Does Aerobic Exercise Increase Skeletal Muscle Mass in Female and Male Adults?

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ABSTRACT

ROSS, R., E. JOHN, C. MCGLORY, L. E. DAVIDSON, and P. J. STOTZ. Does Aerobic Exercise Increase Skeletal Muscle Mass in Female and Male Adults?. *Med. Sci. Sports Exerc.*, Vol. 56, No. 5, pp. 776–782, 2024. **Introduction:** It is uncertain whether aerobic exercise in the form of walking contributes to the preservation or increase in total or regional skeletal muscle mass (SMM). **Purpose:** This study aimed to determine the effects of aerobic exercise on total and regional (upper body verses leg SMM) in male (n = 105) and female (n = 133) adults with overweight and obesity. **Methods:** A retrospective analysis of data from four randomized controlled trials. Participants included those who completed the given trial (control, n = 63; intervention, n = 175) and with complete magnetic resonance imaging (MRI) measured adipose tissue and SMM pre- and postintervention. Macronutrient intake was assessed for a subsample of participants. Supervised exercise was performed by walking on a treadmill for durations ranging from 12 to 24 wk at intensities between 50% and 75% of $\dot{V}O_{2peak}$. **Results:** All MRI-measured adipose tissue depots were reduced, and cardiorespiratory fitness was increased by aerobic exercise compared with controls ($P < 0.001$). Independent of baseline SMM, aerobic exercise was associated with a small reduction (estimated mean difference ± standard error) in whole-body SMM (−0.310 ± 0.150 kg, $P = 0.039$) and upper body SMM (−0.273 ± 0.121 kg, $P = 0.025$) compared with control. No between-group difference was observed for change in leg SMM ($P > 0.10$). A negative association was observed between the relative change in body weight and change in total ($R^2 = 0.37$, $P < 0.001$), upper body ($R^2 = 0.21$, $P < 0.001$), and leg SMM ($R^2 = 0.09$, $P = 0.701$). The SMM-to-adipose tissue ratio increased in response to aerobic exercise and was positively associated with weight loss ($P < 0.001$). Change in SMM was not associated with dietary protein intake ($P > 0.10$). **Conclusions:** Aerobic exercise performed while walking preserves, but does not increase, SMM in exercising muscle of adults. SMM not directly targeted by aerobic exercise may not be maintained. **Key Words:** OBESITY, SKELETAL MUSCLE FUNCTION, WEIGHT LOSS

The strong association between skeletal muscle mass (SMM) and numerous health outcomes underscores the importance of preserving and/or increasing SMM across the lifespan (1). Resistance exercise is recommended as first-line therapy for increasing SMM (2). Whether aerobic exercise provides sufficient stimulus to increase SMM is controversial. Harber and colleagues (3–6) have consistently reported that cycling exercise performed at ~60% to 80% of heart rate reserve is associated with a substantial increase in SMM in the quadricep muscles of older adults, a finding confirmed by others (7,8). The authors argue that their finding of muscle hypertrophy in response to aerobic exercise is observed in generally healthy adults who increased caloric intake to offset the negative energy balance induced by exercise and, thus, maintained body weight (3–6). By contrast, several investigators conclude that aerobic exercise is not associated with increases in SMM regardless of age, biological sex, or weight loss (9–13).

A careful review of the study designs used to investigate the effects of aerobic exercise on SMM reveals limitations that confound interpretation and may partially explain the discrepant findings. With few exceptions, the sample size of aerobic exercise participants was small, increasing the possibility of observing a type 1 error. Many studies did not include a control group, and thus, the observed increase in SMM cannot confidently be attributed to exercise alone (3–8). Whether regional differences in the response of SMM to aerobic exercise exists is also unknown as previous investigations did not include assessment of whole-body SMM using a criterion method. Whether the effects of aerobic exercise on SMM differ according to biological sex is also unclear.

In this secondary analysis, we sought to determine the effects of aerobic exercise consistent with consensus recommendations on whole-body and regional SMM. Whole-body SMM was assessed by magnetic resonance imaging (MRI) in a large sample of middle-aged adults with abdominal obesity, allowing us to address the following questions. Is aerobic

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exercise performed while walking/jogging associated with a preservation or increase in total or regional SMM by comparison with time-matched controls? Is the effect of aerobic exercise restricted to the primary working SMM? Is the exercise effect on SMM different for males and females? Answers to these questions in middle-aged adults are clinically relevant because independent of biological sex, aging is associated with a marked decrease in SMM after the fifth decade of life (14).

METHODS

Study design. A secondary analysis was completed using data from four randomized trials previously conducted within the Lifestyle and Cardiometabolic Research Unit at Queen's University in Kingston, Ontario, Canada. From these trials, we included 238 previously sedentary, adult males and females with overweight and obesity, and with MRI data both pre- and postintervention. The four trials were conducted between 1999 and 2015 and ranged in duration from 3 to 6 months at intensities ranging from 50% to 75% of $\dot{V}O_{2peak}$ (15–18). In all trials, supervised aerobic exercise was performed with participants walking or jogging on a treadmill. Participants in the nonexercise control groups were asked to maintain their normal lifestyle for the duration of the trial.

Participants. Participants were predominately White and were recruited by mass media from the greater Kingston region. Inclusion criteria across all studies were as follows: body mass index >30 kg·m^{-2}, waist circumference >102 cm in men and >88 cm in women, weight stable (±2 kg) for 6 months before the study, physically inactive, not taking medications that would affect the principal outcomes of each trial, and nonsmokers. Waist circumference was obtained at the level of the last (floating) rib. Exclusion criteria included history of heart disease, stroke, diabetes mellitus, or any condition that would prevent the individual from engaging in regular exercise.

Aerobic exercise intervention. For three of the four trials (15–17), participants performed walk/jog exercise consistent with consensus recommendations on a treadmill for the time required to achieve the desired energy expenditure (kcal per session). All participants exercised under supervision 4–5 times per week at the prescribed intensity (relative to $\dot{V}O_{2peak}$) for the duration of the respective trial. Using the heart rate and oxygen consumption data obtained from the baseline exercise test, the heart rate associated with the prescribed $\dot{V}O_2$ (e.g., 50% to 75% of $\dot{V}O_{2peak}$) was prescribed for each participant. Follow-up exercise tests were performed to verify the relationship between heart rate and oxygen consumption. For the remaining trial (18), all participants were asked to walk/jog on a treadmill for 30 min at 60%–75% of their $\dot{V}O_{2peak}$ 5 times per week. For all trials, heart rate was monitored continuously every session to help ensure adherence to the prescribed exercise protocol. Additional detail for each trial can be found elsewhere (15–18)

Diet regimen. For all trials, at baseline, participants were instructed by a nutritionist or dietician to consume a caloric intake that would maintain their baseline body weight and consequently, that any observed weight loss throughout the intervention would be attributed to the negative energy balance induced by exercise. Participants were asked to complete and submit daily food diaries to report the consumption of all self-selected foods. A balanced diet was encouraged (~50% carbohydrate, ~20% protein, and ~30% fat) for all participants regardless of exercise intervention assignment. Participants were counseled by the nutritionist/dietician if their bodyweight deviated substantially from their estimated weight loss according to their prescribed negative energy balance induced by the aerobic exercise program.

To determine whether participants were consuming the recommended dietary allowance of protein (e.g., >0.8 g·kg^{-1}·d^{-1} of total), total caloric and macronutrient intake at baseline and weeks 8, 16, and 24 was assessed for a subsample of 66

TABLE 1. Subject characteristics at baseline.

	Women		Men	
Characteristic	**Control (*n* = 34)**	**Aerobic Exercise (*n* = 99)**	**Control (*n* = 29)**	**Aerobic Exercise (*n* = 76)**
Anthropometric				
Age, yr	55.9 ± 11.3	51.8 ± 11.3	56.6 ± 11.7	52.2 ± 11.2
Weight, kg	82.2 ± 9.6	86.1 ± 12.8	97.1 ± 11.9	100.6 ± 11.0
BMI, kg·m^{-2}	30.3 ± 2.9	32.0 ± 4.2	31. 1 ± 3.0	32.1 ± 3.0
WC, cm	99.0 ± 7.5	100.9 ± 10.2	110.2 ± 8.4	111.8 ± 7.7
MRI				
Whole-body SM, kg	20.8 ± 2.8	21.1 ± 3.1	31.8 ± 3.7	32.9 ± 3.6
Upper body SM, kg	9.7 ± 1.3	9.9 ± 1.5	15.6 ± 2.0	16.4 ± 1.9
Leg SM, kg	11.0 ± 1.7	11.2 ± 1.8	16.2 ± 1.8	16.5 ± 2.0
Whole-body AT, kg	37.9 ± 6.1	41.1 ± 8.8	34.0 ± 8.1	35.9 ± 8.4
Total SAT, kg	31.6 ± 5.5	34.4 ± 7.8	24.3 ± 6.1	26.1 ± 6.8
Total VAT, kg	2.4 ± 0.9	2.4 ± 0.9	4.2 ± 1.4	4.1 ± 1.1
$\dot{V}O_{2peak}$				
L·min^{-1}	2.0 ± 0.3	2.2 ± 0.4	3.3 ± 0.7	3.4 ± 0.7
mL·kg^{-1}·min^{-1}	24.4 ± 3.9	25.1 ± 4.2	33.7 ± 6.6	33.4 ± 6.8
Diet	(*n* = 6)	(*n* = 32)	(*n* = 9)	(*n* = 19)
Total calories, kcal	1699.3 ± 359.6	1883.6 ± 394.8	2448.4 ± 474.3	2407.8 ± 487.2
Total protein, g	70.1 ± 18.5	78.6 ± 18.7	105.0 ± 25.6	99.6 ± 23.3
Total fat, g	65.7 ± 17.7	73.5 ± 18.0	98.2 ± 22.2	91.1 ± 31.1
Total carbohydrates, g	210.9 ± 35.7	220.4 ± 57.4	260.3 ± 51.8	287.1 ± 67.6

Values are presented as mean ± SD.

AT, adipose tissue; BMI, body mass index; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; WC, waist circumference.

participants from a single trial within which complete nutrition intake and MRI data were available (7). Food diaries for these participants were analyzed using a proprietary, Web-based dietary recall program (R24W). Details regarding R24W development and validation are published elsewhere (19,20). Information from the food diaries was entered into the R24W program, which contains a database of 2865 food items and 687 predetermined recipes associated with a corresponding food code established by the Canadian Nutrient File (21) or the USDA Nutrient Database (22).

MRI. Whole-body adipose tissue and SMM distribution was determined using an established MRI protocol (23). Once acquired, the MRI data were transferred to a computer for analysis using a proprietary image analysis software program (Tomovision Inc., Montreal, QC, Canada). Adipose and SMM volume units (L) were converted to mass values (kg) by multiplying the volumes by the assumed constant density for fat (0.92 kg·L^{-1}) and fat-free skeletal muscle (SM; 1.04 kg·L^{-1}). Leg SMM values were derived using the series of 10-mm thick MR images obtained from the femoral head to the foot (~18 images). Upper body SMM was derived by subtracting leg SMM from total SMM.

We have previously reported (24) that that adipose tissue-free SMM estimates by MRI are highly correlated with corresponding cadaver values (r = 0.99, SEE = 3.9 cm^3, $P < 0.001$). Strong associations were also reported for repeat, interobserver measures of adipose tissue-free SMM obtained on two separate days in the leg region (r = 0.99, SEE = 8.8 cm^3, 2.9%).

Data analysis. Differences between groups were determined using ANCOVA with adjustment for baseline values to determine main effects and interaction for anthropometric, cardiorespiratory fitness, and MRI-measured variables. If no group–sex interaction was observed, the analysis was performed again without inclusion of the interaction term. Differences between groups for macronutrient intake were determined using a mixed repeated-measures ANOVA. Statistical significances are presented as Bonferroni-adjusted P values. Multiple linear regression was used to test the relationship between muscle and adipose tissue changes with relative weight change adjusting for baseline values. All analyses were performed using commercially available software (IBM SPSS Statistics, version 28).

RESULTS

Of the 238 predominantly White, middle-aged (mean ± SD: 53.1 ± 11.4 yr) participants (56% female), 175 were randomized to aerobic exercise and 63 to a nonexercise control group. The mean waist circumference value for men and women was 111.4 ± 7.9 and 100.4 ± 9.6 cm, respectively. SMM represented 32.3% and 24.6% of total body weight for men and women, respectively (Table 1).

The increase in $\dot{V}O_{2\text{peak}}$ and the reduction in weight (−4.7 ± 3.8 kg, −5.0% ± 3.8%), waist circumference, and total,

TABLE 2. Anthropometric, cardiorespiratory fitness, and MRI-measured variables pre- and postintervention.

	Control						Aerobic Exercise					
Variable	Women (n = 34)		Men (n = 29)		Combined (n = 63)		Women (n = 99)		Men (n = 76)		Combined (n = 175)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Age, yr	55.9 ± 11.3	–	56.6 ± 11.7	–	56.2 ± 11.4	–	54.0 ± 11.1	–	54.1 ± 11.3	–	51.9 ± 11.2	–
Weight, kg	82.2 ± 9.6	82.5 ± 10.2	97.1 ± 11.9	96.7 ± 12.0	89.0 ± 13.0	89.1 ± 13.1	86.1 ± 13.2	81.7 ± 12.5 *	100.4 ± 11.7	95.9 ± 10.8 *	92.4 ± 14.0	87.7 ± 13.4 *
BMI, kg·m^{-2}	30.3 ± 2.9	30.5 ± 3.1	31.1 ± 3.0	31.0 ± 2.9	30.7 ± 2.9	30.7 ± 3.0	31.9 ± 4.3	30.3 ± 4.2 *	32.0 ± 3.3	30.6 ± 3.1 *	32.0 ± 3.7	30.4 ± 3.7 *
WC, cm	99.0 ± 7.5	99.0 ± 8.4	110.2 ± 8.4	109.6 ± 8.4	104.2 ± 9.7	103.9 ± 9.9	101.1 ± 10.6	95.6 ± 10.0 *	112.7 ± 8.1	106.9 ± 8.1 *	105.7 ± 10.6	100.0 ± 10.4 *
$\dot{V}O_2$ peak, $\text{mL·kg}^{-1}\text{·min}^{-1}$	24.4 ± 3.9	24.2 ± 4.5	33.7 ± 6.6	33.1 ± 7.0	28.7 ± 7.0	28.1 ± 7.2	25.2 ± 3.9	30.7 ± 5.5 *	32.3 ± 6.2	39.2 ± 8.3 *	28.7 ± 6.9	35.1 ± 8.6 *
MRI												
Total SM, kg	20.8 ± 2.8	20.5 ± 3.0	31.8 ± 3.7	31.7 ± 3.8	25.8 ± 6.4	25.6 ± 6.6	21.1 ± 3.1	20.7 ± 2.9	32.3 ± 3.6	31.7 ± 3.6 #	26.2 ± 6.7	25.7 ± 6.6 #
Upper body SM, kg	9.7 ± 1.3	9.4 ± 1.3	15.6 ± 2.0	15.5 ± 2.0	12.4 ± 3.4	12.2 ± 3.5	9.9 ± 1.5	9.4 ± 1.4	16.2 ± 2.0	15.7 ± 2.0	12.7 ± 3.6	12.2 ± 3.6 #
Leg SM, kg	11.0 ± 1.7	11.1 ± 1.8	16.2 ± 1.8	16.2 ± 2.0	13.4 ± 3.1	13.4 ± 3.2	11.0 ± 1.8	11.3 ± 1.8	16.1 ± 1.9	16.1 ± 2.0	13.5 ± 3.2	13.5 ± 3.2
Total AT, kg	37.9 ± 6.1	38.3 ± 6.7	34.0 ± 8.1	33.4 ± 8.1	36.1 ± 7.3	36.0 ± 7.7	41.2 ± 9.1	37.3 ± 9.1 *	36.6 ± 8.9	32.8 ± 8.0 *	38.8 ± 9.0	34.8 ± 9.0 *
Total SAT, kg	31.6 ± 5.5	31.9 ± 6.1	24.3 ± 6.1	24.0 ± 5.9	28.2 ± 6.8	28.3 ± 7.2	34.3 ± 7.9	31.4 ± 8.0 *	26.5 ± 7.2	24.0 ± 6.5 *	30.8 ± 8.4	27.9 ± 8.3 *
Total VAT, kg	2.4 ± 0.9	2.4 ± 1.0	4.2 ± 1.4	4.2 ± 1.5	3.2 ± 1.5	3.2 ± 1.5	2.4 ± 1.0	2.0 ± 0.9 *	4.2 ± 1.1	3.5 ± 1.0 *	3.1 ± 1.3	2.6 ± 1.2 *
SMAT ratio	0.56 ± 0.08	0.54 ± 0.08	0.97 ± 0.21	0.99 ± 0.22	0.75 ± 0.26	0.75 ± 0.28	0.53 ± 0.09	0.58 ± 0.11 *	0.97 ± 0.28	1.09 ± 0.32 *	0.72 ± 0.29	0.80 ± 0.34 *

Data are presented as mean ± SD.

Different from control: * $P < 0.001$, # $P < 0.05$.

AT, adipose tissue; BMI, body mass index; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; WC, waist circumference.

subcutaneous, and visceral adipose tissue in response to aerobic exercise were significant compared with control ($P < 0.001$, Table 2).

No group–sex interaction was observed for change in whole-body, upper body, or leg SMM ($P > 0.10$). For male and female participants combined, independent of baseline value, aerobic exercise was associated with a small reduction (estimated mean difference ± standard error) in whole-body SMM (−0.310 ± 0.150 kg, $P = 0.039$) and upper body SMM (−0.273 ± 0.121 kg, $P = 0.025$) by comparison with control. No between-group difference was observed for change in leg SMM ($P = 0.701$, Table 2).

Independent of baseline body weight, the relative change in body weight for the aerobic exercise group was associated with the corresponding change in total SMM ($R^2 = 0.29$, $P < 0.001$), upper body SM ($R^2 = 0.21$, $P < 0.001$), and leg SM ($R^2 = 0.09$, $P < 0.001$).

To further explore the associations between relative weight loss and change in SMM, exercise participants were divided into tertiles based on relative weight loss (Fig. 1). For males and females combined, after adjustment for baseline values, the reduction in total and upper body SMM observed for tertile 3 (highest weight loss, ~9%) was greater compared with tertile 1 and controls ($P < 0.001$). The change in leg SMM for all tertiles was not different from control ($P > 0.10$). With few exceptions, the observations for the men and women considered separately were not different (Fig. 2).

For aerobic exercise participants, the reduction in SMM (−0.5 ± 1.1 kg) was substantively less than the corresponding reduction in total adiposity (−4.01 ± 3.0 kg, $P < 0.001$). Indeed, the relative contribution of SMM to body weight postintervention increased significantly ($P < 0.001$) for both men and women by about 1%. Accordingly, the SMM-to-adipose tissue (SMAT) ratio increased by comparison with control for both men and women postintervention ($P < 0.001$, Table 2) and was positively associated with relative weight loss for males ($R^2 = 0.38$, $P < 0.001$) and females ($R^2 = 0.33$ $P < 0.001$, Fig. 3).

Total caloric and macronutrient intake at baseline and weeks 8, 16, and 24 for a subsample of 66 participants from a single trial for which complete nutrition intake and MRI data were available is shown in Table 3. No differences were observed for any macronutrient component or total caloric intake between groups for men or women ($P > 0.05$). For aerobic exercise participants, the mean intake of protein at baseline was 96.6 and 78.6 $g \cdot d^{-1}$ for males and females, respectively, at baseline and did not change throughout the intervention ($P > 0.1$, Table 3). The change in SMM was not associated with dietary protein intake ($P > 0.1$)

Finally, we investigated whether components of exercise dose were associated with change in SMM. For men, the mean exercise intensity expressed as a percentage of the $\dot{V}O_{2peak}$, the total kilocalorie expenditure over the course of the intervention, and the total time exercising were not associated with change in SMM ($P = 0.637$, $P = 0.342$, $P = 0.353$, respectively). For women, the mean exercise intensity ($P = 0.008$), the total kilocalorie expenditure ($P < 0.001$), and the total time exercising ($P < 0.001$) were associated with change in SMM (data not shown).

DISCUSSION

The novel finding of this study is that independent of biological sex and weight loss, there are regional differences in the response of SM to aerobic exercise. Although the targeted leg muscle primarily involved in the prescribed exercise was preserved, we identified small reductions in the nontargeted upper body SM. These findings demonstrate that aerobic exercise provides a contraction stimulus sufficient to maintain SMM, but the response is limited to the working muscle. Given that walking is the most popular form of physical activity for adults and that the age associated decrease in SM is explained in large measure by a decrease in lower body muscle (14), our findings underscore the importance of regular exercise as a strategy to mitigate the age-related loss of SM, to

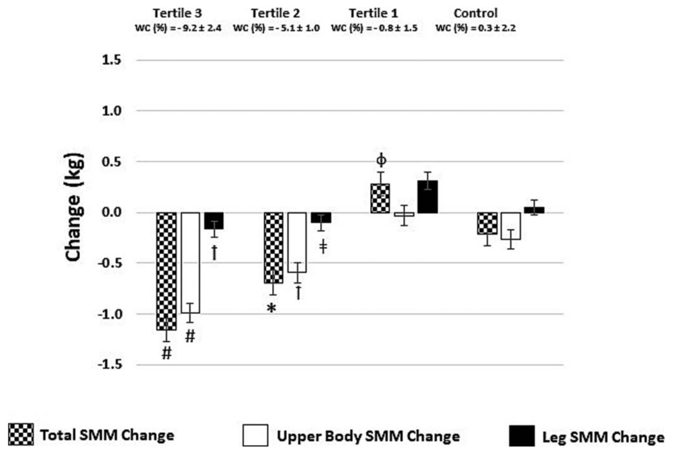

FIGURE 1—Change in total, upper body, and leg SMM according to tertiles of relative weight loss (%) in response to aerobic exercise. # Different from tertile 1 and control ($P < 0.001$) and tertile 2 ($P < 0.05$). * Different from tertile 1 ($P < 0.001$) and control ($P < 0.05$). † Different from tertile 1 ($P < 0.001$). ǂ Different from tertile 1 ($P < 0.05$). ф Different from control ($P < 0.05$). WC (%), weight change (%). Data are presented as estimated means with SEM bars with corresponding baseline value as a covariate. Control, $N = 63$; tertiles 1 and 2, $N = 58$ each; tertile 3, $N = 59$.

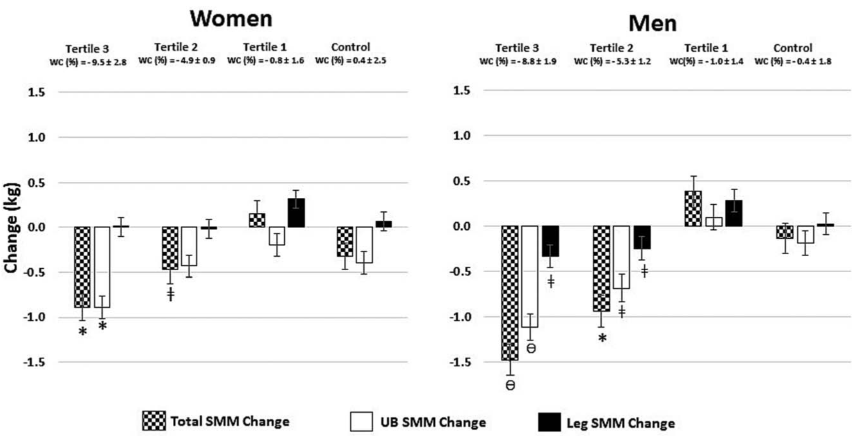

FIGURE 2—Change in total, upper body, and leg SMM according to tertiles of relative weight loss (%) for males and females in response to aerobic exercise. * Different from tertile 1 ($P < 0.001$) and control ($P < 0.05$). ǂ Different from tertile 1 and control ($P < 0.001$). ǂ Different from tertile 1 ($P < 0.05$). WC (%), weight change (%). Data are presented as estimated means with SEM bars with corresponding baseline value as a covariate. ♀ control, $N = 34$; tertiles 1, 2, and 3, $N = 33$ each. ♂ control, $N = 29$; tertiles 1 and 3, $N = 26$ each; tertile 2, $N = 24$.

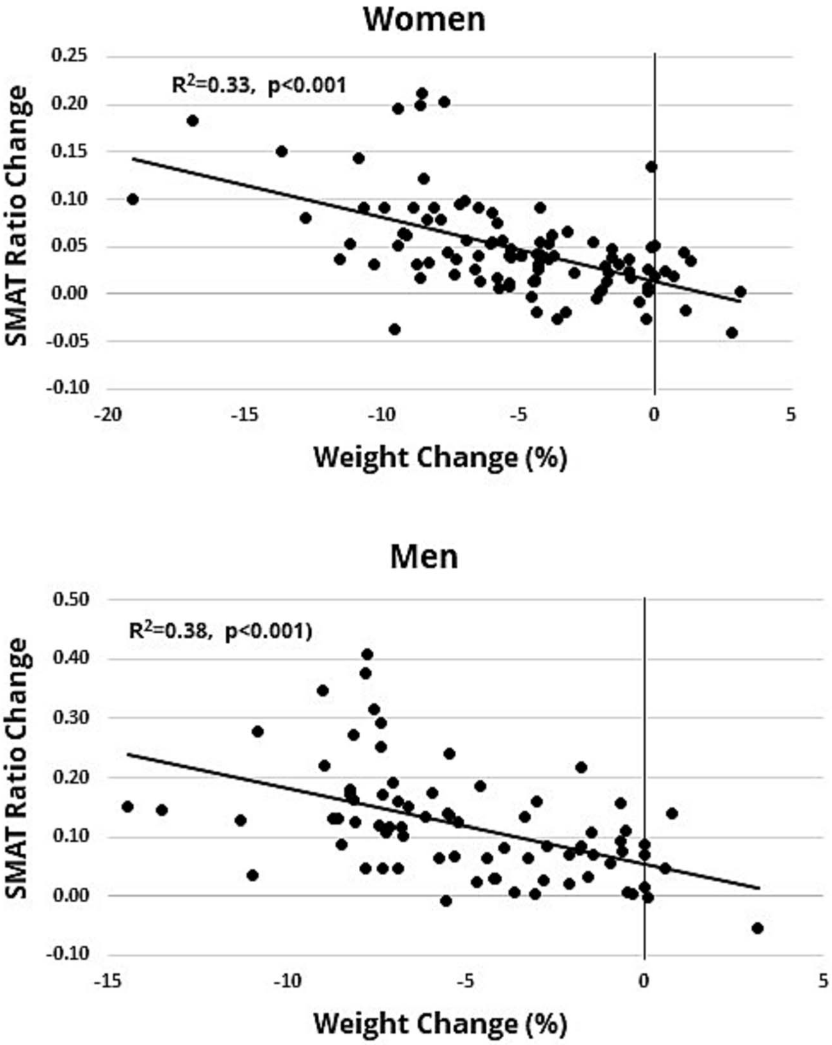

FIGURE 3—SMAT ratio in relation to relative weight loss (%) for women ($N = 99$) and men ($N = 76$) aerobic exercise participants. Regression model included baseline SMAT as a covariate.

TABLE 3. Macronutrient intake over a 24-wk intervention.

	Women			Men		
Macronutrient	**Control (*n* = 6)**	**Aerobic Exercise (*n* = 32)**	***P***	**Control (*n* = 9)**	**Aerobic Exercise (*n* = 19)**	***P***
Protein, g						
Baseline	70.1 ± 18.5	78.6 ± 18.7		105.0 ± 25.6	99.6 ± 23.3	
Week 8	72.4 ± 11.1	72.3 ± 20.7		108.6 ± 25.7	94.4 ± 21.5	
Week 16	66.2 ± 25.0	77.9 ± 19.5		93.3 ± 25.9	92.1 ± 26.1	
Week 24	83.0 ± 14.4	78.1 ± 19.6		99.8 ± 28.9	100.9 ± 27.1	
ΔProtein	12.9 ± 21.7	−0.5 ± 20.0	0.57	−5.2 ± 25.3	1.3 ± 31.4	0.56
Carbohydrate, g						
Baseline	210.9 ± 35.7	220.4 ± 57.4		260.3 ± 51.8	287.1 ± 67.6	
Week 8	188.3 ± 63.3	218.7 ± 63.3		245.7 ± 77.4	298.6 ± 62.8	
Week 16	175.9 ± 53.6	227.1 ± 69.6		255.5 ± 67.7	301.1 ± 72.9	
Week 24	210.3 ± 46.6	225.0 ± 67.6		270.5 ± 63.9	296.9 ± 76.6	
ΔCarbohydrate	−0.6 ± 28.8	4.6 ± 53.0	0.29	10.3 ± 83.9	9.8 ± 68.4	0.09
Fat, g						
Baseline	65.7 ± 17.7	73.5 ± 18.0		98.2 ± 22.2	91.1 ± 31.1	
Week 8	63.5 ± 23.1	67.6 ± 20.8		85.4 ± 25.9	86.4 ± 25.6	
Week 16	52.7 ± 18.2	74.0 ± 24.9		72.1 ± 26.7	80.3 ± 20.4	
Week 24	74.0 ± 25.5	69.1 ± 23.8		87.0 ± 41.3	76.6 ± 19.4	
ΔFat	8.2 ± 29.3	−4.4 ± 23.5	0.34	- 11.2 ± 37.4	- 14.5 ± 32.8	0.80
Calories, kcal						
Baseline	1699.3 ± 359.6	1883.9 ± 394.8		2448.4 ± 474.3	2407.8 ± 487.2	
Week 8	1613.0 ± 276.1	1795.9 ± 425.8		2269.4 ± 449.5	2443.0 ± 500.4	
Week 16	1423.2 ± 418.8	1907.2 ± 522.9		2077.2 ± 426.2	2343.4 ± 525.1	
Week 24	1818.8 ± 397.4	1867.3 ± 481.7		2332.0 ± 573.0	2351.8 ± 566.4	
ΔCalories	119.5 ± 378.4	−16.4 ± 410.0	0.19	−116.4 ± 639.6	−55.9 ± 574.4	0.53

*Values are presented as mean ± SD.

Δ, change from baseline.

improve function, and to decrease health risk independent of biological sex.

Our finding that aerobic exercise does not lead to an increase in the mass of working muscle targeted by aerobic exercise (leg SMM) counters the finding of others who report that 12 wk of cycling exercise (~60%–80% of heart rate reserve) is associated with an increase in SMM ranging from ~5% to 12% in the quadriceps of older, primarily female adults (4–6,8). In the majority of those studies, the participants were asked to consume additional calories to offset the negative energy balance induced by exercise. Although we observed a preservation of leg SMM in participants who did not lose weight, leg SMM did not increase by comparison with controls (see Fig. 3, tertile 1). Cycling at a higher intensity may involve greater muscle activation compared with walking or jogging. The increased muscle activation may lead to a greater high threshold motor unit recruitment, which could potentially enhance the anabolic response to exercise in the absence of weight loss (25). However, considering that most adults in developed countries have overweight or obesity, and a significant number of older adults have sarcopenic obesity, the feasibility and clinical implications of recommending high-intensity cycling exercise combined with an increase in caloric intake is questionable. Accordingly, our finding, and those of others (11–13), that aerobic exercise performed while walking is associated with a preservation of SMM independent of biological sex with or without weight loss is promising.

In this study, the reduction in upper body SMM observed in response to ~10% reduction in body weight was relatively small (~1 kg). For these participants, because of the substantial exercise-induced reduction in total adiposity, the SMAT ratio increased significantly compared with control. Given the well-established benefits of total and visceral adipose tissue reduction across a wide range of health outcomes (26), we suggest that the small decrease observed for SMM for those who reduced body weight by ~10% may be of marginal clinical consequence. Indeed, it is reasonable to suggest that the increase in SMAT ratio would lead to improved whole-body muscle functioning because of an associated improvement in the bodyweight-to-muscle strength ratio. This notion is reinforced by the established observation that aerobic exercise is associated with improvement in SM function (27) and our observed increase in cardiorespiratory fitness, which is associated with SM strength (28).

The participants in all our trials were asked to consume a healthy, balance diet that was monitored continuously throughout the intervention. It is not surprising therefore that the protein consumption for a subset of our participants met the recommended dietary allowance. These observations suggest that that muscle protein synthesis was not restricted because of inadequate dietary protein and the failure to observe an increase SMM in response to aerobic exercise was not a result of insufficient nutrition.

Mechanisms by which aerobic exercise would mitigate the loss of SMM include improvement in insulin sensitivity and increases in protein synthesis. Insulin is a potent inhibitor of muscle protein breakdown (27), and aerobic exercise is an established strategy for improving insulin sensitivity with or without weight loss (15,16). It is also reported that aerobic exercise augments muscle protein synthesis (29–31) and enhances muscle capillarization. Improved capillarization may potentiate insulin and amino acid delivery to SM, suppressing rates of proteolysis and improving rates of muscle protein synthesis, respectively (29).

Strengths of our study include the assessment of a relatively large sample of adult male and female participants. The exercise prescribed was consistent with established physical activity guidelines. MRI measurement of whole-body SMM allowed the investigation of potential regional differences in SMM. Monitoring of daily dietary intake in a subsample helped to interpret the potential effects of dietary protein intake on SMM. Limitations include that we studied a homogeneous sample of abdominally overweight and obese White adults. However, because approximately ~75% of North American adults are overweight or obese, a sizeable proportion of the adult population would likely benefit from the aerobic exercise prescribed.

CONCLUSIONS

In summary, moderate-intensity walking exercise consistent with consensus recommendations maintains but does not increase SMM in adults with overweight and obesity with or without weight loss. The preservation of SMM is restricted to the muscle targeted by the aerobic exercise program. Health care providers are encouraged to counsel clients regarding the potential benefits of aerobic exercise as a means of mitigating the age-related reductions in SMM.

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REFERENCES

1. McLeod M, Breen L, Hamilton DL, Philp A. Live strong and prosper: the importance of skeletal muscle strength for healthy ageing. *Biogerontology*. 2016;17:497–510.
2. American College of Sports Medicine. American College of Sports Medicine Position Stand: progression models in resistance training for healthy adults. *Med Sci Sports Exerc*. 2009;41(3):687–708.
3. Konopka AR, Harber MP. Skeletal muscle hypertrophy after aerobic exercise training. *Exerc Sport Sci Rev*. 2014;42(2):53–61.
4. Harber MP, Konopka AR, Douglass MD, et al. Aerobic exercise training improves whole muscle and single myofiber size and function in older women. *Am J Physiol Regul Integr Comp Physiol*. 2009;297(5):R1452–9.
5. Harber MP, Konopka AR, Undem MK, et al. Aerobic exercise training induces skeletal muscle hypertrophy and age-dependent adaptations in myofiber function in young and older men. *J Appl Physiol (1985)*. 2012;113(9):1495–504.
6. Konopka AR, Douglass MD, Kaminsky LA, et al. Molecular adaptations to aerobic exercise training in skeletal muscle of older women. *J Gerontol A Biol Sci Med Sci*. 2010;65(11):1201–7.
7. Izquierdo M, Hakkinen K, Ibanez J, Kraemer WJ, Gorostiaga EM. Effects of combined resistance and cardiovascular training on strength, power, muscle cross-sectional area, and endurance markers in middle-aged men. *Eur J Appl Physiol*. 2005;94(1–2):70–5.
8. Hudelmaier M, Wirth W, Himmer M, Ring-Dimitriou S, Sanger A, Eckstein F. Effect of exercise intervention on thigh muscle volume and anatomical cross-sectional areas—quantitative assessment using MRI. *Magn Reson Med*. 2010;64(6):1713–20.
9. Short KR, Vittone JL, Bigelow ML, Proctor DN, Nair KS. Age and aerobic exercise training effects on whole body and muscle protein metabolism. *Am J Physiol Endocrinol Metab*. 2004;286(1):E92–101.
10. Sipila S, Suominen H. Effects of strength and endurance training on thigh and leg muscle mass and composition in elderly women. *J Appl Physiol (1985)*. 1995;78(1):334–40.
11. Cuff DJ, Meneilly GS, Martin A, Ignaszewski A, Tildesley HD, Frohlich JJ. Effective exercise modality to reduce insulin resistance in women with type 2 diabetes. *Diabetes Care*. 2003;26(11):2977–82.
12. Ferrara CM, Goldberg AP, Ortmeyer HK, Ryan AS. Effects of aerobic and resistive exercise training on glucose disposal and skeletal muscle metabolism in older men. *J Gerontol A Biol Sci Med Sci*. 2006;61(5):480–7.
13. Weiss EP, Racette SB, Villareal DT, et al. Lower extremity muscle size and strength and aerobic capacity decrease with caloric restriction but not with exercise-induced weight loss. *J Appl Physiol (1985)*. 2007;102(2):634–40.
14. Janssen I, Heymsfield SB, Wang ZM, Ross R. Skeletal muscle mass and distribution in 468 men and women aged 18-88 yr. *J Appl Physiol (1985)*. 2000;89(1):81–8.
15. Ross R, Dagnone D, Jones PJ, et al. Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men. A randomized, controlled trial. *Ann Intern Med*. 2000;133(2):92–103.
16. Ross R, Janssen I, Dawson J, et al. Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial. *Obes Res*. 2004;12(5):789–98.
17. Ross R, Hudson R, Stotz PJ, Lam M. Effects of exercise amount and intensity on abdominal obesity and glucose tolerance in obese adults: a randomized trial. *Ann Intern Med*. 2015;162(5):325–34.
18. Davidson LE, Hudson R, Kilpatrick K, et al. Effects of exercise modality on insulin resistance and functional limitation in older adults: a randomized controlled trial. *Arch Intern Med*. 2009;169(2):122–31.
19. Lafrenière J, Lamarche B, Laramée C, Robitaille J, Lemieux S. Validation of a newly automated web-based 24-hour dietary recall using fully controlled feeding studies. *BMC Nutr*. 2017;3:34.
20. Jacques S, Lemieux S, Lamarche B, et al. Development of a web-based 24-h dietary recall for a French-Canadian population. *Nutrients*. 2016;8(11):724.
21. Health Canada. Canadian Nutrient File [Web page]. http://webprod3.hc-sc.gc.ca/cnf-fce/ language-langage.do?url = t.search. recherche&lang = eng. Accessed April 11, 2018.
22. United States Department of Agriculture. National Nutrient Database for Standard Reference [Web page]. http://ndb.nal.usda.gov/ ndb/search. Accessed April 11, 2018.
23. Ross R, Rissanen J, Pedwell H, Clifford J, Shragge P. Influence of diet and exercise on skeletal muscle and visceral adipose tissue in men. *J Appl Physiol (1985)*. 1996;81(6):2445–55.
24. Mitsiopoulos N, Baumgartner RN, Heymsfield SB, Lyons W, Gallagher D, Ross R. Cadaver validation of skeletal muscle measurement by magnetic resonance imaging and computerized tomography. *J Appl Physiol (1985)*. 1998;85(1):115–22.
25. Bijker KE, de Groot G, Hollander AP. Differences in leg muscle activity during running and cycling in humans. *Eur J Appl Physiol*. 2002;87(6):556–61.
26. Neeland IJ, Ross R, Després J-P, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol*. 2019;7(9):715–25.
27. Cava E, Yeat NC, Mittendorfer B. Preserving healthy muscle during weight loss. *Adv Nutr*. 2017;8(3):511–9.
28. Vaara JP, Kyrolainen H, Niemi J, et al. Associations of maximal strength and muscular endurance test scores with cardiorespiratory fitness and body composition. *J Strength Cond Res*. 2012;26(8):2078–86.
29. Atherton PJ, Smith K. Muscle protein synthesis in response to nutrition and exercise. *J Physiol*. 2012;590(5):1049–57.
30. Pikosky MA, Gaine PC, Martin WF, et al. Aerobic exercise training increases skeletal muscle protein turnover in healthy adults at rest. *J Nutr*. 2006;136(2):379–83.
31. Brightwell CR, Markofski MM, Moro T, et al. Moderate-intensity aerobic exercise improves skeletal muscle quality in older adults. *Transl Sports Med*. 2019;2(3):109–19.