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The Effect of 24 Weeks of Moderate Intensity Walking upon Metabolic Syndrome Risk Factors in Previously Sedentary/Low Active Men

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ABSTRACT

Woolf-May K, Scott A, Kearney E, Jones DW. The Effects of 24 Weeks of Moderate Intensity Walking upon Metabolic Syndrome Risk Factors in Previously Sedentary/Low Active Men. **JEPonline** 2011;14(4):145-156. This study determined the effects of 24 weeks of walking on risk factors and insulin sensitivity associated with metabolic syndrome (MetS) in men. Forty-eight (28.4±3.2 BMI) sedentary/low-active men were randomly selected into controls (n=19, 52.4±8.0) or walkers (n=29, 54.9±8.0 yrs). Over a 24-week period, 156.4±26.5 min-walking-wk⁻¹ was performed in 7.2±2.9 sessions-wk⁻¹ for a mean of 25.1±10.3 min-session⁻¹ at an estimated relative VO₂ max of 50.6±9.1%. GLM statistical analysis using the baseline values as a covariate was employed. Bonferroni correction set significance at P<0.006. Significant post-intervention changes were observed in waist circumference (WC) (walkers -2.0±2.7 vs. controls +1.3±3.1 cm, P<0.0001); waist/hip (W/H) ratio (-0.02 ±0.03 vs. +0.01±0.03, P<0.0001), serum insulin (-1.7±1.5 vs. +2.0±6.0, P<0.006 μU·ml⁻¹), and insulin sensitivity index (ISI) (+2.0±6.0 vs. -0.4±0.7, P<0.002 M·mU⁻¹·L⁻¹, respectively). The post-intervention changes: insulin correlated with WC (R=0.441; P<0.002); ISI negatively with WC (R=-0.433; P<0.003), indicating the changes in body composition to have affected insulin metabolism and insulin sensitivity. The men who walked at an easily attainable intensity for most mobile adults of ~51% of relative VO₂ max showed reduced WC and enhanced insulin sensitivity, thus reducing their risk of MetS and type II diabetes.

Key Words: Insulin Sensitivity, Waist Circumference, Risk Factors

INTRODUCTION

Metabolic syndrome (MetS) is characterized by insulin resistance in the presence of obesity and high levels of abdominal fat, blood glucose, insulin, cholesterol and triglycerides as well as low levels of high-density lipoprotein cholesterol and high blood pressure. For men, MetS is defined when an individual possesses three or more of the following risk factors: waist circumference (WC) >40 inches (102 cm); fasting blood triglyceride (TG) levels $\geq 150 \text{ mg}\cdot\text{dL}^{-1}$ ($1.69 \text{ mmol}\cdot\text{L}^{-1}$) and high-density lipoprotein cholesterol (HDL-C) $\leq 40 \text{ mg}\cdot\text{dL}^{-1}$ ($1.04 \text{ mmol}\cdot\text{L}^{-1}$); blood pressure (BP) ≥ 130 systolic and diastolic 85 mmHg and fasting blood glucose $\geq 110 \text{ mg}\cdot\text{dL}^{-1}$ ($6.1 \text{ mmol}\cdot\text{L}^{-1}$) (1). Individuals with MetS are three times as likely to suffer a myocardial infarction or stroke and twice as likely to die from either. They are also at a fivefold greater risk of developing type II diabetes (37).

Metabolic syndrome is extremely common in western societies (10) and likely to increase with the observed rising rates of obesity (32). Causes of MetS have been recognized to involve factors such as ageing, a pro-inflammatory state, hormonal changes, and physical inactivity; all of which may vary depending upon heredity factors and ethnic origin (17,34). Yet, despite high levels of moderate intensity physical activity and aerobic fitness being inversely associated with MetS (18), there is still little empirical research to determine the direct effect of physical activity or walking intervention on reducing MetS. This is especially the case given the current criterion factors in a single study (17,21,22,27). Given that walking is an easily accessible mode of exercise for the majority of the population, the purpose of this investigation was to determine the effects of 24 weeks of walking (5, 31) on risk factors and insulin sensitivity associated with metabolic syndrome (MetS) (37) in men.

METHODS

Subjects and Recruitment

Volunteers were recruited over 13 months via posters, university website, and local media. They were provided with an information sheet and required to sign informed consent. Their suitability was determined through medical and physical activity/lifestyle screening questionnaires, and their general practitioner's approval. Inclusion criteria allowed men to participate if they engaged in <30 min of accumulated physical activity 5 days of the week, were non-diabetic, non-smoking, free of known cardiovascular disease, with a waist/hip ratio ≥ 0.87 , between 38 and 75 yrs of age, and understood the purpose of the study. Eighty-seven men volunteered, after screening 49 remained. They were randomly selected (using computer generated sequences) into either group controls or walkers. The results presented are from the 48 participants who completed the study, which was approved by the local NHS Research Ethics and Canterbury Christ Church University (CCCU) Faculty Research Ethics Committees.

Variables of Interest

Variables of interest were collected pre- and post-intervention in the Sport Science Laboratory at (SSL) CCCU. These included body mass (BM), waist circumference (WC), hip circumference (HC), waist/hip (W/H) ratio, and samples of fasted venous blood. The venous blood was used to determine insulin and insulin resistance (insulin sensitivity index, ISI), fibrinogen, total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Measures of systolic (SBP), diastolic blood pressure (DBP), and graded treadmill-walking test to predict maximal aerobic capacity (VO_2) were also taken as well as data from walking diaries to determine walking intensity, duration, and compliance.

Aerobic Fitness and Walking Intensity

All participants were familiarized with the treadmill (Mercury Med., HP Cosmos, Nussdorf-Traunstein, Germany) and protocol (Naughton-Balke) on a separate occasion prior to the main tests. Participants were requested not to eat or drink any liquid with caffeine or carbohydrate (water was permitted) at least 2 hrs before the treadmill test or to consume alcohol or perform rigorous physical activity within the previous 24-hr period. After seated for at least 5 min, pre-test blood pressure was measured from the right arm using an aneroid sphygmomanometer (Accoson Limpet, A.C. Cossor & Son Ltd, London, UK). Height and BM were determined using the stadiometer and clinical scales (Seca 052466, Germany), respectively, while participants wore similar sporting clothing on both occasions. Throughout the test each subject wore a facemask (Large 7400 Vmask series oro-nasal with custom adapter for Jaeger Triple-V turbine pneumotach). Expired air was measured breath-by-breath and analyzed every 5 secs (Oxycon Pro, Jäeger, Würzburg, Germany). Oxygen uptake (VO_2 max in $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) was estimated by extrapolating HR versus VO_2 to the age predicted maximum HR of 220-age. Ratings of perceived exertion (RPE) were taken at the end of each 2-min stage (3). Gross energy expenditure at particular walking speeds was calculated from absolute VO_2 (e.g., 5 kcal per liter of VO_2).

Anthropometric Measures

While each subject stood in an anatomical neutral position, waist circumference was measured at the umbilicus level and HC at largest point above the gluteal fold to the nearest 1 mm using a self-tightening circumference tape measure (Seca, Hamburg, Germany). The mean of three measures was taken, and recorded (intra-subject CV 0.5% and between 9.9%); W/H was subsequently derived.

Blood Samples

Each participant's blood was taken following an overnight fast (~14 hrs), collected into vacutainers (BD Systems, Oxford, UK) and dispensed into one tube to form serum (containing 0.105 mm sodium citrate at a ratio of 9 parts blood to 1 part anticoagulant), and two others to form plasma (containing fluoride/oxalate). The samples were then centrifuged at room temperature (Eppendorf 5804, Hamburg, Germany) and stored at $-70\text{ }^\circ\text{C}$ (Revco Ultra, Asheville, NC, USA) for later analysis. The sera were then analysed for TG, TC and HDL-C (all Horiba ABX Diagnostics, Cambridge) using enzymatic colorimetric analyses. From these data LDL-C was calculated (11). The plasma was analyzed for glucose (Horiba ABX, Cambridge). The plasma collected in the sodium citrate tubes were analyzed for fibrinogen (4) at Kent and Canterbury Hospital. Insulin was analyzed from serum using chemiluminescence (Bayer Advia Centaur, Bayer Healthcare, Diagnostics Division, Tarrytown, NY), and ISI (26) was determined at QEQM Hospital, Margate. The samples were analyzed within the same assay series to reduce variability within the biochemical analytical procedure, and were corrected for changes in plasma volume (6). The intra assay coefficient of variations, using low concentration quality control (QC) sera were: TC, 3.82%; HDL-C, 6.90%; TG, 8.04%; glucose, 2.24%; insulin, 1.75%; and fibrinogen, 4.18%.

Intervention

All participants were requested not to significantly change their lifestyle or diet over the 24-week period (i.e., other than the walking for the 'active' group). Walkers were required to walk for 30 min in bouts of no <10 min per session, on at least 5 days of the week, at a 'brisk' pace (defined as an RPE and HR equivalent to around 65% of each individual's HR max). Participants could choose how, where, and when they walked. Though not supervised, walkers were regularly contacted and provided with a diary in which to record the date, duration (min), and intensity (RPE and HR from the wrist) of each session. While acknowledging the limitations of this approach, it was chosen to

achieve a greater level of ecological validity. Using these data and the data from the treadmill test, it was possible to gain an indication of walking intensity for each walker.

Statistical Analysis

Power analysis was performed to determine that an active group of $n=29$ would be sufficient to show post-intervention changes in the variables of interest and would achieve 90% power at an alpha of 0.05. Minitab statistical package (version 15) was employed with variability of data within a distribution given as one standard deviation (mean \pm SD). Bonferroni correction factor was applied, moving the alpha level of significance to 0.006 for between group differences. Data that lacked normal distribution underwent natural logarithmic transformation. Baseline data between groups were compared using a one-way analysis of variance (ANOVA). Post-intervention between group changes were compared employing the Generalised Linear Model (GLM) using baseline data as a covariate. Pearson's Product Moment correlation and multiple regression analyses were used to explore relationships between factors (with an alpha 0.05 level of statistical significance). Statistically significant relationships showing a correlation of less than $R=0.400$ are not reported.

RESULTS

Subject Characteristics

At baseline the two groups did not differ significantly for: (a) age (controls, $n=19$, 52.4 ± 8.0 , range 40 to 69 vs. walkers, $n=29$, 54.9 ± 8.0 , range 38 to 73 yrs, $F=1.17$, $P=0.285$); (b) height (1.77 ± 0.05 , range 1.66 to 1.89 vs. 1.79 ± 0.06 , range 1.69 to 1.92 m, $F=1.60$, $P=0.212$); (c) BMI ($n=19$, 29.3 ± 3.4 , range 23.1 to 34.9 vs. $n=29$, 27.8 ± 3.1 , range 22.0-33.5, $F=2.49$, $P=0.122$, respectively); (d) baseline physical activity levels (i.e., physical activity at >4 METs, controls 0.63 ± 0.68 vs. walkers 0.84 ± 0.61 hrs \cdot wk $^{-1}$, $F=1.16$, $P=0.284$; indicating their pre-intervention levels of physical activity to be sufficiently low); and (e) BM or any of the other measured variables (refer to Tables 1 and 2). The mean group values were within the normal ranges for blood lipids, fibrinogen, glucose, insulin and ISI, and within the 'good' classification for aerobic capacity. Blood pressure readings were at the upper end for normal values, as were WC and subsequent W/H ratio values (Tables 1 and 2). Thus, the participants were considered pre-obese with borderline MetS.

Metabolic Syndrome

At baseline controls and walkers possessed a total of $n=42$ and $n=57$ MetS risk factors respectively, with $n=8$ (42%) and $n=9$ (31%) that would be classified as possessing MetS (1). There was little change post-intervention with a total of $n=44$ and $n=53$ risk factors, and those classified with MetS being $n=9$ (47%) and $n=8$ (28%), respectively.

Pre-Intervention

Combined data from all participants showed that at baseline serum insulin levels correlated with BM ($R=0.586$; $R^2=0.32$; $P\leq 0.0001$), WC ($R=0.518$; $R^2=0.27$; $P\leq 0.0001$) and HC ($R=0.498$; $R^2=0.25$; $P\leq 0.0001$); and ISI negatively with BM ($R=-0.470$; $R^2=0.22$; $P\leq 0.001$), WC ($R=-0.463$; $R^2=0.22$; $P\leq 0.001$) and HC ($R=-0.410$; $R^2=0.17$; $P\leq 0.001$).

Post-Intervention

Insulin correlated with BM ($R=0.557$; $R^2=0.31$; $P\leq 0.0001$), WC ($R=0.519$; $R^2=0.27$; $P\leq 0.0001$), HC ($R=0.486$; $R^2=0.24$; $P\leq 0.001$) and W/H ratio ($R=0.403$; $R^2=0.16$; $P\leq 0.005$); and ISI negatively with BM ($R=-0.432$; $R^2=0.19$; $P\leq 0.003$) and WC ($R=-0.406$; $R^2=0.17$; $P\leq 0.005$). Estimated VO_2 max negatively correlated with SBP ($R=-0.424$; $R^2=0.18$; $P\leq 0.003$).

Table 1. Pre- and post-intervention values for anthropometric measure, VO₂ max, and blood pressure; mean±SD (range).

Variable	Controls (n=19)			Walkers (n=29)			Between group P (F value)
	Pre	Post	Mean difference	Pre	Post	Mean difference	
BM (kg)	91.7±10.6 70.4-106.5	92.0±10.6 71.0-107.8	+3.5±3.1	89.1±9.1 69.0-103.0	88.4±9.4 67.7-103.7	-0.7±1.6	0.148 (2.16)
WC (cm)	102.5±9.5 83.0-119.2	103.8±9.6 84.5-118.0	+1.3±3.1	101.2±8.0 82.2-113.0	99.2±8.4 80.0-111.5	-2.0±2.7*	0.0001 (15.27)
HC (cm)	105.4±5.4 95.0-112.5	104.8±5.4 95.5-115.5	-0.6±2.7	104.4±5.0 93.2-114.5	103.8±4.7 92.5-112.5	-0.6±2.1	0.872 (0.03)
W/H ratio	0.97±0.06 0.87-1.07	0.99±0.07 0.88-1.11	+0.02±0.3	0.95±0.05 0.87-1.05	0.95±0.05 0.86-1.04	-0.02±0.03*	0.000 (19.82)
*VO₂ max (mL·kg ⁻¹ ·min ⁻¹)	35.6±7.5 22.4-51.9	35.9±6.2 24.8-50.7	+0.3±2.7	37.3±6.2 26.8-59.5	39.5±5.6 29.7-52.0	+2.2±3.5	0.011 (7.09)
SBP (mmHg)	132.3±15.4 105.0-163.3	129.2±15.1 110.0-166.7	-3.1±9.3	129.2±14.4 103.3-150.0	122.7±12.6 96.7-145.0	-6.5±10.2	0.119 (2.53)
DBP (mmHg)	87.7±8.2 75.0-101.7	87.2±10.1 75.0-110.0	-0.6±8.2	84.3±9.5 70.0-100.0	82.6±9.2 70.0-108.3	-1.7±6.1	0.322 (1.00)

*Statistically significantly different from controls as determined by GLM using baseline values as a covariate. Bonferroni adjustment factor applied shifting statistical significance to the alpha level to 0.006. #Estimated VO₂ max

Post-Intervention Changes

Statistical significant differences were observed in the amount of post-intervention change in WC, W/H ratio (Table 1), serum insulin and ISI values (Table 2). The amount of change in insulin also significantly correlated with the amount of change in BM ($R=0.412$; $R^2=0.17$; $P\leq 0.004$) and WC ($R=0.441$; $R^2=0.19$; $P\leq 0.002$); and negatively with the amount of change in ISI with WC ($R=-0.433$; $R^2=0.19$; $P\leq 0.003$). Furthermore, baseline measures significantly affected post-intervention degree of change for insulin ($P=0.0001$), fibrinogen ($P\leq 0.0001$), HDL-C ($P\leq 0.0001$), glucose ($P\leq 0.0001$), SBP ($P=0.002$) and estimated VO₂ max ($P\leq 0.0001$). Additionally, the number of walking sessions.wk⁻¹ (Table 3) correlated with degree of change in WC ($R=0.494$; $R^2=0.24$; $P\leq 0.006$). There were no other significant changes or meaningful correlations.

DISCUSSION

Walking Intervention

It was important that the walking intervention was as ecologically valid as possible. As such, the participants were guided to walk at a 'brisk' intensity of about 65% of their estimated HR max. The mean weekly volume of walking performed over the 24 weeks was (156.4±26.5 min.wk⁻¹) within the range recommended by the government (5,31). The walkers recorded a mean walking intensity at an RPE of 11.9±1.4, which related to somewhere between 'fairly light' and 'somewhat hard' on the Borg scale (3). They also walked at 65.2±6.9% of their estimated HR max, and at 50.6±9.1% of their estimated VO₂ max, which is at the upper end of 'light' and within the lower end of the 'moderate' intensity exercise.

Table 2. Pre- and post-intervention measures for blood lipids, insulin resistance and prothrombotic state; mean±SD (range).

Variable	Controls (n=19)			Walkers (n=29)			Between group P (F value)
	Pre	Post	Mean difference	Pre	Post	Mean difference	
TC (mmol·L ⁻¹) C = 18; W = 28	5.4±1.2 2.0-7.1	5.4±1.0 3.7-7.8	-0.5±0.9	5.3±1.0 3.1-6.8	5.1±1.0 3.2-7.4	-0.2±0.7	0.03 (0.16)
TG (mmol·L ⁻¹) W = 28	1.3±0.4 0.6-2.2	1.6±0.8 0.6-4.0	+1.3±0.8	1.3±1.3 0.7-2.3	1.3±1.3 0.43-2.4	-0.03±0.04	0.054 (3.91)
HDL-C (mmol·L ⁻¹)	1.2±0.6 0.4-3.1	1.2±0.4 0.7-2.4	-0.01±0.7	1.2±0.4 0.43-2.13	1.3±0.6 0.52-2.87	-0.1±0.6	0.54 (0.37)
LDL-C (mmol·L ⁻¹) W = 28	3.6±1.0 1.3-5.5	3.5±0.9 0.8-4.8	-0.1±0.8	3.5±0.9 1.6-4.9	3.2±1.1 0.6-5.4	-0.2±0.5	0.574 (0.43)
Glucose (mmol·L ⁻¹) C = 18; W = 28	5.7±0.9 4.2-8.5	5.8±0.7 4.0-7.9	+0.1±0.6	5.4±0.7 3.5-7.2	5.7±0.8 4.4-8.9	-0.3±0.6	0.814 (0.06)
Insulin (μU·mL ⁻¹) W = 27	12.7±12.3 6.0-26.8	14.7±5.7 5.6-23.6	+2.0±6.0	11.7±10.6 3.3-32.3	10.0±9.7 3.1-24.6	-1.7±1.5*	0.006 (8.40)
ISI (M·mU ⁻¹ ·L ⁻¹) W = 27	10.0±0.7 8.7-11.4	9.6±1.0 8.2-12.0	-0.4±0.7	10.2±0.6 9.0-11.3	10.4±0.8 9.3-12.7	+0.2±0.3*	0.002 (10.82)
Fibrinogen (g·L ⁻¹) C = 17; W = 25	2.9±2.8 1.5-5.7	3.0±3.0 2.0-4.4	+0.1±0.7	3.1±3.1 2.4-4.1	2.9±2.9 1.9-4.0	-0.18±0.2	0.206 (1.65)

C = controls, W = walkers. * Significantly different amount of change compared to C as determined by GLM, using baseline values as a covariate. Bonferroni adjustment factor applied shifting statistical significance to the alpha level to 0.006. Due to technical difficulties some of the subject numbers may differ in some of the variables. Therefore, where group sizes differ, group numbers are provided.

Interestingly the participants of this study did however walk at a lower intensity (~51% VO₂ max) than middle-aged men and women of other walking intervention studies (range 65% to 67% VO₂ max) (29,40,41). Furthermore, the intensity of our walkers was generally lower than a group of older women (n=9, aged 75 to 83 yrs) and slightly greater than a group of younger women (n=9, age 20-23 years) who, when asked to walk briskly, walked at ~60% and ~72% of HR max, respectively (9). Adherence to the regimen was relatively high ranging from around 75% to 144% (113 min to 216 min) of the walking duration prescribed.

It is of interest that the number of walking sessions per week correlated with change in WC (R=0.494, P=0.006) with no significant relationship being seen from the 'total minutes walked' or 'mean time of each walking session' within the whole program. Therefore, this suggests that breaking the walking sessions into shorter bouts might have some favourable effect. But, it is important to keep in mind that there were no other strong significant correlations to suggest shorter sessions produced more favourable changes in any of the other measured variables. This latter point seems to agree with Hardman (14), who suggested that neither single nor multiple bouts of exercise of similar volume produced more favourable changes.

Table 3. Walking diary and derivative data; mean±SD (range).

Variable	Walkers (n=29)
Weeks walked	23.7±0.7 (21-24)
Total min walked	3699±640 (2710-5167)
Mean min walked·wk ⁻¹	156.4±26.5 (112.9-220.6)
Percentage of prescribed walking achieved	102.8±17.8 (75.3-143.5)
RPE	11.9±1.4 (9-16)
Palpated HR (beats·min ⁻¹)	108.5±18.5 (75.1-176.3)
Walking intensity % estimated HR max	65.2±6.9 (50.7-78.4)
Walking intensity % estimated VO ₂ max	50.6±9.1 (31.4-65.8)
Estimated walking gross energy expenditure kcal·wk ⁻¹	1314.5±395.8 (511.4-2284.5)
Estimated walking gross energy expenditure MJ·wk ⁻¹	5.6 ±1.7 (2.2-9.7)
Sessions·wk ⁻¹	7.2±2.9 (4.2-15.0)
Session duration (min)	25.1±10.3 (10.1-49.7)

Metabolic Syndrome Related Risk Factors

The walking intervention did not appear to have much impact upon the majority of the risk factors associated with MetS. Perhaps, it is important to remember that the walking intensity was relatively low (50.6±9.1%) compared to other walking intervention studies. Nonetheless, the walking group did significantly reduce their WC, which was accompanied by a reduction in serum insulin and associated increase in ISI. The findings imply that for the 'at risk' men, an additional 1.4 hrs·wk⁻¹ of moderate intensity regular walking, above their baseline physical activity levels, decreased their risk of developing MetS and type II diabetes. In contrast however, the control group actually increased their WC over the intervention period, and showed a reduction in insulin sensitivity (see Table 2). Even though all participants were requested not to change their diet or other physical activity over the intervention, there is always a possibility when dealing with human subjects that this did occur. This is difficult to fully control, and is continually a potential limitation of any study of this type.

Notwithstanding, it was apparent that the observed changes in body composition were linked to the changes in insulin sensitivity. Insulin levels and insulin sensitivity have been shown to change with alterations in fat mass (FM) (24), fat free mass (FFM) (13,30), the ratio of FM/FFM (23), and adaptations in skeletal muscle due to exercise training (16). The link between blood insulin and FM is seen most notably in obese individuals who commonly suffer from hyperinsulinaemia (24). The participants of this study were generally pre-insulin levels and ISI were within the normal range (ISI of ≤ 6.3 M·mU⁻¹·L⁻¹ defined as insulin resistance), and the established relationships between WC, ISI and serum insulin levels were apparent. Given that both groups showed no significant change in BM over the intervention, the findings would suggest an increase in FFM for the walkers and an increase in FM for the controls. Insulin sensitivity has been found to increase when FFM is enhanced (30) and becomes reduced when there is a loss (13). This would corroborate with the increase in insulin sensitivity in the walkers and the reduction in this factor for the controls.

Similar to our study, other researchers have also found reductions in fasting insulin after exercise intervention (7,12,15), which is often associated with adaptations in the trained muscle that results in enhanced insulin sensitivity (16,33). Hickey et al. (15) found reductions in plasma insulin levels in a group of 5 morbidly obese women after 7 days of cycle ergometry and treadmill walking at 65% VO_2 peak for 60 $\text{min}\cdot\text{d}^{-1}$). Their finding was reported as being unrelated to changes in body composition or glucose tolerance. Likewise, Donnelly et al. (7) observed comparable reductions in serum insulin to the findings in the present study. Their subjects consisted of 22 previously sedentary moderately obese women who exercised for 18 months via walking at 60 to 75% VO_2 max with no significant changes in either blood glucose, FM or FFM. This suggests that muscle adaptations to be the cause of the changes in insulin. The data from the present study would however suggest that the increase in insulin levels and reduced ISI for the controls was likely due to the increase in their WC while the enhanced ISI, reduced insulin levels and WC seen in the walkers to be a probable combination of skeletal adaptations from the walking intervention and associated changes in WC, and subsequent ratio of FM/FFM. Given the significance of the relationships between insulin resistance in the presence of abdominal obesity to MetS, and the increased insulin resistance with an enhanced risk of myocardial infarction (38), the outcomes of this study are therefore of interest.

Blood Pressure, Blood Lipids, and Fibrinogen

It is clear that other than the changes stated above the exercise intervention did not have a statistically significant effect on the other measures of MetS. Also, while it is popular to argue from a clinical rather than statistical perspective (i.e., despite the lack of statistical significance the mean change in SBP and DBP for the walkers is important), the fact remains that the intervention did not produce a significant difference in blood pressure. Therefore, it is scientifically incorrect to assume that the walkers would be in position to change their blood pressure classification. Blood lipids also showed no statistically significant change, despite other researchers having observed changes in blood lipids from similar numbers of older aged subjects after exercise intervention of similar intensity (28,36). The findings of this study also showed mean 9% reduction in LDL-C for the walkers compared to 3% in the controls. However, once again, while a 10% reduction in LDL-C has been shown to reduce cardiovascular death by around 10% and cardiovascular events by 25% (25), the statistical fact is that there were no differences in blood lipids between the groups after the intervention.

It is also known that for an increase in HDL-C to occur the gross weekly energy expenditure through physical activity should be in the range of 1200 to 2200 $\text{kcal}\cdot\text{wk}^{-1}$ (~ 4.0 - 9.2 MJ) (8). Considering that the subjects in the walking group engaged in an estimated weekly gross energy expenditure within this range (Table 3), the lack of significant change might be due to the fact that generally their pre-intervention blood lipid profiles were reasonably good. This thinking is in agreement with reports by other researchers, who have argued that pre-intervention blood lipid profiles limit their potential for change (36,40). Furthermore, the subjects walked at around 51% VO_2 max, which was considerably lower than 75% VO_2 max, which has been shown to induce changes in blood lipid profile (42). It would also appear that in order for favourable changes in blood lipids to occur, when baseline values are relatively good, previously low sedentary/low active men may require exercising at a subjective intensity above that of a "brisk" intensity. The lack of statistical change in fibrinogen is more difficult to determine, since research findings in this area have proven to be varied and equivocal (2,19,42,43). However as with the blood lipids our data showed that baseline levels appear to influence the degree and potential for change after the walking intervention.

CONCLUSIONS

Given the importance of insulin resistance in the presence of abdominal obesity in relation to MetS and type II diabetes, the findings of this study demonstrate that for previously sedentary/low active men who engage in 30 min of walking throughout the day on at least 5 days of the week at an easily attainable intensity for most adults (~51% VO₂ max), they can reduce their risk of MetS and type II diabetes.

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REFERENCES

1. ATP III. **National Cholesterol Education Program III expert panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults**. US: National Cholesterol Education Program, National Heart, Lung and Blood Institute, National Institutes of Health, Publication, May, pp. 01-3670, 2001.
2. Banz W J, Maher MA, Thompson WG, Bassett DR, Moore W, Ashraf M, Keefer DJ, Zemel MB. Effects of resistance versus aerobic training on coronary artery disease. **Experi Biolo & Med** 2003;228(4):434-440.
3. Borg GA. Psychophysical bases of perceived exertion. **MSSE** 1982;14(5):377.
4. Clauss A. Gerinnungsphysiologische schnellmethode zur bestimmung des frininogens." **Acta Haemato** 1957;17:237-246.
5. Department of Health (DoH). At least five a week: evidence of the impact of physical activity in relationship to health. A report of the chief medical officer. **DoH**, Reference 2389, p21, 2004.
6. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood plasma and red cells in dehydration. **J Appl Physiol** 1974;37(2):247-248.
7. Donnelly JE, Jacobsen DJ, Heelan KS, Seip R, Smith S. The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. **International Journal of Obes & Rel Meta Dis** 2000;24(5):566-572.
8. Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. **Spor Med** 2001;31(15):1033-1062.

9. Fitzsimons CF, Greig CA, Saunders DH, Lewis SH, Shenkin SD, Lavery C, Young A. Responses to walking-speed instruction: implications for health promotion in older adults. *J Aging & Phys Act J* 2005;13(2):172-183.
10. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey (NHANES). *JAMA* 2002;287(3):356-359.
11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of preparative ultracentrifuge. *Clin Chem* 1972;18:499-502.
12. Greie S, Humpeler E, Gunga HC, Koralewski E, Klingler A, Mittermayr M, Fries D, Lechleitner M, Hoertnagl H, Hoffmann G, Struass-Blasche G, Schobersberger W. Improvement of metabolic syndrome markers through altitude specific hiking vacations. *J Endo Invest* 2006;29(6):497-504.
13. Guillet C, Boirie Y. Insulin resistance: a contributing factor to age-related muscle mass loss? *Diabe & Met* 2005;31(2):S20-5S26.
14. Hardman AE. Issues of fractionization of exercise (short vs long bouts). *MSSE* 2001;33(6):S421-S42.
15. Hickey MS, Gavigan KE, McCammon MR, Tyndall GL, Pories WJ, Israwl RG, Houmard JA. Effects of 7 days of exercise training in insulin action in morbidly obese men. *Clin Ex Phys* 1999;1:24-28.
16. Houmard JA, Shaw D, Hickey MS, Tanner CJ. Effect of short-term exercise training on insulin-stimulated PI 3-kinase activity in human skeletal muscle. *Am J Physiol Endocrinol Metab* 1999;277(6):E1055-E1060.
17. International Diabetes Federation (IDF). Metabolic syndrome. The IDF consensus worldwide definition of the metabolic syndrome. *Diabetes Voice* 2005;50(20):45-47.
18. Irwin ML, Ainsworth BE, Mayer-Davis BJ, Addy CL, Pate RR, Durstine JL. Physical activity and the metabolic syndrome in a tri-ethnic sample of women. *Obes Res* 2002;10(10):1030-1037.
19. Jakicic JM, Wing RR, Butler BA, Robertson RU. Prescribing exercise in multiple short bouts versus on continuous bout: effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. *Int J Obes & Rel Meta Dis* 1995;19(12):893-901.
20. Johnson JL, Slentz CA, Houmanrd JA, Samsa GP, Duscha BD, Aiken LB, McCartney JS, Tanner CJ, Kraus WE. Exercise training amount and intensity effects on metabolic syndrome. *Am J Cardiol* 2007;100(12):1759-1766.
21. Katzmarzyk PT, Leon AS, Wilmore JH, Skinner JS, Rao DC, Rankinen T, Bouchard C. Targeting the metabolic syndrome with exercise: evidence from the HERITAGE family study. *MSSE* 2003;35(10):1703-1709.

22. Kim YH, Yang YP. Effects of walking exercise on metabolic syndrome risk factors and body composition in obese middle school girls." *Taehan Kanho Haknoe Chi* 2005;35(5):858-867.
23. Lear SA, Kohli S, Bondy GP, Tchernof A, Sniderman AD. Ethnic variation in fat and lean body mass and the association with insulin resistance. *Clin Endo Meta* 2009;94(12):4696-4702.
24. Lewis GF, Carpentier A, Adeli K, Giacca A. Disordered fat storage and mobilization in the pathogenesis in insulin resistance and type 2 diabetes. *Endo Rev* 2002;23(2):201-229.
25. Libby P, Theroux P. Pathophysiology of Coronary Artery Disease. *Circulation* 2005;111:3481-3488.
26. McAuley DR, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, Duncan AW. Diagnosing insulin resistance in the general population. *Diabetes Care* 2001;24(3):460-464.
27. Mitsui T, Shimaoka K, Tsuzuku S, Kajioaka T, Sakakibara H. Gentle exercise of 40 minutes with dietary counselling is effective in treating metabolic syndrome. *Tohoku J Experi Med* 2008;215(4):355-361.
28. Motoyama M, Sunami Y, Kinoshita T, Irie T, Sasaki J, Arakawa K, Kiyonaga A, Tanaka H, Shindo M. The effects of long-term low intensity aerobic training and detraining on serum lipid and lipoprotein concentration in elderly men and women. *Euro J App Physiol* 1995;70(2):126-131
29. Murphy MH, Hardman AE. Training effects of short and long bouts of brisk walking in sedentary women. *MESSE* 1998;30(12):152-157.
30. Nam SY, Kim KR, Cha BS, Song YD, Lim SK, Lee HC, Huh KB. Low-dose growth hormone treatment combined with diet restriction decreases insulin resistance by reducing visceral fat and increasing muscle mass in obese type 2 diabetic patients. *Int J Obes & Rel Met Dis: J Int Ass Study Obes* 2001;25 (8)1101-1107.
31. Pate PR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC. Physical activity and public health, A recommendation from the Centre for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273(7):402-407.
32. Prentice AM. The emerging epidemic of obesity in developing countries. *Int J Epidemiol* 2006;35(1):93-99.
33. Ryder JW, Chibalin AV, Zierath JR. Intracellular mechanisms underlying increases in glucose uptake in response to insulin or exercise in skeletal muscle *Acta Physiol Scand* 2001;171(3):249-258.
34. Saad MF, Lilloja S, Nyomna BL, Castillo C, Ferraro R, De Gregorio M, Ravussin E, Knowler WC, Bennett PH, Howard BV. Racial differences in the relation between blood pressure and insulin resistance. *New Eng J Med* 1991;324(11):733-739.
35. Savage MP, Patratis MM, Thomson WH. Exercise training effects on serum lipids or prepubescent boys and adult men. *MSSE* 1986;18(2):197-204.

36. Seals DR, Hagberg JM, Hurley BF, Ehsani AA, Holloszy JO. Effects of endurance training on glucose tolerance and plasma lipid levels in older men and women. **JAMA** 1984;252(5):645-648.
37. Stern M, Williams K, Gonzalez-Villalpando C, Hunt KJ, Haffner SM. Does the Metabolic syndrome improve identification of individuals at risk of type 2 diabetes and/or cardiovascular disease?, **Diabetes Care** 2004;27 (11): 2676-2681.
38. Weikert C, Westphal S, Berger K, Dierkes J, Mohlig M, Spranger J, Rimm EB, Willich SN, Boeing H, Pischon T. Plasma resistin levels and risk of myocardial infarction and ischemic stroke. **J Clin Endo & Meta** 2008;93(7):2647-2653.
39. Wilmore JH. Dose-response: variation with age, sex, and health status. **MSSE** 2001;33(6):S622-S634.
40. Woolf-May K, Kearney EM, Jones DW, Davison RCR, Coleman D, Bird SR. The effect of two different 18-week walking programmes on aerobic fitness, selected blood lipids and factor XIIIa. **J Spor Sci** 1998;16(8):701-710.
41. Woolf-May K, Kearney EM, Owen A, Jones DW, Davsion RC, Bird SR. The efficacy of accumulated short bouts versus single daily bouts of brisk walking in improving aerobic fitness and blood lipid profiles. **Health Ed Res** 1999;14(6):803-815.
42. Stratton JR, Chandler WL, Schwartz TS, Cerqueira MD, Levy WC, Kahn SE, Larson VG, Cain KC, Beard JC, Abrass IB. Effects of physical conditioning on fibrinolytic variables and fibrinogen in young and old healthy adults. **Circulation** 1991;83:1692-1697.
43. Zanettini RD, Bettega O, Agostoni B, Ballestra B, del Rosso G, di Michele R, Mannucci PM. Exercise training in mild hypertension: effects on blood pressure, left ventricular mass and coagulation factor VII and fibrinogen. **Cardiology** 1997;88(5):468-473.

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