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Effect of an 18 month physical activity and weight loss intervention on body composition in overweight and obese older adults

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Abstract

Objective—Our primary objective was to determine the long-term effects of physical activity (PA) and weight loss (WL) on body composition in overweight/obese older adults. Secondarily, we evaluated the association between change in body mass and composition on change in several cardiometabolic risk factors and mobility.

Design and Methods—288 older (X±SD: 67.0±4.8 years), overweight/obese (BMI 32.8±3.8 kg/m^2) men and women participated in this 18 month randomized, controlled trial. Treatment groups included PA+WL (n=98), PA-only (n=97), and a successful aging (SA) health education control (n=93). DXA-acquired body composition measures (total body fat and lean mass), conventional biomarkers of cardiometabolic risk, and 400-m walk time were obtained at baseline and 18 months.

Results—Fat mass was significantly reduced from (X±SE) 36.5±8.9 kg to 31.7±9.0 kg in the PA +WL group (p<0.01), but remained unchanged from baseline in the PA-only (−0.8±3.8 kg) and SA (−0.0±3.9 kg) groups. Lean mass losses were three times greater in the PA+WL group compared to PA-only or SA groups (−2.5±2.8 kg vs. −0.7±2.2 kg or −0.8±2.4 kg, respectively; p<0.01); yet due to a larger decrease in fat mass, percent lean mass was significantly increased over baseline in the PA+WL group (2.1%±2.6%; p<0.01). Fat mass loss was primarily responsible for WL-associated improvements in cardiometabolic risk factors, while reduction in body weight, regardless of compartment, was significantly associated with improved mobility.

Conclusion—This 18 month PA+WL program resulted in a significant reduction in percent body fat with a concomitant increase in percent body lean mass. Shifts in body weight and

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composition were associated with favorable changes in clinical parameters of cardiometabolic risk and mobility. Moderate PA without WL had no effect on body composition.

Keywords

weight loss; physical activity; body composition; cardiometabolic risk; functional decline; aging

Introduction

The number of obese, older adults in the United States is large and growing.[1;2] Both advanced age and obesity are well characterized risk factors for chronic disease and disability.[3] Unless actions are taken to reverse these trends, the individual and societal burden of age- and obesity-related adverse health conditions is projected to increase substantially over the next few decades.[4]

Many health complications associated with higher body mass index (BMI) and greater fat mass are improved with intentional weight loss (WL).[5] Although evidence suggests that lifestyle-based therapies, such as diet and exercise, are successful at promoting weight and fat mass loss in older adults,[6–12] clinical recommendation for WL in aging remains controversial.[13;14] Reluctance stems, at least in part, from the perceived potential for functional limitations associated with loss of both lean and bone mass (known to accompany overall WL[15]), as well as uncertainties surrounding the long-term feasibility and health correlates of intentional WL in this population.

A recent review by Waters and colleagues[13] identified only one study investigating long-term WL and associated health implications in older (≥ 65 years) adults.[16] In this small (n=16) pilot study, which presented 30 month follow-up data to a 12 month WL and physical activity (PA) intervention,[17] older adults maintained, on average, significant WL from baseline at 30 months, despite 18 months of no active intervention (~10% WL reported at 12 months compared to ~7% WL reported at 30 months). Both fat and lean mass, which significantly decreased from baseline to 12 months, remained significantly below baseline at 30 months, and the 7% WL maintenance conferred clinically significant improvements in physical function and insulin sensitivity. Thus, long-term, intentional weight and fat mass loss appears possible in obese, older adults; although confirmation of these findings awaits results from large, long-term randomized controlled trials (RCTs). Furthermore, delineation of the independent contributions of long-term reductions in body, lean, and fat mass on clinically important endpoints for older adults, such as cardiometabolic risk and mobility, has yet to be assessed. Collectively, such information is necessary to (1) comprehensively evaluate the long-term benefits and risks of intentional WL in this population, and (2) optimize intervention strategies by identifying the most clinically relevant target tissue.

Therefore, the primary purpose of this study is to determine the effects of long-term PA and WL on body composition in overweight and obese older adults. A secondary purpose is to evaluate the association between body, lean, and fat mass loss on change in several cardiometabolic risk factors and mobility. We hypothesize that PA+WL will result in significant 18 month reductions in body, lean and fat mass, with loss of fat mass most

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strongly predictive of improvements in cardiometabolic risk factors and mobility when compared to total body mass or lean mass losses.

Methods and Procedures

Study Design

This analysis utilized data from the Cooperative Lifestyle Intervention Program (CLIP), a RCT of PA and WL on mobility in overweight or obese older adults with/at risk for CVD. The study was conducted within North Carolina Cooperative Extension Centers and a total of 288 participants were randomized over a 2.5 year period. Intervention length was 18 months, and treatment groups included PA+WL (n=98), PA-only (n=97), or a successful aging (SA; n=93) education control arm. All outcome assessors were blinded to group assignment. The Wake Forest University institutional review board approved the study, all participants signed an informed consent document, and the primary outcome paper has been published.[12]

Study Participants

Detailed study inclusion and exclusion criteria are published.[12] Briefly, individuals were eligible to participate if they were identified as ambulatory, overweight or obese (BMI >28 but <40 kg/m^2), community-dwelling older (60–79 years) adults who either had CVD or cardiometabolic dysfunction and self-reported limitations in mobility. A Consolidated Standards of Reporting Trials (CONSORT) diagram is published,[12] detailing participant flow through the recruitment and 18 month intervention period.

Interventions

The PA+WL arm involved a PA intervention in conjunction with a dietary WL intervention. The PA program consisted of a combination of daily walking and interactive, group-mediated, behavioral focused sessions (48 total sessions over 18 months), with the primary goal of gradually increasing home-based, moderate intense activity to >150 minutes/week. The PA intervention was divided into two phases: an intensive phase (first six months) and maintenance phase (six-18 months). During the intensive phase, participants attended weekly, supervised behavioral sessions, focused on increasing PA and reducing caloric intake. Three group sessions (90 minutes, consisting of 30–45 minute period of walking followed by a behavioral-focused session) and one individual session (30 minutes) were conducted each month. Individual sessions included one-on-one interactions with staff based on the unique needs of the participant (including review of behavioral tools, execution of techniques or strategies for lifestyle change, brainstorming or problem solving barriers to change, motivation, or simply to touch base with study staff on overall program progress). In addition to the behavioral sessions, participants were asked to walk for 30 minutes on most days of the week at a moderate level of intensity (defined as a self-reported rating of perceived exertion of 13 on the Borg Scale).

During the next 12 months (maintenance phase), frequency of contact was reduced (one group session of 90 minutes and one telephone contact per month), and group discussion focused on PA goals, specific plans of action to be implemented, and the reinforcement of

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self-regulatory skills. The WL goal was a reduction in body mass of approximately 0.3 kilograms (kg) per week for the first six months, for a total loss in mass of 7–10% of initial body mass. During the weight maintenance phase, participants were encouraged to continue WL as long as their BMI was >20 kg/m^2; however, the primary focus was on maintenance of WL.

The PA-only arm consisted of the PA intervention described above. The SA health education intervention was an active control arm. Participants randomized to the SA group met in groups, weekly for the first eight weeks, monthly through the sixth month, and bimonthly until the end of the study (18 sessions total). Sessions included health topics relevant to older adults such as how the body changes with aging, prevention or delaying disease, eating for good health, positive attitudes toward aging, family relationships and care giving, and talking to health care providers. Further details of all treatment arms can be found in the primary outcome paper.[12]

Measurements

Baseline assessments included self-reported demographic, medical history, co-morbidity information, and objectively measured PA energy expenditure (PAEE). Lifecorder-EX accelerometers (New-Lifestyles Inc, Lees Summit, Missouri) were used to assess PA level in all participants. Participants were asked to wear the accelerometer for 7 days at the baseline and 18 month assessment visits. Intensity levels 3 to 9 were classified as “moderate to vigorous”; consistent with the metabolic demands of activity for this age group. Follow-up visits occurred at 6, 12, and 18 months.

Body Composition

Participant height and body mass were assessed at baseline, 6, and 18 months. Height was measured using a stadiometer and body mass was measured on a calibrated electronic scale. BMI was calculated as body mass in kg divided by height in meters squared. Total body fat and lean mass were assessed using dual-energy X-ray absorptiometry (DXA, Hologic Delphi A 11.0 QDR, Bedford, MA) at baseline and 18 months.

Cardiometabolic Risk Factors

Blood was collected in the morning via venipuncture after an overnight fast and abstinence from PA for 24 hours prior to each assessment period. Samples were collected in EDTA-treated vacutainers and separated after centrifugation for 20 min at 4°C. Aliquots of plasma were stored at −70°C until the analyses were conducted. All blood was collected, processed, and analyzed for glucose, serum insulin, total cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) using standardized procedures in a certified laboratory. HOMA-IR was computed (from measures described above) as follows: fasting insulin (μIU/mL) x fasting glucose (mg/dL)/405.

Mobility

The 400-meter walk test was used to assess mobility.[18] Participants were instructed to complete the distance walking “as fast as possible” and time was recorded in seconds.

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Statistical Analysis

Descriptive statistics were calculated overall and by intervention group at baseline. Means and standard deviations were calculated for each measure of body mass and composition, by intervention group and time point. Analysis of covariance was used to determine the overall intervention effect on individual measures of body composition, with results presented as means and standard deviations, after adjustment for recruitment wave, sex, age, and baseline body composition measure. To determine the contribution of PA level on PA+WL intervention effectiveness on body composition, PA level (based on accelerometry) at baseline and 18 months as well as change in PA level from baseline to 18 months were added as covariates to the primary analytic models. To determine the contribution of change in weight and body composition on previously published intervention effects on mobility,[12] change in weight, change in lean mass, and change in fat mass were added separately to the analytic model used in the main outcome paper. Finally, in the PA+WL arm only, 18 month changes in cardiometabolic risk factors per kg change in body mass, lean mass, and fat mass were modeled using linear regression, adjusting for recruitment wave, sex, and baseline risk factor value.

Results

Participant Baseline Characteristics

Baseline demographic data on the CLIP study population (n=288) are published.[12] Briefly, participants were 67.0±4.8 years of age with an average BMI of 32.8±3.8 kg/m^2. Sixty-seven percent of participants were women, 82% were of Caucasian descent, and average PAEE at baseline was 1292±599 kcal/week. Sixty-nine percent of participants presented with hypertension, 17% with type 2 diabetes, and 56% with MetS (classified based on NCEP ATP III criteria). All participants reported limitations in mobility at baseline, and objective measures of physical function revealed an average short physical performance battery score of 9.95±1.46 and time to complete 400-m walk of 352.0±74.9 seconds.

Baseline body composition and cardiometabolic risk factor variables are presented in Table 1. On average, participants were 38.9±7.0% body fat. For most, blood lipid values were ideal or nearly ideal (total cholesterol; TC = 190.3±41.5 mg/dL, low-density lipoprotein cholesterol; LDL-C = 108.6±34.6 mg/dL; high-density lipoprotein cholesterol; HDL-C = 42.0±9.2 mg/dL for men and 54.4±12.3 mg/dL for women; triglyceriges; TG= 158.3±68.1 mg/dL). Fasting blood glucose was modestly elevated (107.8±19.6 mg/dL), although insulin values fell within the normal range (17.2±10.5 μIU/mL). No significant differences in body composition or cardiometabolic risk were seen by random assignment to treatment group.

Intervention Adherence and Effect on Change in Body Mass and Composition

As previously reported,[12] 86.5% of initially enrolled participants completed the 18-month follow-up visit. On average, participants in the PA+WL arm attended 88.2±25.2% of sessions, participants in the PA group attended 79.8±24.6% of sessions, and participants in the SA group attended 70.9±26.5% of sessions.

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Change in body mass for participants with baseline and follow-up data (n=253) observed during the first 6 months of intervention (intensive phase) was −7.3±7.1 kg, −1.3±5.1 kg, and −1.0±6.2 kg for the PA+WL, PA-only, and SA groups, respectively (p<0.01), with no difference observed between PA-only and SA groups (p=0.84). Regardless of group assignment, average body mass remained stable from the 6 to 18 month visits. Eighty-four percent of participants in the PA+WL group weighed less at 18 months than at baseline, although 30% regained at least 2 kg of lost weight from the 6 month visit. On average, this group experienced a sustained reduction in baseline body mass of 7.7±6.8%.

Unadjusted body composition variables by treatment group at 18-months are presented in Table 2. Fat mass was significantly reduced from 36.5±8.9 kg to 31.7±9.0 kg in the PA+WL group (p<0.01, PA+WL vs PA p<0.01, PA+WL vs. SA p<0.01), but remained virtually unchanged in the PA-only (−0.8±3.8 kg) and SA (−0.0±3.9 kg) groups, with no difference between the two (p=0.27). All groups lost a significant amount of lean mass over the 18 month period (all p≤0.01); however, the PA+WL group experienced a reduction in lean mass that was roughly three times the amount lost in by the PA-only and SA groups (−2.5±2.8 kg versus −0.7±2.2 kg or −0.8±2.4 kg, respectively; both p<0.01). Of note, despite this absolute reduction in lean mass, percent lean mass increased in the PA+WL group (2.1±2.6% PA+WL versus 0.3±2.3% PA-only or −0.3±1.9% SA; p<0.01) with no difference observed in PA-only or SA groups.

Also presented in Table 2, are the intervention effects on body composition at 18 months, adjusted for recruitment wave, sex, age, and baseline outcome measure. Even after adjustment, all measures of body composition were significantly reduced in the PA+WL group compared to PA-only or SA groups (p<0.01). Of note, all body composition comparisons between PA-only and SA groups were non-significant (all p>0.05). To examine the relative contributions of PA on changes in body composition observed in the PA+WL group, associations between 18 month body composition changes and weekly PAEE (via accelerometry) were modeled (data not shown). When modeled separately, and using the same covariate adjustment, baseline, 18 month, or change in PAEE from baseline to 18 months did not predict change in body, fat, or lean mass (all p>0.05).

Relationships between Changes in Body Composition and Changes in Cardiometabolic Risk and Mobility

Table 3 presents the 18 month changes in several cardiometabolic risk factors per unit change in body mass (kg), lean mass (kg), and fat mass (kg) in the PA+WL group only. Reduction in body mass was associated with lowered diastolic blood pressure, triglycerides, glucose, insulin and HOMA-IR, and elevations in HDL-C (all p≤0.02). Changes in diastolic blood pressure, glucose and HDL-C were driven by changes in fat (rather than lean) mass, while changes in triglycerides, insulin and HOMA-IR were related to changes in both fat and lean mass compartments. When modeled together, there was a significant interaction between change in fat mass and change in lean mass for models involving insulin (β=0.11 SE [0.03]; p<0.01) and HOMA-IR (β=0.03 SE [0.01]; p=0.02), but not triglycerides (p>0.05). For both insulin and HOMA-IR, loss of both fat and lean mass attenuated improvements observed when just one compartment was reduced.

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As previously published, the PA+WL group improved their 400-meter walk time (adjusted mean [SE], 323.3 [3.7] seconds) compared with both PA (336.3 [3.9] seconds; p=0.02) and SA ((341.3 [3.9] seconds; $p<0.01$); overall $p<0.01$) groups.[12] When changes in body weight, lean mass, or fat mass were added individually to the primary analytic model, the previously significant overall treatment effect was attenuated to non-significance, while change in weight and lean mass were significant predictors of follow-up 400-m walk time (β [SE]: 0.87 [0.49] s/kg, $p=0.02$ and 2.92 [1.27] s/kg, $p=0.02$, respectively), and change in fat mass was marginally significant (1.39 [0.74] s/kg, $p=0.06$). When change in body weight, lean mass, fat mass were added in lieu of treatment effect, all body weight and composition variables were significant and direct predictors of follow-up 400 meter walking time.

Discussion

The clinical recommendation for intentional WL to treat obesity in older adults remains controversial, partially due to lack of data demonstrating the long-term efficacy and safety of WL programs to reduce body and fat mass in this population. Primary findings from this study confirm and extend the external validity of the work presented by Villareal et al.[17], demonstrating that an 18 month, behaviorally based, PA+WL intervention is successful in achieving and maintaining clinically significant WL in most overweight and obese, older adults.

Examination of intervention related changes in body composition reveal that two-thirds of lost weight is from fat mass and one-third is from lean mass, which is in general agreement with findings reported in other studies.[15] Importantly, despite a significant reduction in lean mass, individuals in the PA+WL group experienced an increase in percent body lean mass (along with a concomitant decrease in percent body fat mass), evidence of a favorable shift in body composition. Also of interest, PA without WL was not found to significantly alter body composition when compared to control. Secondary results from this study provide clinical translation of change in body composition, suggesting that fat mass loss is primarily responsible for WL-associated improvements in cardiometabolic risk, and that reduction in body weight, regardless of compartment, is significantly associated with improved mobility.

Overall, WL success reported in this trial is encouraging; with 84% of participants in the PA +WL group weighing less at 18 months than at baseline and sustaining an average WL of 7.7%. Long-term WL maintenance of this magnitude is in general agreement with the pilot study findings in frail, obese, older adults reported by Waters et al.[16] and notably, is clinically meaningful.[19] Nevertheless, we observed considerable variability about this average WL estimate (SD=6.8%), with 30% of participants in the PA+WL group experiencing weight regain of at least 2 kg during the 12 month “weight maintenance” period. Therefore, the PA+WL program utilized in this trial was capable of inducing and preserving clinically meaningful weight loss for most, but not all, CLIP participants. Given the potential negative consequences of weight regain on body composition and cardiometabolic health,[20;21] future WL studies in overweight and obese, older adults should focus on identifying barriers to (and consequences of not) maintaining WL beyond the intervention timeframe.

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Better understanding of how long-term intentional WL and associated shifts in body composition affect risk of chronic disease and disability in older adults is necessary to comprehensively evaluate the clinical recommendation for WL in this population. Although definitive outcomes were not assessed, our results show that improvements in several risk factors for cardiovascular and metabolic disease correlate with the magnitude of WL, and are influenced primarily by fat mass loss. Improvements in insulin and insulin resistance were influenced by loss of both fat and lean mass, although interestingly, individual improvements were attenuated when both compartments were reduced. Though difficult to explain, the effect of loss of fat mass and lean mass on insulin and insulin resistance do not appear to act independently. It may be that the PA+WL intervention induced reductions in intermuscular fat (and subsequent reductions in total muscle size), thereby improving insulin sensitivity.[22] However, the imaging methodology utilized to capture body composition in this study is limited it its ability to quantify the quality of lean mass loss. Future WL studies including more sophisticated measures of body composition, such as computed tomography (CT), may help to clarify this finding. At present, our data suggest that targeted reduction of fat mass, while preserving lean mass, should provide maximal cardiometabolic benefit for overweight and obese, older adults.

The beneficial effect of fat mass loss on cardiometabolic risk may be mediated by changes in inflammatory biomarkers. Epidemiological and clinical studies have shown strong and consistent relationships between markers of inflammation and risk of future cardiovascular events.[23] Specifically, high concentrations (i.e., >3.0 pg/mL) of the inflammatory cytokine interleukin-6 (IL-6) predict increased risk of CVD-related mortality in older adults.[24;25] The CLIP intervention effect on serum levels of IL-6 is published,[26] with the PA+WL group achieving significant reductions over the 18 month period (2.9±2.0 pg/mL to 2.5±1.8 pg/mL), with no change observed in the PA-only and SA groups. Thus, it is possible that reduced IL-6 may partially underlie the observed relationship between reduced fat mass and improvements in cardiometabolic risk factors.

While clinical implications of WL-induced reductions in lean mass are not fully elucidated, results presented here suggest that loss of weight, regardless of compartment, is associated with the improvements in mobility seen in the PA+WL group. Our findings likely reflect greater absolute loss of fat mass compared to lean mass (evidenced by an increase in percent lean mass), and are illustrative of an emerging body of research suggesting that factors other than absolute muscle mass, such as muscle strength and quality,[31] fat infiltration into muscle,[28;32;33] and inflammation,[34] are stronger predictors of functional status in older adults. Indeed, a related publication from our group (including additional physical function outcome data from CLIP) systematically examined the effect of lean and fat mass loss on physical function in several studies of intentional WL in overweight and obese, older adults.[27] Results demonstrate that loss of total body mass – including losses of both fat and lean mass compartments - is associated with significant improvement in self-reported mobility disability and walking speed; with fat mass loss conferring greater predictive power of change in physical function than lean mass loss. In fact, lean mass loss was not independently associated with changes in any functional measures assessed, including self-reported mobility disability, walking speed, chair stand time, or short physical performance battery score. Likewise, improvements in muscle strength and power have also been seen in

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older adults following intentional WL, despite significant reductions in lean tissue size.[29;30] Taken together, these findings help to temper the negative clinical interpretations of WL-associated loss of lean mass in older adults, notwithstanding the fact that the attenuation of lean mass loss in older adults seems prudent and is currently recommended.[35]

The moderate level of aerobic PA achieved with this study design was insufficient to influence shifts in body composition; therefore, practitioners aiming to maximize fat mass losses while minimizing lean mass losses in obese older adults are advised to focus first on caloric restriction and then select an intensity and modality of PA (i.e., heavy resistance training) shown to attenuate lean mass losses associated with intentional WL.[10;29;36] Although few in number, studies designed specifically to examine the effects of PA+WL compared to WL alone in older adults consistently show more beneficial effects on body composition and physical function when WL is combined with PA.[17;36;37]

This study is not without limitations. Body composition data were only available at baseline and 18 months; however, in the PA+WL group, intervention modality changed from an intensive WL (0–6 months) to a WL maintenance (6–18 months) phase. Information on body composition at the end of the intensive phase would allow for examination of how body composition changes as a function of the rate of WL, or with regard to weight regain. Additionally, measures of body composition are highly correlated (i.e. r= 0.70–0.95 in our study), and teasing apart the effects of change in lean mass and fat mass on cardiometabolic risk and mobility is a challenge only crudely accounted for by statistical adjustments. Lastly, although data from this study provided some clinical translation of change in body composition to cardiovascular outcomes, only intermediate cardiometabolic endpoints were assessed. Forthcoming data from the Look AHEAD trial,[38] a large (n= 5,145) RCT designed to examine the effects PA+WL on risk of CVD mortality in obese adults with type 2 diabetes, should provide more definitive evidence on the effects of change in body weight and composition on hard clinical outcomes, along with long-term feasibility of behavioral WL interventions.

In sum, we demonstrated that an 18 month behaviorally-based, PA+WL program resulted in clinically significant loss of weight in overweight and obese, older adults and shifted body composition to a lower percentage of total body fat mass and a higher percentage of total body lean mass. In contrast, PA alone did not influence change in body composition. Fat mass loss was associated with improvement in several clinical cardiometabolic risk factors, although changes in triglycerides, insulin and insulin resistance were associated with loss of both fat and lean mass. Loss of body mass, regardless of compartment, was associated with improvements in mobility.

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What is already known about this subject?

- Many health complications associated with higher body mass index and greater fat mass are immediately improved with weight loss.
- Overall weight loss encompasses loss of both fat and fat-free mass, the latter of which may exacerbate functional limitations for the older adult.

What this study adds?

- Results from this large, randomized controlled trial show that an 18 month, physical activity and weight loss intervention was successful in achieving clinically significant weight loss in overweight and obese older adults.
- Despite a significant absolute reduction in lean mass, individuals in the physical activity and weight loss group experienced an increase in percent body lean mass (along with a concomitant decrease in percent body fat mass), indicative of a favorable shift in body composition.
- Physical activity without a weight loss intervention did not significantly alter body composition when compared to control.

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Table 1

Baseline body composition and cardiometabolic risk factors according to treatment group.

Baseline Body Composition and Cardiometabolic Risk Factors	PA+WL (n=73–98)	PA-only (n=73–97)	SA (n=68–93)
Body Composition			
Body Mass (kg)	92.8±16.1	91.7±13.1	91.2±15.1
BMI (kg/m^2)	33.1±4.1	32.8±3.9	32.6±3.5
Fat Mass (kg)	36.5±8.9	36.3±8.9	35.3±7.5
Lean Mass (kg)	57.3±12.2	56.9±11.3	56.7±11.7
% Fat (%)	39.0±6.8	39.0±7.6	38.6±6.6
% Lean (%)	58.5±6.5	58.5±7.3	58.9±6.4
Cardiometabolic Risk Factors			
Systolic Blood Pressure (mmHg)	136.5±16.5	133.1±16.5	133.4±15.3
Diastolic Blood Pressure (mmHg)	76.2±10.0	74.1±10.4	73.2±10.8
Total Cholesterol (mg/dL)	190.1±38.3	186.9±40.1	194.3±45.7
LDL (mg/dL)	107.2±33.1	106.7±31.4	112.1±39.5
HDL (mg/dL)			
Men	41.2±8.1	43.8±11.3	40.7±7.7
Women	55.7±11.9	51.9±11.5	55.5±13.5
Triglycerides (mg/dL)	160.6±72.2	155.7±66.8	158.8±66.0
Glucose (mg/dL)	107.3±15.1	107.7±20.6	108.5±22.9
Insulin (μIU/mL)	17.9±9.8	17.1±11.2	16.6±10.6
HOMA-IR (μIU/mL* mg/dL)	4.8±2.7	4.8±4.5	4.8±4.1

Data are presented as means ± SD or n (%). Baseline sample sizes varied by outcome measures for each treatment group. PA+WL: n=98 for body weight and BMI; n=95 for DXA variables, 73 for blood lipids and 92 for glucoregulatory markers; PA: n=97 for body weight and BMI; n=96 for DXA variables, 73 for blood lipids and 90 for glucoregulatory markers; SA: n=93 for body weight and BMI; n=91 for DXA variables, 68 for blood lipids and 83 for glucoregulatory markers. Abbreviations: kg = kilograms; BMI = body mass index; m = meters; % = percentage; PA = physical activity; WL = weight loss; SA = successful aging; n = sample size; BL = baseline; 18M = 18 months; SD = standard deviation.

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Table 2

Overall intervention effects on body composition measures at 18 months.

Body Composition Outcome Measures at 18 Months	PA+WL (n=81–88)	PA-only (n=75–83)	SA (n=77–82)	*p*-value			
				Group × Time	PA+WL vs. SA	PA-only vs. SA	PA+WL vs. PA-only
Body Mass (kg)	85.7±15.5	90.9±14.4	90.3±16.0	<.0001	<.0001	0.9848	<.0001
BMI (kg/m^2)	30.7±4.2	32.6±4.5	32.5±3.9	<.0001	<.0001	0.9999	<.0001
Fat Mass (kg)	31.7±9.0	35.7±9.6	35.4±7.7	<.0001	<.0001	0.5787	<.0001
Lean Mass (kg)	55.1±11.6	56.1±11.2	55.7±11.6	<.0001	0.0001	0.9995	0.0001
% Fat (%)	36.4±7.5	38.8±7.6	39.0±6.6	<.0001	<.0001	0.3386	<.0001
% Lean (%)	60.8±7.2	58.7±7.3	58.4±6.4	<.0001	<.0001	0.3052	<.0001

Raw body composition data are presented as means ± SD. Presented *p*-values are adjusted for recruitment wave, sex, age, and baseline outcome measure. Sample sizes varied by outcome measures for each treatment group. PA+WL: n=88 for body weight and BMI; n=81 for DXA variables; PA: n=83 for body weight and BMI; n=75 for DXA variables; SA: n=82 for body weight and BMI; n=77 for DXA variables. Abbreviations: kg = kilograms; BMI = body mass index; m = meters; % = percentage; PA = physical activity; WL = weight loss; SA = successful aging.

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Table 3

18 month changes in cardiometabolic risk factors per unit change in weight, lean mass, and fat mass in PA+WL group.

Cardiometabolic Risk Factor	Δ Body Mass (kg)			Δ Lean Mass (kg)			Δ Fat Mass (kg)		
	B	SE	*p*-value	B	SE	*p*-value	B	SE	*p*-value
Δ Systolic Blood Pressure (mmHg)	0.31	0.23	0.19	1.17	0.60	0.05	0.20	0.37	0.60
Δ Diastolic Blood Pressure (mmHg)	0.32	0.13	0.02	0.55	0.34	0.10	0.40	0.20	0.05
Δ Total Cholesterol (mg/dL)	0.81	0.65	0.22	1.26	1.76	0.48	1.11	1.01	0.27
Δ LDL (mg/dL)	0.60	0.56	0.29	0.38	1.50	0.80	0.95	0.85	0.27
Δ HDL (mg/dL)	−0.36	0.11	<0.01	−0.43	0.31	0.17	−0.58	0.16	<0.01
Δ Triglycerides (mg/dL)	2.56	0.91	0.01	5.52	2.46	0.03	3.34	1.39	0.02
Δ Glucose (mg/dL)	0.66	0.28	0.02	1.18	0.80	0.14	0.86	0.44	0.06
Δ Insulin (μIU/mL)	0.52	0.10	<0.01	1.47	0.27	<0.01	0.72	0.16	<0.01
Δ HOMA-IR (μIU/mL* mg/dL)	0.15	0.03	<0.01	0.41	0.08	<0.01	0.21	0.05	<0.01

Model-adjusted estimates control for recruitment wave, sex, and baseline risk factor value. Abbreviations: mmHg = millimeters mercury; mg = milligrams; dL = deciliter; μIU = microunits; kg = kilogram; B = parameter estimate; SE = standard error.