Review

# The effect of walking on risk factors for cardiovascular disease: An updated systematic review and meta-analysis of randomised control trials

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## A R T I C L E   I N F O

## A B S T R A C T

*Objective.* To conduct a systematic review and meta-analysis of randomised control trials that examined the effect of walking on risk factors for cardiovascular disease.

*Methods.* Four electronic databases and reference lists were searched (Jan 1971–June 2012). Two authors identified randomised control trials of interventions ≥4 weeks in duration that included at least one group with walking as the only treatment and a no-exercise comparator group. Participants were inactive at baseline. Pooled results were reported as weighted mean treatment effects and 95% confidence intervals using a random effects model.

*Results.* 32 articles reported the effects of walking interventions on cardiovascular disease risk factors. Walking increased aerobic capacity (3.04 mL/kg/min, 95% CI 2.48 to 3.60) and reduced systolic ($-3.58$ mm Hg, 95% CI $-5.19$ to $-1.97$) and diastolic ($-1.54$ mm Hg, 95% CI $-2.83$ to $-0.26$) blood pressure, waist circumference ($-1.51$ cm, 95% CI $-2.34$ to $-0.68$), weight ($-1.37$ kg, 95% CI $-1.75$ to $-1.00$), percentage body fat ($-1.22\%$, 95% CI $-1.70$ to $-0.73$) and body mass index ($-0.53$ kg/m$^2$, 95% CI $-0.72$ to $-0.35$) but failed to alter blood lipids.

*Conclusions.* Walking interventions improve many risk factors for cardiovascular disease.
This underscores the central role of walking in physical activity for health promotion.

## Contents

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## Introduction

Physical inactivity is the fourth leading cause of global mortality (World Health Organisation, 2009) responsible for 6–10% of the major non-communicable diseases of coronary heart disease, type 2 diabetes, and breast and colon cancers (Lee et al., 2012). Whilst sport, running and vigorous gym based exercise are often seen as counter measures, walking offers a natural, widely accepted, low cost, low injury risk (Hootman et al., 2001) and environmentally friendly approach to physical activity which can be incorporated into activities of daily living and/or undertaken recreationally. Walking is also likely to be more accessible and suitable to a considerable portion of the higher-risk population who may be obese, sedentary, at high risk of cardiovascular disease and for whom strenuous forms of exercise may be unsuitable. Walking at a self-selected pace is moderate intensity for most adults (Ainsworth et al., 2000; Murtagh et al., 2002). Indeed it is estimated that walking at 3 mph would be vigorous intensity for approximately 20% of the population (Kelly et al., 2011). Systematic reviews have indicated that inactive people can be encouraged to walk more by tailored interventions (Ogilvie et al., 2007) and the National Institute for Health and Clinical Excellence has recently produced guidelines to promote walking for travel and recreational purposes (National Institute for Health and Clinical Excellence, 2012).

Whilst it is unsurprising that walking has become a cornerstone of physical activity promotion strategies, a challenge faced by healthcare professionals and patients is knowing the effects of walking on health, especially as many published walking interventions employ relatively small samples and findings are often inconsistent between studies. Conversely, the use of meta-analysis increases the precision and accuracy of the estimates of the effects of walking, quantifies the inconsistency between studies and enhances generalizability to a larger population. We previously reported a meta-analysis of walking interventions published up to 2004, that included aerobic fitness, blood pressure, and body composition (Murphy et al., 2007). Since then there has been an increase in the number of published interventions examining the effects of walking on risk factors for cardiovascular disease. In addition, an increased range of outcome measures have been included in these studies, such as blood lipids and several measures of adiposity. Whilst there is now greater evidence of the concomitant dangers of these factors to public health (Physical Activity Guidelines Advisory Committee, 2008) a recent comprehensive synthesis of evidence from randomised control trials on the effect of walking on health is lacking. This updated meta-analysis therefore expands our understanding of the treatment–effect relationship between walking and health.

The objective of this study was to assess the effect of walking interventions on risk factors for cardiovascular disease in previously inactive adults. This updates our previous review and provides healthcare professionals with a synthesis of the effects accruing when inactive adults undertake a walking programme.

## Methods

We followed the PRISMA statement (preferred reporting items for systemic reviews and meta-analyses) in conducting and reporting the meta-analysis (Moher et al., 2009). A review protocol has not been published separately.

*Data sources and searches*

The following electronic databases were searched: PubMed, Web of Science, ScienceDirect and the Cochrane Central Register of Controlled Trials. In addition, we hand-searched reference lists from review and original articles. Authors were contacted, if necessary, to confirm eligibility criteria. The following search terms were used: walking, exercise, health, and cardiovascular risk. Date limits of Sept 2004–Sept 2012 were applied.

*Study selection*

The study selection process is summarised in Fig. 1. Initial eligibility assessment was performed by one author by reviewing the title and abstracts. The full text versions of 48 articles were then reviewed independently by two authors. Disagreements between reviewers were resolved by consensus by reassessing each of the eligibility criteria for the study.

The following eligibility criteria were used: randomised, controlled trials studying the effect of walking on one or more cardiovascular risk factors; trials with at least one group who completed walking as the only intervention; training for a minimum of four weeks; no-exercise control group; participants aged 18 years or older who were reported as being apparently sedentary but otherwise healthy at baseline; selected cardiovascular disease risk factors assessed pre- and post-intervention (or change from pre- to post-intervention reported); and English language articles published in peer-reviewed journals between January 1971 and June 2012.

*Data extraction and quality assessment*

We used a modified version of the data extraction sheet developed for the previous meta-analysis. Two individuals extracted the data from included studies and a second author checked the extracted data. Disagreements were resolved by consensus. Two of the selected studies were suspected to be reports from the same participants — this was confirmed by contacting the authors. The authors of eight articles were contacted for further information (Aldred and Rohalu, 2011; Baker et al., 2008; Osei-Tutu and Campagna, 2005; Stensel et al., 1993, 1994; Tully et al., 2005, 2007; Woolf-May et al., 2011). All responded and provided numerical data (Aldred and Rohalu, 2011; Osei-Tutu and Campagna, 2005; Tully et al., 2005, 2007) or clarifications regarding the study protocol (Aldred and Rohalu, 2011; Baker et al., 2008; Stensel et al., 1993, 1994) that were not detailed in the published paper. Previously unpublished numerical data was obtained from the original researchers of three articles (Osei-Tutu and Campagna, 2005; Tully et al., 2005, 2007).

The previous meta-analysis extracted data on:

1) Participant characteristics (age, sex, number of men and women)
2) Intervention characteristics (duration, frequency, intensity of walking, duration of the intervention)
3) Outcome measures (aerobic fitness, body weight, body fat percentage, body mass index, systolic blood pressure and diastolic blood pressure).
4) Study design.

In addition to the above items, the following outcome measures were extracted from all included studies: total cholesterol, HDL cholesterol, LDL cholesterol, waist circumference, and waist-to-hip ratio.

The Cochrane Collaboration 'risk of bias' assessment tool was employed. Two authors, with adequate reliability, evaluated studies for sequence generation, allocation concealment, blinding, incomplete outcome data, selective outcome reporting and other potential threats to validity (Higgins et al., 2011a).
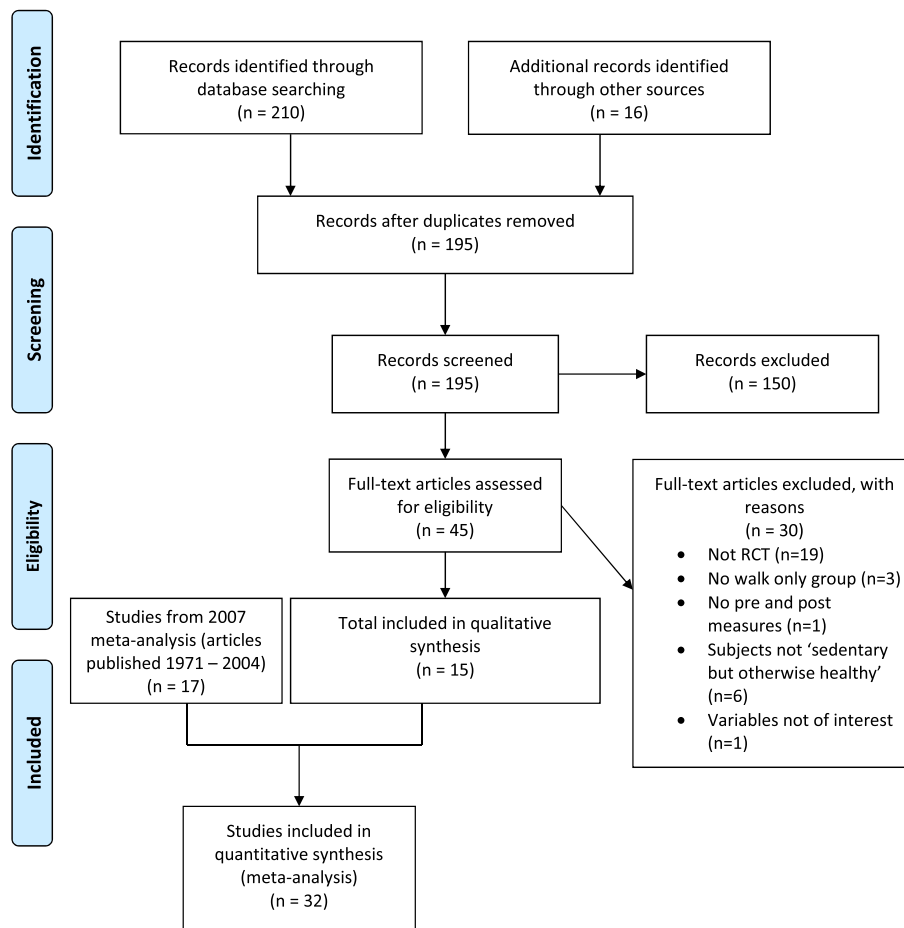
**Fig. 1.** Study selection process. Databases were searched for articles published between Jan 1971 and June 2012.

*Data synthesis and statistical analysis*

Treatment effect was calculated by subtracting pre-intervention mean from the post-intervention mean (post − pre) for both exercise (delta 1) and control (delta 2) groups. Treatment effect was then obtained as delta 1 − delta 2 for each study. Six studies reported data on standard deviation of change between pre- and post-intervention measurements (Butcher et al., 2008; Murphy and Hardman, 1998; Murphy et al., 2006; Tully et al., 2005, 2007; Woolf-May et al., 2011). If not reported, standard deviation of the individual delta values was calculated using pre- and post-standard deviations and the mean pre/post correlation from studies which reported sufficient detail to calculate correlation (Higgins et al., 2011b). To assess the effect of this assumption, sensitivity analyses were performed using minimum and maximum available pre–post correlations. In trials that had more than two intervention arms the description of the interventions were checked to ensure that they only varied in the level of exercise undertaken and if so all intervention arms were collapsed into a single treatment arm (Higgins and Deeks, 2011).

Seven studies reported weight and height as outcomes but did not report body mass index (Braith et al., 1994; Duncan et al., 1991; Hinkleman and Nieman, 1993; Moreau et al., 2001; Osei-Tutu and Campagna, 2005; Santiago et al., 1995; Woolf-May et al., 2011). For these studies, an approximation for the mean and standard deviation of body mass index was derived using formulae for the product and ratio of random variables (Stuart and Ord, 1987). A sensitivity analysis was performed on the body mass index outcome by including and excluding these approximated data. Similarly, waist-to-hip ratio was approximated using separately reported waist and hip circumference data from the three studies (Anderson et al., 2006; Murtagh et al., 2005; Serwe et al., 2011).

Pooled results were reported as weighted mean treatment effects and 95% confidence intervals using a random effects model (DerSimonian and Laird method (DerSimonian and Laird, 1986)). Statistical heterogeneity

was evaluated using the $I^2$ statistic. Publication bias was appraised by visual inspection of the funnel plots of treatment effect against standard error (to identify asymmetry, which can indicate evidence of non-publication of small trials with negative results) and also by Egger's test (Egger et al., 1997). To investigate possible sources of heterogeneity across studies we performed a meta-regression analysis to investigate the effects of the following study specific characteristics on treatment effect: mean age of participants, mean pre-intervention weight, gender (proportion of male participants) and duration of intervention. A p-value < 0.05 was considered statistically significant. All analyses were conducted using Stata, Version 12 (StataCorp, College Station, TX).

## Results

*Study selection*

A total of 210 articles were identified by electronic searches and 16 articles from hand-searching. Thirty one duplicates were then excluded. After reviewing the title and abstract of the 195 articles identified, 150 were excluded as they did not meet the inclusion criteria. The full text versions of 45 articles were then reviewed. Fifteen studies were deemed eligible for inclusion in the study and combined with studies reported in our previous meta-analysis (Murphy et al., 2007) (n = 17). Therefore in total 32 articles are included in this systemic review and updated meta-analysis. See Fig. 1 for further information. Two articles reported different outcome measures from the same subjects in a larger study (Stensel et al., 1993, 1994) and so the results from these two articles were combined to represent one study in the meta-analysis.

*Study characteristics*

All studies selected for this review are randomised controlled trials published in English.

Subject and intervention characteristics are summarised in Table 1. Participants ranged in age from 30 to 83 years. Sixteen studies include females only, 14 included both males and females and three included males only. The mean length of the walking interventions was 18.7 weeks (range: 8–52 weeks). Duration of walking per day was 20–60 min at 2–7 days per week. Twenty one studies reported exercise intensity as either percentage of maximum heart rate (range: 56–86% $HR_{max}$), percentage of $VO_{2max}$ or $VO_{2peak}$ (45–62%), or percentage heart rate reserve (54–85%). Using the generally accepted definition of moderate intensity exercise as 64–76% $HR_{max}$/ 46–63% $VO_{2max}$/40–59% HRR (Ewing Garber et al., 2011), 19 of these studies included a moderate intensity walking group (Aldred et al., 1995; Aldred and Rohalu, 2011; Anderson et al., 2006; Asikainen et al., 2002a,b; Duncan et al., 1991; Hamdorf and Penhall, 1999; Hinkleman and Nieman, 1993; Jette et al., 1988; Murphy and Hardman, 1998; Murtagh et al., 2005; Osei-Tutu and Campagna, 2005; Probart et al., 1991; Ready et al., 1995, 1996; Santiago et al., 1995; Stensel et al., 1993, 1994; Woolf-May et al., 1999). Three studies incorporated a vigorous intensity (Ewing Garber et al., 2011) walking group (Braith et al., 1994; Duncan et al., 1991; Serwe et al., 2011) and three studies included a light intensity group (Asikainen et al., 2002a; Duncan et al., 1991; Murphy et al., 2006). Additionally, five studies reported that walking was self-paced (Bell et al., 2010; Butcher et al., 2008; Moreau et al., 2001; Morgan et al., 2010; Simons and Andel, 2006) and four noted that walking intensity was at a brisk pace (Baker et al., 2008; Brandon and Elliott-Lloyd, 2006; Tully et al., 2005, 2007). It was a criterion for inclusion that studies included a no-exercise control group. All authors were contacted to confirm the data extracted in the study characteristics table and responses were received from the authors of 21 articles.

Table 2 describes the number of studies and participants and summary baseline values for all outcome measurements. Also shown in this table are the weighted mean treatment effect and its 95% confidence interval, % change from baseline, $I^2$ and Egger's test bias coefficients. For each outcome there is a separate appendix containing a table of results from the individual studies pooled, a forest plot and a funnel plot (Appendices 1–11). Appendix 12 details the results of meta-regression analyses of treatment effect against mean age of participants, mean pre-intervention weight, proportion of male participants and duration of intervention. A summary of the effect of the walking intervention on each outcome variable is given below.

*Aerobic fitness*

Observed $VO_{2max}$ and predicted $VO_{2max}$ and $VO_{2peak}$ were utilised as the outcome measure of aerobic fitness. Twelve studies reported $VO_{2max}$ (Aldred and Rohalu, 2011; Asikainen et al., 2002a,b; Braith et al., 1994; Duncan et al., 1991; Hinkleman and Nieman, 1993; Jette et al., 1988; Murphy and Hardman, 1998; Osei-Tutu and Campagna, 2005; Probart et al., 1991; Ready et al., 1995; Santiago et al., 1995), four reported predicted $VO_{2max}$ (Brandon and Elliott-Lloyd, 2006; Butcher et al., 2008; Woolf-May et al., 1999, 2011) and two reported $VO_{2peak}$ (Bell et al., 2010; Ready et al., 1996). There was a statistically significant improvement in $VO_{2max}$ of 3.04 mL/kg/min (95% CI 2.48 to 3.60) in the participants who followed the walking intervention. There was, however, evidence of significant heterogeneity between the results ($I^2 = 71\%$, p < 0.001).

*Anthropometric measures*

Sixteen trials examined the effect of walking on body mass index (Aldred and Rohalu, 2011; Anderson et al., 2006; Asikainen et al.,

2002a,b; Baker et al., 2008; Bell et al., 2010; Brandon and Elliott-Lloyd, 2006; Butcher et al., 2008; Murphy et al., 2006; Probart et al., 1991; Ready et al., 1995, 1996; Serwe et al., 2011; Stensel et al., 1994; Tully et al., 2005, 2007) and a further seven trials reported height and weight, allowing body mass index to be approximated (Braith et al., 1994; Duncan et al., 1991; Hinkleman and Nieman, 1993; Moreau et al., 2001; Osei-Tutu and Campagna, 2005; Santiago et al., 1995; Woolf-May et al., 2011). The pooled effect of the walking intervention was a statistically significant reduction in body mass index of 0.53 kg/m² (95% CI −0.72 to −0.35). A sensitivity analysis considering only those studies which reported body mass index directly as an outcome found a similar estimate of effect (0.49 kg/m² reduction (95% CI −0.70 to −0.27)). There was substantial heterogeneity between the studies ($I^2 = 70\%$, p < 0.001). The funnel plot showed some asymmetry indicating that smaller studies which found an increase in body mass index in the intervention group appeared to be absent (Egger's test, p = 0.005).

Eleven studies measured waist circumference (Anderson et al., 2006; Baker et al., 2008; Bell et al., 2010; Murphy and Hardman, 1998; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1996; Serwe et al., 2011; Tully et al., 2005, 2007; Woolf-May et al., 2011). Overall a significant reduction of 1.51 cm (95% CI −2.34 to −0.68) was found when the studies were pooled. The funnel plot showed some asymmetry indicating an absence of smaller studies (Egger's test, p = 0.02).

Waist-to-hip ratio was an outcome in eleven trials (Aldred et al., 1995; Baker et al., 2008; Bell et al., 2010; Brandon and Elliott-Lloyd, 2006; Murphy et al., 2006; Ready et al., 1995, 1996; Stensel et al., 1994; Tully et al., 2005, 2007; Woolf-May et al., 2011) and a further three trials gave enough information for this outcome to be approximated (Anderson et al., 2006; Murtagh et al., 2005; Serwe et al., 2011). A small non-significant reduction in waist-to-hip ratio of 0.01 (95% CI −0.02 to 0.00) was found. A sensitivity analysis of only those trials directly reporting waist-to-hip ratio found a similar estimate of effect (−0.01 (95% CI −0.02 to 0.00)). There was substantial heterogeneity between studies ($I^2 = 60\%$, p < 0.001).

Twenty-five studies presented data on body weight (Aldred et al., 1995; Aldred and Rohalu, 2011; Anderson et al., 2006; Asikainen et al., 2002a,b; Baker et al., 2008; Bell et al., 2010; Braith et al., 1994; Brandon and Elliott-Lloyd, 2006; Butcher et al., 2008; Duncan et al., 1991; Hinkleman and Nieman, 1993; Moreau et al., 2001; Murphy and Hardman, 1998; Murphy et al., 2006; Murtagh et al., 2005; Osei-Tutu and Campagna, 2005; Ready et al., 1995, 1996; Santiago et al., 1995; Serwe et al., 2011; Stensel et al., 1994; Tully et al., 2005, 2007; Woolf-May et al., 2011). With the exception of one study (Baker et al., 2008), all reported a negative treatment effect (intervention group lost more or gained less weight than the control group). Pooling of the data resulted in an overall reduction in body weight of 1.37 kg (95% CI −1.75 to −1.00) in those participating in a walking intervention. There was significant heterogeneity between studies ($I^2 = 66\%$, p < 0.001). Meta-regression analyses found that studies of only female participants had a greater overall treatment effect (−1.96 kg (95% CI −2.52 to −1.37)) than those studies including some male participants (−0.71 kg (95% CI −1.04 to −0.37)), although substantial heterogeneity remained for the female only studies. There was also evidence of publication bias indicating that studies reporting a positive treatment effect (greater weight gain in the treatment group) may be less likely to reach publication.

Data for the effect of walking on body fat percentage was available from fourteen trials (Anderson et al., 2006; Asikainen et al., 2002a,b; Baker et al., 2008; Brandon and Elliott-Lloyd, 2006; Butcher et al., 2008; Duncan et al., 1991; Hinkleman and Nieman, 1993; Moreau et al., 2001; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1995; Santiago et al., 1995; Stensel et al., 1994). Pooling of these data found an overall reduction in body fat of 1.22% (95% CI −1.70 to −0.73). There was substantial heterogeneity between studies ($I^2 = 68\%$, p < 0.001).

**Table 1**
Characteristics of included studies examining the effect of walking interventions on risk factors for cardiovascular disease.

| | Participants | | | | Details of Intervention | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Groups | n | Age (mean ± SD) | Sex | No. of weeks | Duration (min/session) | Freq (days/week) | Intensity | Notes |
| Jette et al. (1988) | Walk | 13 | | 14 m 12 f | | 30 | 3 | 60% $VO_{2max}$ | |
| | Con | 13 | | | | | | | |
| Duncan et al. (1991) | Stroll | 18 | 20–40 | f | 24 | 60 | 5 | 56% $HR_{max}$ | |
| | Brisk | 12 | | | | 45 | 5 | 67% $HR_{max}$ | |
| | Aerobic | 16 | | | | 36 | 5 | 86% $HR_{max}$ | |
| | Con | 13 | | | | | | | |
| Probart et al. (1991) | Walk | 10 | 72.0 ± 1.9 | f | 26 | 20 | 3 | 70% $HR_{max}$ | |
| | Con | 6 | 72.0 ± 1.7 | | | | | | |
| Hinkleman and Nieman (1993) | Walk | 18 | 36.0 ± 6.8 | f | 15 | 45 | 5 | 62 ± 2% $VO_{2max}$ | |
| | Con | 18 | 32.4 ± 6.4 | | | | | | |
| Braith et al. (1994) | Mod | 19 | 66.0 ± 5.0 | m f | 26 | 45 | 3 | 70 HRR | Duration increased by 5 min every 2 weeks until 40 min. Weeks 14–26: 45 min for mod and 35 for high intensity group. Both groups progressed to 70% HRR by week 8. Mod group continued at 70% for last 13 weeks. The high group progressed to 80–85 HRR for the last 13 weeks. |
| | High | 14 | 65.0 ± 4.0 | | | 35 | 3 | 80–85 HRR | |
| | Con | 11 | 66.0 ± 5.0 | | | | | | |
| Stensel et al. (1994); Stensel et al. (1993) | Walk | 42 | 50.3 ± 5.2 | m | 52 | 28 | 7 | 68% $HR_{max}$ | |
| | Con | 23 | 51.6 ± 4.8 | | | | | | |
| Aldred et al. (1995) | Walk | 11 | 49.6 ± 4.7 | f | 12 | 33 | 5.6 | 74% $HR_{max}$ | Duration of sessions progressed from 24 ± 1 to 33 ± 1 by week 12. Number of sessions progressed from 3.5 ± 0.3 to 5.6 ± 0.3 by week 12. Mean age for walk group is for the 13 subject who began the study. |
| | Con | 13 | 49.1 ± 4.7 | f | | | | | |
| Ready et al. (1995) | Walk | 15 | 60.9 ± 4.6 (group mean) | f | 24 | 54.3 ± 7.7 | 4.9 | 54% HRR | |
| | Con | 10 | | | | | | | |
| Santiago et al. (1995) | Walk | 16 | 30.1 ± 5.3 | f | 40 | | 4 | 72% $HR_{max}$ | 4.8 km × 4 times per week. Progressive programme: weeks 3–10: 5.1 kph walk at 5% grade (68% $HR_{max}$); weeks 11–25: 5.4 kph walk at 6% grade (71% $HR_{max}$); weeks 26–40: 5.8 kph walk at 7% grade (76% $HR_{max}$). |
| | Con | 11 | 31.5 ± 6.1 | | | | | | |
| Ready et al. (1996) | 3 day | 19 | 61.3 ± 5.8 | f | 24 | 60 | 3 | 60% $VO_{2peak}$ | |
| | 5 day | 17 | (group | | | 60 | 5 | | |
| | Con | 20 | mean) | | | | | | |
| Murphy and Hardman (1998) | Short | 12 | 44.8 ± 8.4 | f | 10 | 3 × 10 | 5 | 73% $HR_{max}$ | |
| | Long | 12 | 48.0 ± 5.5 | | | 30 | 5 | 75% $HR_{max}$ | |
| | Con | 10 | 47.3 ± 4.1 | | | | | | |
| Hamdorf and Penhall (1999) | Walk | 18 | 82.4 ± 2.8 | f | 26 | 25 | 2 | 73% $HR_{max}$ | Duration increased progressively (1 min/week) from 5 min during the first week to 25 min by week 22 |
| | Con | 20 | 83.1 ± 3.1 | | | | | | |
| Woolf-May et al. (1999) | LW | 19 | 50.1 ± 6.3 | m f | 18 | 34.8 ± 1.0 | 4.4 ± 0.3 | 73.4 ± 4.8% $HR_{max}$ | Frequency of session noted is number of sessions per week |
| | IW | 10 | 57.7 ± 6.1 | | | 14.5 ± 0.8 | 10.6 ± 1.2 | 74.8 ± 3.8% $HR_{max}$ | |
| | SW | 14 | 54.3 ± 7.4 | | | 9.9 ± 0.3 | 15.4 ± 1.2 | 74.6 ± 4.1% $HR_{max}$ | |
| | Con | 13 | 54.7 ± 7.0 | | | | | | |
| Moreau et al. (2001) | Walk | 15 | 53.0 ± 7.8 | f | 24 | | 7 | Self-paced | Goal to increase activity by 3 km every day of the week |
| | Con | 9 | 55.0 ± 3.0 | | | | | | |
| Asikainen et al. (2002a) | Walk 1 | 20 | 57.0 ± 3.8 | f | 24 | 54.0 ± 5.6 | 5 | 55% $VO_{2max}$ | Mean (SD) age is for subjects numbers at pre-test: W1 = 21, W2 = 21, W3 = 18 and W4 = 21, control = 40 |
| | Walk 2 | 21 | 55.0 ± 3.7 | | | 65.0 ± 7.8 | 5 | 45% $VO_{2max}$ | |
| | Walk 3 | 16 | 54.0 ± 3.5 | | | 38.0 ± 3.9 | 5 | 55% $VO_{2max}$ | |
| | Walk 4 | 21 | 55.0 ± 4.2 | | | 46.0 ± 6.2 | 5 | 45% $VO_{2max}$ | |
| | Con | 38 | 56.0 ± 3.8 | | | | | | |
| Asikainen et al. (2002b) | Single bout | 44 | 57.8 ± 4.4 | f | 15 | 47.9 ± 14.2 | 5 | 65% $HR_{max}$ | Daily sessions for accumulated bout groups were divided into two equally long sessions with at least a 5 hour interval |
| | Accum bout | 43 | 57.6 ± 4.2 | | | 25.0 ± 3.2 | 5 | | |
| | Con | 43 | 56.5 ± 4.2 | | | | | | |
| Murtagh et al. (2005) | Single bout | 16 | 45.7 ± 9.4 | 15 m 17 f | 12 | 20 | 3 | 73.1 ± 8.7% $HR_{max}$ | Mean age is for the 48 subjects who began the study. |
| | Accum bout | 9 | | | | 2 × 10 | | 72.1 ± 7.7% $HR_{max}$ | |
| | Con | 7 | | | | | | | |
| Osei-Tutu and Campagna (2005) | Long bout | 15 | 35.1 ± 4.6 | 8 m 7 f | 8 | 1 × 30 | 5 | 60–79% $HR_{max}$ | Progressive programme: week 1: 30 min × 3 days; week 2: 30 min × 4 days; weeks 3–8: 30 min × 5 days week |
| | Short bout | 15 | 35.4 ± 8.3 | 8 m 7 f | | 3 × 10 | | | |

**Table 1** (*continued*)

| | Participants | | | | Details of Intervention | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| | Groups | n | Age (mean ± SD) | Sex | No. of weeks | Duration (min/session) | Freq (days/week) | Intensity | Notes |
| Tully et al. (2005) | Con | 10 | 31.6 ± 4.8 | 5 m 5 f | | | | | Mean age is for baseline subject numbers. |
| | Walk | 21 | 55.5 ± 4.0 | 9 m 12 f | 12 | 30 | 5 | Brisk | |
| Anderson et al. (2006) | Con | 10 | 57.8 ± 4.6 | 4 m 6 f | | | | | Additional group not included in meta-analysis. |
| | Walk | 10 | 38.1 ± 9.3 (all groups, n = 37) | f | 8 | 30 | 5 | 74% HR$_{max}$ | |
| Brandon and Elliott-Lloyd (2006) | Con | 9 | | | | | | | Instruction to walk as briskly as possible for 3 miles with goal of 3.5 mph |
| | Walk AA | 15 | 34.0 ± 7.2 | f | 18 | | 3 | Brisk | |
| | Walk W | 13 | 40.5 ± 7.1 | | | | | | |
| | con AA | 12 | 36.0 ± 8.4 | | | | | | |
| | Con W | 12 | 42.0 ± 9.7 | | | | | | |
| Murphy et al. (2006) | Walk | 21 | 41.4 ± 7.5 | m f | 8 | 45 | 2 | 62 ± 7.1% HR$_{max}$ | Progressive programme: week 1: 25 min × 2 days; week 2: 35 min × 2 days; weeks 3–8: 45 min × 2 days |
| | Con | 12 | 40.8 ± 10.0 | | | | | | |
| Simons and Andel (2006) | Walk | 18 | 81.6 ± 3.3 | 7 m 11 f | 16 | | 2 | Self-paced | The initial walk length and pace were determined at the beginning of the study based on a pre-assessment performance during a timed, 880-yard walk. Participants were encouraged to gradually increase the distance of their walks and reduce the elapsed time. |
| | Con | 21 | 84.0 ± 3.3 | 3 m 18 f | | | | | |
| Tully et al. (2007) | 3 day | 44 | 47.8 ± 6.0 | 21 m 23 f | 12 | 30 | 3 | Brisk | Participants could choose to complete their walking in a single bout, or in multiple bouts of at least 10 min. Intention-to-treat analysis performed, substituting baseline data for those at 12 weeks for the participants who withdrew during the study. |
| | 5 day | 42 | 46.4 ± 4.8 | 16 m 26 f | | 30 | 5 | | |
| | Con | 20 | 49.1 ± 6.3 | 5 m 15 f | | | | | |
| Baker et al. (2008) | Walk | 39 | 47.3 ± 9.3 | 8 m 31 f | 12 | | | Brisk | Goal to increase steps by 3000 on at least 5 days/week. Intention to treat analysis conducted. |
| | Con | 40 | 51.2 ± 7.9 | 8 m 32 f | | | | | |
| Butcher et al. (2008) | Walk | 17 | 44.9 ± 10.0 | 9 m 8 f | 8 | | 3 | Self-paced | 10,000 steps 3 times a week on treadmill |
| | Con | 17 | 46.1 ± 12.2 | 9 m 8 f | | | | | |
| Bell et al. (2010) | Walk | 43 | m: 49 ± 11; f: 50 ± 9 | m f | 24 | | 7 | Self-paced | Progressive increase to 9221 ± 1429 steps per day by week 17 |
| | Con | 45 | | | | | | | |
| Morgan et al. (2010) | Walk | 14 | 57.4 ± 6.5 | 3 m 11 f | 15 | | 7 | Self-paced | Progressive increase to 10,000 steps per day by week 3 and maintained for additional 12 weeks |
| | Con | 15 | 62.1 ± 4.0 | 4 m 11 f | | | | | |
| Aldred and Rohalu (2011) | Walk | 12 | 68.1 ± 2.6 | 6 m 6 f | 8 | 30 | 3 | 50% W$_{max}$ | |
| | Con | 9 | 67.7 ± 2.9 | 4 m 5 f | | | | | |
| Serwe et al. (2011) | SB | 20 | 38.2 ± 7.3 | f | 8 | 30 | 5 | 60–70 HRR | |
| | LB | 20 | 37.1 ± 7.2 | | | 10 × 3 | | | |
| | Con | 20 | 36.3 ± 8.1 | | | | | | |
| Woolf-May et al. (2011) | Walk | 29 | 54.9 ± 8.0 | m | 24 | 25.1 ± 10.3 | 7.2 ± 2.9 | 65.2 ± 6.9% HR$_{max}$ | Frequency of session noted is number of sessions per week |
| | Con | 19 | 52.4 ± 8.0 | m | | | | | |

Abbreviations: Accum = accumulated, AA = African American, Con = control group, f = female, HR$_{max}$ = maximum heart rate, HRR = heart rate reserve, IW = intermediate walkers, LW = long walkers, m = males, min = minutes, mod = moderate, SW = short walkers, VO$_{2max}$ = maximal oxygen consumption, VO$_{2peak}$ = peak oxygen consumption, W = White, walk = walk group, W$_{max}$ = maximal performance.
Databases were searched for articles published between Jan 1971 and June 2012.
Note: Subject numbers are those included in the analysis unless otherwise stated.

## Blood pressure

The weighted mean treatment effect of the sixteen trials which measured systolic blood pressure (Baker et al., 2008; Bell et al., 2010; Braith et al., 1994; Brandon and Elliott-Lloyd, 2006; Butcher et al., 2008; Duncan et al., 1991; Hamdorf and Penhall, 1999; Murphy and Hardman, 1998; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1996; Serwe et al., 2011; Simons and Andel, 2006; Tully et al., 2005, 2007; Woolf-May et al., 2011) was −3.58 mm Hg (95% CI −5.19 to −1.97) indicating a reduction in systolic blood pressure in those who followed a programme of walking. Diastolic blood pressure was also reported by sixteen studies (Baker et al., 2008; Bell et al., 2010; Braith et al., 1994; Brandon and Elliott-Lloyd, 2006; Butcher

et al., 2008; Duncan et al., 1991; Hamdorf and Penhall, 1999; Moreau et al., 2001; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1996; Serwe et al., 2011; Simons and Andel, 2006; Tully et al., 2005, 2007; Woolf-May et al., 2011) and the weighted mean treatment effect of the walking intervention was a significant reduction of 1.54 mm Hg (95% CI −2.83 to −0.26).

## Lipids

Data on the effect of walking on total cholesterol were available from sixteen studies (Baker et al., 2008; Bell et al., 2010; Butcher et al., 2008; Duncan et al., 1991; Hinkleman and Nieman, 1993; Morgan et al., 2010; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1995, 1996;

**Table 2**
Pre-intervention mean (SD) and weighted mean treatment effect by outcome.

| Outcome | No. of studies | No. of participants | Mean (SD) | Pooled analysis | | | % change | Heterogeneity | | Egger's test | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Weighted mean TE | (95% CI) | p | | $I^2$ (%) | p | Bias coefficient | p |
| $VO_{2max}$ (mL/kg/min) | 18 | 894 | 29.02 (5.19) | 3.04 | (2.48 to 3.60) | <0.001 | 10.5 | 71 | <0.001 | 0.47 | 0.45 |
| BMI (kg/m$^2$) | 23 | 1201 | 27.15 (5.01) | −0.53 | (−0.72 to −0.35) | <0.001 | −2.0 | 70 | <0.001 | −1.56 | 0.005 |
| Systolic BP (mm Hg) | 16 | 816 | 124.66 (14.50) | −3.58 | (−5.19 to −1.97) | <0.001 | −2.9 | 39 | 0.05 | −0.91 | 0.29 |
| Diastolic BP (mm Hg) | 16 | 806 | 77.54 (9.52) | −1.54 | (−2.83 to −0.26) | 0.02 | −2.0 | 29 | 0.13 | −0.60 | 0.63 |
| Total cholesterol (mmol/L) | 16 | 758 | 5.58 (1.07) | −0.12 | (−0.27 to 0.04) | 0.14 | −2.2 | 42 | 0.04 | −0.09 | 0.94 |
| HDL cholesterol (mmol/L) | 15 | 725 | 1.35 (0.47) | 0.01 | (−0.04 to 0.07) | 0.65 | 0.7 | 0 | 0.47 | 0.05 | 0.95 |
| LDL cholesterol (mmol/L) | 14 | 664 | 3.72 (1.00) | −0.05 | (−0.17 to 0.07) | 0.39 | 1.3 | 0 | 0.59 | 0.22 | 0.79 |
| Waist circumference (cm) | 11 | 574 | 90.56 (14.15) | −1.51 | (−2.34 to −0.68) | <0.001 | −1.7 | 38 | 0.10 | 2.27 | 0.02 |
| Waist-to-hip ratio | 14 | 706 | 0.85 (0.10) | −0.01 | (−0.02 to 0.00) | 0.07 | −1.2 | 60 | 0.001 | 1.04 | 0.28 |
| Body weight (kg) | 25 | 1275 | 75.40 (14.75) | −1.37 | (−1.75 to −1.00) | <0.001 | −1.8 | 66 | <0.001 | −1.75 | 0.001 |
| Body fat (%) | 14 | 719 | 34.29 (6.07) | −1.22 | (−1.70 to −0.73) | <0.001 | −3.5 | 68 | <0.001 | −0.78 | 0.38 |

$VO_{2max}$ = maximal oxygen consumption, BMI = body mass index.
Databases were searched for articles published between Jan 1971 and June 2012.

Santiago et al., 1995; Stensel et al., 1993; Tully et al., 2005, 2007; Woolf-May et al., 1999, 2011), HDL cholesterol from fifteen studies (Baker et al., 2008; Bell et al., 2010; Butcher et al., 2008; Duncan et al., 1991; Morgan et al., 2010; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1995, 1996; Santiago et al., 1995; Stensel et al., 1993; Tully et al., 2005, 2007; Woolf-May et al., 1999, 2011), and LDL cholesterol from fourteen studies (Bell et al., 2010; Butcher et al., 2008; Duncan et al., 1991; Hinkleman and Nieman, 1993; Murphy et al., 2006; Murtagh et al., 2005; Ready et al., 1995, 1996; Santiago et al., 1995; Stensel et al., 1993; Tully et al., 2005, 2007; Woolf-May et al., 1999, 2011). None of the pooled treatment effects for blood lipid outcomes were statistically significant. There was no evidence of publication bias, nor was any between-study heterogeneity found.

For all outcomes, sensitivity analyses to assess the impact of the correlation coefficient used for imputing standard deviation of change score did not result in any variation in significance of the pooled treatment effect. Similarly, meta-regression analyses were conducted to assess whether heterogeneity could be explained by age, baseline weight, gender or duration of intervention. With the exception of body weight, where a gender effect was found to explain some of the heterogeneity, the majority of the slope parameters were found to be non-significant (see Appendix 12).

*Risk of bias of individual studies*

Details of the risk-of-bias assessment appear in Table 3. Only two studies were judged to be at low risk of bias. Many studies did not provide sufficient information to make firm judgements about bias.

**Discussion**

This updated systematic review and meta-analysis suggests that walking programmes improve several markers of cardiovascular risk, including aerobic capacity, systolic and diastolic blood pressure, and adiposity (waist circumference, body mass, percentage body fat and body mass index) in previously inactive but apparently healthy adults. However there was no evidence of changes in total cholesterol, HDL cholesterol, LDL cholesterol or waist-to-hip ratio. In this updated meta-analysis we analysed five additional outcome variables not included in our earlier publication (Murphy et al., 2007), namely total cholesterol, HDL cholesterol, LDL cholesterol, waist-to-hip ratio and waist circumference, and identified an additional 15 relevant studies. Overall, this body of evidence supports the role of walking as a central feature of individual and population health promoting strategies to ameliorate the escalating global burden of cardiovascular disease.

*Aerobic fitness*

Aerobic fitness is an independent risk factor for cardiovascular disease and a stronger risk factor than physical activity alone (Blair et al., 2001; Blair and Jackson, 2001). $VO_{2max}$, considered the "gold standard" for the assessment of aerobic fitness (Murphy et al., 2007), was measured or predicted in 18 of the studies included and showed a significant weighted mean treatment effect (TE) of 3.04 mL/kg/min. Consequences of improvement in aerobic capacity include greater ease of performance of everyday physical activities and improved quality of life for the individual (Murphy et al., 2007). From a population perspective an improvement of this magnitude (approx 10%) is likely to result in a 15% reduction in mortality (Dunn et al., 1999), irrespective of the baseline fitness level (Blair et al., 1995). Healthcare professionals should be mindful however that walking needs to be of at least moderate intensity (64–76% $HR_{max}$) (American College of Sports Medicine, 2010) in order to improve aerobic fitness. Evidence has demonstrated that even greater fitness improvements can be attained from walking at vigorous intensity (Duncan et al., 1991; Nicklas et al., 2009).

*Blood pressure*

Hypertension is a major risk factor for cardiovascular disease (Egan et al., 2010). A significant reduction in systolic blood pressure of 3.58 mm Hg and in diastolic blood pressure of 1.54 mm Hg was observed in this meta-analysis. This weighted mean treatment effect is similar to the findings of an earlier meta-analysis focussing on walking and resting blood pressure (Kelley et al., 2001). A previous prospective study reported that a 2 mm Hg reduction in systolic blood pressure would result in 10% lower stroke mortality and 7% lower mortality from ischaemic heart disease or other vascular causes in middle age (Prospective Studies Collaboration, 2002), thus highlighting the clinical significance of even small changes in resting blood pressure. Given that hypertension has wide-scale population prevalence, affecting approximately 19–32% of men and women in UK (Maryon-Davis, 2005) and 29% of US adults (Egan et al., 2010), policy makers should consider the central role walking could play in population blood pressure control strategies.

*Lipids*

Elevated total cholesterol levels have been associated with coronary heart disease mortality (Menotti and Lanti, 2003) and also found to be independently predictive of ischemic heart disease (Yarnell et al., 2001). In the current review 16 studies reported total cholesterol and a non-significant weighted mean treatment effect of −0.12 mmol/L was found. The weighted mean treatment effect for HDL cholesterol of

**Table 3**
Assessment of risk of bias.

| Trial | Adequate sequence generation? | Allocation concealment? | Blinding? | Incomplete outcome data addressed? | Free of selective reporting? | Free of other bias? |
|---|---|---|---|---|---|---|
| Jette et al. (1988) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Duncan et al. (1991) | Unclear | Unclear | Unclear | No | Yes | Yes |
| Probart et al. (1991) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Hinkleman and Nieman (1993) | Unclear | Unclear | Unclear | No | Yes | Yes |
| Braith et al. (1994) | Unclear | Unclear | Unclear | Unclear | Yes | Yes |
| Stensel et al. (1994); Stensel et al. (1993) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Aldred et al. (1995) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Ready et al. (1995) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Santiago et al. (1995) | Yes | Yes | Unclear | Yes | Yes | Yes |
| Ready et al. (1996) | Unclear | Unclear | Unclear | No | Yes | Yes |
| Murphy and Hardman (1998) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Hamdorf and Penhall (1999) | Yes | Yes | Unclear | Yes | Yes | Yes |
| Woolf-May et al. (1999) | Unclear | Unclear | No | Unclear | Yes | Yes |
| Moreau et al. (2001) | Unclear | Unclear | No | Yes | Yes | Yes |
| Asikainen et al. (2002a) | Yes | Yes | No | Yes | Yes | Yes |
| Asikainen et al. (2002b) | Unclear | Unclear | Unclear | Yes | Yes | No |
| Murtagh et al. (2005) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Osei-Tutu and Campagna (2005) | Unclear | Unclear | No | Yes | Yes | Yes |
| Tully et al. (2005) | Yes | Yes | Yes | Yes | Yes | Yes |
| Anderson et al. (2006) | Yes | Unclear | Unclear | Yes | Yes | Yes |
| Brandon and Elliott-Lloyd (2006) | Unclear | Unclear | Unclear | Unclear | Yes | Yes |
| Murphy et al. (2006) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Simons and Andel (2006) | Unclear | Unclear | Yes | Yes | Yes | Yes |
| Tully et al. (2007) | Yes | Yes | No | Yes | Yes | Yes |
| Baker et al. (2008) | Yes | Yes | Yes | Yes | Yes | Yes |
| Butcher et al. (2008) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Bell et al. (2010) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Morgan et al. (2010) | Unclear | Unclear | Unclear | Yes | Yes | Yes |
| Aldred and Rohalu (2011) | Unclear | Unclear | Unclear | Unclear | No | Yes |
| Serwe et al. (2011) | Unclear | Unclear | No | Yes | Yes | Yes |
| Woolf-May et al. (2011) | Yes | Yes | Unclear | Unclear | Yes | Yes |

Databases were searched for articles published between Jan 1971 and June 2012.

0.01 mmol/L did not reach statistical significance, which may be linked to the optimal mean pre-intervention values of these subjects (Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults, 2001). Whilst HDL cholesterol levels are strongly, independently and inversely associated with coronary heart disease (Cooney et al., 2009), evidence from a systematic review and regression analysis suggests that simply increasing the amount of circulating HDL cholesterol does not reduce the risk of coronary heart disease events, coronary heart disease deaths, or total deaths (Briel et al., 2009). Existing evidence supports reduction in LDL cholesterol as the primary goal for lipid modifying interventions (Briel et al., 2009; Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults, 2001). LDL cholesterol was reported in 14 studies in this meta-analysis; however the mean TE of −0.05 mmol/L was not significant. Some authors have suggested that changes in apolipoprotein A or B may reduce cardiovascular risk by increasing the LDL-C particle size without changing LDL levels and therefore monitoring apolipoproteins rather than just the cholesterol of lipoproteins might be a more sensitive measure of exercise induced changes in lipoprotein function (Holme et al., 2007).

*Anthropometric measures*

Obesity is a major influence on the development of cardiovascular diseases (Kumanyika et al., 2008). Several measures of overweight and obesity were utilised in the studies included in this review and significant effects were observed for body mass, percentage body fat, body mass index and waist circumference. The mean decrease in body mass and body fat percentage of 1.37 kg and 1.22% respectively was significant and reinforces the critical role that walking can play in the management of overweight. Body mass index, a strong predictor of overall mortality both above and below the apparent optimum of about 22.5–25 kg/m$^2$ (Prospective Studies Collaboration, 2009), demonstrated

a TE of −0.53 km/m$^2$. Waist circumference, which may be the best single indicator of other individual and multiple cardiovascular risk factors (Dobbelsteyn et al., 2001), also demonstrated a significant TE of −1.51 cm. Although the treatment effect observed in waist circumference may be small, a previous meta-regression analysis of prospective studies demonstrated that a 1 cm increase in waist circumference was associated with a 2% increase in the relative risk of a cardiovascular disease event (de Koning et al., 2007). So even small improvements in waist circumference resulting from walking interventions may have substantial public health gains.

*Limitations*

Whilst this meta-analysis provides useful updated information for healthcare providers and policy-makers, the results should be interpreted with the following limitations in mind. First, this meta-analysis pooled data from several studies in order to calculate mean weighted treatment effects. However the walking intervention employed, in terms of dose of exercise and support provided, is not the same across studies. Second, risk of bias, assessed using the Cochrane Collaboration assessment tool (Higgins et al., 2011a), revealed that many studies did not provide sufficient information to make firm judgements about bias. Third, observed changes in cardiovascular risk factors may be mediated by a change in another risk factor. For example change in VO$_{2max}$ measured per unit of body mass may be affected by change in body mass; therefore, the impact of the intervention on a single outcome measure is difficult to isolate. Fourth, the search strategy was restricted to English-language publications. Fifth, several studies did not fully report outcomes of interest and not all authors who were contacted for further information provided missing data. Additionally, for many of the studies standard deviation of change from baseline was not reported and these data were imputed using the mean correlation coefficient from the available data. Sensitivity analyses were performed using a range of correlation

coefficients which did not show any changes to the pooled effect sizes. Sixth, publication bias was evident for some outcomes including body mass index, weight and waist circumference. Asymmetrical funnel plots suggested that there was an absence of small studies with negative findings (favouring the control group). Finally the majority of subjects in the studies were middle-aged and female. Whilst this may reflect the appeal that this form of physical activity has for women (Murphy et al., 2007), the applicability of these findings to a male population may therefore be limited.

## Conclusions

Our findings confirm the important role that regular walking can play in the prevention of cardiovascular disease. The meta-analysis revealed positive changes for multiple cardiovascular disease risk factors, including aerobic fitness, systolic and diastolic blood pressure, and several measures of adiposity in previously inactive adults who walked as part of a randomised controlled trial. Clinicians and health-care providers can prescribe walking with confidence that it will evoke health benefits. As in our previous meta-analysis, few studies have investigated the effects of walking interventions on non-traditional cardiovascular disease risk factors, such as markers of inflammation, oxidative stress and coagulation (Murphy et al., 2007). Future research should incorporate these measures and further examine the effect of walking in specific populations including ethnic minorities and older adults. The results and recommendations of this meta-analysis are of relevance for health professionals involved in the primary prevention of cardiovascular disease and should underscore the central role of walking as a cornerstone in physical activity for health promotion.

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### Conflict of interest statement

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