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REVIEW ARTICLE

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Effects of interventions on sedentary behaviour and cardiovascular disease biomarkers in individuals with spinal cord injury: a systematic review

Daniel L. Cooper[a,b], Alyson Warland[a,c], Emma Norris[c], Cherry Kilbride[a,c], Sue Paddison[d] and Daniel P. Bailey[a,b]

[a]Centre for Physical Activity in Health and Disease, College of Health, Medicine and Life Sciences, Brunel University of London, Uxbridge, United Kingdom; [b]Department of Sport, Health and Exercise Sciences, Brunel University of London, Uxbridge, United Kingdom; [c]Department of Health Sciences, Brunel University of London, Uxbridge, United Kingdom; [d]London Spinal Cord Injury Centre, Royal National Orthopaedic Hospital NHS Trust, Stanmore, United Kingdom

ABSTRACT

Purpose: Reducing sedentary behaviour may be an intervention target to improve cardiovascular health in individuals with spinal cord injury. The aim of this study was to systematically review the effects of interventions on sedentary behaviour and cardiovascular disease biomarkers in individuals with paraplegia.

Materials and methods: Following prospective protocol registration (CRD42023420260), eleven sources were searched to identify articles, which were screened by two reviewers. Eligible articles included participants with paraplegia, interventions targeting physical activity and/or sedentary behaviour and studies that measured sedentary behaviour and cardiovascular disease biomarkers. Quality of evidence was assessed for each outcome.

Results: Two interventions targeting sedentary behaviour and six targeting physical activity were included. One intervention targeting sedentary behaviour and one targeting physical activity reduced sedentary behaviour. Two interventions targeting sedentary behaviour and three targeting physical activity improved cardiovascular disease biomarkers. Quality of evidence was very low for sedentary behaviour and moderate for cardiovascular disease biomarker outcomes.

Conclusions: Sedentary behaviour was not improved by physical activity interventions but these interventions may improve cardiovascular disease biomarkers in individuals with paraplegia. Interventions targeting sedentary behaviour, although limited, show potential effectiveness for improving cardiovascular disease biomarkers; such interventions require further investigation to inform public health and clinical care guidelines.

IMPLICATIONS FOR REHABILITATION

- Physical activity interventions are not effective for reducing sedentary behaviour in individuals with paraplegia
- Evidence regarding interventions targeting sedentary behaviour is limited, but such interventions show some potential effectiveness
- Interventions targeting sedentary behaviour in paraplegia should be investigated further to inform their relevance for rehabilitation

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Cardiometabolic health; cardiovascular disease; spinal cord injury; paraplegia; sedentary behaviour; physical activity

Introduction

A spinal cord injury (SCI) is a life-changing event that can cause loss of motor, sensory and autonomic function across and below the site of the injury [1]. Consequently, many individuals with SCI become manual wheelchair users [2,3]. A number of physical changes occur due to SCI, such as increased body mass and abdominal body fat [4], which are adversely associated with cardiovascular disease (CVD) biomarkers (e.g. glycaemia and lipid profile) in this population group [5]. This combination of factors makes the SCI population highly susceptible to cardiovascular disease (CVD) [6].

CONTACT Daniel P Bailey daniel.bailey@brunel.ac.uk Centre for Physical Activity in Health and Disease, College of Health, Medicine and Life Sciences, Brunel University of London, Kingston Lane, Uxbridge UB8 3PH, United Kingdom.

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Physical activity has been defined as "any bodily movement produced by skeletal muscles that results in energy expenditure" [7]. Greater levels of physical activity are associated with a decreased risk of CVD in non-disabled individuals [8]. Evidence-based guidelines recommend 40 min of moderate-to-vigorous-intensity physical activity (MVPA) per week to improve fitness and cardiometabolic health in individuals with SCI [9]. Paraplegia is described as damage to the spinal cord at the first thoracic vertebrae (T1) or below, resulting in trunk and lower limb dysfunction [10]. Retained function in the upper limbs gives greater capability for physical activity compared to individuals with tetraplegia who additionally have loss in upper limb function [11]. The majority of physical activity interventions in individuals with SCI have involved regular arm crank ergometry exercise [12]. These interventions have generally been successful for increasing MVPA in individuals with paraplegia [13–16]. Improvements in CVD biomarkers have also been reported in such interventions [17,18], but with mixed findings [13,18,19]. However, the SCI population generally engage in low levels of physical activity, with 44% of individuals reporting no participation in MVPA whatsoever [20].

Reducing sedentary behaviour ("an energy expenditure of ≤ 1.5 metabolic equivalents (MET) while sitting, lying or reclined during waking hours") [21], could be an alternative intervention target to MVPA for improving cardiometabolic health [22]. Individuals with SCI have been reported to spend more than 11 h per day being sedentary when measured using accelerometry [23]. This population group inherently spend long periods of time sitting due to regular or complete reliance on a wheelchair for mobility. Higher levels of sedentary behaviour are associated with increased risk of CVD in the general population, independent of physical activity [24,25]. Interventions targeting sedentary behaviour have been effective in reducing daily sedentary behaviour [26] and improving CVD biomarkers [27] in non-disabled individuals. The positive effects of reducing sedentary behaviour in non-disabled individuals could be of clinical relevance in those with SCI. Reducing and breaking up sedentary behaviour may also be more achievable than MVPA for individuals with long-term conditions [28].

A systematic review is needed to understand the potential effectiveness of interventions for reducing sedentary behaviour and improving CVD biomarkers in individuals with SCI. This could help to inform sedentary behaviour guidelines. A focus on individuals with paraplegia may be particularly relevant as this type of SCI provides a greater opportunity to break up sedentary behaviour due to retained upper limb function. The aims of this study were to systematically review (1) the effects of interventions on sedentary behaviour, and (2) the effects of these interventions on CVD biomarkers, in individuals with paraplegia.

Methods

The systematic review protocol was registered prospectively on the International Prospective Register of Systematic Reviews database (CRD42023420260) and is reported in accordance with the Preferred Reporting Items for Systematic reviews and Meta-analyses (PRISMA) guidelines (Supplementary file 1) [29].

Eligibility criteria

The Population, Intervention, Comparators, Outcomes and Study design (PICOS) process [30] was used as the framework to guide the eligibility criteria (Table 1). Articles were excluded if the publication was not in English. Published journal articles, conference papers, theses and pre-printed papers were eligible for inclusion. Conference abstracts were only eligible if the review team could obtain the necessary data from the abstract or study authors.

Table 1. Population, intervention, comparator, outcomes, and study design criteria for the review.

PICOS component	Eligibility criteria
Population	Adult participants (≥ 18 years old) of any sex with paraplegia.
Intervention	Any intervention that targeted a reduction in sedentary behaviour, or targeted an increase in physical activity and included a measurement of sedentary behaviour as an outcome.
Comparator	Studies with or without comparators, including either passive control or active control.
Outcomes	Aim 1: Sedentary behaviour as measured by self-report or device. Aim 2: ≥ 1 cardiovascular disease biomarker.
Study design	Randomised controlled trials, uncontrolled trials, crossover trials, quasi-experimental studies, pre-post studies, pilot studies, and feasibility studies.

Population

Studies with adults (≥ 18 years old) with paraplegia were eligible. It was anticipated that articles would vary in their definition of paraplegia; therefore, this review kept the definition purposefully broad to capture all articles that may provide data relevant to the review aims. For example, studies that provided brief or detailed definitions of paraplegia, or where individual level of injury were included. Studies that included participants with paraplegia and tetraplegia were included irrespective of the proportion of the sample with paraplegia as the outcomes would be relevant to the review's target population. Studies were excluded if participants were predominantly ambulant in order to ensure a focus on the target population of this review i.e., manual wheelchair-users who have the opportunity to break up sedentary behaviour with upper-limb movement.

Intervention

Interventions that targeted a reduction in sedentary behaviour and/or an increase in physical activity (if sedentary behaviour was reported as an outcome) were eligible. As such, interventions targeting physical activity, which could lead to displacement of sedentary behaviour, were included. Interventions were not limited with respect to any characteristic, delivery mode or dose.

Comparator

Studies with or without comparators were eligible, including passive or active control.

Outcome

Interventions that specifically target sedentary behaviour and/or include a quantitative measure of sedentary behaviour (self-reported, device-assessed) (Aim 1). Interventions that are eligible for aim 1 and report outcomes for ≥ 1 CVD biomarker (Aim 2).

Study design

Randomised controlled trials, uncontrolled trials, crossover trials, quasi-experimental studies, pre-post studies, pilot studies and feasibility studies were eligible for inclusion.

Search strategy

Searches were conducted on 12 June 2023, followed by updated searches on 13 August 2024 and 2 September 2025, using the following databases: CINAHL Plus (*via* EBSCO Host), ClinicalTrials.gov, Cochrane library, ISRCTN Registry, MEDLINE (*via* EBSCO Host), PsycInfo (*via* EBSCO Host), Physiotherapy Evidence Database, PubMed, Scopus, SPORT Discus (*via* EBSCO Host), and Web of Science. There were no search restrictions on publication date or publication type. The full list of search strings is included within Supplementary File 2. Reference lists of eligible articles were searched to identify any further potential studies for inclusion.

Study selection

Identified articles were exported to an online systematic review data management system (www.covidence.org) for screening, following removal of duplicates. Two independent reviewers screened titles and abstracts. Full texts were obtained for potentially eligible studies. The same reviewers independently reviewed the full texts. The reviewers reached a consensus through discussion in cases of disagreement during each stage of screening.

Quality appraisal

Risk of bias was assessed using the Cochrane Risk of Bias 2 tool for randomised trials (RoB-2) and relates to five domains: randomisation, deviation from intended intervention, missing outcome data, measurement of the outcome and selection of the reported results [31]. Risk of bias was determined as low, high or some concerns [31].

Data extraction

Two reviewers independently extracted items into a custom data extraction spreadsheet using Microsoft Excel (Redmond, WA, USA). Extracted data included publication details, study design, participant characteristics (age, sex, and level of injury), intervention characteristics (type, duration, frequency, intensity, delivery mode, setting, provider, use of behaviour change theory), the methods used to measure outcomes, and outcome results (effects on sedentary behaviour and CVD biomarkers).

Quality of evidence

The Grading of Recommendations, Assessment, Development and Evaluation (GRADE) criteria were used to assess certainty of evidence for each outcome (sedentary behaviour and CVD biomarkers) across five domains: risk of bias, inconsistency of results, indirectness of evidence, imprecision and publication bias [32]. Overall certainty of evidence was rated as high, moderate, low or very low for each outcome [32].

Data synthesis

There was wide heterogeneity in study design and intervention characteristics. Therefore, a meta-analysis was not undertaken. Instead, a qualitative synthesis was undertaken following Synthesis Without a Meta-analysis guidelines (Supplementary File 3) [33]. Eligible studies were grouped into suitable categories to interpret the results. When neither P-values nor confidence intervals (CI) were reported in a study, 95% CIs for mean differences were calculated to enable determination of statistical significance and aid interpretation. When SD for the mean difference was not available, this was estimated following Cochrane guidelines with a conservative *r* correlation of 0.5 assumed between baseline and follow-up SDs [34]. The 95% CIs were then calculated using a paired-samples *t* distribution, with adjustment for width of the interval according to sample size [35]. Studies were synthesised in relation to:

- Nature of the intervention i.e. the target behaviour.
- Intervention characteristics (dose, setting, delivery mode, duration, use of behaviour change theory).
- Study population.
- Outcome measurement method.
- Risk of bias appraisal.

Results

Study identification

A total of 11,221 records were identified, from which 5,290 duplicates were removed (Figure 1). Titles and abstract screening of the remaining 5,931 records resulted in 5,868 exclusions. The full-text of the 63 remaining records were assessed for eligibility, resulting in the removal of 54 records. Nine articles were included in the final synthesis. Nightingale et al. [14,17] and Nooijen et al. [36,37] reported findings from a single study across two separate articles; therefore, these were each considered one single intervention. Martinez et al. [38] reported findings from two different interventions within a single article; therefore, these were considered two distinct interventions. As a result, the review comprised eight distinct interventions that were investigated in seven studies. The findings of these studies are reported across nine articles.

Study characteristics

Characteristics of the included studies are shown in Table 2. Three of the seven studies were undertaken in the United Kingdom [14,18,39], two in the United States [38,40], one in the Netherlands [37] and one in Norway [41]. Five studies were randomised controlled trials [14,18,37,40,41], one was a randomised comparative effectiveness trial [38] and one employed a randomised controlled crossover design [39].

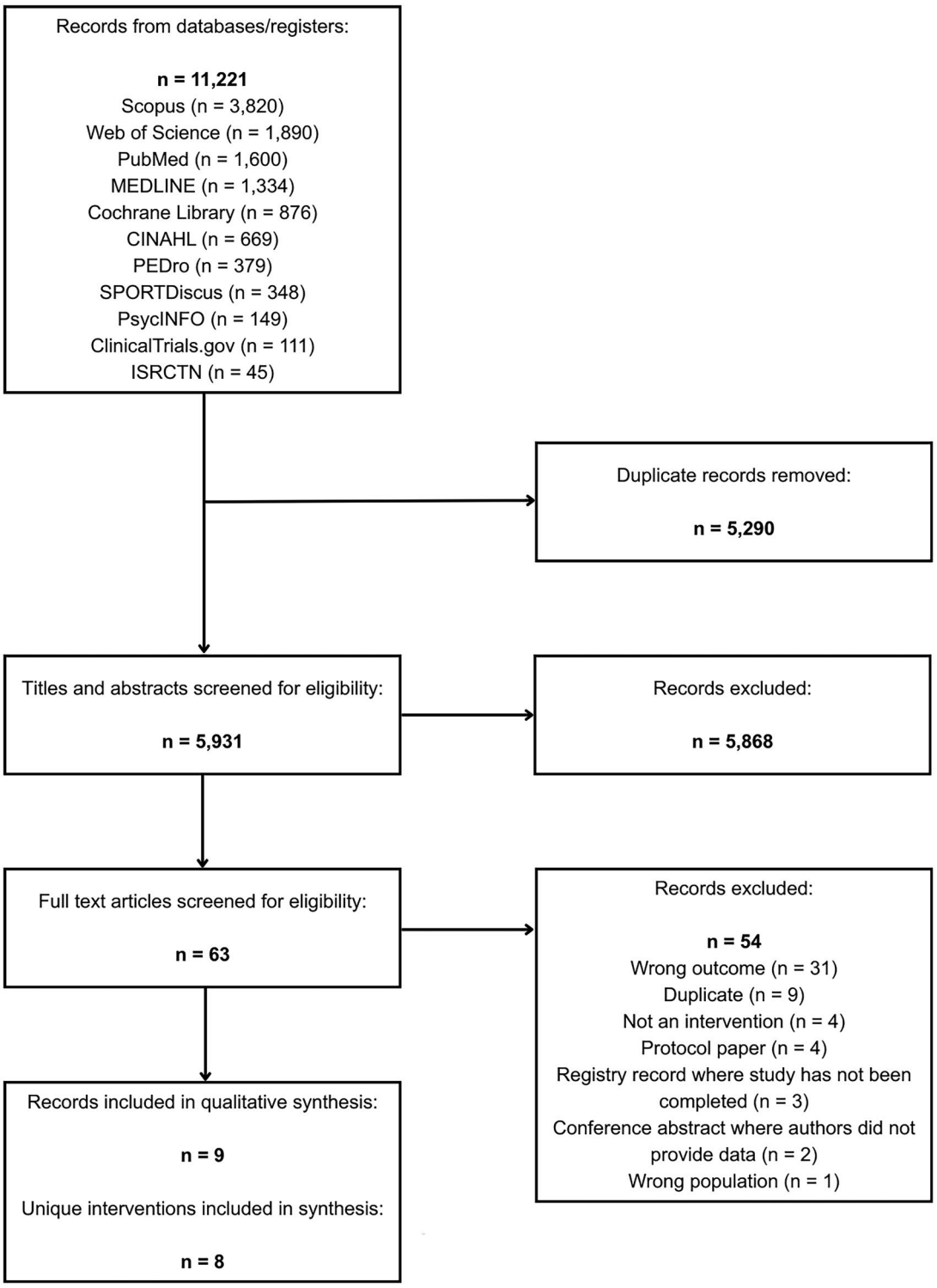

Figure 1. PRISMA flowchart for article identification.

Sample size ranged from 14 [39] to 168 [40] participants (Table 2). Four studies had a sample including only individuals with paraplegia [14,18,38,39]. Three studies included participants with paraplegia and tetraplegia, in which outcomes were reported for the whole sample [37,40,41]. None of the study sample sizes were powered a-priori to detect changes in sedentary behaviour. One study was powered a-priori to detect changes in physical activity [40], and one was underpowered for detecting changes in physical activity as the target sample size was not achieved [37]. Two studies were powered to detect changes in CVD biomarkers, namely postprandial glucose [39] and fasting insulin [17]. One study was

Table 2. Characteristics of included studies.

Study, country	Study design	Participants	Intervention and control characteristics	Intervention delivery mode and setting
Bailey et al. [39], United Kingdom.	Two-condition randomised controlled crossover design.	$N=14$ (8 F, 6 M). Mean age = 51 ± 9 years. Paraplegia between T6 - L5 (plus one participant with post-polio syndrome). Wheelchair user $N=9$. Ambulatory $N=5$.	Intervention: Participants performed moderate-intensity PA for 2 min every 20 min over 5.5 h using an arm ergometer. No behaviour change theory reported. Control: Participants remained seated and sedentary in a wheelchair over 5.5 h.	Controlled condition undertaken individually within University Sport and Exercise Science Laboratories.
Farrow et al. [18], United Kingdom.	Randomised controlled trial.	$N=27$ (14 F, 13 M). Mean age 46 ± 8 years. Paraplegia between T2 and L2. Self-reported use of a wheelchair for >75% of their waking day.	Intervention: Four intervention sessions per week for six weeks involving 10 × 60 s intervals at 80 - 90% of peak HR. Intensity increased by 5% every two weeks. No behaviour change theory reported Control: Participants were asked to maintain their habitual diet and physical activity routine.	Home-based exercise training undertaken individually.
Froehlich-Grobe et al. [40], United States.	Randomised, wait-list controlled trial.	$N=168$ (72 F, 96 M). Mean age 49.6 ± 12.3 years. Paraplegia $N=100$. Tetraplegia $N=66$. Not known $N=2$. Manual wheelchair user $N=104$. Power wheelchair user $N=60$. Scooter user $N=4$.	Intervention: 16-week programme providing participants (a) unlimited website access with exercise information, resources, and 16 skill-building modules; (b) virtual 60-minute, group-based 1x/week meetings, (c) unlimited access to a starter package of exercise equipment. The programme was founded upon Social Cognitive Theory and the Relapse Prevention model. Control: Participants underwent testing twice before being invited to participate in the intervention programme after a 4-month delay.	Home and community. Online educational resources and learning modules, group-based meetings, access to personal exercise equipment.
Martinez et al. [38], United States.	Randomised comparative effectiveness trial.	Sedentary behaviour intervention: $N=21$ (5 F, 16 M). Mean age 41 (range 27–57) years. Paraplegia between T2 and L3. All manual wheelchair users. Physical activity intervention: $N=28$ (5 F, 23 M). Mean age 41 (range 22–61) years. Paraplegia between T2 and L3. All manual wheelchair users.	Sedentary behaviour intervention: Designed to decrease sedentary time and increase overall PA by measuring and accumulating activity throughout the day. Given a wrist-worn activity monitor and phone app to view and track physical activity. Individualised goal-setting to progressively increase daily physical activity and decrease sedentary time and review of activity data with a physical therapist. Given home-based shoulder flexibility and strengthening exercises and recommendations for movement techniques that reduce shoulder demands associated with PA and daily activities. Physical activity intervention: Planned arm-crank ergometry. Individualised goal-setting to progressively increase daily physical activity and review of activity data with a physical therapist. Participants were asked to perform 3 × 15 min cycling sessions each week at 70% of maximum heart rate. Participants were encouraged to progressively increase session duration from 15 to 30 min between Weeks 2 and 4 and to 33 min by Week 5. Last, participants were instructed to exercise for 33 - 35 min per sessions, but at a higher intensity (target 85% of maximum heart rate) between week 5 and intervention end. Given home-based shoulder flexibility and strengthening exercises and recommendations for movement techniques that reduce shoulder demands associated with PA and daily activities.	Sedentary behaviour intervention: Home- and community-based. Physical activity intervention: Home- and community-based.

(*Continued*)

Table 2. Continued.

Study, country	Study design	Participants	Intervention and control characteristics	Intervention delivery mode and setting
Nightingale et al. [14, 17], United Kingdom.	Randomised controlled trial.	$N=24$ (9 F, 15 M). Mean age 47 ± 8 years. Participants had paraplegia below the T4 level. Those with an incomplete injury used a wheelchair >75% of their waking day.	Intervention: 4 × 45-minute moderate-intensity (60%–65% peak oxygen uptake) arm-crank exercise sessions per week for 6 weeks. No behaviour change theory reported. Control: Participants were asked to maintain their habitual physical activity behaviour.	Portable desktop arm-crank ergometer set up in their own home for individual exercise training.
Nooijen et al. [36, 37], Netherlands.	Single-blind, multicentre, randomised controlled trial.	$N=39$ (6 F, 33 M). Mean age 44 + 15 years. Paraplegia $N=26$. Tetraplegia $N=13$. All wheelchair users.	Intervention: Behavioural intervention promoting an active lifestyle after discharge. Intervention involved 13 individual 1-hour sessions delivered by a coach trained in motivational interviewing beginning 2 months before and ending 6 months after discharge from inpatient rehabilitation. Motivational interviewing was based on the transtheoretical model. Control: Participants in both groups received usual care, which included a handcycle training program and advice on physical activity after discharge	Specialised rehabilitation Centres administered the rehabilitation. Face-to-face, individual sessions with a coach were planned for intervention group. Some sessions after discharge were conducted remotely by telephone.
Piira et al. [41], Norway.	Two parallel independent single-blinded randomised controlled trials.	$N=37$ (14 F, 23 M). Mean age 50 ± 13 years. Paraplegia $n=20$. Tetraplegia $n=17$. Wheelchair-dependent for ambulation $N=24$.	Intervention: 60 training days of body weight supported locomotor training, either with manual or robotic assistance 60–90 min per day, 3–5 days per week over 6 months. Telephone follow-up secured compliance. No behaviour change theory reported. Control: Usual care, typically one-on-one, by their local physical therapists 1–3 times per week (range 0–5). Telephone follow-up secured compliance.	Body weight supported locomotor training individual exercise training programme, either with manual or robotic assistance. Two inpatient rehabilitation facilities and one outpatient clinic in Norway.

Notes: F, female; HR, heart rate; L, lumbar; M, male; PA, physical activity, T, thoracic.

underpowered to detect changes in fasting insulin due to an insufficient sample size [18], whilst the remaining three studies did not conduct power calculations for CVD biomarker outcomes [36,38,40].

Intervention characteristics

Of the eight interventions identified across the seven studies, six interventions targeted increases in physical activity [14,18,37,38,40,41], whilst two targeted sedentary behaviour [38,39]. Interventions varied from one day [39] to eight months [37] in duration. Three interventions were home-based exercise training protocols [14,18,38], one was a home-based online programme [40], one used an exercise training protocol within a rehabilitation centre [41], one used motivational interviewing within a rehabilitation centre [37], one involved a whole-day approach to replace sedentary behaviour with physical activity [38], and one involved supervised breaks in sedentary behaviour within a controlled laboratory setting [39]. Four interventions were explicitly informed by behaviour change theory, including Social Cognitive Theory and the Relapse Prevention Model [40], Brief Action Planning [38] and motivational interviewing based on the Transtheoretical Model [37].

Risk of bias

Risk of bias was consistent for both sedentary behaviour and CVD biomarker outcomes across studies. Therefore, risk of bias in each domain is presented as a single judgement for each study (Figure 2). With regards to bias arising from the randomisation process, six of seven studies were deemed low risk [14,18,37–39,41], with one deemed high risk as allocation sequence was not concealed [40]. For deviations from the intended intervention protocol, five studies had a low risk of bias [14,18,37–39] and two had a high risk due to concerns around participant adherence [40,41]. Five of seven studies had a low risk of bias arising from missing outcome data [14,18,39–41], one study raised some concerns as a result of unexplained missing data points [37], and one study was high risk due to missing data points [38]. Regarding measurement of outcome variables, five of seven studies were judged to have low risk of bias [14,18,37,39,40], while two studies were deemed high risk due to the findings being pooled from two separate trials undertaken in different settings [41] or the use of non-validated methods for outcome measurement [38]. Four studies were assessed as low risk of bias arising from the selection of the reported outcome variables [14,18,39,41], with the remaining three studies raising some concerns as they did not follow a pre-specified data analysis plan [37,38,40]. Overall bias was deemed low risk in three studies [14,18,39], high risk in three studies [38,40,41] and of some concern in one study [37].

Study outcomes

Sedentary behaviour outcomes

Of the seven interventions that included a measurement of sedentary behaviour (Table 3) [14,18,37,38,40,41], two led to improvements [38,40]. In one intervention where there was an improvement [40], self-reported daily sitting was the sedentary behaviour outcome, measured using the International Physical Activity Questionnaire (IPAQ) [42]. The other intervention that led to a reduction in sedentary behaviour used METs derived from heart rate zones [38]. The remaining interventions, in which sedentary behaviour was not reduced, employed accelerometry [14,18,37], heart rate zones [38] and the IPAQ [41].

Cardiovascular disease biomarker outcomes

Seven of eight interventions included a measurement of one or more CVD biomarkers. Thirty CVD biomarkers were assessed, with improvements in eight of these (systolic blood pressure, diastolic blood pressure, fasting insulin, postprandial glucose, insulin resistance, Matsuda insulin sensitivity index, total cholesterol and low-density lipoprotein [LDL] cholesterol) reported in five out of seven interventions (Table 3) [17,18,36,38,39]. In the other interventions that included CVD biomarker outcomes, there were no improvements [38,40].

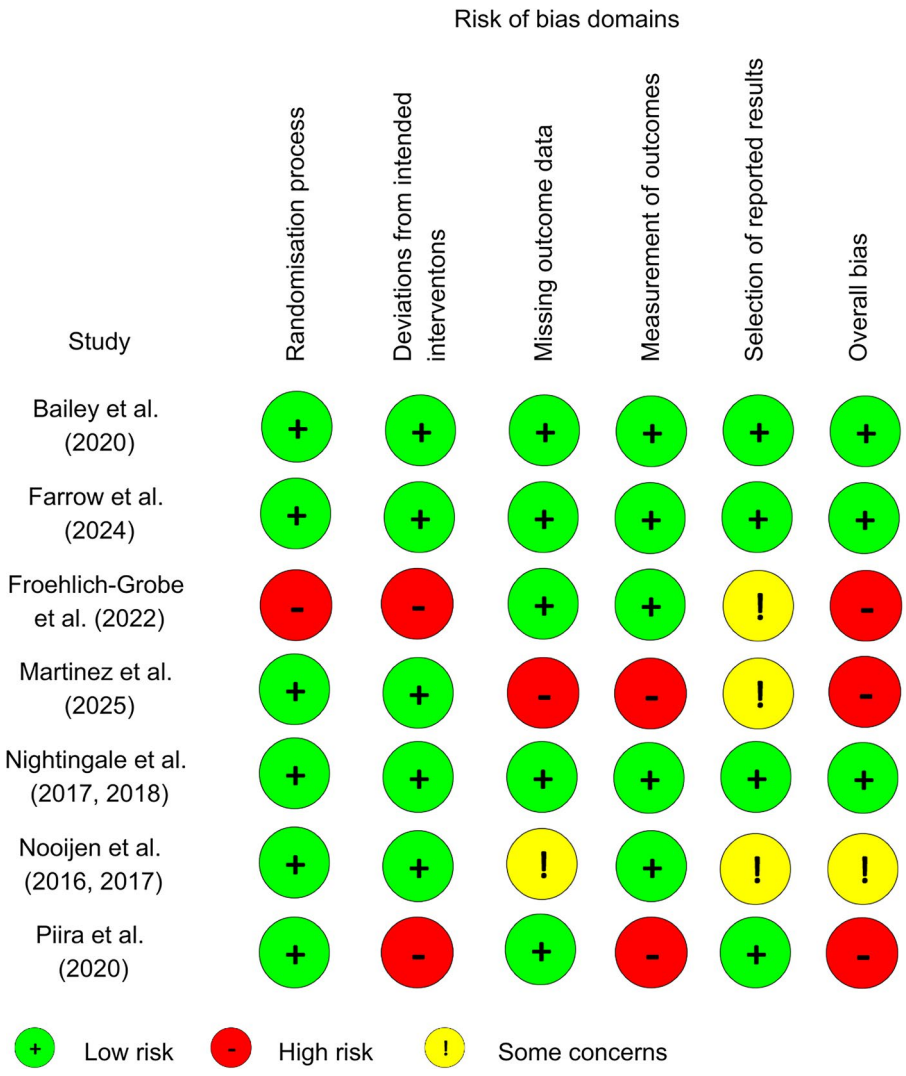

Figure 2. Risk of bias for included studies.

Nightingale et al. [13,16] reported an improvement in insulin resistance and fasting insulin, but with no reduction in sedentary behaviour. There were no improvements in these biomarkers in one other intervention that also did not reduce sedentary behaviour [18], but there was an improvement in insulin sensitivity [18]. Another intervention found no improvements in fasting insulin, insulin resistance or insulin sensitivity, despite reductions in sedentary behaviour [38].

In the two interventions where postprandial glucose was evaluated, one led to an improvement following supervised breaks in sedentary behaviour [39], whereas there was no change in postprandial glucose or sedentary behaviour in response to the other intervention [18]. One intervention led to an improvement in total cholesterol and LDL cholesterol [36], but with no reduction in sedentary behaviour [37]. Lipid outcomes did not change in response to one intervention that reduced sedentary behaviour [38], nor three interventions that did not affect sedentary behaviour [14,18,38].

Diastolic blood pressure was reduced in one study [36] in which the intervention did not affect sedentary behaviour [37]. Six other interventions had no effect on diastolic blood pressure [17,18,38–40], despite two of these reporting reductions in sedentary behaviour [38,40], and one involving supervised breaks in sedentary behaviour [39]. There was a reduction in systolic blood pressure and sedentary behaviour in one [38] of the seven interventions measuring this outcome [17,18,36,38–40].

Body composition outcomes (body mass, body fat, body mass index, waist circumference and waist to hip ratio) did not improve in any of the four interventions that assessed these outcomes [17,18,36,40].

Table 3. Outcomes of included studies.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Bailey et al. [39], United Kingdom.	N/A	N/A	Capillary blood samples at baseline, 30, 60, 90, 120, 180, 210, 240, 300, and 330 min. YSI analyser used to analyse blood glucose (mmol/L), Reflotron Plus used to measure triglycerides (mmol/L), ELISA used to measure insulin (μU/mL). Glucose, insulin and triglycerides were reported as total and incremental AUC. Blood pressure measured at baseline, 60, 120, 180, 240 and 300 min.	Data presented as mean (95% confidence intervals) for Intervention vs. Control *denotes significant difference between conditions ($p < 0.05$). **Total 5 h postprandial period:** • **Mean arterial pressure (**mmHg) 97.1 (96.7, 97.6) vs. 96.8 (96.4, 97.3) $p = 0.310$ • **Systolic blood pressure** (mmHg) 125.9 (121.5, 130.3) vs. 123.9 (119.4, 128.3) $p = 0.366$ • **Diastolic blood pressure** (mmHg) 76.6 (74.4, 78.9) vs. 76.8 (74.5, 79.0) $p = 0.934$ • **Glucose iAUC** (mmol/*L*·5.5 h) 5.1 (2.8, 7.4) vs. 6.5 (4.2, 8.8) $p = 0.275$ • **Glucose tAUC** (mmol/*L*·5.5 h) 33.7 (31.4, 36.0) vs. 35.1 (32.8, 37.4) $p = 0.276$ • **Insulin iAUC (**μU/mL·5.5 h) 217.1 (165.5, 268.8) vs. 202.9 (121.3, 284.5) $p = 0.753$ • **Insulin tAUC** (μU/mL·5.5 h) 285.4 (232.6, 338.1) vs. 262.7 (175.7, 349.8) $p = 0.980$ • **Triglycerides iAUC** (mmol/*L*·5.5 h) 3.5 (1.6, 5.4) vs. 2.1 (0.3, 4.0) $p = 0.194$ • **Triglycerides tAUC** (mmol/*L*·5.5 h) 16.2 (14.3, 18.1) 14.8 (13.0, 16.6) $p = 0.194$ **Breakfast postprandial period:** • **Glucose iAUC** (mmol/*L*·3 h) 4.9 (2.3, 7.5) vs. 5.0 (2.4, 7.6) $p = 0.905$ • **Glucose tAUC** (mmol/*L*·3 h) 19.8 (17.2, 22.4) vs. 20.0 (17.4, 22.6) $p = 0.905$ • **Insulin iAUC** (μU/mL·3 h) 129.0 (101.9, 156.3) vs. 115.8 (70.3, 161.3) $p = 0.594$ • **Insulin tAUC** (μU/mL·3 h) 164.6 (136.4, 192.8) vs. 147.0 (98.1, 195.9**)** $p = 0.509$ • **Triglycerides iAUC** (mmol/*L*·3 h) 0.8 (−0.2, 1.7) vs. −0.01 (−0.9, 0.9) $p = 0.172$ • **Triglycerides tAUC** (mmol/*L*·3 h) 7.4 (6.4, 8.3) vs. 6.6 (5.7, 7.5) $p = 0.172$ **Lunch postprandial period:** • ***Glucose iAUC** (mmol/*L*·2.5 h) 1.9 (1.0, 2.7) vs. 3.0 (2.1, 3.9) **(**$p = 0.015$, $f = 0.34$**)** • ***Glucose tAUC** (mmol/*L*·2.5 h) 15.3 (14.4, 16.1) vs. 16.4 (15.5, 17.2) **(**$p = 0.015$, $f = 0.34$) • **Insulin iAUC** (μU/mL·2.5 h) 38.0 (−8.9, 84.8) vs. 57.7 (10.8, 104.5) **(**$p = 0.122$) • **Insulin tAUC** (μU/mL·2.5 h) 128.5 (101.4, 155.5) vs. 127.7 (100.7, 154.7) **(**$p = 0.949$) • **Triglycerides iAUC** (mmol/*L*·2.5 h) 1.1 (0.23, 2.0) vs. 1.4 (0.6, 2.3) ($p = 0.482$) • **Triglycerides tAUC** (mmol/*L*·2.5 h) 8.3 (7.4, 9.2) vs. 8.6 (7.8, 9.5) ($p = 0.482$).
Farrow et al. [18], United Kingdom.	Participants wore a PA monitor (ActiheartTM) for 7-days after the baseline visit, and in the final week of the intervention period. Defined as <1.5 METs. Measured as min/day.	Data presented as mean (95% CI) for Intervention and Control groups pre- vs. post-intervention. **Sedentary time (min/day):** Intervention 642 (598, 687) vs. 687 (595, 780) Control 723 (628, 818) vs. 522 (404, 639) ($p = 0.040$)	Body mass was measured using platform wheelchair scales. DEXA scan was used to measure total fat mass (kg), total fat free mass (kg), and total body fat percentage. Supine length was measured using a non-elastic tape measure. Waist and hip circumferences were taken in duplicate, using a non-metallic tape measure. Resting blood pressure was measured in triplicate using an automated blood pressure monitor. Serum insulin, leptin and adiponectin were determined using ELISA. Plasma glucose and serum triglycerides, NEFA, total cholesterol, HDL cholesterol, LDL cholesterol concentrations were determined using an automated analyser. Insulin and glucose tAUC and iAUC were determined using the trapezoidal rule to characterise responses to the OGTT. The Matsuda C-ISI, HOMA2-IR and HOMA-ß were calculated to give fasting measures of insulin resistance, insulin sensitivity and pancreatic ß-cell function.	Data presented as means (95% CI) or percentage change (%) for Intervention and Control groups pre- vs. post-intervention. *denotes significant difference ($p < 0.05$). • ***Matsuda ISI:** Intervention 10.3 ± 29.7% Control −12.6 ± 24.8% ($p = 0.036$) • **Systolic blood pressure (mmHg):** Intervention 125 (120, 129) vs. 118 (114, 123) Control 114 (106, 123) vs. 117 (111, 124) ($p = 0.774$) • **Diastolic blood pressure (mmHg):** Intervention 81 (78, 84) vs. 78 (75, 81) Control 77 (72, 82) vs. 78 (74, 82) ($p = 0.942$) • **Body mass (kg):** Intervention 76.5 (67.7, 85.3) vs. 74.9 (74.1, 75.7) Control 75.5 (60.8, 90.2) vs. 70.6 (59.6, 81.7) ($p = 0.103$) • **BMI (kg/m²):** Intervention 26.6 (23.9, 29.3) vs. 26.1 (25.8, 26.3) Control 24.6 (21.6, 27.7) vs. 25.7 (25.2, 26.1) ($p = 0.115$) • **Waist circumference (cm):** Intervention 87.7 (79.7, 95.7) vs. 85.2 (83.8, 86.6) Control 83.8 (76.2, 91.4) vs. 84.8 (82.8, 86.8) ($p = 0.778$) • **Waist:hip ratio:** Intervention 0.85 (0.81, 0.89) vs. 0.85 (0.83, 0.86) Control 0.86 (0.81, 0.92) vs. 0.82 (0.79, 0.84) ($p = 0.089$) • **Fat mass (kg):** Intervention 30.2 (24.5, 36.0) vs. 29.4 (28.4, 30.3) Control 25.0 (19.7, 30.4) vs. 28.5 (27.1, 29.8) ($p = 0.286$) • **Body fat (%):** Intervention 39.5 (34.7, 44.2) vs. 39.3 (38.3, 40.3) Control 36.4 (29.9, 42.9) vs. 38.5 (37.0, 40.0) ($p = 0.365$)

(*Continued*)

Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
				• **Fasting glucose** (mmol/L) Intervention 5.52 (4.23, 6.80) vs. 5.73 (5.27, 6.19) Control 4.77 (4.14, 5.40) vs. 5.80 (5.19, 6.41) ($p = 0.849$) • **HOMA2-IR:** Intervention 1.07 (0.54, 1.60) vs. 0.85 (0.70, 1.00) Control 0.71 (0.49, 0.94) vs. 1.00 (0.80, 1.21) ($p = 0.224$) • **Glucose iAUC** (mmol/L x 120 min): Intervention 433 (320, 545) vs. 411 (319, 503) Control 350 (192, 508) vs. 383 (221, 545) ($p = 0.765$) • **Glucose tAUC** (mmol/L x 120 min): Intervention 1096 (859, 1333) vs. 1053 (982, 1123) Control 908 (787, 1029) vs. 1028 (903, 1153) ($p = 0.728$) • **Insulin iAUC** (pmol/L x 120 min): Intervention 44.5 (27.1, 61.9) vs. 48.1 (40.7, 55.5) Control 69.4 (−5.4, 144.3) vs. 57.1 (44.0, 70.1) ($p = 0.242$) • **Insulin tAUC** (pmol/L x 120 min): Intervention 51.2 (33.8, 68.6) vs. 53.3 (45.7, 60.9) Control 74.9 (−1.60, 151.3) vs. 63.3 (51.0, 77.7) ($p = 0.164$) • **NEFA** (mmol/L): Intervention 0.61 (0.47, 0.76) vs. 0.56 (0.46, 0.66) Control 0.58 (0.35, 0.81) vs. 0.55 (0.43, 0.69) ($p = 0.953$) • **Leptin** (µg/L): Intervention 10.7 (6.0, 15.4) vs. 15.0 (11.3, 16.9) Control 11.4 (6.4, 16.3) vs. 11.1 (5.3, 16.9) ($p = 0.264$) • **Adiponectin** (µg/L): Intervention 9.21 (6.89–11.5) 9.58 (8.69–10.5) Control 10.6 (4.91–16.3) 10.1 (8.88–11.3) ($p = 0.491$) **Fasting insulin** (pmol/L): • **($p = 0.415$)** **Total cholesterol (mmol/L):** Intervention 4 5.03 (4.51, 5.56) vs. 5.47 (5.16, 5.77) Control 5.47 (4.73, 6.21) vs. 5.13 (4.72, 5.54) ($p = 0.193$) • **HDL cholesterol (mmol/L):** Intervention 1.12 (0.91, 1.32) vs. 1.30 (1.21, 1.39) Control 1.27 (0.99, 1.54) vs. 1.23 (1.11, 1.35) ($p = 0.329$) • **LDL cholesterol (mmol/L):** Intervention 3.46 (3.00, 3.92) vs. 3.71 (3.38, 4.05) Control 3.65 (2.85, 4.44) vs. 3.33 (2.88, 3.78) ($p = 0.173$) • **Triglycerides (mmol/L):** Intervention 1.13 (0.80, 1.46) vs. 1.04 (0.87, 1.21) Control 1.20 (0.70, 1.70) vs. 1.25 (1.02, 1.48) ($p = 0.142$)

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Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Froehlich-Grobe et al. [40], United States.	IPAQ-SF question to assess sitting time (hours/day).	**Wait-list control:** Data presented as mean ± SD for Intervention and wait-list control combined pre-post results between 0 m vs. 4 m, and 0 m vs. 6 m. *denotes significant group x time interaction effect. ***Sit time (min/day):** 0 m 616.22 ± 229.19 vs. 4 m 567.05 ± 264.86 ($p = 0.076$) 0 m 616.22 ± 229.19 vs. 6 m 555.13 ± 252.28 ($p = 0.017$)	Resting blood pressure (mmHg) and heart rate were obtained before (0 months) and after (4 month) the exercise intervention. Body weight (kg) measured using a digital wheelchair scale.	**RCT:** Data presented as means ± SD for Intervention and Control groups pre- vs. post-intervention • **Systolic blood pressure (mmHg):** Intervention 114.1 ± 19.6 vs. 115.5 ± 18.0 Control 109.2 ± 21.5 vs. 114.4 ± 20.1 Group x time interaction $p = 0.374$ • **Diastolic blood pressure (mmHg):** Intervention 73.7 ± 12.8 vs. 71.8 ± 12.9 Control 73.2 ± 11.2 vs. 71.3 ± 11.3 Group x time interaction $p = 0.644$ • **Body weight (kg):** Intervention 86.2 ± 22.0 vs. 85.2 ± 23.8 kg Control 81.6 ± 20.8 vs. 81 ± 21.8 kg Group x time interaction $p = 0.563$ • **BMI (kg/m²):** Intervention 28.3 ± 6.2 vs. 28.4 ± 7.2 Control 27.2 ± 6.5 vs. 26.7 ± 6.9 Group x time interaction $p = 0.104$ **Wait-list control:** Data presented as means ± SD for Intervention and wait-list control combined pre-post results between 0 m vs. 4 m. • **Systolic blood pressure (mmHg):** 0 m 112.4 ± 20.3 vs. 4 m 115.0 ± 18.7 ($p = 0.706$) • **Diastolic blood pressure (mmHg):** 0 m 73.5 ± 12.1 vs. 4 m 71.6 ± 12.0 ($p = 0.125$) • **Body weight (kg):** 0 m 84.6 ± 21.5 vs. 4 m 83.3 ± 22.7 ($p = 0.722$) • **BMI (kg/m²):** 0 m 27.9 ± 6.3 vs. 4 m 27.6 ± 7.0 ($p = 0.475$)

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Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Martinez et al. [38], United States.	Participants wore a Fitbit device on the wrist over 7 days to measure baseline sedentary behaviour in min/day based on heart rate zones (1 MET for > 10 min in duration). The same device was worn throughout the whole intervention, with the average daily sedentary behaviour over the final month used to determine post-intervention values.	Data presented as Δ mean difference (95% CI) for pre- vs post-intervention. * denotes significant difference. **Sedentary behaviour intervention:** ***Sedentary behaviour (min/day):** Intervention A Δ −118 (−222 to −14[a]) **Physical activity intervention:** **Sedentary behaviour (min/day):** Intervention B Δ −47 (−109 to 15[a]) Between group difference in change p = n.s.	Diastolic and systolic blood pressure (mmHg) were measured after a 6-minute push test. Fasted blood samples were taken to measure glucose, insulin, HOMA-%B, HOMA-%S, HOMA-IR, lipid profile and triglycerides (mg/dL).	Data presented as Δ mean difference (95% CI) for diastolic blood pressure and median (interquartile range) for triglycerides, pre- vs. post-intervention. *denotes significant difference. **Sedentary behaviour intervention:** • ***Diastolic blood pressure (mmHg):** Intervention Δ −6 (−11 to −1a) Between group difference in change p = 0.019 • **Systolic blood pressure (mmHg):** Between group difference in change p > 0.05. • **Glucose (mg/dL):** Between group difference in change p > 0.05 • **Insulin (mg/dL):** Between group difference in change p > 0.05 • **HOMA-%B:** Between group difference in change p > 0.05 • **HOMA-%S:** Between group difference in change p > 0.05 • **HOMA-IR:** Between group difference in change p > 0.05 • **Lipid profile:** Between group difference in change p > 0.05 • **Triglycerides (mg/dL):** Intervention Δ 7 (7, 46) Between group difference in change p = 0.092 **Physical activity intervention:** • **Diastolic blood pressure (mmHg):** Intervention Δ 2 (−3 to 7a) Between group difference in change p = 0.019 • **Systolic blood pressure (mmHg):** Between group difference in change p > 0.05. • **Glucose (mg/dL):** Between group difference in change p > 0.05 • **Insulin (mg/dL):** Between group difference in change p > 0.05 • **HOMA-%B:** Between group difference in change p > 0.05 • **HOMA-%S:** Between group difference in change p > 0.05 • **HOMA-IR:** Between group difference in change p > 0.05 • **Lipid profile:** Between group difference in change p > 0.05 • **Triglycerides (mg/dL):** Intervention Δ −5 (−29, 9) Between group difference in change p = 0.092

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Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Nightingale et al. [14,17], United Kingdom.	Participants wore a chest-mounted Actiheart device to measure sedentary behaviour (< 1.5 METs) in min/day.	Data presented as means ± SD for Intervention and Control groups pre- vs. post-intervention. *denotes significant difference. **Sedentary time (min/day):** Intervention: 1232 ± 118 vs. 1179 ± 124 (Δ − 53, −126 to 20[a]) Control: 1220 ± 115 vs. 1191 ± 139 (Δ − 29, −136 to 78[a])	Blood pressure (mmHg), supine height (m), body mass, fat mass and lean mass (kg) were measured fasted OGTT to measure metabolic regulation, glucose, insulin (mmol/L), post-load glucose, post-load insulin (mmol/120 min/L), NEFA, triacylglycerol, HOMA-2ß (%), HOMA2-IR, Matsuda C-ISI and total cholesterol, HDL cholesterol and LDL cholesterol (mmol/L).	Data presented as Δ mean difference (95% CI) for Intervention and Control groups pre- vs. post-intervention. *denotes significant difference ($p < 0.05$). • ***Insulin (pmol/L):** Intervention Δ −12.7 (−24.0, −1.4) ($p = 0.031$) Control Δ 3.1 (−5.9, 12.0) (N.S.) Between group difference in change $p \leq 0.044$ • ***HOMA2-IR:** Intervention Δ −0.24 (−0.45, −0.02) ($p < 0.035$) Control Δ 0.06 (−0.10, 0.23) (N.S.) Between group difference in change $p \leq 0.044$ • **Systolic blood pressure (mmHg):** Intervention Δ −3 (−10, 5) Control Δ −2 (−8, 2) • **Diastolic blood pressure (mmHg):** Intervention Δ −1 (−8, 6) Control Δ −4 (−9, 2) • **Body mass (kg):** Intervention Δ −1.1 (−2.1, −0.0) Control Δ −0.7 (−2.2, 1.0) Between group difference in change $p = 0.6$ • **Fat mass (kg):** Intervention Δ −0.6 (−1.4, 0.2) Control Δ −0.0 (−0.7, 0.7) • **Glucose (mmol/L):** Intervention Δ 0.0 (−0.2, 0.2) Control Δ 0.0 (−0.2, 0.2) Group x time interaction $p \geq 0.3$ • **HOMA2-ß (%):** Intervention Δ −14 (−26, −2) Control Δ 1 (−10, 13) Group x time interaction $p = 0.066$ • **Matsuda ISI:** Intervention Δ 0.3 (−0.7, 1.2) Control Δ −0.7 (−2.6, 1.2) Group x time interaction $p \geq 0.3$ • **Glucose response (mmol/L·120 min):** Intervention Δ 19 (−48, 86) Control Δ −25 (−153, 104) Group x time interaction $p \geq 0.3$ • **Insulin response (nmol/L·5.5 h):** Intervention Δ −4.4 (−19.0, 10.2) Control Δ 2.2 (−11.6, 16.0) Group x time interaction $p \geq 0.3$ • **NEFA (mmol/L):** Intervention Δ 0.3 (−0.2, 0.8) Control Δ −0.1 (−0.8, 0.6) Group x time interaction $p \geq 0.3$ • **Triacylglycerol (mmol/L):** Intervention Δ −0.1 (−0.2, 0.1) Control Δ 0.5 (−2.0, 1.2) Group x time interaction $p = 0.054$ • **Total cholesterol (mmol/L):** Intervention Δ −0.1 (−0.5, 0.4) Control Δ 0.1 (−0.5, 0.5) Group x time interaction $p \geq 0.3$ • **HDL cholesterol (mmol/L):** Intervention Δ 0.1 (−0.1, 0.1) Control Δ −0.0 (−0.1, 0.1) Group x time interaction $p \geq 0.3$ • **LDL cholesterol (mmol/L):** Intervention Δ −0.0 (−0.4, 0.3) Control Δ −0.2 (−0.6, 0.2) Group x time interaction $p \geq 0.3$

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Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Nooijen et al. [36,37], Netherlands.	Body-fixed accelerometers measured the total duration of sedentary daytime bouts longer than 30 min. Defined as sitting and lying during the day without interruption by physical activity for a minimum of 5 s (min/day).	Data presented as mean difference (95% CI) in change from baseline to discharge, baseline to month 6, baseline to month 12 and overall change between Intervention and Control groups. Means are adjusted for rehabilitation centre, gender and age. **Sedentary daytime (min/day):** Baseline to discharge: −14 (−69, 40) Baseline to 6 m: −50 (−134, 33) Baseline to 12 m: −21 (−119, 77) Overall intervention vs. control: −34 (−97, 29)	BMI (kg/m^2) was calculated from height (m) and body mass (kg). Resting diastolic and systolic blood pressure (mmHg) were measured by a physician. Fasting blood samples were taken for total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides and glucose (mmol/L). Comparisons are "baseline vs. discharge," "baseline vs. month 6," and "baseline vs. month 12."	Data presented as mean ± SD for Intervention vs. Control groups at each timepoint: baseline, discharge, month 6 and month 12. P values are adjusted for rehabilitation centre, sex and age. *denotes significant between-group effect ($p < 0.05$). • ***Diastolic blood pressure (mmHg):** Baseline: 72 ± 9 vs. 77 ± 13 Discharge: 73 ± 9 vs. 77 ± 8 ($p = 0.52$) 6 m: 74 ± 13 vs. 84 ± 11 ($p = 0.04$) 12 m: 74 ± 12 vs. 83 ± 18 ($p = 0.01$) Overall model: ($p = 0.02$) • ***Total cholesterol (mmol/L):** Baseline: 4.47 ± 0.84 vs. 4.96 ± 1.19 Discharge: 4.47 ± 0.92 vs. 5.17 ± 1.00 ($p = 0.39$) 6 m: 4.63 ± 0.85 vs. 5.55 ± 1.29 ($p = 0.17$) 12 m: 4.17 ± 0.51 vs. 5.21 ± 0.83 ($p = 0.01$) Overall model ($p = 0.06$) • ***LDL cholesterol (mmol/L):** Baseline: 2.76 ± 0.85 vs. 3.22 ± 0.91 Discharge: 2.63 ± 0.73 vs. 3.39 ± 0.86 ($p = 0.34$) 6 m: 2.95 ± 0.54 vs. 3.46 ± 1.10 ($p = 0.40$) 12 m: 2.46 + 0.75 vs. 3.13 ± 0.63 ($p = 0.05$) Overall model ($p = 0.08$) • **Systolic blood pressure (mmHg):** Baseline: 123 ± 19 vs. 127 ± 21 Discharge: 120 ± 15 vs. 124 ± 14 ($p = 0.75$) 6 m: 128 ± 28 vs. 132 ± 14 ($p = 0.62$) 12 m: 125 ± 19 vs. 130 ± 18 ($p = 0.36$) Overall model ($p = 0.46$) • **BMI (kg/m^2):** Baseline: 25.43 ± 5.23 vs. 23.90 ± 4.68 Discharge: 25.60 ± 5.56 vs. 24.60 ± 5.18 ($p = 0.56$) 6 m: 25.66 ± 5.53 vs. 26.00 ± 5.53 ($p = 0.41$) 12 m: 25.36 ± 5.59 vs. 27.13 ± 5.20 ($p = 0.36$) Overall model ($p = 0.29$) • **Glucose (mmol/L):** Baseline: 4.97 ± 0.69 vs. 5.52 ± 2.20 Discharge: 5.16 ± 1.28 vs. 6.61 ± 3.02 ($p = 0.06$) 6 m: 5.00 ± 0.57 vs. 6.25 ± 1.27 ($p = 0.58$) 12 m: 5.66 ± 1.93 vs. 7.13 ± 3.55 ($p = 0.38$) Overall model ($p = 0.05$) • **HDL cholesterol (mmol/L):** Baseline: 1.37 ± 1.21 vs. 1.23 ± 1.02 Discharge: 1.02 ± 0.31 vs. 1.01 ± 0.15 ($p = 0.61$) 6 m: 1.08 ± 0.48 vs. 0.97 ± 0.15 ($p = 0.80$) 12 m: 1.09 ± 0.31 vs. 1.04 ± 0.31 ($p = 0.23$) Overall model ($p = 0.62$) • **Triglycerides (mmol/L):** Baseline: 1.40 ± 0.76 vs. 1.84 ± 0.93 Discharge: 1.50 ± 0.97 vs. 1.93 ± 0.93 ($p = 0.71$) 6 m: 1.80 ± 1.71 vs. 2.24 ± 1.23 ($p = 0.17$) 12 m: 1.12 ± 0.65 vs. 2.37 ± 1.38 ($p = 0.36$) Overall model ($p = 0.71$)

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Table 3. Continued.

Study, country	Sedentary behaviour outcome measures	Sedentary behaviour outcomes	Cardiovascular disease biomarker outcome measures	Cardiovascular disease biomarker outcomes
Piira et al. [41], Norway.	Measured using the IPAQ-SF questionnaire (with no adaptation for wheelchair dependent individuals), reported in min/day.	Data presented as means ± SD for Intervention and Control groups pre- vs. post-intervention. * denotes significant difference. **Sitting time (min/day):** Intervention: 553.1 ± 265.4 vs. 457.9 ± 292.1 (Δ −95.2, −244.2 to 53.8[a]) Control: 554.3 ± 323.6 vs. 504.0 ± 229.0 (Δ −50.3, −182.5 to 81.9[a])	N/A	N/A

BMI, body mass index; CI, confidence interval; C-ISI, composite insulin sensitivity index; DEXA, Dual-energy X-ray absorptiometry; ELISA, enzyme-linked immunoassay; HDL, high density lipoprotein; HOMA-ß, homeostatic model assessment for ß cell function; HOMA-IR, homeostatic model assessment for insulin resistance; HOMA-%S, homeostatic model assessment for insulin sensitivity; iAUC, incremental area under the curve; IPAQ, international physical activity questionnaire; LDL, low density lipoprotein; MET, metabolic equivalent of task; NEFA, non-esterified fatty acids; N.S., non-significant; OGTT, oral glucose tolerance test; PA, physical activity; QUICKI, quantitative insulin sensitivity check index; RCT, randomised controlled trial; SD, standard deviation; tAUC, total area under the curve.

[a]Within-group 95% confidence interval estimated from study data to determine statistical significance.

Outcomes in the context of study population

In the two interventions that led to reductions in sedentary behaviour, one included participants with paraplegia and tetraplegia [40], whereas the other included participants with paraplegia only [38]. Of the five interventions that had no effect on sedentary behaviour, two included participants with paraplegia and tetraplegia [37,41], and three included paraplegia only [14,18,38]. Overall, the proportion of individuals with paraplegia in the study sample did not appear to influence sedentary behaviour outcomes.

Four interventions that led to improvements in CVD biomarkers included only individuals with paraplegia [17,18,38,39], whilst one studied a sample comprising individuals with paraplegia and tetraplegia [36]. Of the two interventions that did not improve CVD biomarkers, one included only individuals with paraplegia [38] and the other included both people with paraplegia and tetraplegia [40]. Intervention effects, therefore, appeared to be more consistent in studies that included only participants with paraplegia.

Outcomes in the context of intervention characteristics

Targeting physical activity or sedentary behaviour. Of the two interventions that reduced sedentary behaviour, one targeted reductions in sedentary behaviour *via* a whole-day approach [38], whilst the other targeted increased physical activity using an online programme [40]. The five other interventions that targeted increases in physical activity (four using structured exercise training and one using motivational interviewing) found no effect [14,18,37,38,41]. The remaining intervention included supervised breaks in sedentary behaviour and, therefore, reduced sedentary time but did not report sedentary behaviour as an outcome [39]. It appears that interventions targeting physical activity are not effective for reducing sedentary behaviour in participants with SCI.

Three of the five interventions that improved CVD biomarkers targeted increases in physical activity, but did not reduce sedentary behaviour [17,18,37]. Two interventions that reported biomarker improvements targeted sedentary behaviour [38,39]; these studies led to reduced sedentary behaviour [38] or were supervised breaks in sedentary behaviour [39]. The two interventions that did not improve any CVD biomarker both targeted physical activity [38,40]; one reduced sedentary behaviour [40] and the other did not [38]. One intervention that targeted physical activity did not include any CVD biomarker outcomes [41]. There appears to be some evidence for CVD biomarkers being improved in interventions that target sedentary behaviour.

Intervention duration. The two interventions that led to reductions in sedentary behaviour were each 16 weeks [38,40] in duration. The interventions that had no effect on sedentary behaviour were six weeks [14,18], sixteen weeks [38], six months [41] and eight months [37] in duration. It is unclear whether intervention duration affects sedentary behaviour outcomes.

One of the five interventions that improved CVD biomarkers lasted one day [39], two lasted six weeks [17,18], one lasted one lasted sixteen weeks [38] and one lasted eight months [36] (Table 2). The two interventions that had no effect on any CVD biomarker were 16 weeks in duration [38,40]. It is unclear whether intervention duration affects CVD biomarker outcomes.

Intervention setting and delivery mode. One of the two interventions that reduced sedentary behaviour was a home-based online programme [40] and one was a home- and community-based intervention targeting the whole day [38]. Three of the five interventions that did not reduce sedentary behaviour were home- and community-based exercise training protocols [14,18,38], whilst the others included motivational interviewing within a rehabilitation centre [37] and a structured exercise training protocol in a rehabilitation centre [41]. It appears that structured exercise training protocols are not effective for reducing sedentary behaviour.

Two of the five interventions that improved CVD biomarkers included home-based exercise training protocols [17,18], one involved a home- and community-based intervention targeting the whole day [38], one involved motivational interviewing within a rehabilitation centre [36] and one involved supervised breaks in sedentary behaviour in a controlled laboratory setting [39]. The two interventions that did not improve CVD biomarkers included a home-based online programme [40] and a home- and community-based structured exercise training protocol [38]. Cardiovascular disease biomarker outcomes appear to be improved across a range of intervention settings and delivery modes.

Use of behaviour change theory. Four interventions were either underpinned by, or employed, behaviour change theory [37,38,40]. There was no mention of behaviour change theory in the other four

interventions [14,18,39,41]. Both of the interventions that reduced sedentary behaviour utilised behaviour change theory [38,40]. Two of the five interventions that did not affect sedentary behaviour utilised behaviour change theory [37,38]. It is not clear whether the use of behaviour change theory was beneficial to sedentary behaviour outcomes.

Two of the five interventions that led to improvements in CVD biomarkers were informed by behaviour change theory [36,38], whereas the remaining three interventions were not [17,18,39]. Behaviour change theory was included in both interventions that had no effect on CVD biomarkers [38,40]. It is not clear whether use of behaviour change theory influenced CVD biomarker outcomes.

Outcomes in context of measurement methods

One of the two interventions that reduced sedentary behaviour measured this outcome *via* self-report (Table 3) [40]. Metabolic equivalent of task derived from heart rate zones was used in the other intervention that reduced sedentary behaviour [38]. Of the five interventions that had no effect on sedentary behaviour, three measured this outcome using accelerometry [14,18,37], one used the IPAQ [41] and one used heart rate zones [38]. Improvements in sedentary behaviour appear to occur less consistently when measured *via* accelerometry.

One of the five interventions that improved CVD biomarkers found improvements in fasted outcome measures (insulin and insulin resistance), despite also measuring postprandial outcomes [17]. Another study only measured biomarkers in a fasted state, with the intervention leading to improvements in diastolic blood pressure, LDL cholesterol and total cholesterol [37]. One intervention reported improvements in postprandial glucose [39], whilst another reported improvement in the Matsuda index, which is measured using fasted and postprandial measurements [18]. One intervention led to improvements in systolic blood pressure [38], but blood pressure was measured after a six-minute push test, not at rest [38]. Other CVD biomarkers were assessed similarly across studies, making it challenging to recognise differences in outcomes according to the method of measurement. In summary, it appears that the method of measurement does not affect CVD biomarker outcomes.

Outcomes in context of risk of bias

Both interventions that led to reductions in sedentary behaviour were in studies with high risk of bias (Figure 2) [38,40]. Two of the five interventions that did not affect sedentary behaviour were in a study with low risk of bias [14,18], two were in studies with high risk of bias [38,41], and one was in a study that raised some concerns [37]. It appears that sedentary behaviour reductions were reported more frequently in studies with high risk of bias.

Three of the five interventions that improved CVD biomarkers were in studies with low risk of bias [17,18,39], one with high risk of bias [38] and one that raised some concerns [36]. The interventions that did not report an improvement in any CVD biomarker were in studies with high risk of bias [38,40]. CVD biomarker outcomes were, therefore, improved more consistently in studies with low risk of bias.

Quality of evidence

Overall quality of evidence for sedentary behaviour was deemed very low (Table 4), with quality downgraded due to risk of bias, inconsistency of results, indirectness of evidence and imprecision. Overall quality of evidence for CVD biomarkers was deemed moderate, with quality downgraded due to risk of bias and inadequate sample sizes.

Discussion

The findings of this review indicate that interventions targeting increases in physical activity are not effective for reducing sedentary behaviour in individuals with paraplegia, but show some effectiveness for improving CVD biomarkers. There was a scarcity of interventions targeting sedentary behaviour, but these interventions may have potential for improving CVD biomarkers.

The majority of interventions targeted physical activity, as opposed to sedentary behaviour, with only one leading to an improvement in the sedentary behaviour outcome. These findings suggests that

Table 4. Assessment of overall quality of the evidence.

Outcome		Risk of bias	Inconsistency of results	Indirectness of evidence	Imprecision	Publication bias[f]	Quality of evidence
Sedentary behaviour	Overall	−2[ab]	−1[c]	−1[d]	−1[e]	0	0 – very low
	Device-measured	0	−1[c]	0	−1[e]	0	3 – moderate
	Estimated from heart rate zone	−1[b]	0	−1[d]	−1[e]	0	2 – low
	Self-reported	−2[ab]	0	−1[d]	−1[e]	0	1 – very low
Cardiovascular disease biomarkers	Overall	−1[b]	0	0	−1[e]	0	3 – moderate
	Blood pressure	−1[b]	0	0	−1[e]	0	3 – moderate
	Body composition	−1[b]	0	0	−1[e]	0	3 – moderate
	Glycaemic biomarkers	−1[b]	0	0	−1[e]	0	3 – moderate
	Lipid biomarkers	−1[b]	0	0	−1[e]	0	3 – moderate

[a]One study does not use a randomised controlled design for this outcome.
[b]One or more study has a high risk of bias.
[c]Large differences in means between studies.
[d]One or more study uses surrogate measurements.
[e]One or more study has an inadequate sample size to ensure sufficient statistical power.
[f]Funnel plots not generated as < 10 studies included in the systematic review.

physical activity interventions may not be effective for reducing sedentary behaviour in individuals with paraplegia. This is in disagreement with a meta-analysis that found interventions targeting physical activity were effective for reducing sedentary behaviour in non-disabled individuals [26]. The contrasting findings may be due to the majority of physical activity interventions in the present review focusing on structured exercise training, rather than non-exercise physical activity accumulated throughout the day [26], which is more likely to displace sedentary behaviour [22] and overcome physical impairments that may present a barrier to some exercise interventions in individuals with disabilities [28]. The intervention in the present review that targeted sedentary behaviour *via* a whole-day approach and measured sedentary behaviour as an outcome was found to be effective [38]. It could be postulated that interventions targeting sedentary behaviour would be most effective as interventions focusing on physical activity may not utilise the most appropriate behaviour change techniques for sedentary behaviour [43] or appropriate activities for individuals with paraplegia. Further interventions targeting sedentary behaviour, tailored for individuals with paraplegia, require development and evaluation in order to determine their effectiveness and inform public health and clinical care guidelines.

The quality of evidence in the present review was deemed very low and, as a result, there is very little confidence with respect to sedentary behaviour outcomes [32]. The lack of quality is due to unexplained variability in sedentary behaviour changes across studies, small sample sizes and the measurement techniques employed [44]. Sedentary behaviour was assessed *via* self-report using the IPAQ "time spent sitting" question in one of the two interventions that reduced sedentary behaviour. The validity of this IPAQ question has not been evaluated in individuals with SCI and may not be appropriate for wheelchair-users. One intervention that reduced sedentary behaviour measured this outcome using METs derived from heart rate zones, which is not validated [38]. In addition, sample heterogeneity was high, with just one intervention that reduced sedentary behaviour including a sample comprising only individuals with paraplegia. Studies may choose to adopt broad inclusion criteria, such as including individuals with paraplegia and tetraplegia in their sample, due to difficulty recruiting and retaining individuals with SCI [45]. As a result, the effects on sedentary behaviour cannot be isolated to individuals with paraplegia. The findings with respect to interventions targeting sedentary behaviour potentially being effective may, therefore, be generalisable to paraplegia and tetraplegia. However, differences in upper-limb function between individuals with paraplegia and tetraplegia means that the types of physical activities they are able to engage in differ. Interventions will, therefore, need to be tailored to different injury levels. High-quality studies that address the sources of bias and heterogeneity identified in this review are needed to inform definitive conclusions regarding sedentary behaviour intervention effectiveness.

The influence of behaviour change theory on sedentary behaviour outcomes is unclear. However, only half of included interventions employed behaviour change theory [37,38,40]. Limited use of behaviour change theory has been reported in reviews of sedentary behaviour interventions in non-disabled individuals [46,47]. The lack of behaviour change theory is partly due to the nature of some included interventions, with half employing prescribed exercise training protocols in which behaviour change theory may have limited added value [14,18,38,41]. Integration of theory within interventions may be important to support behaviour change *via* identification of precursors to behaviour and causal factors of change,

which can be selectively targeted with specific behaviour change techniques [48,49]. Indeed, interventions grounded in behaviour change theory yield greater improvements in physical activity in individuals with physical disabilities, such as SCI [50]. Time spent in sedentary behaviour was more strongly correlated with engagement in light physical activity and activities of daily living than with MVPA and structured exercise [51], which could explain why sedentary behaviour was unaffected by the exercise training interventions in the current review. Therefore, the effects of sedentary behaviour interventions grounded in behaviour change theory, that utilise strategies to increase light physical activity and activities of daily living, should be evaluated in individuals with paraplegia as they are likely to be more effective.

There were mixed effects for CVD biomarkers in response to interventions that reduced sedentary behaviour. Interventions that reduced sedentary behaviour or involved supervised breaks in sedentary behaviour led to improvements in systolic blood pressure and postprandial glucose [38,39]. Previous reviews in non-disabled individuals [27] and clinical populations [52] also found that reducing sedentary behaviour improved CVD biomarkers. However, there was no change in sedentary behaviour in three interventions that improved CVD biomarkers in the present review [17,18,36]. This may indicate that improvements in cardiovascular health in these studies were due to increases in physical activity [14,18,37]. A previous meta-analysis found that physical activity interventions improved CVD biomarkers in individuals with SCI [53], supporting their inclusion in healthcare for this population group. Two interventions reduced sedentary behaviour and increased physical activity in the present review [38,40], but only one of these led to improvements in CVD biomarkers [38]. A combination of changes in sedentary behaviour and physical activity may, therefore, not always be necessary to achieve optimal effects [54]. Targeting physical activity or sedentary behaviour separately may, therefore, yield cardiovascular benefits; this supports literature demonstrating that these are distinct behaviours related to CVD risk [24,25]. The quality of evidence for CVD biomarker outcomes was deemed moderate. Future studies that address limitations of the current literature and evaluate interventions targeting sedentary behaviour are needed to provide stronger evidence regarding the effects of such interventions on CVD biomarkers in individuals with paraplegia.

The majority of interventions led to an improvement in at least one CVD biomarker. Biomarkers related to glycaemia were often improved, including fasting insulin [17], postprandial glucose [39], insulin resistance [17] and insulin sensitivity [18]. Studies with glycaemic outcomes had a moderate quality of evidence, meaning there is some confidence in these findings. However, two of these interventions had no effect on sedentary behaviour [17,18]. Also, both interventions that reduced sedentary behaviour did not improve glycaemic biomarkers, suggesting limited causality between these outcomes [38,40]. The evidence for interventions improving blood pressure and lipids in individuals with paraplegia was mixed, while there was consistent data that body composition was unaffected. These findings extend those of previous reviews and meta-analyses in individuals with SCI, which found that physical activity interventions had no effect on blood pressure or lipids [53]. Research has also shown exercise training may not improve body composition in individuals with SCI [55]. Lack of improvement in these biomarkers could be a result of changes in blood pressure and body composition that occur because of SCI, limiting responsiveness to reduced sedentary behaviour and/or increased physical activity [56,57]. Another plausible explanation is that intervention durations were too short (one day to sixteen weeks) [17,18,38–40]. The one intervention, which increased physical activity and improved lipids, was delivered over eight months [36]. Moreover, the magnitude of change in sedentary behaviour (ranging from 118 min/day decrease to 45 min/day increase) or physical activity (8 to 97 min/day increase) across studies may have been insufficient to bring about consistent changes in blood pressure, lipid profile or body composition. Future research should assess whether interventions that target sedentary behaviour over the long-term can produce greater changes in sedentary behaviour and, subsequently, affect CVD biomarkers.

This is the first systematic review to assess the effectiveness of interventions to reduce sedentary behaviour and improve CVD biomarkers in individuals with paraplegia. Key areas for future research have been identified to improve the quality of evidence, which will benefit the development of sedentary behaviour guidelines for this population group. Further strengths include the application of frameworks to guide the reporting, risk of bias and grading of evidence to ensure rigour within the review. Potential limitations include some eligible studies having a high risk of bias and the overall quality of evidence generally being low. Three of the included studies comprised samples of individuals with paraplegia and tetraplegia, which may influence conclusions being made specifically for individuals with paraplegia. In

addition, no studies were powered to detect changes in sedentary behaviour and only two studies were sufficiently powered to detect changes in CVD biomarkers. It is, therefore, recommended that further high-quality studies in individuals with paraplegia are conducted with sufficient power to detect changes in sedentary behaviour and CVD biomarkers.

Conclusion

In conclusion, interventions that target increases in physical activity appear to be ineffective for reducing sedentary behaviour in individuals with paraplegia, but do have beneficial effects on CVD biomarkers. The literature examining interventions that target reductions and breaks in sedentary behaviour in individuals with paraplegia is limited, yet shows potential effectiveness for improving CVD biomarkers. Investigating interventions that focus on changing sedentary behaviour in individuals with paraplegia is an important avenue for future research to inform recommendations for public health and clinical care.

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ORCID

Daniel L. Cooper http://orcid.org/0000-0002-8755-3489
Alyson Warland http://orcid.org/0000-0002-4629-0395
Emma Norris http://orcid.org/0000-0002-9957-4025
Cherry Kilbride http://orcid.org/0000-0002-2045-1883
Sue Paddison http://orcid.org/0009-0005-0895-9480
Daniel P. Bailey http://orcid.org/0000-0003-3772-630X

Data availability statement

All data associated with this review can be found within the included published articles.

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