 5. SCH was explicitly defined as a raised TSH with normal FT4 in 7 studies, standard and reliable measurement could be confirmed in only 3, and the response among those invited was not reported in any study (Figure 6).

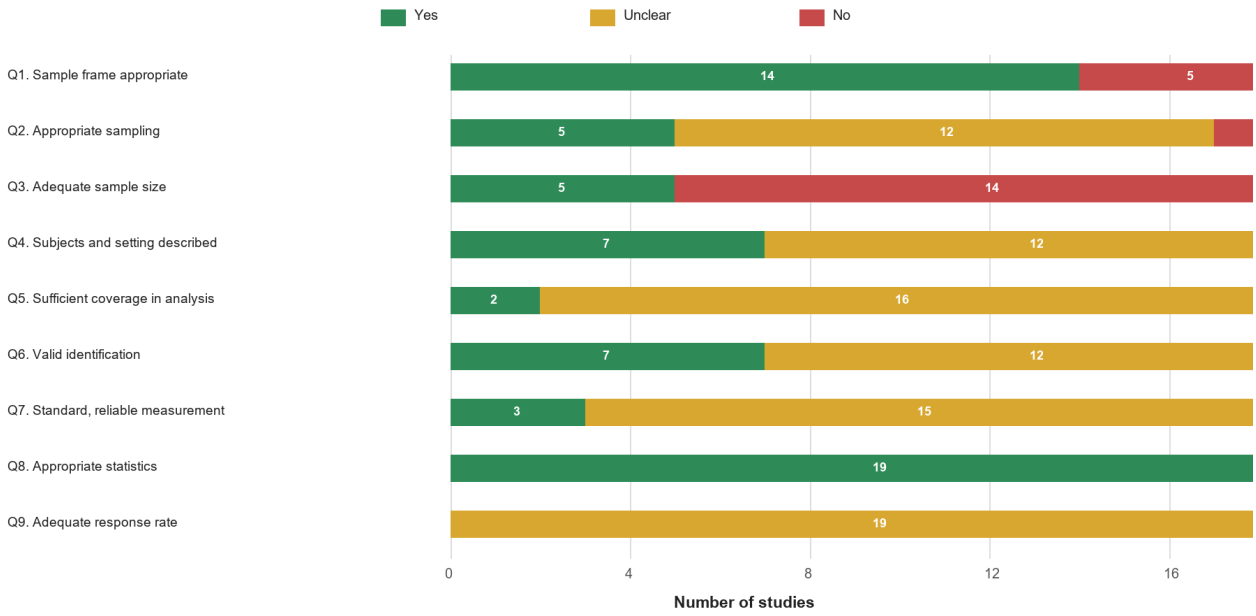


Figure 6. Methodological quality of the studies in the meta-analysis (JBI checklist for prevalence studies)

Numbers in the bars are numbers of studies.

Cardiometabolic correlates

Eight studies compared cardiometabolic factors between adults with SCH and euthyroid adults with overweight or obesity (Table 3). Five reported a less favourable profile in SCH: higher triglycerides, total or LDL cholesterol, fasting glucose or insulin resistance, or a higher frequency of MetS. Two found no overall differences in lipids, energy expenditure or glucose metabolism, and one reported descriptive data only. The outcomes, group definitions and statistics differed too much for pooling.

Table 3. Cardiometabolic factors in adults with overweight or obesity with and without subclinical hypothyroidism

Study	Country	Population	SCH / euthyroid, n	Findings in SCH compared with euthyroid participants
Tagliaferri 2001 [48]	Italy	Obese adults, BMI-matched groups	108 / 131	No differences in energy intake, resting energy expenditure, body composition, leptin or lipids; lower energy expenditure per kg fat-free mass only with TSH >5.7 mIU/L
Adamarczuk-Janczyszyn 2016 [49]	Poland	Obese women aged 50–70 years	100 / 42	Higher total cholesterol, LDL-C, triglycerides, LDL-C/HDL-C ratio and Castelli index
Wright 2016 [50]	USA	Overweight and obese adults	35 / 269a	No differences in fasting glucose, insulin, HOMA-IR or glucose tolerance
Zhang 2018 [38]	China	Obese women of reproductive age	108 / 426a	Higher triglycerides (1.7 vs 1.5 mmol/L, p = 0.002); similar insulin resistance
Salman 2021 [42]	Egypt	Sleeve gastrectomy candidates	30 / 98a	Higher waist circumference, diastolic blood pressure, cholesterol and triglycerides (all p ≤ 0.02); more often women
Stoica 2022 [51]	Romania	Obese women with and without prediabetes	22 / 27	HOMA-IR 5.68 vs 3.59 with prediabetes and 2.33 vs 2.46 without; no between-group test reported
Hewage 2024 [52]	Sri Lanka	Non-diabetic women 18–35 years, BMI ≥23 kg/m2	71 / 69	MetS 84.5% vs 7.2%; insulin resistance 77.5% vs 34.8% (both p ≤ 0.001)
Jameel 2025 [53]	Pakistan	Obese adults, incident SCH over 6 months	82 / 118	Higher fasting glucose, HOMA-IR, total cholesterol and LDL-C, lower HDL-C (all p ≤ 0.004)

Note. a Number of euthyroid participants calculated as the total minus the SCH group. HOMA-IR, homeostasis model assessment of insulin resistance; LDL-C and HDL-C, low- and high-density lipoprotein cholesterol; MetS, metabolic syndrome; SCH, subclinical hypothyroidism.

Discussion

In this meta-analysis of 19 studies with 5,847 adults with obesity, about one in eight had SCH (12.6% (95% CI 10.2–15.4)). This is about three times the prevalence of SCH in the general population of the United States, 4.3% [2], and higher than the 4.94% reported for undiagnosed hypothyroidism in Europe [3]; in direct comparisons, obesity carried a 1.7-fold risk of SCH [10]. The prediction interval of 5.0–28.3% indicates that the prevalence in a new obese population may vary several-fold; the setting, the TSH threshold and the handling of known thyroid disease did not significantly explain this variation.

The prevalence was similar in bariatric surgery candidates, other clinical samples and community or volunteer samples, and no association with mean BMI was detected in the 9 studies that reported it, although TSH correlates with BMI within individual cohorts [7, 8, 54]. Part of the TSH elevation in obesity may be functional: thyroid antibodies were less frequent in obese than in normal-weight patients with SCH (32.1% vs 66.1%), and the authors questioned whether a raised TSH alone is enough for the

diagnosis in morbid obesity [11]. However, in one community study the excess of SCH in obesity was confined to adults with thyroid antibodies (22.4% in obese vs 11.7% in non-obese antibody-positive adults) [44], obesity is associated with thyroid peroxidase antibodies [10], and leptin may link adiposity with thyroid autoimmunity [55]. After bariatric surgery, TSH decreased and SCH resolved in 87% of patients in a meta-analysis [12], in 87% after Roux-en-Y gastric bypass in the Dutch cohort [35] and in all patients in two cohorts [31, 33]; complete resolution at 12 months was also reported in a third cohort, in which half of the patients with SCH received thyroxine [39], and the prevalence fell from 23.4% to 7.8% one year after sleeve gastrectomy [42].

The cardiometabolic correlates were inconsistent. Five of eight comparative studies found higher lipid or glucose levels, more insulin resistance or more MetS in SCH, with the strongest differences in young women [52] and in adults with incident SCH [53], whereas the only matched comparison found no differences except a lower energy expenditure per kilogram of fat-free mass when TSH exceeded 5.7 mIU/L [48]. In the wider population, SCH is associated with MetS (odds ratio 1.28) [5] and with slightly higher systolic and diastolic blood pressure (by 1.9 and 0.8 mm Hg) [56]. In obesity, these associations are difficult to separate from the effect of adiposity itself, which raises both TSH and cardiometabolic risk.

These findings support a differentiated approach. A raised TSH should be confirmed by repeating TSH and FT4, together with thyroid peroxidase antibodies, after 2–3 months [57, 58]. Levothyroxine is recommended for younger patients with TSH above 10 mU/L, and a trial may be considered in younger symptomatic patients with lower values [57], whereas a guideline panel issued a strong recommendation against thyroid hormones for most adults with SCH [59]. In obese patients, adding thyroxine to sleeve gastrectomy did not improve outcomes in a randomised comparison [39], and follow-up alone was considered sufficient for most patients after gastric bypass [35]. When treatment is needed, the levothyroxine requirement relates more closely to lean body mass than to fat mass [60]. Levothyroxine lowered total cholesterol by only about 0.2 mmol/L in mild thyroid failure, and by 0.14 mmol/L in previously untreated patients [61].

For servicemen with obesity, these data suggest that TSH testing could be considered at periodic medical examinations and that weight reduction should come first, because SCH often resolved after substantial weight loss, whereas exercise capacity is reduced in SCH [62] and only partly restored by hormone replacement [63, 64]. In Uzbekistan, 23.5% of adults aged 18–69 years were obese in 2019 [65] and iodine status is borderline in some groups [66], so national data on thyroid function in obese service members are needed.

The strengths of this review are the restriction of the prevalence analysis to adults with obesity, the checking of every value used against the source by a second reviewer and the exploration of heterogeneity. Its limitations are the search of a single database, the unregistered protocol, the changes made to it and the analyses that were not specified in it, the reliance on abstracts for 11 of 19 studies, the lack of a stated TSH threshold in 13 studies, the single TSH measurement in most studies, which overestimates persistent SCH, the appraisal of quality by a single reviewer and the absence of studies in military personnel. The comparative evidence was too heterogeneous to be pooled.

Conclusion

SCH affects about one in eight adults with obesity; its prevalence did not differ significantly between bariatric, clinical and community samples, was not associated with mean BMI in the studies that reported it, and often fell after surgically induced weight loss. Its association with an unfavourable cardiometabolic profile is inconsistent. In military personnel with obesity, TSH testing could be considered at periodic examinations; a raised value should be confirmed with FT4 and thyroid antibodies after 2–3 months, weight reduction should be the first step, and levothyroxine should be considered only for younger patients with TSH persistently above 10 mU/L or, as a trial, for younger symptomatic patients.

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