Association between total daily sedentary time and cardiometabolic biomarkers in older adults: A systematic review and meta-analysis

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ABSTRACT

Background: Older adults engage in the highest levels of sedentary behaviour across all age groups. Yet, the extent to which sedentary time is associated with cardiometabolic health in older adults is unclear. This systematic review and meta-analysis examined associations between daily sedentary time and cardiometabolic biomarkers in older adults. **Methods:** Peer-reviewed articles in participants aged ≥60 years that studied the association between daily sedentary time and ≥1 cardiometabolic biomarker were eligible. Five electronic databases (PubMed, CINAHL, Medline, Web of Science and PsycINFO) were searched. Screening, data extraction and study quality were undertaken independently by two reviewers. Meta-analyses were undertaken using random effects models based on correlation and regression coefficients. Methodological quality was assessed using the Joanna Briggs Institute checklist. **Results:** Twenty-eight articles were included with sample sizes ranging from 30 to 62,754 participants. Increasing daily sedentary time was adversely associated with body mass index (Hedge's *g*: 0.32; P=0.001), waist circumference (Hedge's *g*: 0.45; P<0.001), body fat percentage (Hedge's *g*: 0.61; P=0.012) and fat mass (Hedge's *g*: 0.30; P=0.018). There were also unfavourably associations with systolic blood pressure (Hedge's *g*: 0.37; P=0.047), blood glucose (Hedge's *g*: 0.30; P=0.044), triglycerides (Hedge's *g*: 0.36; P=0.039) and HDL cholesterol (Hedge's *g*: 0.34; P=0.034). **Conclusions:** Increased daily sedentary time is adversely associated with body composition, systolic blood pressure and blood biomarkers in older adults. Therefore, limiting sedentary behaviour should be considered an important target in this population group for improved cardiometabolic health.

1. INTRODUCTION

The global prevalence of diabetes is estimated at 10.5% (536.6 million people) in adults aged 20 to 79 years[1]. There are an estimated 523 million prevalent cases of cardiovascular disease (CVD) and 19.8 million deaths worldwide due to CVD per annum[2,3]. The risk of CVD and CVD-mortality increases with age[4]. Older adults have the highest prevalence of diabetes, affecting 24% of individuals aged 75 to 79 years[1], and nearly half of all individuals living with Type 2 diabetes mellitus (T2DM) aged ≥65 years[5]. Prevalence of hypertension also increases with age, affecting 75% of adults aged 60 years and over in the National Health and Nutrition Examination Survey[6]. Clinical guidelines emphasise lifestyle management as a priority for those with an elevated risk of cardiometabolic disease[7]. Despite the promotion of moderate to vigorous-intensity physical activity (MVPA)[8], large proportions of older adults are physically inactive[9–11].

Limiting sedentary behaviour may be more achievable than increasing MVPA and is now recommended in global physical activity guidelines for older adults[8]. Older adults engage in the highest levels of sedentary behaviour across all age groups[12,13], with studies demonstrating that this population group spend between 62 and 80% of their waking day sedentary[14,15]. A large body of literature suggests that higher volumes of sedentary time are associated with an elevated risk of T2DM and CVD in the general population, and older adults[16,17]. The increased risk of cardiometabolic diseases associated with higher sedentary time may be independent of physical activity[16–18]. To understand the mechanisms though which sedentary time increases cardiometabolic disease risk and to inform targeted interventions, it is pertinent to explore associations of this behaviour with individual and clustered CVD and T2DM biomarkers (i.e., metabolic syndrome risk factors). In community-dwelling adults aged ≥55 years, daily sedentary time was unfavourable associated with diastolic blood pressure (DBP) and high-density lipoprotein (HDL) cholesterol but was not associated with waist circumference or fasting glucose[19]. Inconsistent findings have also been reported in other studies of older adults for individual biomarkers and the metabolic syndrome[20,21]. A synthesis of evidence is, therefore, warranted to overcome the limitations of drawing conclusions from individual studies and provide precise effects regarding the relationship between sedentary behaviour and cardiometabolic biomarkers in older adults.

An overview of systematic reviews examining sedentary behaviour and health in adults found that reducing or breaking up sedentary time may benefit markers of cardiometabolic risk[22]. That said, this included only one systematic review specific to older adults[23], which found mixed evidence across 26 studies with respect to

cardiometabolic biomarkers, and none included a meta-analysis. Furthermore, the metabolic and vascular dysfunction that occurs with ageing may mean that findings in adults are not directly relevant to older adults[24]. Thus, the association between sedentary time and cardiometabolic biomarkers in older adults remains unclear. Understanding this relationship is important to identify if sedentary behaviour should be a target for reducing CVD and T2DM risk in this population.

The primary aim of this study was to provide an up-to-date systematic review and meta-analysis of evidence concerning the association of daily sedentary time with traditional cardiometabolic biomarkers in older adults. A secondary aim was to assess whether the associations observed were influenced by the method of exposure measurement, i.e. self-report versus device-assessed sedentary time.

2. METHODS

2.1. Review Protocol

This review was conducted and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis guidelines [25]. Ethical approval was obtained from the Institutional Ethics Committee (application number: ***anonymised***). The study protocol was registered with PROSPERO (***anonymised***).

2.2 Eligibility Criteria

The search criteria used the Population, Intervention, Comparator, Outcomes and Study design framework [26] (**Table 1**). Articles needed to report the association of daily sedentary time with at least one cardiometabolic biomarker in older adults aged ≥60 years to be eligible. The cardiometabolic biomarkers of focus for this review were traditional clinical metabolic syndrome risk factors and additional lipoprotein and body composition outcomes. Cross-sectional or prospective studies were eligible, in addition to studies that undertook analysis of baseline data from randomised controlled trials. Review articles, conference abstracts, and grey literature were excluded. Articles were limited to English language only.

2.3 Information Sources

Five electronic databases (PubMed, CINAHL, Medline, SPORTDiscus and PsycINFO) were searched to identify original research articles published in peer-reviewed journals in the last 20 years up to 13th June 2024. A systematic

block search of Boolean terms was developed in PubMed and implemented in three blocks: sedentary time, cardiometabolic biomarkers and older adults (**Supplementary Material 1**). The reference lists of relevant articles and review articles were hand-searched to identify any further studies and were added to full-text screening manually. The results of the search were imported into rayyan[27] for eligibility screening.

2.4 Study Selection and Extraction of Data

Three reviewers (RLJ, LDC and DPB) undertook eligibility screening and data extraction. Following the removal of duplicates, each article title and abstract was screened independently by two reviewers. Full text articles were then assessed for eligibility independently by two reviewers. Discrepancies were resolved through discussion between the first and second reviewer, with any further disagreements being resolved by consulting a third reviewer. The following data was extracted from each eligible article independently by two authors: author, year of publication, study design, sample characteristics (age and sex), country of study, method of measuring sedentary time, cardiometabolic biomarker(s) assessed, confounders adjusted for in the analysis and results (correlation or regression coefficient including odds ratio, β coefficient, and *r*). Corresponding authors were contacted by email to acquire relevant data if necessary.

2.5. Study Quality

The methodological quality of the papers was assessed independently by two reviewers (DLC and JKZ) using the Joanna Briggs Institute checklist for analytical cross-sectional studies[28]. Study quality criteria focused on definition of the inclusion criteria, description of the study sample and setting, measurements and outcomes being recorded in valid and reliable ways, identification of confounding factors, and appropriateness of statistical analysis. Each criterion was recorded as 'Yes', 'No', 'Unclear' or 'Not applicable' (**Table 3**). If more than 50% of items (>4 criterion) were recorded as 'No' or 'Unclear', papers were considered high risk of bias and excluded[29].

2.6. Data Synthesis

Data for the most adjusted correlation or regression coefficient was used for the meta-analysis. Where SD was not provided, this was estimated from standard error or 95% confidence intervals (CI). Standardised mean differences (SMDs) and Hedge's *g* effect sizes were calculated, which enabled dichotomised and continuous outcome data to be pooled[30]. Hedge's *g* effect sizes of 0.2, 0.5 and 0.8 were considered small, moderate and large, respectively[31]. Data was pooled for meta-analysis when at least three studies reported data for the same cardiometabolic

122 biomarker. Random effects meta-analyses were conducted for each eligible outcome using Jamovi for Windows
123 (Version 2.3, Sydney, Australia). Meta-analyses were conducted for all available data (overall effect), as well as
124 separate models for self-reported and device-assessed sedentary time (subgroup analysis). Heterogeneity
125 (I^2 statistic) for each outcome was categorised as low (≥ 30%) moderate (≥ 50%) or high (≥ 75%)[32]. High
126 heterogeneity was also indicated from the pooled data with a Q statistic of $P \leq 0.05$. To assess publication bias,
127 forest and funnel plots were developed with asymmetry being assessed using Egger's Regression Test (> 10
128 studies) or visual inspection (< 10 studies[28]). Data is reported as Hedge's *g* effect sizes. Statistical significance
129 was accepted at $P \leq 0.05$.
130
131 For the narrative synthesis, including biomarker outcomes that were not meta-analysed, data is presented in terms
132 of the number of studies that did or did not observe significant associations between sedentary time and
133 cardiometabolic biomarker outcomes. To provide a more coherent analysis, cardiometabolic biomarkers were
134 grouped into body composition, blood pressure, glycaemic, lipid and other cardiometabolic biomarkers.

3. RESULTS

3.1 Article selection

A total of 38,808 articles were identified from the search. Following duplicate removal, 23,174 articles remained for title and abstract screening, from which 23,064 studies were excluded. Full texts were retrieved for 110 articles and assessed for eligibility. Twenty-eight articles, including 82,806 participants, met the eligibility criteria and were included in this review (**Figure 1**).

3.2 Study quality [33–36]

All 28 studies meeting eligibility criteria were also eligible for inclusion based on study quality assessment (**Table 2**). Common methodological issues across the studies included a) a lack of information about inclusion criteria, exclusion criteria or both (k = 18[20,33,35,37–51]), and b) poor quality of exposure (k = 6[33,37–39,52,53]) or outcome (k = 4[42,43,45,52]) assessment (e.g., including a small number of valid days required for device-measured sedentary time assessment and self-reported measures to calculate BMI). The range for 'yes' responses was 4 to 8, median of 7. Reviewer agreement was 95%.

3.3 Study characteristics

Study characteristics are displayed in **Table 3**. All studies used a cross-sectional design and were conducted across 15 different countries (Australia [k = 4]; United States [k = 4]; Brazil [k = 3]; Portugal [k = 3]; United Kingdom [k = 3]; Finland [k = 2]; Sweden [k = 2]; Belgium and Hong Kong [k = 1]; China [k = 1]; Japan [k = 1]; Korea [k = 1]; Netherlands [k = 1]; Spain [k = 1]; Taiwan [k = 1]). Sample size ranged from 30 to 62,754 participants. Females and males were included in all but three studies, in which two reported only female data[40,54] and one reported only male data[44]. Mean age of the samples ranged from 65[55] to 84 years[56]. Mean age was not stated in two studies, instead reporting $\geq$ 65 years[45] or sub-grouped into 60-69 years[41]. All but one study[56] investigated younger older adults i.e., ages 60-80 years. A wide array of cardiometabolic biomarkers were assessed, the most common being BMI (k = 14), waist circumference (k = 10), HDL cholesterol (k = 7), metabolic syndrome (k = 6), triglycerides (k = 6), DBP (k = 5), and SBP (k = 5). The method of sedentary time measurement also varied across the 28 studies, with 19 studies using accelerometery to provide device-assessed data (only three used activPal to capture changes in posture) and nine studies employing a self-report assessment (the majority, six out of nine, used the International Physical Activity Questionnaire [IPAQ]). The procedures used to measure each outcome are shown in **Supplementary Material 3.**

3.4 Sedentary time and body composition

Meta-analyses revealed that daily sedentary time was adversely associated with BMI (Hedge's *g*: 0.32; P = 0.001), waist circumference (Hedge's *g*: 0.45; P < 0.001), body fat percentage (Hedge's *g*: 0.61; P = 0.012) and fat mass (Hedge's *g*: 0.30; P = 0.018). There was significant evidence of publication bias for BMI, waist circumference and body fat percentage (all P ≤ 0.021), but not fat mass (P = 0.512). High levels of heterogeneity (all $I^2 \geq 99.7\%$; P < 0.001) were evident across body composition outcomes. In the subgroup analysis, there was a significant association between device-assessed sedentary time being unfavourably associated with waist circumference (Hedge's *g*: 0.22; P < 0.021) and a trend for an adverse association with BMI (Hedge's *g*: 0.25; P = 0.061). Self-reported sedentary time was unfavourably associated with BMI (Hedge's *g*: 0.36; P = 0.004) and waist circumference (Hedge's *g*: 0.64; P < 0.001); **Figure 2**.

For outcomes not included in the meta-analysis, sedentary time was not associated with limb fat, subcutaneous fat, trunk fat and visceral fat mass[54]. Sedentary time was positively associated with fat mass index[44].

Fourteen of 23 studies that assessed body composition outcomes observed a significant adverse association with sedentary time[37,38,41,42,44–46,48,50,52,53,57,58]; see **Table 4** for individual study outcomes. Adjustment of physical activity did not appear to influence the presence of significant associations between sedentary time and body composition. Eight of 14 studies that reported significant associations adjusted for physical activity[37,38,41,45,46,52,55,57]. Physical activity was adjusted for in four of nine studies that found no associations[43,47,54,59] (**Supplementary Material 2**).

3.5 Sedentary time and blood pressure

Increasing daily sedentary time was unfavourably associated with SBP (Hedge's *g*: 0.37; P = 0.047) but not associated with DBP (Hedge's *g*: 0.18; P = 0.150). High levels of heterogeneity (I^2 = 100%; P < 0.001) and publication bias (P < 0.001) were evident. Device-assessed sedentary time subgroup analysis revealed non-significant associations with both SBP (Hedge's *g*: 0.32; P = 0.215) and DBP (Hedge's *g*: 0.24; P = 0.382). Subgroup analysis for self-reported sedentary time revealed an unfavourable association with DBP (Hedge's *g*: 0.13; P < 0.001) but not SBP (Hedge's *g*: 0.39; P = 0.202); **Figure 3**. Men arterial pressure was reported in one study, which found no association with sedentary time[53].

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196 Two of seven studies that included blood pressure as an outcome observed an unfavourable association with
197 sedentary time[41,47]; these two studies included adjustment for physical activity[41,47] (**Supplementary Material 2)**.
198 Two of the five studies that did not observe significant associations adjusted for physical activity[46,56].

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200 **3.6 Sedentary time and glycaemic biomarkers**

201 Meta-analyses revealed that daily sedentary time was unfavourably associated with blood glucose (Hedge's *g*:
202 0.30; P = 0.044); **Figure 4**. Heterogeneity ($I^2 \geq 93.8\%$; $P < 0.001$) and publication bias ($P < 0.001$) were high.

203

204 For outcomes not meta-analysed, sedentary time was positively associated with fasting insulin[44] and glucose 120
205 min[51]. Sedentary time was positively associated with HbA1c in males, but not in females[41].

206

207 Four of eight studies that investigated sedentary time and glycaemic biomarkers reported significant adverse
208 associations[41,44,51,53]; **Table 4**. There was no clear indication that adjustment for physical activity influenced the
209 associations observed; two of four studies observed significant associations[41,51] and three of four studies did not
210 observe associations[37,46,47] adjusted for physical activity.

211

212 **3.7 Sedentary time and lipid biomarkers**

213 Daily sedentary time was unfavourably associated with triglycerides (Hedge's *g*: 0.36; P = 0.039) and HDL
214 cholesterol (Hedge's *g*: 0.34; P = 0.034); **Figure 4**. Heterogeneity ($I^2 \geq 100\%$; $P < 0.001$) and publication bias (P
215 < 0.001) were high for both outcomes. In subgroup analysis, self-reported sedentary time was unfavourably
216 associated with triglycerides (Hedge's *g*: 0.50; P = 0.031) and HDL cholesterol (Hedge's *g*: 0.45; P = 0.022).
217 There was insufficient data for subgroup analysis of device-assessed sedentary time.

218

219 Sedentary time was unfavourably associated with lipid biomarkers that were not meta-analysed, including total
220 cholesterol[49], non-HDL cholesterol in males[41] and cholesterol ratio[37]. There was no association with LDL[49], non-
221 HDL cholesterol in females[41], apolipoprotein A-1, apolipoprotein B and apolipoprotein B:A-1 ratio[20].

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223 There were significant adverse associations between sedentary time and lipid biomarkers in five of eight
224 studies[37,41,46,47,49]; **Table 4**. Adjustment for physical activity did not appear to influence associations as this was
225 adjusted for in four of five studies that reported significant associations[37,41,46,47].
226
227 **3.8 Sedentary time and metabolic syndrome**
228 Increasing sedentary time was unfavourably associated with metabolic syndrome (Hedge's *g*: 0.56; P = 0.003);
229 **Figure 4.** There were high levels of heterogeneity ($I^2 \geq 100\%$; $P < 0.001$) and publication bias ($P < 0.001$). Device-
230 assessed sedentary time subgroup analysis revealed a non-significant association (Hedge's *g*: 0.44; P = 0.07).
231
232 There were significant adverse associations between sedentary time and metabolic syndrome in three of seven
233 studies [21,44,46]. Only two studies adjusted for physical activity; one reported a significant association[46] and the
234 other reported no association with metabolic syndrome[33].

4. DISCUSSION

This is the first meta-analysis investigating the associations of daily sedentary time with cardiometabolic biomarkers in older adults. Increasing daily sedentary time was adversely associated with body composition, SBP, lipid and glycaemic biomarkers, and metabolic syndrome. Where there was sufficient data for exposure measurement subgroup analysis (BMI, waist circumference and blood pressure), self-reported sedentary time yielded stronger and more consistent associations with cardiometabolic biomarkers than device-assessed sedentary time.

The current study extends, and updates findings reported in a previous systematic review in which there was mixed evidence for associations between sedentary time and cardiometabolic biomarkers in older adults[23]. Detrimental associations were reported more consistently across biomarkers in this review. This may be due to inclusion only of studies that measured daily sedentary time as the exposure, as opposed to multiple domains or contexts, such as TV viewing or leisure time[23]. The increase in available evidence in recent years permitted meta-analyses for multiple biomarkers to provide an accurate estimate of effect in relation to risk associated with sedentary time. Despite there being high heterogeneity, significant detrimental associations were found for all cardiometabolic biomarkers, except DBP. These findings emphasise the potential importance of limiting daily sedentary time for promoting cardiometabolic health in older adults and supports the focus on sedentary behaviour in physical activity guidelines[60].

Unfavourable associations with biomarkers were present across 67% of studies that adjusted for physical activity, suggesting that sedentary time may be an independent risk factor related to cardiometabolic risk in older adults. However, measurement of physical activity varied widely (e.g. moderate-intensity, light-intensity, leisure time and METs), which may affect outcomes across studies. Engaging in high daily volumes of MVPA (60-75 and 30-40 minutes per day according to self-report and device-based methods, respectively) may offset the adverse association between high sedentary time and mortality in mixed samples of middle- and older-aged adults[61,62]. It was not possible to investigate the joint effects of sedentary time and physical activity in the current review due to the nature of the data reported within the included studies; therefore, the mediating role of MVPA in older adults remains unclear. Engaging in 30 to 75 minutes per day of MVPA is unfeasible for large proportions of the population, especially older adults who have unique barriers to physical activity such as pain and perceived risk

of injury[63]. Therefore, limiting sedentary behaviour may be a more achievable strategy, initially, to improve cardiometabolic health[64].

As the meta-analyses demonstrated unfavourable associations with cardiometabolic biomarkers regardless of how sedentary time is measured, this provides strong support for limiting daily sedentary time in older adults. Yet, the subgroup analysis revealed stronger effects with self-reported versus device-assessed sedentary time for BMI, waist circumference and DBP. The high heterogeneity across studies could be a contributing factor explaining this disparity between exposure measurement methods. For example, there were two datasets with much stronger effect sizes for waist circumference when sedentary time was measured by self-report, therefore inflating the overall effect. It has also been widely reported among adults that self-report methods underestimate daily sedentary time compared with device methods, which may weaken the strength of associations[65]. Further, the majority of the studies employing self-report (six out of nine) used the IPAQ, which neglects any time spent sedentary that does not align with sitting, potentially further contributing to under-reporting of total sedentary time. When comparing accelerometer and IPAQ data among older adults specifically, it has been suggested under-reporting of sedentary behaviour at the individual-level may be improved by providing additional detail of types of daily activities that this population might undertake to improve recall, alongside examples of typical activities performed across the day[66]. Although 19 of the 28 studies used accelerometery, only three of these studies used the activPal to capture changes in posture and, therefore, better discriminate sedentary behaviour from light, moderate or vigorous physical activity. The determination of posture is important as definitions of sedentary behaviour include both an energy expenditure element (≤1.5 METs) and a postural element (i.e., sitting, reclining or lying)[67]. Further, a systematic review of accelerometery studies suggested that more data regarding the validity of accelerometery to determine sedentary time is needed in older adults, including population-specific recommendations for non-wear time classifications and the required number of hours and days for valid sedentary time estimates[68]. As such, further research using activPal with consideration for older adult-specific sedentary time analysis methods would be useful in confirming the strength of associations with cardiometabolic biomarkers in this population.

Older adults have a higher prevalence of physical and psychological chronic conditions[69], yet the impact of chronic conditions on outcomes within the current review remains unclear partly due to the disparity in participant inclusion criteria. For example, Chastin et al.,[50] aligned to the 'healthy' definition of older adults proposed by

294 Greig et al.,[70] including the presence of metabolic disease, yet clear definitions were not presented across the
295 literature. This heterogeneity could contribute to differences in findings between studies and, therefore, the effects
296 sizes reported. Additionally, factors concerning the management of long-term health conditions requires
297 consideration, especially in a population where medication is prevalent. Treatments for mental health conditions,
298 for example, can cause fatigue or drowsiness, leading to increased sedentary behaviour[71]. Understanding the
299 impact of chronic conditions and their management on the relationship between sedentary time and
300 cardiometabolic health should be considered in future research.
301
302 The mechanisms through which sedentary time increases cardiometabolic risk may include prolonged periods of
303 muscular inactivity, leading to reduced production of metabolites (e.g., nitric oxide) involved with downstream
304 vasodilatory effects[72]. With respect to adiposity, sedentary behaviours require minimal energy expenditure, which,
305 without a corresponding reduction in caloric intake, may result in an energy surplus[73]. The consequential
306 accumulation of body fat can act as a mediating pathway to impaired cardiometabolic health[73]. High volumes of
307 sedentary behaviour may also result in insufficient muscular activity to stimulate contraction-mediated glucose
308 uptake pathways[74]. Similarly, reduced muscle contractile activity has an inhibitory effect on the production of
309 lipoprotein lipase, which is an essential enzyme in the lipolysis of triglycerides and production of HDL
310 cholesterol[75].
311
312 **Strengths and limitations**
313 A strength of this study is the novel meta-analytic insight demonstrating unfavourable associations between daily
314 sedentary time and cardiometabolic biomarkers in older adults. The findings strengthen the importance of
315 including recommendations to limit sedentary time in physical activity and clinical care guidelines. Furthermore,
316 rigorous methods were employed to assess article eligibility and risk of bias of included studies. A potential
317 limitation was the variation across studies with regards to sample size. To account for this, Hedge's *g* effect sizes
318 were calculated to adjust for small sample size bias[76]. Heterogeneity remained significant, even with the
319 application of random effect models. Study quality was acceptable for all eligible articles. Yet, the quality
320 checklist revealed some notable limitations, including a lack of detailed description of the sample and settings to
321 help determine which populations the findings are applicable. Sedentary time and outcomes were not always
322 recorded in valid and reliable ways; for example, BMI was self-reported in some studies[45] and the device
323 (accelerometery) methods did not consistently align with recommendations for valid wear time, with some studies

only requiring one valid day to be included in their analysis[37,56]. There was also wide variation in the confounding factors included in the statistical models employed. Lastly, all but one study investigated younger older adults (60-80 years). Preliminary data in nonagenarians and centenarians showed that 91-94% of wake time was spent in sedentary behaviour. The findings of this review may, therefore, not be generalisable to the 'oldest' old adults[77].

Conclusion

This review demonstrates that increasing daily sedentary time is adversely associated with cardiometabolic biomarkers in older adults. The associations observed were present regardless of how sedentary time was measured, but with stronger effects for self-report versus device assessment. The unfavourable associations appeared to be largely independent from physical activity, suggesting that sedentary behaviour is an independent risk factor that should be targeted in public health initiatives for promoting cardiometabolic health in older adults. To generate more precise effects, future studies should employ recommended criteria for valid wear time when using device-based methods. Research investigating associations between sedentary time and cardiometabolic biomarkers in the oldest old is also needed to inform recommendations for this segment of the population.

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571 **Table 1.** Population, intervention or exposure, comparator, outcomes, and study design (PICOS)
572 criteria

Population, intervention or exposure, comparator, outcomes, and study design (PICOS) criteria	
Population	Individuals were required to be aged ≥ 60 years with no upper age limit. Studies were excluded where older adults were included but did not report data distinctly for that age group.
Intervention or Exposure	Daily sedentary time assessed by self-report or device methods. Studies were excluded if there was no differentiation between sedentary time and other types of activity (e.g., lying down, sleeping, light physical activity).
Comparator	Studies of all older adults living in any setting and of any health status were eligible.
Outcomes	Studies reported associations (correlation or regression coefficient) of daily sedentary time with at least one cardiometabolic biomarker regardless of assessment method for the exposure or outcome variables. Studies were excluded when no statistical test assessing the association between sedentary time and a cardiometabolic biomarker was present.
Study design	All study designs were included; as such, data was extracted from observational studies that were cross-sectional or prospective, and randomised controlled trial studies in which associations within baseline data were reported.

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Table 2. Check list for study quality from Joanna Briggs Institute for Cross-sectional studies; scoring yes = +, no = -, unsure = ?

Study	Were the criteria for inclusion in the study clearly defined?	Were the study subjects and the setting described in detail?	Was the exposure measured in a valid and reliable way?	Were objective; standard criteria used for measurement of the condition	Were confounding factors identified?	Were strategies to deal with confounding factors stated	Were the outcomes measured in a valid and reliable way?	Was appropriate statistical analysis used?	Overall appraisal
Bankoski et al.[33]	-	+	?	+	+	+	+	+	**Include**
Chastin et al.[50]	-	-	+	+	-	-	+	+	**Include**
Chen et al.[54]	+	+	+	+	+	+	+	+	**Include**
Cheng et al.[34]	+	+	+	+	-	-	+	+	**Include**
Danésio de Souza et al.[49]	-	+	+	+	+	+	+	+	**Include**
van Dyck et al.[48]	-	+	+	+	+	+	+	+	**Include**
Figueiró et al.[47]	-	+	+	+	+	+	+	+	**Include**
Freire et al.[21]	+	+	+	+	+	+	+	+	**Include**
Gardiner et al.[46]	?	+	+	+	+	+	+	+	**Include**
Gianoudis et al.[59]	+	+	+	+	+	+	+	+	**Include**
Howard et al.[20]	?	+	+	+	+	+	+	+	**Include**
Hsueh et al.[45]	-	+	+	+	+	+	?	+	**Include**
Jefferis et al.[44]	-	+	+	+	+	+	+	+	**Include**
Júdice et al.[35]	-	+	+	+	+	+	+	+	**Include**
Júdice, Silva, and Sardinha[43]	-	?	+	+	+	+	?	+	**Include**
Koolhaas et al.[42]	-	+	+	+	+	+	?	?	**Include**
Koyama et al.[41]	-	?	+	+	+	+	+	?	**Include**
Lansitie et al.[51]	-	?	+	+	+	+	+	+	**Include**
Nilsson et al.[40]	?	+	+	+	+	+	+	+	**Include**
Park and Larson[53]	+	+	?	+	+	+	+	+	**Include**
Reid et al.[57]	+	+	+	+	+	+	+	?	**Include**
Rosenberg et al.[52]	+	+	?	+	+	+	?	+	**Include**
Rosenberg et al.[56]	+	+	+	+	+	+	+	+	**Include**

Rosique-Esteban et al.[55]	+	+	+	+	+	+	+	+	**Include**
Savikangas et al.[58]	+	+	+	+	+	-	+	-	**Include**
Silva et al.[39]	-	+	?	+	+	+	+	+	**Include**
Sohn et al.[38]	-	+	?	+	+	+	+	+	**Include**
Stamatakis et al.[37]	-	+	?	+	+	+	+	+	**Include**

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575 **Table 3.** Study characteristics

Study, country	Study design	Sample	Sedentary time measurement method	Cardiometabolic biomarker outcomes
Bankoski et al.[33], United States	Cross-sectional	**Metabolic Syndrome:** N = 665 (61.6% female) Mean age = 71 ± 7.4 years **No Metabolic Syndrome:** N = 702 (50.7% female) Mean age = 71 ± 8 years	Accelerometery, ActiGraph AM-7164	Metabolic syndrome (NCEP ATP III definition)
Chastin et al.[50], United Kingdom	Cross-sectional	**Females:** N = 14 Mean age = 79.3 ± 3.4 years **Males:** N = 16 Mean age = 79 ± 3.9 years	Accelerometery, activPAL	Body fat (%)
Chen et al.[54], China	Cross-sectional	N = 1105 (100% female) Mean age = 65 years	Accelerometery, ActiGraph wGT3X-BT	BMI (kg/m^2), Body fat (%), Body fat mass (kg), Limb fat mass (kg), Subcutaneous fat mass (kg), Trunk fat mass (kg), Visceral fat mass (kg)
Cheng et al.[34], Australia	Cross-sectional	N = 39 (49% female) Mean age = 74 ± 10 years	Accelerometry, activPAL3	BMI (kg/m^2)
Danésio de Souza et al.[49], Brazil	Cross-sectional	N = 402 (60.4% female) Mean age = 72.2 ± 7 years	Self-reported, IPAQ	HDL cholesterol (mg/dL), LDL cholesterol (mg/dL), Total cholesterol (mg/dL), Triglycerides (mg/dL)

van Dyck et al.[48], Belgium, Hong Kong	Cross-sectional	N = 829 (61.35% female) Mean age = 74.83 ± 6.18 years	Accelerometery, ActiGraph GT3X and GT3X+	BMI (kg/m^2)
Figueiró et al. [47], Brazil	Cross-sectional	N = 425 (59.8% female) Mean age = 73.9 years	Accelerometery, ActiGraph GT3X and GT3X+	DBP (mmHg), Fasting glucose (mg/dL), HDL cholesterol (mg/dL), SBP (mmHg), Triglycerides (mg/dL), Waist circumference (cm)
Freire et al.[21], Brazil	Cross-sectional	N = 248 (78% female) Mean age = 66 ± 4.6 years	Accelerometery, ActiGraph GT3X+	Metabolic syndrome (number)
Gardiner et al. [46], Australia	Cross-sectional	**Whole sample:** N = 1958 (54% female) Mean age = 69 years **Females with Metabolic Syndrome:** N = 642 Mean age = 68.9 years **Females with Metabolic Syndrome:** N = 460 Mean age = 69.3 years **Males without Metabolic Syndrome:** N = 487 Mean age = 69.7 years	Self-reported, IPAQ	Blood pressure (mmHg), Fasting glucose (mmol/L), HDL cholesterol (mmol/L), Metabolic syndrome (number), Triglycerides (mmol/L), Waist circumference (cm)

		Males with Metabolic Syndrome: N = 409 Mean age = 69.4 years		
Gianoudis et al. [59], Australia	Cross-sectional	N = 162 (74% female) Mean age = 67.5 ± 6 years	Self-reported, validated seven-day recall questionnaire	BMI (kg/m^2), Body fat mass (kg)
Howard et al.[20], Sweden	Cross-sectional	N = 364 (72% female) Mean age = 74 ± 6.8 years	Self-reported, IPAQ modified for the elderly	ApoA-1 (g/L), ApoB (g/L), ApoB : ApoA-1 ratio (g/L), BMI (kg/m^2), DBP (mmHg), HDL cholesterol (mmol/L), High sensitivity CRP (g/L), LDL cholesterol (mmol/L), SBP (mmHg)
Hsueh et al.[45], Taiwan	Cross-sectional	N = 1046 (53.1% female) Age > 65 years	Self-reported, validated seven-day recall questionnaire	BMI (kg/m^2)
Jefferis et al.[44], United Kingdom	Cross-sectional	N = 1078 (0% female) Mean age = 78.5 ± 4.7 years	Accelerometery, ActiGraph GT3X	BMI (kg/m^2), Fasting insulin (mmol/L), Fat mass index (kg/m^2), Metabolic syndrome (number) Waist circumference (cm)
Judice et al.[35], Portugal	Cross-sectional	N = 301 (63.1% female) Mean age = 75 ± 6.8 years	Accelerometery, ActiGraph GT1M	Waist circumference (cm)
Judice, Silva & Sardinha[43], Portugal	Cross-sectional	N = 351 (65.5% female) Mean age = 74.6 ± 7 years	Accelerometery, ActiGraph GT1M	Waist circumference (cm)
Koolhaas et al.[42], Netherlands	Cross-sectional	N = 1210 (51.9% female) Mean age = 77.5 ± 5 years	Accelerometery, Activinsights GeneActiv	BMI (kg/m^2)

Koyama et al.[41], Japan	Cross-sectional	**60-69 years:** N = 62,754 (55.5% female) Age range = 60 – 69 years	Self-reported, IPAQ	BMI (kg/m^2), DBP (mmHg), HbA1c (%), HDL cholesterol (mg/dL), non-HDL cholesterol (mg/dL), SBP (mmHg), Triglycerides (mg/dL)
Lansitie et al.[51], Finland	Cross-sectional	N = 702 (58.3% female) Mean age = 68.9 $\pm$ 0.6 years	Accelerometery, Polar Electro Polar Active	Glucose 120 min (mmol/L)
Nilsson et al.[40], Sweden	Cross-sectional	N = 120 (100% female) Mean age = 67.5 $\pm$ 1.6 years	Accelerometery, ActiGraph GT3X	DBP (mmHg), Fasting glucose (mmol/L), HDL cholesterol (mmol/L), Metabolic syndrome (z-score), Metabolic syndrome minus Waist circumference (z-score), SBP (mmHg), Triglycerides (mmol/L), Waist circumference (cm)
Park and Larson [53], United States	Cross-sectional	N = 223 (48.9% female) Mean age = 70.1 $\pm$ 8.7 years	Accelerometery, ActiGraph 7164	Fasting glucose (mmol/L), HDL cholesterol (mmol/L), MAP (mmHg), Metabolic syndrome (number), Triglycerides (mmol/L), Waist circumference (cm)
Reid et al.[57], Australia	Cross-sectional	N = 124 (63% female) Mean age = 70.9 $\pm$ 4.2 years	Accelerometery, activPAL 3	Body fat (%), Body fat mass (kg)
Rosenberg et al.[52], United States	Cross-sectional	N = 3538 (49% female) Mean age = 72.6 $\pm$ 6 years	Self-reported, IPAQ	BMI (kg/m^2)
Rosenberg et al.[56], United States	Cross-sectional	N = 307 (72.3% female) Mean age = 83.6 $\pm$ 6.4 years	Accelerometery, ActiGraph GT3X	DBP (mmHg), SBP (mmHg)
Rosique-Esteban et al.[55], Spain	Cross-sectional	N = 1539 (48% female) Mean age = 65.3 $\pm$ 5 years	Self-reported, Nurses' Health Study questionnaire for Sedentary Behaviours	BMI (kg/m^2), Body fat mass (kg), Waist circumference (cm)
Savikangas et al.[58], Finland	Cross-sectional	N = 293 (58% female) Mean age = 74.4 $\pm$ 3.8 years	Accelerometery, UKK RM42	Body fat (%)

Silva et al.[39], Portugal	Cross-sectional	N = 83 (67.5% female) Mean age = 72.1 ± 5.6 years	Accelerometery, ActiGraph GT1M	BMI (kg/m^2)
Sohn et al.[38], South Korea	Cross-sectional	**Females:** N = 906 Mean age = 70 ± 7 years **Males:** N = 656 Mean age = 69.4 ± 6.7 years	Self-reported, IPAQ	BMI (kg/m^2), Waist circumference (cm)
Stamatakis et al.[37], United Kingdom	Cross-sectional	**Sitting time lowest tertile:** N = 213 (61 % female) Mean age = 67.8 ± 6.3 years **Sitting time middle tertile:** N = 217 (60.4% female) Mean age = 69.3 ± 6.9 years **Sitting time highest tertile:** N = 216 (43.5 % female) Mean age = 72.5 ± 8.1 years	Accelerometery, ActiGraph GT1M	BMI (kg/m^2), Cholesterol Ratio (mmol/L), HbA1c (%), Waist circumference (cm)

ApoA-1, Apolipoprotein A-1; ApoB, Apolipoprotein B; BMI, Body Mass Index; CRP, C-reactive protein; DBP, Diastolic blood pressure; HbA1c, Glycated haemoglobin A1c; HDL, High-density lipoprotein; IPAQ, International Physical Activity Questionnaire; LDL, Low-density lipoprotein; MAP, Mean arterial pressure; NCEP ATP III, National Cholesterol Education Program Adult Treatment Panel III; SBP, Systolic blood pressure.

579 **Table 4.** Associations between daily sedentary time and cardiometabolic biomarkers for each included study

Study	Sample size and grouping	Cardiometabolic biomarkers	Statistic	Effect size (95% CI)	P value
Bankoski et al.[33]	Whole sample (n = 1,367)	Metabolic syndrome (NCEP ATP III definition)	Odds ratio	1.16 (0.77, 1.74)	0.25
Chastin et al.[50]	Females (n = 14)	Body fat (%)	Pearson's correlation	0.382	0.276
	Males (n = 16)	Body fat (%)	Pearson's correlation	0.382	**0.042**
Chen et al.[54]	Whole sample (n = 1,105)	BMI (kg/m^2)	Standardised β	-0.09 (-0.26, 0.30)	0.292
		Body fat (%)		0.01 (-0.30, 0.31)	0.970
		Body fat mass (kg)		0.02 (-0.33, 0.38)	0.894
		Limb fat mass (kg)		0.06 (-0.09, 0.20)	0.442
		Subcutaneous fat mass (kg)		0.03 (-0.23, 0.29)	0.827
		Trunk fat mass (kg)		-0.03 (-0.25, 0.19)	0.774
		Visceral fat mass (kg)		-0.01 (-0.10, 0.09)	0.880
Cheng et al.[34]	Whole sample (n = 39)	BMI (kg/m2)	Simple correlation	-0.01	> 0.05
Danésio de Souza et al.[49]	Whole sample (n = 402)	HDL cholesterol (mg/dL)	Standardised β	-0.01 (-0.10, 0.08)	Not reported
		LDL cholesterol (mg/dL)	Standardised β	-0.09 (-0.13, 0.06)	0.10
		Total cholesterol (mg/dL)	Standardised β	-0.09 (-0.18, 0.009)	**0.03**
		Triglycerides (mg/dL)	Standardised β	-0.09 (-0.25, 0.06)	Not reported
van Dyck et al.[48]	Whole sample (n = 829)	BMI (kg/m^2)	Standardised β	0.493 (0.299, 0.686)	**< 0.001**
Figueiró et al.[47]	Whole sample (n = 425)	DBP (mmHg)	Standardised β	-0.01 (-0.01, 0.01)	0.672
		Fasting glucose (mg/dL)		0.01 (-0.01, 0.03)	0.229

		HDL cholesterol (mg/dL)		-0.02 (-0.02, 0.01)	**0.019**
		SBP (mmHg)		-0.03 (-0.05, -0.01)	**0.017**
		Triglycerides (mg/dL)		0.04 (-0.01, 0.08)	0.179
		Waist circumference (cm)		0.02 (-0.01, 0.03)	0.079
Freire et al.[21]	Whole sample (n = 248)	Metabolic syndrome (number)	Standardised β	0.09 (0.03, 0.15)	**0.03**
Gardiner et al.[46]	Females (n = 1,062)	High blood pressure (mmHg)	Odds ratio	1.29 (0.88, 1.87)	> 0.05
		Glucose intolerance (mmol/L)		1.17 (0.81, 1.71)	> 0.05
		Low HDL cholesterol (mmol/L)		1.27 (0.81, 1.99)	> 0.05
		Metabolic syndrome (number)		1.56 (1.09, 2.24)	**< 0.05**
		High triglycerides (mmol/L)		1.66 (1.14, 2.41)	**< 0.01**
		High waist circumference (cm)		1.81 (1.21, 2.70)	**< 0.01**
	Males (n = 896)	High blood pressure (mmHg)	Odds ratio	0.87 (0.55, 1.39)	> 0.05
		Glucose intolerance (mmol/L)		0.92 (0.60, 1.40)	> 0.05
		Low HDL cholesterol (mmol/L)		1.78 (1.05, 3.02)	**< 0.05**
		Metabolic syndrome (number)		1.57 (1.02, 2.41)	**< 0.05**
		High triglycerides (mmol/L)		1.61 (1.01, 2.58)	**< 0.05**
		High waist circumference (cm)		1.52 (0.94, 2.45)	> 0.05
Gianoudis et al.[59]	Whole sample (n = 162)	BMI (kg/m^2)	Standardised β	0.04 (-0.30, 0.22)	> 0.05
		Body fat mass (kg)		0.29 (-0.24, 0.82)	> 0.05
Howard et al.[20]	Whole sample (n = 364)	ApoA-1 (g/L)	Standardised β	-0.02 (-0.10, 0.06)	0.676
		ApoB (g/L)		-0.03 (-0.09, 0.03)	0.374
		ApoB:ApoA-1 ratio (g/L)		-0.03 (-0.08, 0.02)	0.255
		BMI (kg/m^2)		1.12 (-0.01, 2.25)	0.056
		DBP (mmHg)		0.13 (-3.09, 3.35)	0.938

		HDL cholesterol (mmol/L)		0.95 (0.88, 1.01)	0.120
		LDL cholesterol (mmol/L)		-0.12 (-0.40, 0.16)	0.408
		SBP (mmHg)		1.04 (0.99, 1.09)	0.085
Hsueh et al.[45]	Whole sample (n = 1,046)	High BMI (kg/m^2)	Odds ratio	1.51 (1.03, 2.20)	**0.03**
Jefferis et al.[44]	Sub-sample (n = 1,019)	BMI (kg/m^2)	Standardised β	0.011 (0.008, 0.013)	**< 0.05**
	Sub-sample (n = 966)	Fasting insulin (mmol/L)	Standardised β	0.009 (0.004, 0.014)	**< 0.05**
	Sub-sample (n = 962)	Fat mass index (kg/m^2)	Standardised β	0.009 (0.006, 0.011)	**< 0.05**
	Sub-sample (n = 907)	Metabolic syndrome (number)	Odds ratio	1.004 (1.002, 1.006)	**< 0.05**
	Sub-sample (n = 1,023)	Waist circumference (cm)	Standardised β	0.034 (0.025, 0.042)	**< 0.05**
Júdice et al.[35]	Females (n = 190)	High waist circumference (cm)	Odds ratio	1.00 (0.99, 1.04)	0.297
		Waist circumference (cm)	Unstandardised β	0.01 ± 0.01	0.297
	Males (n = 111)	High waist circumference (cm)	Odds ratio	1.00 (0.99, 1.01)	0.251
		Waist circumference (cm)	Unstandardised β	0.001 ± 0.001	0.251
Júdice, Silva, & Sardinha[43]	Whole sample (n = 351)	Waist circumference (cm)	Standardised β	0.02 (-0.12, 0.16)	0.789
Koolhaas et al.[42]	Females (n = 582)	BMI (kg/m^2)	Standardised β	0.90 ± 0.14	**< 0.001**
	Males (n = 628)	BMI (kg/m^2)	Standardised β	0.96 ± 0.18	**< 0.001**
Koyama et al.[41]	Females - 60-69 years (n = 11,510)	BMI (kg/m^2)	Standardised β	0.034	**0.004**
		DBP (mmHg)		0.057	**< 0.001**
		HbA1c (%)		-0.003	0.815
		HDL cholesterol (mg/dL)		0.042	**0.001**
		non-HDL cholesterol (mg/dL)		0.021	0.080

		SBP (mmHg)		0.056	**< 0.001**
		Triglycerides (mg/dL)		0.045	**< 0.001**
	Males - 60-69 years	BMI (kg/m^2)	Standardised β	0.058	**< 0.001**
	(n = 10,994)	DBP (mmHg)		0.047	**< 0.001**
		HbA1c (%)		0.024	**0.044**
		HDL cholesterol (mg/dL)		-0.016	0.171
		non-HDL cholesterol (mg/dL)		0.039	**0.001**
		SBP (mmHg)		0.042	**< 0.001**
		Triglycerides (mg/dL)		0.054	**< 0.001**
Lansitie et al.[51]	High waist circumference tertile (men > 104 cm; women > 94 cm) (n = 189)	Glucose 120 min (mmol/L)	Standardised β	0.14 (0.02, 0.26)	**0.022**
Nilsson et al.[40]	Whole sample	DBP (mmHg)	Standardised β	-0.06 (-0.30, 0.18)	> 0.05
	(n = 113)	Fasting glucose (mmol/L)		0.01 (-0.01, 0.03)	> 0.05
		HDL cholesterol (mmol/L)		0.00 (-0.02, 0.00)	> 0.05
		Metabolic syndrome (z-score)		0.02 (0.00, 0.03)	> 0.05
		Metabolic syndrome without waist circumference (z-score)		0.02 (0.00, 0.03)	> 0.05
		SBP (mmHg)		0.12 (-0.31, 0.54)	> 0.05
		Triglycerides (mmol/L)		0.00 (-0.02, 0.00)	> 0.05
		Waist circumference (cm)		0.09 (-0.20, 0.39)	> 0.05
Park and Larson[53]	Whole sample	Fasting glucose (mg/dL)	Unstandardised β	0.02	**< 0.05**
	(n = 223)	HDL cholesterol (mmol/L)	Unstandardised β	0.01	> 0.05
		MAP (mmHg)	Unstandardised β	0.01	> 0.05
		Metabolic syndrome (number)	Odds ratio	2.46 (0.79, 7.64)	> 0.05

		Triglycerides (mmol/L)	Unstandardised β	-0.08	> 0.05
		Waist circumference (cm)	Unstandardised β	0.02	**< 0.05**
Reid et al.[47]	Whole sample	Body fat (%)	Unstandardised β	1.07 (0.21, 1.92)	**< 0.05**
	(n = 123)	Body fat mass (kg)		1.93 (0.71, 3.15)	**< 0.01**
Rosenberg et al.[52]	Whole sample	BMI (kg/m^2)	Unstandardised β	0.82 (0.53, 1.10)	**< 0.001**
	(n = 3,538)				
Rosenberg et al.[56]	Whole sample	DBP (mmHg)	Standardised β	0.77 ± 0.58	0.19
	(n = 307)	SBP (mmHg)		0.78 ± 1.10	0.48
Rosique-Esteban et al.[55]	Whole sample	BMI (kg/m^2)	Standardised β	0.25 (0.16, 0.35)	**< 0.001**
	(n = 1, 539)	Body fat mass (kg)		0.47 (0.30, 0.65)	**< 0.001**
		Waist circumference (cm)		0.60 (0.35, 0.83	**< 0.001**
Savikangas et al.[58]	Whole sample	Body fat (%)	Pearson's correlation	0.251	**< 0.001**
	(n = 293)				
Silva et al.[39]	Whole sample	BMI (kg/m^2)	Pearson's correlation	0.146	0.187
	(n = 83)				
Sohn et al.[38]	Female	High BMI (kg/m^2)	Odds ratio	1.19 (0.86, 1.51)	> 0.05
	(n = 906)	BMI (kg/m^2)	Standardised β	0.100 ± 0.035	**0.002**
		High waist circumference (cm)	Odds ratio	1.19 (0.87, 1.53)	> 0.05
		Waist circumference (cm)	Standardised β	0.033 ± 0.100	0.318
	Male	High BMI (kg/m^2)	Odds ratio	1.54 (1.09, 2.16)	**< 0.05**
	(n = 656)	BMI (kg/m^2)	Standardised β	0.105 + 0.035	**0.007**
		High waist circumference (cm)	Odds ratio	1.38 (0.88, 1.81)	> 0.05
		Waist circumference (cm)	Standardised β	0.109 + 0.109	**0.006**
Stamatakis et al.[37]	Whole sample (n = 649)	BMI (kg/m^2)	Unstandardised β	0.160 (-0.021, 0.342)	> 0.05
	Sub-sample (n = 333)	Cholesterol Ratio (mmol/L)	Unstandardised β	0.060 (0.000, 0.121)	**< 0.05**
			Unstandardised β		> 0.05

	Sub-sample (n = 333)	HbA1c (%)	Unstandardised β	0.008 (-0.024, 0.040)	**< 0.05**
	Whole sample (n = 649)	Waist circumference (cm)		0.633 (0.173, 1.093)	

Bold indicates significant association. ApoA-1, Apolipoprotein A-1; ApoB, Apolipoprotein B; BMI, Body Mass Index; DBP, Diastolic blood pressure; HbA1c, Glycated haemoglobin A1c; HDL, High-density lipoprotein; LDL, Low-density lipoprotein; MAP, Mean arterial pressure; NCEP ATP III, National Cholesterol Education Program Adult Treatment Panel III; SBP, Systolic blood pressure.

583 **Figure captions.**

584

585 **Fig. 1** Flow diagram of the article selection process

586

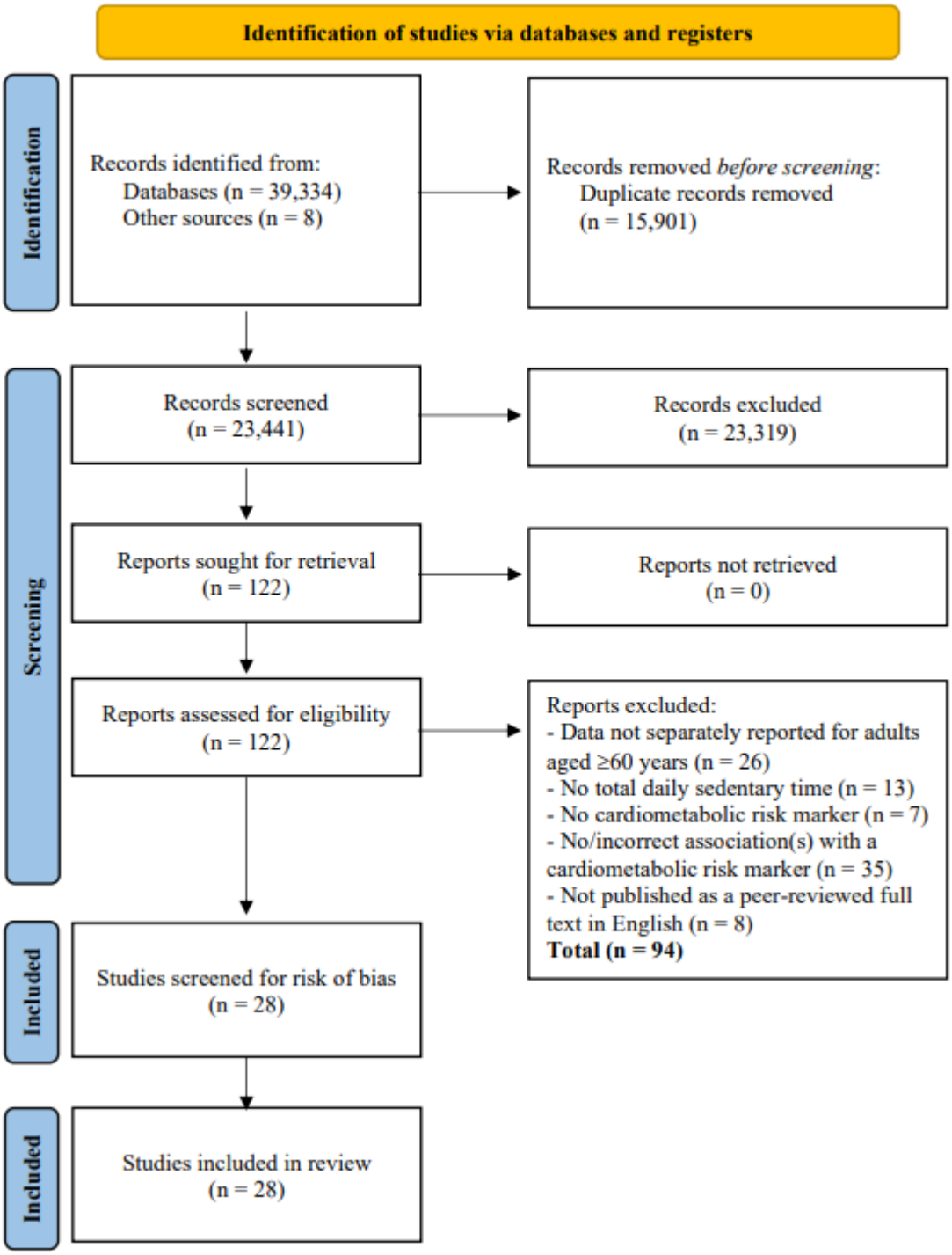
587 **Fig. 2** Forest plot for the random-effect meta-analysis for body mass index, waist circumference, body fat
588 percentage and fat mass. SE, standard error; CI, confidence interval.

589

590 **Fig. 3** Forest plot for the random-effect meta-analysis for diastolic and systolic blood pressure. SE, standard
591 error; CI, confidence interval.

592

593 **Fig. 4** Forest plot for the random-effect meta-analysis for blood glucose, triglycerides, high-density lipoprotein
594 cholesterol and metabolic syndrome. SE, standard error; CI, confidence interval.


Identification of studies via databases and registers
Identification
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Databases (n = 39,334)
Other sources (n = 8)
Records removed *before screening*:
Duplicate records removed
(n = 15,901)
Screening
Records screened
(n = 23,441)
Records excluded
(n = 23,319)
Reports sought for retrieval
(n = 122)
Reports not retrieved
(n = 0)
Reports assessed for eligibility
(n = 122)
Reports excluded:
- Data not separately reported for adults aged ≥60 years (n = 26)
- No total daily sedentary time (n = 13)
- No cardiometabolic risk marker (n = 7)
- No/incorrect association(s) with a cardiometabolic risk marker (n = 35)
- Not published as a peer-reviewed full text in English (n = 8)
Total (n = 94)
Included
Studies screened for risk of bias
(n = 28)
Included
Studies included in review
(n = 28)

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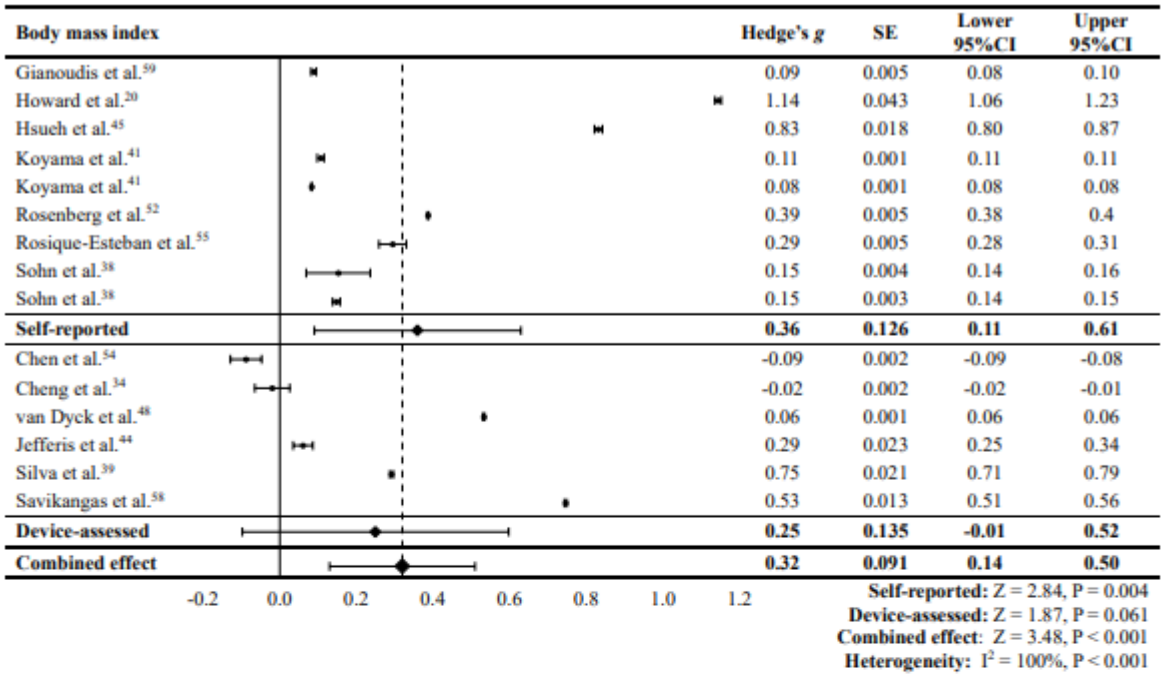
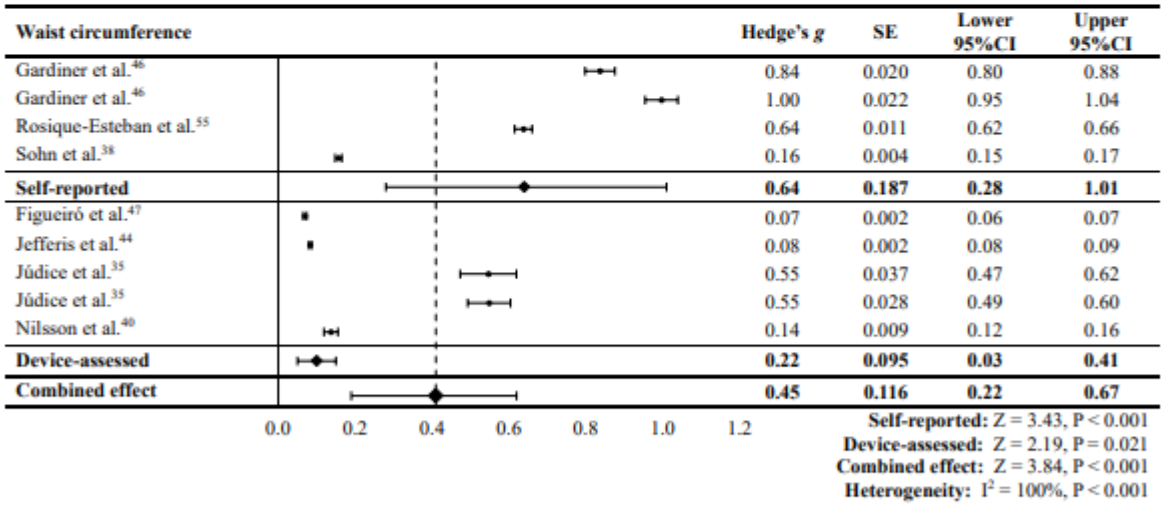
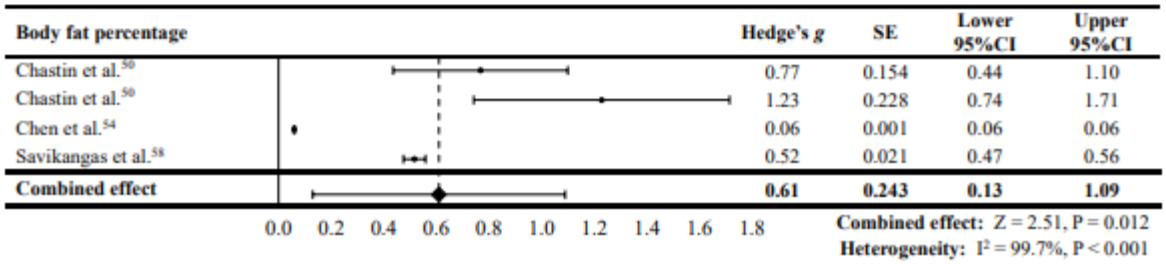

Body mass index
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Gianoudis et al.[59] 0.09 0.005 0.08 0.10
Howard et al.[20] 1.14 0.043 1.06 1.23
Hsueh et al.[45] 0.83 0.018 0.80 0.87
Koyama et al.[41] 0.11 0.001 0.11 0.11
Koyama et al.[41] 0.08 0.001 0.08 0.08
Rosenberg et al.[52] 0.39 0.005 0.38 0.4
Rosique-Esteban et al.[55] 0.29 0.005 0.28 0.31
Sohn et al.[38] 0.15 0.004 0.14 0.16
Sohn et al.[38] 0.15 0.003 0.14 0.15
Self-reported 0.36 0.126 0.11 0.61
Chen et al.[54] -0.09 0.002 -0.09 -0.08
Cheng et al.[34] -0.02 0.002 -0.02 -0.01
van Dyck et al.[48] 0.06 0.001 0.06 0.06
Jefferis et al.[44] 0.29 0.023 0.25 0.34
Silva et al.[39] 0.75 0.021 0.71 0.79
Savikangas et al.[58] 0.53 0.013 0.51 0.56
Device-assessed 0.25 0.135 -0.01 0.52
Combined effect 0.32 0.091 0.14 0.50
-0.2 0.0 0.2 0.4 0.6 0.8 1.0 1.2
Self-reported: Z = 2.84, P = 0.004
Device-assessed: Z = 1.87, P = 0.061
Combined effect: Z = 3.48, P < 0.001
Heterogeneity: I² = 100%, P < 0.001


Waist circumference
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Gardiner et al.[46] 0.84 0.020 0.80 0.88
Gardiner et al.[46] 1.00 0.022 0.95 1.04
Rosique-Esteban et al.[55] 0.64 0.011 0.62 0.66
Sohn et al.[38] 0.16 0.004 0.15 0.17
Self-reported 0.64 0.187 0.28 1.01
Figueiró et al.[47] 0.07 0.002 0.06 0.07
Jefferis et al.[44] 0.08 0.002 0.08 0.09
Júdice et al.[35] 0.55 0.037 0.47 0.62
Júdice et al.[35] 0.55 0.028 0.49 0.60
Nilsson et al.[40] 0.14 0.009 0.12 0.16
Device-assessed 0.22 0.095 0.03 0.41
Combined effect 0.45 0.116 0.22 0.67
0.0 0.2 0.4 0.6 0.8 1.0 1.2
Self-reported: Z = 3.43, P < 0.001
Device-assessed: Z = 2.19, P = 0.021
Combined effect: Z = 3.84, P < 0.001
Heterogeneity: I² = 100%, P < 0.001


Body fat percentage
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Chastin et al.[50] 0.77 0.154 0.44 1.10
Chastin et al.[50] 1.23 0.228 0.74 1.71
Chen et al.[54] 0.06 0.001 0.06 0.06
Savikangas et al.[58] 0.52 0.021 0.47 0.56
Combined effect 0.61 0.243 0.13 1.09
0.0 0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8
Combined effect: Z = 2.51, P = 0.012
Heterogeneity: I² = 99.7%, P < 0.001

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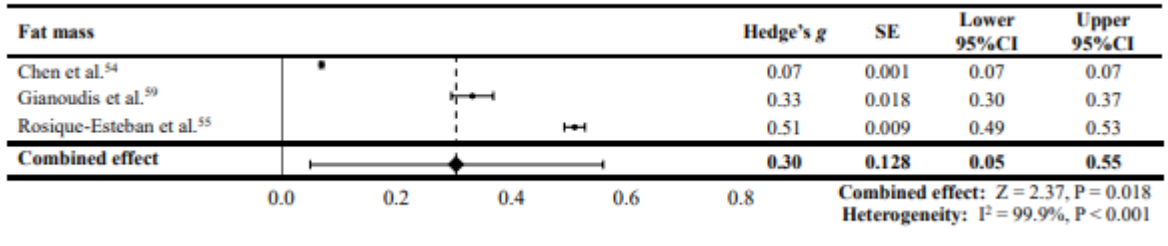
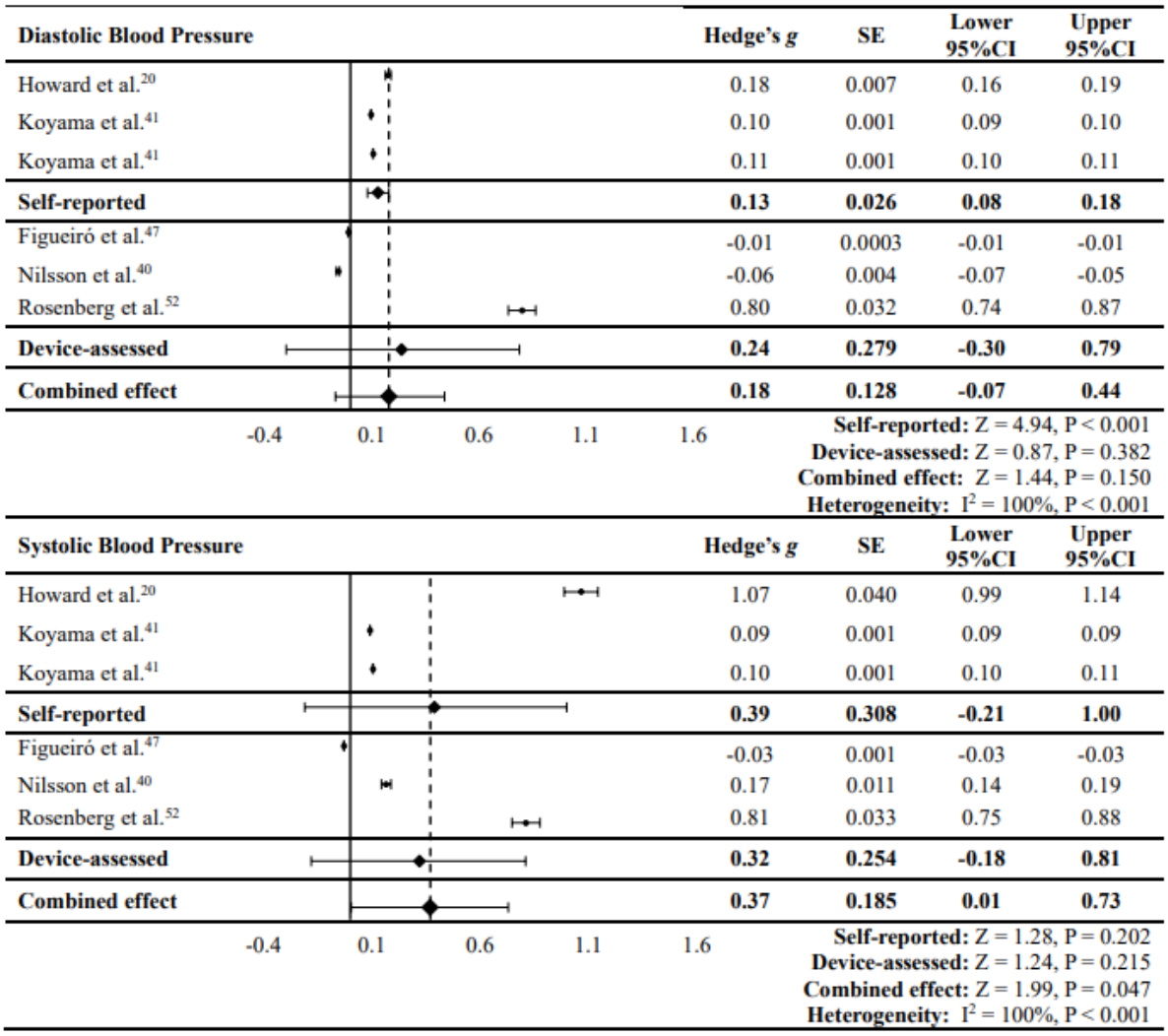
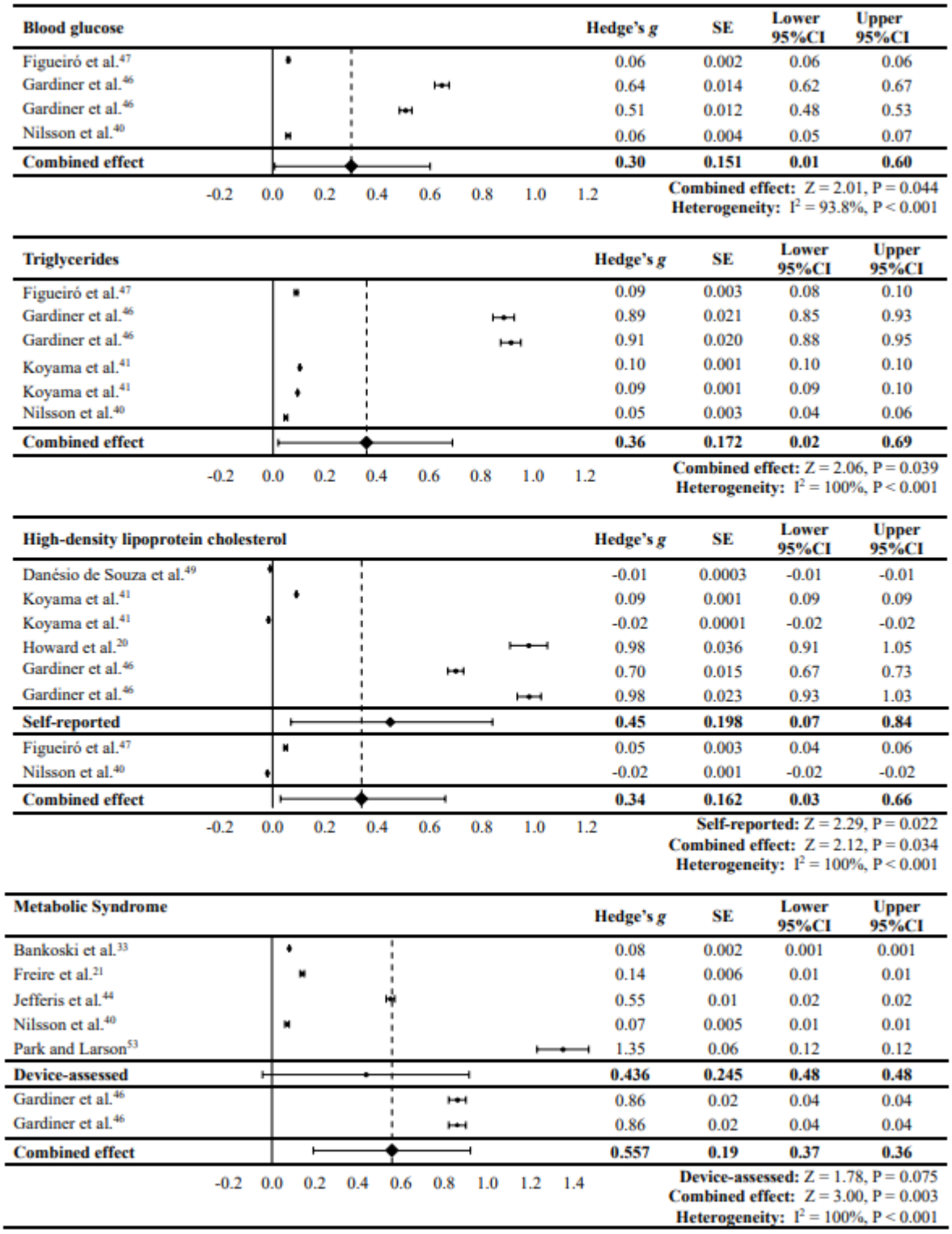

Fat mass
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Chen et al.[54] 0.07 0.001 0.07 0.07
Gianoudis et al.[59] 0.33 0.018 0.30 0.37
Rosique-Esteban et al.[55] 0.51 0.009 0.49 0.53
Combined effect 0.30 0.128 0.05 0.55
0.0 0.2 0.4 0.6 0.8
Combined effect: Z = 2.37, P = 0.018
Heterogeneity: I² = 99.9%, P < 0.001

597

598


Diastolic Blood Pressure
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Howard et al.[20] 0.18 0.007 0.16 0.19
Koyama et al.[41] 0.10 0.001 0.09 0.10
Koyama et al.[41] 0.11 0.001 0.10 0.11
Self-reported 0.13 0.026 0.08 0.18
Figueiró et al.[47] -0.01 0.0003 -0.01 -0.01
Nilsson et al.[40] -0.06 0.004 -0.07 -0.05
Rosenberg et al.[52] 0.80 0.032 0.74 0.87
Device-assessed 0.24 0.279 -0.30 0.79
Combined effect 0.18 0.128 -0.07 0.44
-0.4 0.1 0.6 1.1 1.6
Self-reported: Z = 4.94, P < 0.001
Device-assessed: Z = 0.87, P = 0.382
Combined effect: Z = 1.44, P = 0.150
Heterogeneity: I² = 100%, P < 0.001
Systolic Blood Pressure
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Howard et al.[20] 1.07 0.040 0.99 1.14
Koyama et al.[41] 0.09 0.001 0.09 0.09
Koyama et al.[41] 0.10 0.001 0.10 0.11
Self-reported 0.39 0.308 -0.21 1.00
Figueiró et al.[47] -0.03 0.001 -0.03 -0.03
Nilsson et al.[40] 0.17 0.011 0.14 0.19
Rosenberg et al.[52] 0.81 0.033 0.75 0.88
Device-assessed 0.32 0.254 -0.18 0.81
Combined effect 0.37 0.185 0.01 0.73
-0.4 0.1 0.6 1.1 1.6
Self-reported: Z = 1.28, P = 0.202
Device-assessed: Z = 1.24, P = 0.215
Combined effect: Z = 1.99, P = 0.047
Heterogeneity: I² = 100%, P < 0.001

599


Blood glucose
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Figueiró et al.[47] 0.06 0.002 0.06 0.06
Gardiner et al.[46] 0.64 0.014 0.62 0.67
Gardiner et al.[46] 0.51 0.012 0.48 0.53
Nilsson et al.[40] 0.06 0.004 0.05 0.07
Combined effect 0.30 0.151 0.01 0.60
-0.2 0.0 0.2 0.4 0.6 0.8 1.0 1.2
Combined effect: Z = 2.01, P = 0.044
Heterogeneity: I² = 93.8%, P < 0.001
Triglycerides
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Figueiró et al.[47] 0.09 0.003 0.08 0.10
Gardiner et al.[46] 0.89 0.021 0.85 0.93
Gardiner et al.[46] 0.91 0.020 0.88 0.95
Koyama et al.[41] 0.10 0.001 0.10 0.10
Koyama et al.[41] 0.09 0.001 0.09 0.10
Nilsson et al.[40] 0.05 0.003 0.04 0.06
Combined effect 0.36 0.172 0.02 0.69
-0.2 0.0 0.2 0.4 0.6 0.8 1.0 1.2
Combined effect: Z = 2.06, P = 0.039
Heterogeneity: I² = 100%, P < 0.001
High-density lipoprotein cholesterol
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Danésio de Souza et al.[49] -0.01 0.0003 -0.01 -0.01
Koyama et al.[41] 0.09 0.001 0.09 0.09
Koyama et al.[41] -0.02 0.0001 -0.02 -0.02
Howard et al.[20] 0.98 0.036 0.91 1.05
Gardiner et al.[46] 0.70 0.015 0.67 0.73
Gardiner et al.[46] 0.98 0.023 0.93 1.03
Self-reported 0.45 0.198 0.07 0.84
Figueiró et al.[47] 0.05 0.003 0.04 0.06
Nilsson et al.[40] -0.02 0.001 -0.02 -0.02
Combined effect 0.34 0.162 0.03 0.66
-0.2 0.0 0.2 0.4 0.6 0.8 1.0 1.2
Self-reported: Z = 2.29, P = 0.022
Combined effect: Z = 2.12, P = 0.034
Heterogeneity: I² = 100%, P < 0.001
Metabolic Syndrome
Hedge's g
SE
Lower 95%CI
Upper 95%CI
Bankoski et al.[33] 0.08 0.002 0.001 0.001
Freire et al.[21] 0.14 0.006 0.01 0.01
Jefferis et al.[44] 0.55 0.01 0.02 0.02
Nilsson et al.[40] 0.07 0.005 0.01 0.01
Park and Larson[53] 1.35 0.06 0.12 0.12
Device-assessed 0.436 0.245 0.48 0.48
Gardiner et al.[46] 0.86 0.02 0.04 0.04
Gardiner et al.[46] 0.86 0.02 0.04 0.04
Combined effect 0.557 0.19 0.37 0.36
-0.2 0.0 0.2 0.4 0.6 0.8 1.0 1.2 1.4
Device-assessed: Z = 1.78, P = 0.075
Combined effect: Z = 3.00, P = 0.003
Heterogeneity: I² = 100%, P < 0.001

600