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The effect of walking on fitness, fatness and resting blood pressure: A meta-analysis of randomised, controlled trials

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Abstract

Objective. The purpose of this review was to perform a meta-analysis on walking intervention studies in order to quantify the magnitude and direction of walking-induced changes that may alter selected cardiovascular risk factors.

Method. Twenty-four randomised controlled trials of walking were assessed for quality on a three-point scale. Data from these studies were pooled and treatment effects (TEs) were calculated for six traditional cardiovascular risk variables: body weight, body mass index (BMI), percentage body fat, aerobic fitness (VO2 max in ml kg−1 min−1) and resting systolic and diastolic blood pressure. Weighted TEs were analysed using a random effects model with weights obtained using the inverse of the individual TE variances. Random effects models were used to investigate the influence of both study quality and exercise volume (<150 vs. 150 min week−1).

Results. Random effects modelling showed that walking interventions increased VO2 max and decreased body weight, BMI, percent body fat and resting diastolic blood pressure in previously sedentary adults ($p<0.05$ for all).

Conclusion. The results of this study provide evidence that healthy but sedentary individuals who take up a programme of regular brisk walking improves several known risk factors for cardiovascular disease.

Keywords: Walking; Cardiovascular risk; Fitness; Blood pressure; Fatness; Meta-analysis.

Introduction

It is widely accepted that physical inactivity is a risk factor for cardiovascular disease (Blair and Connelly, 1996). Increasing physical activity has been repeatedly associated with a decrease in cardiovascular risk. Despite the strength of this association the ideal type of physical activity for gaining health benefits remains a matter of substantial debate (Bouchard, 2001; Haskell, 2001).

Current physical activity guidelines recommend the accumulation of at least 30 min of moderate intensity physical activity on most days of the week (Pate et al., 1995). Walking is the most popular physical activity among the EU population (Vaz De Almeida et al., 1999). One of its appeals lies in the fact that it is accessible to all, requires little skill and has a low risk of injury. Walking can be performed at a variety of speeds (and therefore intensities), in a group or alone and without the need for special equipment or clothing. Research from our group, among others, has shown that for middle-aged and older adults, as well as for sedentary younger individuals, walking at a self-selected pace is at least a moderate intensity (Murtagh et al., 2002). The importance of walking for an individual attempting to increase their daily physical activity is underscored by the results of a study by Dunn et al. (1998); when individuals were asked to voluntarily incorporate 30 min of additional physical activity into their daily lives they did so by increasing their walking activity by 19–20 min per day (Dunn et al., 1998). In other words, walking appears to be a preferred activity among sedentary individuals taking up physical activity. Consequently therefore walking has become an important cornerstone in many physical activity promotion campaigns.

Despite the intuitive appeal of walking as a method of reducing aspects of cardiovascular risk and the palatability of this type of exercise with the sedentary population most at risk of cardiovascular disease, interventions which have considered the effects of a programme of

brisk walking on the risk factors of body fatness, fitness and blood pressure have yielded equivocal results (Aldred et al., 1995; Duncan et al., 1991). Some clinicians promoting physical activity are often unconvinced of the efficacy of such 'lifestyle' physical activities on health outcomes. The conflicting results in walking intervention studies may be attributable to small sample sizes and underpowered studies. A meta-analysis of randomised controlled trials can provide a more reliable estimate of treatment effects due to increased statistical power afforded by larger sample sizes. The purpose of this study was to perform a meta-analysis on walking intervention studies in order to quantify the magnitude and direction of walking induced changes on selected cardiovascular risk factors.

Methods

Study selection

The inclusion criteria for this study were as follows: (1) randomised, controlled trials, (2) walking as the only intervention, (3) intervention a minimum of 4 weeks duration, (4) selected cardiovascular disease risk factors assessed pre- and post-intervention, (5) subjects apparently sedentary but otherwise healthy at baseline, and (6) subjects aged 18 years or older.

Data sources

Studies were identified using computerised literature searches of the following databases: Medline and Web of Science. The main keywords used in these searches were: walking, health, exercise, and cardiovascular risk. Extensive hand-searching and cross-referencing from review and original articles was also performed. English language articles published from 1971 to September 2004 were included. We chose 1971 as the starting date since this

appears to be the first year in which a study examining the effects of walking on fatness, fitness or resting blood pressure was published (Pollock et al., 1971).

Data abstraction

The following variables were extracted: number, age and gender of participants who completed the trial, intervention characteristics (intensity, frequency, duration and volume of walking), and pre- and post-intervention values for variables of interest (VO2 max, body weight, BMI, body fat, resting systolic and diastolic blood pressure).

Assessment of quality

The methodological quality of the studies reviewed was assessed using the scheme described in the Cochrane Collaboration Handbook (1995) and used by Halbert et al. (1999a,b) (Halbert et al., 1999a,b). This scheme involves assessing the quality of the allocation (control of selection bias at entry). A three-point rating scale was employed with gradings of: (A) if the effort to control selection bias had been maximal (that is, central randomisation by an independent third party); (B) if there had been some effort to control selection bias; and (C) if there had been little or no effort to control selection bias at entry. Halbert and coworkers noted that the score allocated to each study may reflect the quality of reporting rather that the methodological quality of the trials as information was often lacking about the randomisation methods. Two authors (MHM and EMM) coded each study independently and disagreements were resolved by consensus.

Statistical methods

For all baseline measures and descriptors of the walking interventions, means and standard deviations (SD) were calculated by combining the mean values from the intervention and control groups, weighted by the number of participants from each study.

The effect of brisk walking on the six cardiovascular risk variables were identified by calculating a 'treatment effect', as described by Kelley and Tran (1995). In brief, treatment effects were calculated by subtracting the pre-exercise value from the post-exercise value (post−pre) for both the exercise (delta 1) and control (delta 2) groups. The treatment effect (TE) was then obtained as delta 1−delta 2 for each study and then averaged to give the unadjusted mean TE. The variances of the individual delta values were calculated from the pre and post-variances and the pre/post correlations. These were then used inversely as the weights for individual TE in obtaining the weighted mean TEs. Random effects models were then used to estimate the between study variances (SDb2). These were then combined with the weighted mean TE variances to form the weighted mean TE SDc given in Table 2. The random effects model controls (statistically) for between-study heterogeneity, a feature confirmed by the significant Cochran's Q statistics also reported in Table 2 (Hardy and Thompson, 1998). Funnel plots were used to assess publication bias. A random effects model was used to compare TEs when grouped according to study quality and exercise volume (<150 vs. ⩾150 min week−1) using the methods described by Lipsey and Wilson (2001). Statistical significance was established at $p<0.05$. Weights for individual TEs were calculated using an Excel worksheet. These were transferred into the MINITAB statistical software and a mixture of a MINITAB macro and hand calculations were used to obtain the statistics reported in Table 2. Multiple intervention groups within the same study were treated independently.

Results

Studies selected

Initial searches yielded over 40 studies using walking as an intervention. Twenty-four of these studies met the inclusion criteria (see Table 1) (Aldred et al., 1995; Asikainen, 2002; Asikainen et al., 2002; Braith et al., 1994; Duncan et al., 1991; Hamdorf, 1992; Hamdorf and Penhall, 1999; Hardman and Hudson, 1994; Hardman et al., 1992; Hinkleman and Nieman, 1993; Jette et al., 1988; Keller and Trevino, 2001; Kobayashi et al., 2001; Kukkonen-Harjula et al., 1998; Moreau et al., 2001; Murphy and Hardman, 1998; Pollock et al., 1971; Probart et al., 1991; Ready et al., 1995, 1996; Santiago et al., 1995; Stensel et al., 1994; Whitehurst and Menendez, 1991; Woolf- May et al., 1999) No study was categorised as A quality, 9 and 15 studies were categorised as B and C respectively. Table 1 summarises the characteristics of the studies included in the analysis and the pre to post intervention alterations in the cardiovascular risk factors selected for this analysis. All studies were parallel-designed with a control group using 1, 2, 3 or 4 intervention groups to compare different volumes, intensities or patterns of walking. Funnel plots for result in no significant publication bias for any of the 6 variables of interest (VO2 max, body weight, BMI, body fat, systolic and diastolic blood pressure).

Subjects

Subject populations varied in size from 16 to 130 with a mean of 45 (median 36) subjects. Overall 1128 participants were evaluated with 698 completing walking interventions and

430 acting as control subjects. Weighted mean age of the subjects was 51.6 years (12.7 years). Eighteen studies involved exclusively female participants, two exclusively male and four used a mixed subject sample. In total 193 men and 935 women were participants in the

studies. Twenty of the studies reported data on subject drop out. Mean percentage drop out was 20.2% (15.3%, range 0–53.8%) and 12.4% (13.2%, range 0–38)% among walkers and controls, respectively.

Characteristics of walking interventions

The mean length of the walking programme was 34.9 weeks (4.9 weeks, range 8–104 weeks) and the mean frequency of days on which walks were performed was 4.4 days week−1 (range 2–7 days week−1). The mean total volume of walking was 188.8 min week−1 (range 50–270 min week−1). The mean duration of walking bouts was 38.3 min (14.4 min, range 9.9– 65 min). The intensity of brisk walking was described by 20 studies using a variety of relative exercise intensity measures including percentage of actual or predicted VO2 max or VO2 peak (6 studies) and percentage of predicted maximum heart rate or heart rate reserve (14 studies). Using these estimates, the mean relative intensity of the walking interventions was 70.1% (9.1%, range 50–86%) of predicted maximum heart rate or 56.3% VO2 max (7.1% VO2 max, range 45–65% VO2 max). Compliance, expressed as a percentage of the prescribed walking bouts completed was 87.8% (9.9%, range 57–98%).

Cardiovascular fitness

Table 2 summarises pooled estimates for TEs for cardiovascular risk factors. Thirteen studies, consisting of 24 intervention groups, used a measure of cardiovascular fitness before and after the walking intervention. Mean weighted baseline VO2 max for the 713 subjects in these studies was 30.0 ml kg−1 min−1 (5.0 ml kg−1 min−1). All 13 studies reported an increase in VO2 max ranging from 0.6 to 6.9 ml kg−1 min−1. When the data were pooled we observed a statistically significant weighted TE increase of 2.73 ml kg−1 min−1 (0.35 ml kg−1 min−1) after the walking programmes. This represents a 9% increase from baseline.

Body composition

Eighteen studies measured body weight, with a mean baseline weight of 70.4 kg (6.7 kg). A weighted mean TE of −0.95 kg (0.61 kg) was observed after the walking programmes. Body Mass Index at baseline was reported in 20 studies with a mean body mass index of 25.9 kg m−2 (1.0 kg m−2). BMI at both pre- and post-intervention was reported in 16 studies with a weighted mean TE of −0.28 kg m−2 (0.2 kg m−2). Body fat percentage before and after walking intervention was determined in 12 studies with a mean baseline body fat of 32.4% (5.8%). A weighted mean TE of −0.63% (0.66%) was observed following walking interventions. As can be seen in Table 2, decreases in body weight, BMI and percent body fat were all statistically significant. These decreases represent a relative reduction of 1.4% in body weight a 1.1% in BMI and a 1.9% in percent body fat from baseline.

Blood pressure

Nine studies measured resting systolic blood pressure before and after the walking interventions. At baseline, resting systolic blood pressure was 127.0 mm Hg (11.7 mm Hg). No significant TE was found for systolic blood pressure. Six studies recorded resting diastolic blood pressure before and after the walking interventions. At baseline, resting diastolic blood pressure was 77.7 mm Hg (4.5 mm Hg). A significant mean weighted TE or −1.54 mm Hg (0.79 mm Hg) was found for diastolic blood pressure. These decreases represent a relative reduction of 0.8% in systolic and 2% diastolic blood pressure from baseline.

Treatment effects by study quality and volume of walking

Random effects models identified no differences in TE for any variable according to study quality (B vs. C) or total walking time per week (<150 min vs. >150 min). All p values were greater than 0.05.

Table 1
Characteristics of the walking intervention studies selected for meta-analysis ($n=24$) and percentage change in cardiovascular risk factors pre to post intervention

First author/year	Quality	Study design	Subjects				Details of intervention				% Change from pre- to post-intervention					
			Group	*N*	Sex	Age	Days per week	Intensity	Min per week	Weeks	V_{O_2} max	Body weight	Body fat (%)	BMI	Systolic BP	Diastolic BP
Aldred 1995	B	2 groups	Walk	11	f	49.6	5.6	74% HR max	33	12		−1.6				
		Parallel design walk vs. control	Control	13	f	49.1						+1.4				
Asikainen 2002	B	5 groups	Walk 1	20	f	57	5	55% V_{O_2} max	54	24	+3.3	−1.2	−3.0	−1.2		
		Parallel design	Walk 2	21	f	55	5	45% V_{O_2} max	65	24	+1.9	−0.4	−2.7	−0.4		
		2 intensities, 2 volumes+ control	Walk 3	16	f	54	5	55% V_{O_2} max	38	24	+4.8	−0.8	−1.4	−0.8		
			Walk 4	21	f	55	5	45% V_{O_2} max	46	24	+1.0	+0.2	−2.5	0		
			Control	38	f	56					−5.8	+0.1	+0.3	0		
Asikainen 2002	B	3 groups	Single bout	44	f	57.8	5	65% V_{O_2} max	48	15	+12.5	−1.8	−6.8	−1.9		
		Parallel design	Accumulated	43	f	57.7	5	65% V_{O_2} max	2×25	15	+12.7	−1.8	−5.6	−1.5		
		2 patterns of walking+ control	Control	43	f	56.5					+4.3	0	−1.1	0		
Bmith 1994	B	3 groups	Moderate	19	20m	66	3	70% HRR	45	26	+16.0	−1.4		−1.4	−7.4	−11.1
		Parallel design	High	14	24f	65	3	80–85% HRR	30	26	+26.9	−2.6		−2.6	−6.7	−9.3
		2 intensities+control	Control	11		66					0	+1.4		+1.4	+1.7	−1.4
Duncan 1991	B	4 groups	Stroll	18	f	30	3	56% HR max	4.8 km/day	24	+4.4	+1.3	−6.1	+1.3	−2.8	0
		Parallel design	Brisk	12	f	30	3	67% HR max		24	+9.3	+0.2	−5.3	+1.8	+0.9	−1.4
		3 intensities+control	Aerobic	16	f	30	3	86% HR max		24	+16.3	+1.8	−4.0	+1.8	0	0
			Control	13	f	30					−5.8	+6.3	+4.0	+6.3	+1.9	+1.4
Hamdorf 1999	B		Walk	18	f	82.4	2	73% HR max	25	24					+5.2	+4.8
			Control	20	f	83.1									−0.7	+5.0
Hamdorf 1992	C		Walk	30	f	64.1	2	40–60% HRR	45	26					−3.0	−6.6
			Control	36	f	64.1									−2.2	−5.3
Hardman 1994	C		Walk	10	f	47.3	>3	Brisk	180–315 pw	24		+0.6				
			Control	10	f	41.6						+2.0				
Hardman 1992	C		Walk	28	f	44.9	>3	Brisk	157 pw	52		+0.5	+2.8	−0.4		
			Control	16	f	44.4						+0.8	+1.4	+0.3		
Hinkleman 1993	C		Walk	18	f	36.0	5	62% V_{O_2} max	45	15	+2.3		−0.5			
			Control	18	f	32.4					−1.2		+0.9			
Jette 1988	C		Walk	13	7m 6f	44	3	60% V_{O_2} max	30	12	+14.9					
			Control	13	7m 6f	30				+1.7						
Keller 2001	B		Low	12	f	32	3	50% HRR max	30	24		−1.2		−0.8		
			High	12	f	30	5	50% HRR max	30			+1.7		+2.1		
			Control	12	f	30						+3.1		+7.8		
Kobayashi 2001	C		Walk	14	f	54	4–5	11–13 RPE	30	24		−2.5	−4.8	−2.5		
			Control	9	f	49						+1.0	+6.4	+1.0		
Kukkonen Harjula 1998	B		Walk	53	2 5 m 28f	42.1	4	65–75% V_{O_2} max	50	15	+13.9			−1.6		
			Control	55	2 6 m 29f	40					+6.8			+0.8		
Moreau 2001	C		Walk	15	f	53	7	3 km self-paced		24		−1.6	−0.2	−1.6	−7.7	−3.4
			Control	9	f	55						+0.8	−0.2	+0.8	+0.7	+4.8
Murphy 1998	C		Short	12	f	44.8	5	73% HR max	3x10	10	+8.3	−2.6		−2.6	−5.9	
			Long	12	f	48.0	5	75% HR max	30		+8.5	−0.1		−0.1	−3.7	
			Control	10	f	47.3					−2.0	+0.8		+0.8	−1.6	
Pollock 1971	C		Walk	16	m	48.9	3	76% HR max	20	26		−1.7	−5.0		−2.4	+1.3
			Control	8	m	30						0	+2.2		+0.6	+1.3
Probart 1991	C		Walk	10	f	72	3	70% HR max	20	26	+7.5			−1.6		
			Control	6	f	72					−5.6			+0.9		
Ready 1996	C		3 days	19	f	61.3	3	60% V_{O_2} peak	60	24	+12.1	−0.8		0	−5.2	
			5 days	17	f	61.3	5	60% V_{O_2} peak	60	24	+13.9	0		+0.4	−3.8	
			Control	20	f	61.3					0	+1.1		+1.5	−1.5	
Ready 1995	C		Walk	15	f	60.9	5	54% HRR max	54	24	+10.9	−2.5	−2.9	−3.1		
			Control	10	f	60.9					+1.7	+.7	+0.5	+0.9		
Santiago 1995	C		Walk	16	f	30.1	4	72% HR max	3 miles	40	+21.9	−1.6	−6.3	−1.6		
			Control	11	f	31.5					−2.3	+3.1	+5.5	+3.1		
Stensel 1994	C		Walk	42	m	50.3	7	68% HR max	28	52		−0.3	−3.8	0		
			Control	23	m	51.6						+0.9	−1.0	+0.8		
Whitehurst 1991	C		Walk	18	f	68.0	3	70–80% HR max	40	8		−1.7	+3.2	−1.7	−2.8	
			Control	13	f	69.6						+1.3	+1.4	+1.3	+0.9	
Woolf-May 1999	B		Long	19	6m 13f	50.1	4.4	70–75% HR max	34.8	18	+13.7					
			Intermediate	10	3m 7f	57.7	10.6	70–75% HR max	14.5	18	+14.0					
			Short	14	5m 9f	54.3	15.4	70–75% HR max	9.9	18	+13.3					
			Control	13	5m 8f	54.7					−3.9					

Sixteen studies were 2-group-parallel designs comparing controls and walkers, 3 studies included a comparison of two or more walking intensities, 3 studies compared patterns of walking (accumulated vs. continuous) and 2 studies compared differing volumes of walking.

Table 2
The treatment effect (TE) and weighted treatment effects of brisk walking on selected cardiovascular risk factors

Variable	*N*	Unadjusted mean TE	Weighted mean TE	SD_b (*Q*)	SD_t	*z*	*P*
V_{O_2} max (ml kg^{-1} min^{-1})	24	3.66	2.73	0.75 (86.4*)	0.35	7.70	<0.001
Body weight (kg)	27	−1.72	−0.95	0.61 (241.7*)	0.25	−3.78	<0.001
Fat (%)	18	−1.52	−0.63	0.66 (222.0*)	0.35	−1.81	0.035
BMI (kg m^{-2})	16	−0.67	−0.28	0.20 (99.0*)	0.13	−2.16	0.015
SBP (mm Hg)	14	−4.16	−1.06	6.21 (256.1*)	2.23	−0.48	0.316
DBP (mm Hg)	12	−2.63	−1.54	1.37 (67.9*)	0.79	−1.95	0.026

Weighted TE, weights obtained using the inverse of the individual TE variances.
SD_b = Between-study standard deviation of TEs, together with Cochran's *Q* statistic. Note that all Cochran's *Q* statistics were significant ($P < 0.01$).
SD_t = Weighted mean TE standard deviation, obtained by combining the weighted TE variance with the between study TE variance.
z = standardized weighted mean TE.
P = level of significance of *z*, assuming a normal distribution.

Discussion

This meta-analysis attempted to determine the changes in selected cardiovascular risk factors following a walking programme in sedentary individuals. The findings suggest that walking is sufficient stimulus to increase cardiovascular fitness, reduce body weight, BMI and body fat, and decrease resting diastolic blood pressure in previously sedentary but otherwise healthy individuals.

Cardiovascular fitness

Cardiovascular fitness has been shown to be an independent risk factor for cardiovascular disease and indeed a stronger risk factor than physical activity level alone (Blair and Jackson, 2001). Maximum oxygen uptake (VO2 max) is regarded as the gold standard for the assessment of cardiovascular fitness. VO2 max was measured or predicted in 13 of the studies reviewed. All of these studies reported an increase in VO2 max ranging from 0.6 to 6.9 ml kg−1 min−1. When the data were pooled we observed a statistically significant weighted mean TE of 2.7 ml kg−1 min−1 (0.35 ml kg−1 min−1). This improvement in functional capacity is likely to result in greater ease of performance of everyday physical activities and improved quality of life. In addition, such an increase in maximal oxygen uptake may equate to important reductions in CVD mortality risk (Blair et al., 1995). Although some epidemiological studies have suggested that walking may not be a sufficient stimulus to increase cardiovascular fitness (Lee and Paffenbarger, 1997), it is likely that

walking speed and the baseline level of cardiovascular fitness, will determine whether a walking programme will result in a significant increase in VO2 max. The exercise intensity of the walking interventions ranged from 50% to 85% of maximum Heart Rate Reserve which falls within ACSM recommendations for improvement of cardiovascular fitness (American College of Sports Medicine, 2006). It would appear therefore that for sedentary but otherwise healthy middle-aged individuals, walking at a self-selected brisk pace can result in modest but meaningful increases in VO2 max.

Body weight, BMI and body fat

Overweight and obesity increases the risk of many diseases including cardiovascular disease (Rashid et al., 2003). Body weight is one of the simplest methods of assessing overweight and obesity. In the current review 18 studies reported mean body mass at baseline and post intervention. Fourteen of these studies reported decreases in body mass ranging from 0.2 to 2.0 kg following the walking programme. In the studies selected for this review, walking was the only change (no dietary change) and weight loss was not a program goal so it is likely that this reduction is the result of the increased energy expenditure associated with walking. Although the reduction in body weight is modest, it may represent an important reduction in cardiovascular risk (Lavie and Milani, 1997). In all but 1 study the body weight of the control group increased (range +0.1 to +4.2 kg). This highlights the role of walking in opposing age-related weight gain an important strategy in the management of cardiovascular risk (Christou et al., 2005).

Body mass index (BMI) which is a simple indicator for weight for height which commonly used to determine overweight and obesity. At baseline, subjects in the studies included in this analysis ranged from normal weight to obese with the mean reported BMI indicating overweight ($\geqslant$25). In 16 of the studies included in this review BMI was reported before and

after walking interventions. A significant mean weighted TE of −0.28 kg m−2 (0.13 kg m−2) was represents a modest reduction in BMI, which on an individual basis, may have little effect in altering cardiovascular risk. The modest reductions in both body weight and BMI may also illustrate the role that a walking programme plays in the maintenance of lean body mass. Decreases in BMI resulting from an exercise programme are likely to be due to a reduction in fat mass and therefore potentially more significant in terms of cardiovascular risk than equivalent reductions due to calorie restriction which can reduce lean body weight.

Twelve studies utilised skinfold measurements to estimate changes in percentage of body fat from baseline to post intervention. Of these, 11 showed a decrease in percentage body fat ranging from 0.2% to 2.5%. Although skinfold measurements provide an estimate of the total amount of subcutaneous body fat, it provides little information on visceral fat which is known to be particularly important in terms of cardiovascular risk (Despres et al., 2001). Three of the studies included in this meta-analysis measured waist circumferences, a practical assessment method that has been used as a proxy measure of abdominal fat distribution (Hardman et al., 1992; Murphy and Hardman, 1998; Stensel et al., 1994). However only one of these studies report reductions in waist circumference as a result of walking (Murphy and Hardman, 1998).

Blood pressure

Elevated resting blood pressure significantly increases cardiovascular disease risk (He and Whelton, 1999). Estimates suggest that on a population level even small reductions in resting diastolic blood pressure of 2mmHg can reduce CHD risk by 6% (Cook et al., 1995). A recent meta-analysis focusing on the effects of walking on resting blood pressure noted reductions in both systolic and diastolic blood pressure of the magnitude of 2% (Kelley et al., 2001). In the current review data pooled from the 9 studies that included blood pressure measurement before and after walking, intervention revealed no effect on systolic but there was a

significant reduction in diastolic blood pressure (weighted mean TE −1.5 mm Hg (0.8 mm Hg)). This represents a relative reduction of 3.4%. Given that at baseline the weighted mean blood pressure was 127mm Hg (11.7mmHg) systolic and 77.7 mm Hg (4.5 mm Hg) diastolic, the reduction underlines the potential of a walking programme in reducing diastolic blood pressure even in individuals whose blood pressure is already within normal range. The association between blood pressure and cardiovascular risk has no lower threshold and therefore reductions of this magnitude in individuals with normal blood pressure at baseline may still have clinical significance (Mcinnes, 2005).

Total walk volume

Current physical activity recommendations have been translated to suggest that for health benefits, adults should undertake 30 min of activity on at least 5 days week−1. Not all studies included in this analysis fulfilled this threshold of 150 min week−1.We were interested to see if pooling data from studies which employed amounts of walking below this 150 min week−1 would yield different TEs than those employing an amount of walking above this threshold. In six of the studies included in this review, participants walked between 60 and 150 min week−1. Two studies compared interventions which were less and more than this 150-min threshold (Keller and Trevino, 2001; Woolf-May et al., 1999). In the remaining 17 studies participants walked more than 150 min week−1 (range 150–325 min week−1). Random effects models revealed no difference in weighted TEs for any of the measured variables between participants who walked less than 150 min week−1 and those who walked more than 150 min week−1. Given the hypothesised dose–response relationship between the volume of physical activity and health benefits (Haskell, 1994), these findings are perhaps somewhat unexpected. Closer examination of the relative intensity of brisk walking may help to explain this finding. Studies which employed the lower-volume walking interventions tended to prescribe walking at a higher relative intensity (70–85% HRR) compared to those using

higher volumes of walking (55–75% HRR). The notion is that for health benefit there is an intensity vs. volume trade-off. In other words, more intense exercise performed for a shorter duration confers the same benefit as lower intensity exercise performed for longer periods. A prospective study by Manson and colleagues supports this notion. Women who walked 3 h week−1 and those who exercised vigorously for 1.5 h week−1 showed a similar (30– 40%) reduction in CHD (Manson et al., 1999).

Limitations

The meta-analysis presented is not without limitations. The predominance of female subjects in the walking studies included in this analysis may indicate the intuitive appeal that this form of activity has for women. Many physical activity surveys report higher levels of inactivity in women at most ages compared to men (Activity and Health Research, 1992). Indeed, the cited rationale for several of the studies in this analysis includes reference to the role of walking in overcoming inactivity among females. Provided that walking is performed at the same relative exercise intensity, it is not implausible to suggest that similar cardiovascular benefits might accrue for men.

All of the studies included in the current analysis reported or inferred subject drop out from initial recruitment to post-intervention testing. Analysis based upon those who successfully completed the brisk walking intervention, rather than an ‘intention to treat’ approach is a weakness in the literature and reduces the degree to which our findings can be applied to a population setting.

The risk factors included in the present analysis represent the traditional markers of cardiovascular disease. More recently, markers of inflammation, oxidative stress and coagulation have been identified as potentially potent indicators of risk of a cardiovascular

event (Maas and Boger, 2003). Although it is possible that walking would affect these risk factors, few studies have been conducted to investigate these effects.

Conclusion

Despite the limitations described above, the results of this study provide evidence that healthy but sedentary individuals who take up a programme of regular brisk walking will improve several cardiovascular disease risk factors. It reinforces the centrality of walking in health promotion and underlines the efficacy of this type of physical activity for enhancing health among the sedentary majority.

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