Effects of a 12-Month Physical Activity Intervention on Prevalence of Metabolic Syndrome in Elderly Men and Women

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Background. There is a lack of information on whether exercise training alone can reduce the prevalence of metabolic syndrome (MetS) in elderly men and women.

Methods. This study was an ancillary to the Lifestyle Interventions and Independence for Elders Pilot Study, a four-site, single-blind, randomized controlled clinical trial comparing a 12-month physical activity (PA) intervention (N = 180) with a successful aging intervention (N = 181) in elderly (70–89 years) community-dwelling men and women at risk for physical disability. The PA intervention included aerobic, strength, and flexibility exercises, with walking as the primary mode. MetS was defined using the National Cholesterol Education Program criteria.

Results. There was no significant change in body weight or fat mass after either intervention. The trend of MetS prevalence over the intervention period was similar between PA and successful aging groups (p = .77). Overall, the prevalence of MetS decreased significantly from baseline to 6 months (p = .003) but did not change further from 6- to 12-month visits (p = .11). There were no group differences in any individual MetS components (p > .05 for all group by visit interactions). However, in individuals not using medications at any visit to treat MetS components, those in the PA intervention had lower odds of having MetS than those in the successful aging group during follow-up (odds ratio = 0.28, 95% confidence interval = 0.08–0.96).

Conclusions. In this sample, a 12-month PA intervention did not reduce the prevalence of MetS more than a successful aging intervention, perhaps due to the large proportion of individuals taking medications for treating MetS components.

Key Words: Metabolic syndrome—Elderly—Physical activity.

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Metabolic syndrome (MetS) is a condition characterized by the clustering of three or more metabolic abnormalities, including abdominal obesity, hypertriglyceridemia, low high-density lipoprotein cholesterol (HDL-C), hypertension, and high fasting glucose in an individual (1). The presence of MetS is associated with considerable risk for the development of cardiovascular disease, diabetes, disability, and mortality (2–11). MetS prevalence increases with age (12); using the National Cholesterol Education Program criteria (1), it approaches 47% and 58% for men and women older than 70 years in the United States, respectively (12).

Lifestyle interventions are the initial therapies recommended for the prevention, as well as treatment, for MetS (1). There are sizable benefits of weight loss, with and without exercise training, on MetS components in young and middle-aged persons (13–15). The evidence for such a benefit is less strong in elderly people due to the smaller number of studies. Some data on older adults suggest that exercise training combined with weight loss interventions may improve several MetS components (16–19), reduce the number of MetS components (16,19), decrease the incidence of MetS, and eliminate the syndrome in some individuals (19,20). However, not all individuals with MetS are overweight or
obese (21) and would be recommended for weight loss, especially among older individuals. Thus, it would be important to know the benefits of exercise intervention in the absence of weight loss on MetS in elderly individuals.

Unfortunately, only a few studies examined the effects of exercise training alone on MetS in elderly individuals, and results are not consistent. In females older than 65 years, a 12-month high-intensity aerobic and resistance exercise program reduced the number of MetS components (22). A 12-week exercise training reduced abdominal obesity, blood pressure (BP), triglycerides, and insulin resistance in 24 older (>60 years) men and women (17). Additionally, a 6-month resistance and aerobic exercise program reduced the prevalence of MetS and the number of MetS components in older adults (61–65 years) with elevated BP (23). On the contrary, a recent study in men and women (67–76 years) did not show significant metabolic benefit after 12-week exercise training compared with control (24).

Despite this evidence, there is still a lack of information from randomized controlled trials with elderly people, especially those older than 70 years, examining whether exercise training without weight loss is beneficial for simultaneously improving at least three of the MetS components enough to reduce the prevalence of this disorder. The purpose of this analysis was to determine the prevalence of MetS at baseline and following a 12-month physical activity (PA) and a nonexercise successful aging (SA) health education intervention in elderly (70–89 years) men and women. We hypothesized that the PA intervention would decrease the prevalence and severity (individual and number of MetS components) of MetS compared with the SA intervention.

METHODS

Data reported here are from an ancillary study to the Lifestyle Interventions and Independence for Elders Pilot (LIFE-P) Study, a four-site (Wake Forest University, Cooper Institute, University of Pittsburgh, and Stanford University), single-blinded, randomized controlled clinical trial comparing a 12-month PA intervention with an SA intervention in elderly, nondisabled community-dwelling men and women at risk for physical disability (25,26). The institutional review board at each site approved the study, and all study participants gave written informed consent.

Detailed inclusion and exclusion criteria were described previously (25). Briefly, the participants were aged 70–89 years, sedentary, with low functional performance, and willing to be randomized to either treatment group. The exclusions were designed to identify persons for whom PA would be unsafe and those who were unlikely to be able to participate fully in the study because of comorbid conditions or cognitive difficulties. A total of 361 participants had information available to determine the presence of MetS at baseline. Among these, MetS information was available from 332 to 328 participants at the 6-month and 12-month follow-up, respectively.

Interventions

The PA intervention consisted of a combination of aerobic, strength, balance, and flexibility exercises (25). During weeks 1–8 (adoption phase), participants attended center-based exercises (40–60 minutes) three times each week in a supervised setting. During weeks 9–24 (transition phase), center-based exercises reduced to two times and home-based exercises increased to at least three times each week. Week 25 to the end of trial (maintenance phase) consisted of home-based exercise with one optional center-based exercise and monthly phone contacts. The PA intervention included one per week group-based behavioral counseling sessions for the first 10 weeks that focused on PA participation and encouraging participants to increase all forms of PA.

Walking was the primary mode of PA with a goal of walking on 5 or more days of the week and at least 150 minutes/week. Each session was preceded by a brief warm-up and followed by a brief cooldown period. Participants also performed lower extremity strengthening exercises and a stretching regimen. The intensity of training was gradually increased in the first 2–3 weeks. Participants were asked to walk at an intensity of 13 (SOMewhat HARD) based on the Borg’s ratings of perceived exertion scale (27). Strengthening exercises were performed at an intensity of 15–16 (two sets of 10 repetitions).

The SA health education intervention included weekly small group meetings for the first 24 weeks and monthly thereafter. Health topics were relevant to older adults, such as nutrition, medications, recommended preventive services, and screenings at different ages, etc. A 5- to 10-minute instructor-led program of upper extremity stretching exercises was also delivered. Telephone calls were made after each missed session to problem-solve barriers to attendance and to encourage regular participation.

Laboratory Assays

Blood samples were collected in early morning, after a 12-hour fast at baseline, 6, and 12 months. The 6- and 12-month blood samples were collected at least 24 hours after the last exercise session. Blood sampling was postponed in the event of an acute infection. All blood was collected, processed, divided into aliquots, and stored locally at −80°C until shipment to the Wake Forest University, where samples were stored for long term at −80°C until analysis.

The insulin assays were determined using an automated chemoluminescent immunoanalyzer (IMMULITE, Siemens Healthcare Diagnostic, Los Angeles, CA) at the biogerontology laboratory at Wake Forest University. Serum glucose and lipoprotein lipids were measured by the Esoterix Clinical
Trial Services, a Division of LabCorp (Cranford, NJ), using enzymatic method. Homeostasis model assessment (HOMA) score was calculated by fasting plasma insulin (μIU/mL) × glucose (mmol/L)/22.5 (28).

**Body Composition and Other Measurements**

Body weight, height, and waist circumference were measured at each clinic visit. Waist circumference was obtained horizontally at the midpoint between the highest point of the iliac crest and the lowest part of the costal margin in the mid-axillary line. Total body fat mass and lean mass were measured using dual x-ray absorptiometry at baseline and 12 months at three sites (Wake Forest University, Cooper Institute, and University of Pittsburgh).

At each assessment time point, participants were asked to bring to the clinic containers of prescription and nonprescription medicines that they were taking. They were asked about the frequency and dose. Sociodemographic factors including age, race, education, income, and smoking were assessed at the screening interview. The Community Healthy Activities Model Program for Seniors (CHAMPS) was used to assess self-reported PA (29).

**Definition of MetS and Calculation of z-MetS**

The diagnosis of the MetS was based on the criteria of the National Cholesterol Education Program (Adult Treatment Panel III, NCEP ATP III) (1). The MetS was diagnosed when three or more of the following were present: waist circumference greater than or equal to 102 cm in men and greater than or equal to 88 cm in women (≥90 cm in Asian men and ≥80 cm in Asian women), triglycerides greater than or equal to 150 mg/dL or drug treatment for elevated triglycerides, HDL-C less than 40 mg/dL in men and less than 50 mg/dL in women or drug treatment for low HDL-C, fasting glucose greater than or equal to 100 mg/dL or drug treatment for elevated glucose, and systolic BP greater than or equal to 130 mmHg or diastolic BP greater than or equal to 85 mmHg or on antihypertensive drug treatment with a history of hypertension.

MetS z-score, used previously by other investigators (30), was calculated. It takes into account continuous changes in each component, such as a change in triglycerides from 200 to 160 mg/dL that cannot be reflected using the MetS criteria. For example, the formula for non-Asian men was:

\[
\text{z-MetS} = \frac{(40 - \text{HDL-C})}{SD_{\text{HDL-C}}} + \frac{(TG - 150)}{SD_{\text{TG}}} + \frac{(\text{Glucose} - 100)}{SD_{\text{glucose}}} + \frac{(\text{Waist circumference} - 102)}{SD_{\text{WC}}} + \frac{(\text{systolic BP} - 130)}{SD_{\text{SBP}}} + \frac{(\text{diastolic BP} - 85)}{SD_{\text{DBP}}},
\]

where \( SD_{\text{HDL-C}}, SD_{\text{TG}}, SD_{\text{glucose}}, SD_{\text{WC}}, SD_{\text{SBP}}, \) and \( SD_{\text{DBP}} \) were gender-specific SDs for HDL-C, triglycerides, glucose, waist circumference, systolic BP, and diastolic BP, respectively, from all participants at baseline.

**Statistical Analyses**

Statistical analyses were performed using the SAS 9.2 (SAS Institute, Inc., Cary, NC). Means and SDs were computed for continuous variables, and proportions were calculated for discrete variables. Comparisons between intervention groups for continuous measures were performed using two-sample t test and for discrete measures were performed using chi-square test. Triglycerides, HDL-C, and HOMA score were log transformed to best approximate the conditional normality assumption and to minimize the heterogeneity of variance. The outcome variables in this study include metabolic syndrome as a binary variable, the number of MetS components, and z-MetS as continuous variables. Statistical significance was set as \( p \leq .05 \).

Marginal models were used to assess the longitudinal effect of intervention on MetS. Generalized estimating equations were used to account for dependency between repeatedly measured discrete outcome. Differences in mean MetS components between groups over time were estimated using mixed effects models. In generalized estimating equation and mixed effects models, the baseline measure, gender (stratifying variable for randomization), clinic site, race, diabetes, intervention assignment, visit, and an intervention by visit interaction were included to determine whether the two interventions changed the outcome differently over time. Hypothesis tests for intervention effects at the 6- and 12-month visits were performed using contrasts of the 6- and 12-month means. Overall comparisons between groups for each component across follow-up visits were obtained using a contrast to compare average effects across both follow-up visits.

We also examined whether effects of the interventions were different in participants who were obese (body mass index ≥ 30 kg·m⁻²) versus nonobese at baseline, those who lost weight (≥5% weight loss) during intervention versus those who did not, and those using medications for treating MetS components at any visit versus those not using these medications at all visits. An interaction term between intervention arm and each of these subgroups was included in above-mentioned adjusted models. In addition to performing analysis using an “intention-to-treat” approach, we also assessed the longitudinal effect of interventions based on actual PA attendance, that is, 80% or more attendance in PA, less than 80% attendance in PA, and SA intervention.

**RESULTS**

**At Baseline**

Table 1 includes participant characteristics, body composition measures, and medication use relevant to the diagnosis of MetS. The two groups were similar at baseline, except
that a higher proportion of participants in the PA group were using glucose-lowering medications ($p = .003$) and body mass index was slightly lower in the SA group ($p = .029$). There were no differences between groups in the prevalence of the MetS (PA: 65.6%; SA: 61.3%), number of MetS components, or Z-MetS. There were also no group differences in individual MetS components, insulin, or HOMA score at baseline (Table 2). Overall, fat mass and HOMA score were positively associated with the presence of MetS after adjustment for gender, race, clinic site, and diabetes, so that for every SD increase in fat mass and log-transformed HOMA score, the odds of having MetS were 1.73 (95% confidence interval [CI] = 1.21–2.47) and 4.26 (95% CI = 2.91–6.23) times greater, respectively.

**Effects of Interventions on MetS**

In the PA group, attendance rates to the adoption and transition phases were 70% and 61%, respectively; during the maintenance phase, participants engaged in an average of 3.8 walking sessions per week and walked an average of 141 minutes per week. In the SA group, attendance rates were 70% for weeks 1–26 and 75% for weeks 27–52. At baseline, the number of bouts of moderate-intensity PA and estimated calories expended in such activities were similar in the two groups. At 6 and 12 months, PA participants reported a higher frequency of PA bouts and expended more exercise calories than SA participants (both $p < .01$). In the SA group, calories spent in moderate-intensity PA were ~25% and ~15% greater at 6 and 12 months, respectively, than at baseline. In the PA group, calories spent in moderate-intensity PA almost doubled at 6 month and were ~60% greater than baseline at 12 month. Body weight did not change in either group (PA: 6 month = 82.9 ± 18.0 kg, 12 month = 82.9 ± 18.7 kg and SA: 6 month = 80.8 ± 16.9 kg, 12 month = 79.5 ± 16.8 kg). Total fat mass did not change in the subset with body composition measures.

The trend of change in the prevalence of MetS over the intervention period was similar in the PA and SA groups. In the entire sample, the prevalence of MetS decreased significantly from baseline to 6 months ($p = .003$) but did not change further from 6 to 12 month ($p = .11$). It was not different between groups after 6 or 12 months of intervention after adjustment for baseline prevalence, gender, race, clinic site, and diabetes (Figure 1). The number of components meeting MetS criteria did not change in either group, and there were no differences between groups throughout the 12-month intervention, after adjustment for gender, race, clinic site, diabetes, and baseline value (PA: baseline = 2.98 ± 1.22; 6 month = 2.94; 12 month = 2.82 and SA: baseline = 2.81 ± 1.29; 6 month = 2.91; 12 month = 2.83; $p = .67$ for visit by intervention interaction). Similarly, the interventions had no significant effect on z-MetS (PA: 6 month = −1.37; 12 month = −1.63 and SA: 6 month = −1.23; 12 month = −1.56; $p = .79$ for visit by intervention interaction). Table 2 presents the individual MetS components, insulin, and HOMA score by group at baseline and months 6 and 12. The changes in these parameters were not different between

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**Table 1. Baseline Characteristics by Intervention Group**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>PA ($n = 180$)</th>
<th>SA ($n = 181$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>76.9 ± 4.1</td>
<td>77.7 ± 4.3</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>125 (69.4)</td>
<td>121 (66.9)</td>
</tr>
<tr>
<td>White, n (%)</td>
<td>137 (76.1)</td>
<td>137 (75.7)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>82.9 ± 18.2</td>
<td>81.0 ± 16.8</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>30.8 ± 6.0</td>
<td>29.5 ± 5.3*</td>
</tr>
<tr>
<td>Total fat mass, kg†</td>
<td>30.1 ± 8.8</td>
<td>28.0 ± 8.3</td>
</tr>
<tr>
<td>Body fat percent†</td>
<td>37.5 ± 6.9</td>
<td>36.5 ± 7.7</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>7 (3.9)</td>
<td>4 (2.2)</td>
</tr>
<tr>
<td>Medication use, n (%)</td>
<td>141 (78.3)</td>
<td>133 (73.5)</td>
</tr>
<tr>
<td>Antihypertensive</td>
<td>15 (8.3)</td>
<td>14 (7.7)</td>
</tr>
<tr>
<td>Cholesterol lowering</td>
<td>42 (23.3)</td>
<td>21 (11.6)</td>
</tr>
</tbody>
</table>

*Notes: Values are $M$ ± SD. PA = physical activity; SA = successful aging.

†$p = .029$ between groups from a two-sample $t$ test.

§$n = 103$ for PA and $n = 97$ for SA.

1$p = .005$ between groups from a two-sample $t$ test.

**Table 2. Metabolic Syndrome Components, Serum Insulin, and HOMA Score at Baseline and After 6 and 12 Months**

<table>
<thead>
<tr>
<th></th>
<th>PA</th>
<th>SA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist circumference, cm</td>
<td>103.0 ± 14.9</td>
<td>101.1 ± 14.0</td>
</tr>
<tr>
<td>6 mo</td>
<td>101.5 ± 14.3</td>
<td>99.1 ± 14.3</td>
</tr>
<tr>
<td>12 mo</td>
<td>101.1 ± 15.4</td>
<td>98.5 ± 13.2</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.49 ± 0.62</td>
<td>1.49 ± 0.76</td>
</tr>
<tr>
<td>6 mo</td>
<td>1.47 ± 0.77</td>
<td>1.46 ± 0.78</td>
</tr>
<tr>
<td>12 mo*</td>
<td>1.46 ± 0.76</td>
<td>1.36 ± 0.65</td>
</tr>
<tr>
<td>HDL-cholesterol, mmol/L</td>
<td>1.30 ± 0.37</td>
<td>1.32 ± 0.42</td>
</tr>
<tr>
<td>6 mo</td>
<td>1.35 ± 0.42</td>
<td>1.33 ± 0.42</td>
</tr>
<tr>
<td>12 mo</td>
<td>1.31 ± 0.37</td>
<td>1.33 ± 0.43</td>
</tr>
<tr>
<td>Systolic BP, mmHg</td>
<td>131.9 ± 17.6</td>
<td>133.1 ± 17.3</td>
</tr>
<tr>
<td>6 mo</td>
<td>130.1 ± 15.8</td>
<td>130.6 ± 16.2</td>
</tr>
<tr>
<td>12 mo*</td>
<td>128.2 ± 17.8</td>
<td>128.1 ± 16.8</td>
</tr>
<tr>
<td>Diastolic BP, mmHg</td>
<td>68.7 ± 10.7</td>
<td>69.5 ± 10.2</td>
</tr>
<tr>
<td>6 mo</td>
<td>68.9 ± 9.7</td>
<td>70.1 ± 9.7</td>
</tr>
<tr>
<td>12 mo</td>
<td>67.5 ± 9.4</td>
<td>69.1 ± 11.9</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L</td>
<td>5.61 ± 1.18</td>
<td>5.49 ± 1.13</td>
</tr>
<tr>
<td>6 mo</td>
<td>5.65 ± 1.19</td>
<td>5.58 ± 1.17</td>
</tr>
<tr>
<td>12 mo</td>
<td>5.61 ± 1.20</td>
<td>5.47 ± 1.27</td>
</tr>
<tr>
<td>Insulin, pmol/L</td>
<td>79.2 ± 57.6</td>
<td>75.7 ± 50.7</td>
</tr>
<tr>
<td>6 mo</td>
<td>76.4 ± 45.1</td>
<td>74.3 ± 49.3</td>
</tr>
<tr>
<td>12 mo</td>
<td>82.6 ± 56.9</td>
<td>77.1 ± 58.3</td>
</tr>
<tr>
<td>HOMA score</td>
<td>3.1 ± 2.9</td>
<td>2.8 ± 2.4</td>
</tr>
<tr>
<td>6 mo</td>
<td>2.9 ± 2.1</td>
<td>2.9 ± 2.8</td>
</tr>
<tr>
<td>12 mo</td>
<td>3.1 ± 2.5</td>
<td>2.9 ± 3.1</td>
</tr>
</tbody>
</table>

*Notes: Values are $M$ ± SD. n = 181, 166, and 172 at baseline, 6, and 12 months, respectively. PA = physical activity; SA = successful aging.

†Significantly changed from baseline value in the entire sample.
PA and SA. In the entire sample, triglycerides and systolic BP were significantly lower at 12 months than at baseline ($p = .004$ and .002, respectively), after adjustment for gender, race, clinic site, diabetes, and baseline value.

The presence of MetS over the 12-month follow-up was not associated with gender, race, or clinic site ($p = .40$, .14, and .61, respectively) but was associated with diabetes (odds ratio $= 3.48$, 95% CI $= 1.60$–7.58) when all covariates and baseline MetS information were included in the model. Fat mass change and HOMA score change were not associated with the presence of MetS during the intervention (odds ratio $= 0.86$, 95% CI $= 0.73$–1.01; odds ratio $= 1.07$, 95% CI $= 0.97$–1.19, respectively) after adjustment for baseline MetS status, gender, race, clinic site, diabetes, and intervention.

In addition, those who had MetS and those who did not have MetS at baseline had similar dropout rates during follow-up ($p = .94$). The assignment to PA and SA groups did not modify the effect of having versus not having MetS at baseline on dropout rates during follow-up ($p = .25$ for baseline MetS by group interaction). There were no differences in MetS prevalence ($p = .29$), number of MetS components ($p = .97$), and z-MetS ($p = .84$) among participants with 80% or more PA attendance, less than 80% PA attendance, and SA intervention over time, adjusted for race, sex, clinic, visit, diabetes, and corresponding baseline values.

**Subgroup Analyses**

The interactions between intervention and baseline obesity (body mass index $\geq 30$ kg/m$^2$) or weight loss ($\geq 5\%$ weight loss) during intervention were not significant for MetS prevalence ($p = .61$ and .95, respectively) or for the number of MetS components ($p = .56$ and .54 respectively).

Figure 1. The prevalence of metabolic syndrome at baseline (raw values) and 6 and 12 months (values adjusted for gender, race, clinic site, diabetes, and baseline values) by intervention group. The trend of change was similar for the physical activity (PA) and successful aging (SA) interventions ($p = .77$ for visit by intervention interaction from generalized estimating equation model), and the prevalence was not different between PA and SA at follow-up visits ($p = .74$). The prevalence of MetS decreased significantly from baseline to 6 months ($p = .003$) but did not change further from 6 to 12 months ($p = .11$).

Those who had 5% or more weight loss during intervention had lower odds of having MetS (odds ratio $= 0.39$, 95% CI $= 0.21$–0.75) and lower number of MetS components (difference in adjusted means across the intervention period: $\sim 0.38$, $SE = 0.11$) than those who had less than 5% weight loss. There was a significant interaction between intervention and medication use such that during the follow-up period, in individuals not using any medications for treating MetS components, those in PA had lower odds of having MetS over those in SA (Figure 2). The number of MetS components was also significantly influenced by the interaction of medication use and the intervention ($p = .009$). The PA and SA groups were very similar for those who used medication at any visit; however, for those did not use any medication, the number of MetS components was lower in those in the PA group at follow-up visits (adjusted mean across the follow-up period [$SE$], PA: 2.28 [0.14] and SA: 2.68 [0.12]).

**DISCUSSION**

This randomized controlled trial showed that, in this group of elderly men and women, a 12-month PA intervention and an SA intervention reduced the prevalence of MetS, but the PA intervention did not reduce the prevalence of MetS, the average number of MetS components, or the z-MetS more than the SA intervention. However, in the subgroup of participants not taking any medication for treating MetS components, those in the PA intervention had lower odds of having MetS compared with the SA intervention. This suggests that the effect of PA in this group of elderly men and women may be blunted by the use of medications to treat MetS components.

The findings of no significant differential effects of the 12-month PA intervention compared with SA intervention.
were somewhat unanticipated considering the beneficial effects of PA shown in previous studies (17,22,23). However, in a recent randomized controlled study in 67- to 76-year-old men and women (24), 12 weeks of aerobic exercise training did not significantly change a composite metabolic risk score (systolic and diastolic BP, 2-hour postprandial glucose, fasting insulin, HDL-C, triglycerides, and waist circumference) or its constituents (except waist circumference). Our results were in line with their findings but with a much longer intervention. However, our results do not exclude metabolic effects of the PA intervention because in the entire sample, the prevalence of MetS was reduced and triglycerides and systolic BP were also reduced after 12 months.

In comparison with other studies in older adults (17,22–24), our participants were older, and a large proportion (78.1%) were taking medications for treating at least one of the MetS components. Because using medication for treating MetS components was considered having met the criterion, it is not sensitive to changes in medication dose or the number of medications taken for the same MetS component. It is also unlikely that a participant’s physician would discontinue the medication during the PA intervention. In fact, we observed greater beneficial effects of PA compared with SA in those not taking any medication for treating MetS. Also, the exercise intensity and volume in our study were lower than in the few studies showing beneficial effects of exercise training alone on metabolic risks and MetS in older adults (17,22,23).

Moreover, in the above-mentioned studies showing metabolic effects of exercise training, two had a small degree of exercise-induced weight loss (17,23), and one showed approximately 4% fat mass loss (22). Another study in middle-aged and older adults showed that 16-week moderate-intensity continuous aerobic exercise and high-intensity interval training both improved MetS, and both groups had small but significant weight loss (3%–4%) (15). In the study that did not show exercise improved metabolic composite, the decreases in body weight (from 77.2 to 77.0 kg) and waist circumference (from 98.6 to 97.8 cm) in the exercise group were almost negligible (24). In our study, the PA intervention did not affect body weight, fat mass, or waist circumference overall, and the odds of having MetS were lower in those who lost 5% or more weight than those who did not, regardless of intervention assignment. Additionally, a recent study in obese postmenopausal women showed that those who lost more fat were more likely to resolve MetS (19). Thus, the metabolic benefits of exercise training may be less clear in the absence of weight loss or fat loss.

Importantly, the SA intervention in our study was not a no-intervention control. Although the PA intervention did not result in greater changes in MetS than the SA and both interventions decreased the prevalence of MetS, it should not be interpreted as the PA intervention had no effect. Perhaps in this group of elderly men and women, the health education SA intervention enabled some unmeasured behavioral changes in those participants that led to beneficial health effects.

At baseline, higher HOMA score and greater amount of fat mass were associated with greater odds of having MetS. Fat mass change and HOMA score change, however, were not associated with the presence of MetS during the intervention after adjusting for baseline MetS and covariates. It is widely accepted that insulin resistance and adipose tissue play important roles in the development of MetS (1). However, HOMA score is an index, not a direct measure for insulin resistance, and may not be adequate to detect changes in insulin resistance. Similarly, more specific measures of adiposity, such as visceral adipose tissue, may be more sensitive to show changes due to intervention.

In summary, this study showed that in a group of men and women aged 70–89 years old, a PA intervention did not result in lower prevalence of MetS, above beyond that of an SA health education intervention. Our findings suggest that medication use may override any beneficial metabolic effects of PA in this age group. It should be noted that the SA group also demonstrated slightly increased PA calories during follow-up visits, which may have contributed to the reduced MetS prevalence at 6 months in this group. Further studies are needed to determine whether a greater PA volume and/or intensity that elicits weight loss will result in greater beneficial metabolic effects in this age group.

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References

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