Physical activity, protein intake, and appendicular skeletal muscle mass in older men

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ABSTRACT

Background: Aging is associated with physical inactivity, low energy intake, and loss of skeletal muscle mass. It is not clear whether regular physical activity and adequate dietary protein intake can attenuate the loss of skeletal muscle mass.

Objective: We hypothesized that the maintenance of physical activity and dietary protein intake would attenuate the age-related decline in total appendicular skeletal muscle mass.

Design: Total appendicular skeletal muscle mass was determined by dual-energy X-ray absorptiometry in 44 healthy, older white men aged 49–85 y. Physical activity level was determined by using a uniaxial accelerometer over a 9-d period. Dietary protein intake was estimated from a 3-d food record.

Results: Aging was inversely associated with total appendicular skeletal muscle mass in older men ($r = -0.43$; slope: $-0.119 \pm 0.039$ kg/y; $P < 0.01$). An effect of age on appendicular skeletal muscle mass persisted after standing height and physical activity were controlled for ($r = -0.34$; slope: $-0.120 \pm 0.052$ kg/y; $P = 0.03$). Furthermore, an effect of age on appendicular skeletal muscle mass persisted after standing height and dietary protein intake per kilogram body mass was controlled for ($r = -0.41$; slope: $-0.127 \pm 0.045$ kg/y; $P < 0.01$).

Conclusions: Maintaining regular physical activity and adequate protein intake may not offset the age-related loss of appendicular skeletal muscle mass in older men. Prospective studies are needed to confirm these results and to determine whether anabolic physical activity (eg, strength training) can attenuate the age-related loss of muscle mass in the elderly.

KEY WORDS Aging, sarcopenia, uniaxial accelerometer, dual-energy X-ray absorptiometry, DXA, elderly men, skeletal muscle, appendicular muscle mass, protein intake

INTRODUCTION

The etiology of age-related skeletal muscle mass loss is unclear (1). Physical activity (2) and dietary protein intake (3) decrease with aging and may influence the loss of skeletal muscle mass. Recent data suggest that increased protein intake (4) and physical activity, in the form of resistance training (5), stimulate muscle protein synthesis in the elderly, although these results are divergent (6, 7). Thus, physical inactivity and low protein intake may be partially related to the loss of muscle mass with aging.

The lack of accurate, noninvasive measures of skeletal muscle mass (8) has limited the systematic examination of factors modulating muscle loss with aging. The adaptation of dual-energy X-ray absorptiometry (DXA) to measure fat and bone-free lean tissue masses over the past decade has allowed a timely and noninvasive assessment of appendicular skeletal muscle mass (9). Bone-free lean tissue mass of the arms and legs can be determined and used as an estimate of total appendicular skeletal muscle mass. A strong concordance has been shown between DXA and computed tomography for estimating total appendicular skeletal muscle mass (10).

Although other studies have examined the effects of age and sex on appendicular skeletal muscle mass determined from DXA (11–13), few studies have considered the effects of physical activity and dietary protein intake on the age-related loss of skeletal muscle. Therefore, the primary aims of this study were to determine the rate of decline in total appendicular skeletal muscle mass of older, white men and to examine whether variations in physical activity and dietary protein intake influence this loss.

SUBJECTS AND METHODS

Subjects

Subjects were 44 healthy, older white men between 49 and 85 y of age recruited from greater Burlington, VT, via local advertisements. All participants were healthy and had no history or evidence of physical examination of 1) coronary heart disease (eg, ST segment depression > 1 mm at rest or exercise), 2) hypertension (resting blood pressure > 140/90 mm Hg), 3) medications that could affect cardiovascular function or metabolism, 4) diabetes, 5)
body mass fluctuation > 2 kg in the past year, exercise-limiting noncardiac disease (arthritis, peripheral vascular disease, or cerebral vascular disease), or smoking. Each subject signed a consent form approved by the Institutional Review Board of the University of Vermont before participating in the study.

Testing protocol and measurements

All subjects were tested at the General Clinical Research Center at the University of Vermont. Body composition was assessed by using DXA and a 12-h fasting blood sample was obtained. Each subject completed a 3-d dietary record and wore a uniaxial Caltrac accelerometer (Muscle Dynamics Fitness Network, Torrance, CA) for a 9-d period to measure physical activity. Specific details about data collection are provided below.

**Body composition and appendicular skeletal muscle**

Body mass was measured to the nearest 0.1 kg by using an electronic scale (Scale-Tronix Inc, Wheaton, IL) and standing height was determined with a wall-mounted stadiometer. Fat mass, lean tissue mass, and bone mineral content were measured with a whole-body DXA scan made with use of a Lunar DPX-L densitometer (Lunar Radiation Corp, Madison, WI). Total appendicular skeletal muscle mass was determined as described by Heymsfield et al (9). Briefly, the identification of anatomic landmarks and the use of system software (Lunar version 1.3y) allowed the quantification of total fat mass and lean tissue mass (minus bone mineral content) for the arms (ie, center of the arm socket to the tip of the distal phalanx) and legs (ie, femoral socket to the tip of the distal phalanges). Total appendicular skeletal muscle mass was calculated as the sum of lean tissue mass of the arms and legs. The CV for test-retest of total appendicular skeletal muscle mass within 1 wk in 7 older women from our laboratory is 0.8% for arm and 1.0% for leg lean tissue mass.

**Physical activity**

Physical activity energy expenditure was determined with a uniaxial accelerometer. This accelerometer was worn during all waking hours over a 9-d period; it was firmly attached to a belt or the waistband of clothing, directly inferior to the greater trochanter. The accelerometer used measures of walking and running energy expenditure in addition to calculating non-weight-bearing activities such as weightlifting, bicycling, rowing, and strenuous upper-body motions, by using unpublished correction factors. All accelerometers were concurrently agitated with a mechanical shaker under standardized conditions over a 24-h period and those accelerometers with activity energy counts >2.5% of the mean reading for the group were not used. Average daily physical activity energy expenditure (kJ/d) over the 9-d measurement period was used for data analyses.

**Dietary intake**

Each subject was instructed to maintain his normal dietary intake throughout the study as described previously (14). A dietitian provided each subject with a 5-lb (11-kg) food scale and instructed him on how to complete a 3-d dietary record. Diets were recorded on 2 weekdays and 1 weekend day. Dietary analyses were completed by using FOOD PROCESSOR software (EHSA Research, Salem, OR) to determine daily energy and protein intakes. Protein intake was expressed as g/kg body mass and as a percentage of daily energy intake for all data analyses.

**Serum albumin**

Serum albumin concentrations were determined by using a standardized bromcresol green colorimetric assay.

**Statistical analyses**

All data are expressed as means ± SDs. Regression analyses were used to determine the rate of decline in total appendicular skeletal muscle mass per decade. Partial correlation analyses were used to examine the relation between age and total appendicular skeletal muscle mass (also arm and leg separately) after standing height, physical activity, and dietary protein intake were controlled for. Standing height was used in the partial correlation analyses to remove the body size influence on muscle mass (13); however, body mass was not used in partial analyses because of its covariance with appendicular muscle mass (ie, muscle mass is a direct component of body mass) as described previously (15). Secondary correlational analyses were also done to examine the relations between total appendicular skeletal muscle mass and serum albumin and dietary protein intake. Significance was accepted at the \( P < 0.05 \) level.

**RESULTS**

Descriptive characteristics for all subjects are presented in Table 1. The men participating in this study covered an age span of 35 y, were lean to moderately overweight, and had low-to-moderate cardiorespiratory fitness. Total appendicular skeletal muscle mass, physical activity, dietary intake, and serum albumin data are presented in Table 2.

Age was inversely correlated with total appendicular skeletal muscle mass in older men (Figure 1). Partial correlation data between age and total appendicular skeletal muscle mass (also arm and leg data separately) adjusted for standing height, physical activity, and dietary protein intake are presented in Table 3. After standing height and physical activity level were controlled for, the inverse relation between age and total appendicular skeletal muscle mass persisted. Furthermore, a significant inverse relation persisted between age and total appendicular skeletal muscle mass after standing height and dietary protein (g/kg body mass) or dietary protein expressed as a percentage of daily energy intake were controlled for \( (r = -0.38, P = 0.01) \). The inverse association between age and total appendicular skeletal muscle mass after adjustment for standing height and physical activity \( (r = -0.46, P = 0.03; n = 23) \) or dietary protein intake \( (r = -0.50, P = 0.03; n = 23) \) also persisted when individuals who may have underestimated protein intake were excluded.

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**Table 1**

Descriptive characteristics of healthy, older, white men

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>( 67 ± 11 ) (49–85)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>( 174 ± 6 ) (160–185)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>( 79 ± 14 ) (58–139)</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>( 26.2 ± 4.3 ) (18–41)</td>
</tr>
<tr>
<td>Lean tissue mass (kg)</td>
<td>( 58 ± 7 ) (43–79)</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>( 16 ± 5 ) (11–25)</td>
</tr>
<tr>
<td>Body fat (% of lean tissue)</td>
<td>( 21 ± 7 ) (6–39)</td>
</tr>
<tr>
<td>( VO_2 \text{max} ) (L/min)</td>
<td>( 2.67 ± 0.76 ) (1.27–4.47)</td>
</tr>
</tbody>
</table>

\( n = 44 \), \( VO_2 \text{max} \), maximal aerobic treadmill capacity.

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[ie, those whose resting metabolic rate was < 1.4 \times \text{predicted resting metabolic rate} (16)].

Total appendicular skeletal muscle mass was associated with absolute dietary protein intake ($r = 0.45, P < 0.01$), although protein intake expressed as a percentage of energy intake ($r = 0.12, P = 0.43$) or per kilogram body mass ($r = 0.12, P = 0.44$) was not associated with total appendicular skeletal muscle mass. When men with a daily dietary protein intake < 0.80 g/kg body mass were excluded, there was no relation between total appendicular skeletal muscle mass and dietary protein intake expressed per kilogram body mass ($r = -0.08, P = 0.66; n = 36$) after standing height and physical activity were controlled for. Moreover, there was no association between total appendicular skeletal muscle mass and serum albumin in older men after physical activity and dietary protein intake were controlled for ($r = 0.20, P = 0.20$).

### DISCUSSION

We hypothesized that the maintenance of physical activity and dietary protein intake would attenuate the age-related decline in total appendicular skeletal muscle mass in healthy, older white men. Contrary to our hypothesis, the results suggest that the maintenance of physical activity and protein intake may exert relatively little influence on the loss of appendicular skeletal muscle mass in older men, although longitudinal data are needed to confirm these results.

### Aging and total appendicular skeletal muscle mass

Although sarcopenia is considered a significant clinical problem, there is a paucity of data about it. This may be partly attributable to the lack of accurate methods for quantifying skeletal muscle mass and the absence of longitudinal studies. Several methods, each with inherent limitations, have been reported to measure skeletal muscle mass (17). We used DXA to quantify total appendicular skeletal muscle mass of the arms and legs. DXA provides adequate precision (18) and close agreement with computed tomography for quantification of muscle mass (10).

Our cross-sectional data suggest that appendicular skeletal muscle mass declines at a rate of \( \approx \) 1.2 kg/decade in older men. This rate of decline is less than that reported previously in older men (11), but greater than that in a recent report on middle-aged men (13). Baumgartner et al (11) reported a 1.8-kg/decade decline in appendicular skeletal muscle mass for men between 60 and 95 y of age. In contrast, Gallagher et al (13) showed a decline of 0.8 kg/decade for men between 20 and 90 y of age. The greater loss of appendicular skeletal muscle mass in the current study, compared with that found by Gallagher et al, may be due to the older age of our cohort.
Appendicular skeletal muscle mass loss in individuals after the age of 50 y may be accelerated because of reductions in muscle fiber area (19), in myosin heavy-chain synthesis (20), and in strength (21). A methodologic limitation of previous studies examining muscle mass loss with aging was the failure to control for body size because the number of elderly individuals participating in moderate to high-intensity activities, which may attenuate muscle mass loss, is minimal. Our results support the cross-sectional data of Klitgaard et al (25), who showed that regular strength training and anabolic activities, such as strength training, have been shown to improve muscle strength (24) and protein synthesis (5), although these findings are controversial (7). Most subjects in our study were not regularly participating in strength training but were doing general aerobic activities (eg, walking and gardening), which are less anabolic in nature. Regular physical activity, as measured in the current study, may be useful in offsetting age-related comorbidities (eg, type 2 diabetes mellitus and obesity) because of its energy-expending properties; however, its effects on maintenance of appendicular skeletal muscle mass are probably minimal. Our results support the cross-sectional data of Klitgaard et al (25), who showed that regular strength training and not regular swimming or running attenuated the decline in muscle fiber area and strength in a small group of elderly men. We suggest that greater efforts should be focused on developing exercise prescriptions to accrue maximal anabolic and cardiovascular benefits in the elderly. This becomes especially important because the number of elderly individuals participating in higher-intensity activities, which may attenuate muscle mass loss, decreases with increasing age (26).

In addition to physical inactivity, low dietary protein intake may augment the loss of skeletal muscle mass with aging. The current recommended dietary allowance (RDA; 27) for protein intake is 0.80 g/kg body mass; however, some data suggest that the dietary protein requirements for the elderly may be as high as 1.25 g/kg body mass·d (28), although these results are controversial and need further scientific examination. Nonetheless, protein intake in the present cohort of older men (1.20 g · kg· d) was higher than the current RDA. We found an inverse relation between age and appendicular skeletal muscle mass after controlling for standing height and grams of dietary protein per kilogram body mass. That is, variations in protein intake had virtually no effect in modifying the age-related decline in appendicular skeletal muscle mass. Even when the protein intake data were expressed as a percentage of daily energy intake, aging was still associated with loss of appendicular muscle mass (r = −0.38; slope: −0.103 ± 0.040 kg/y; P = 0.01). In contrast, more recent data suggest that exogenous amino acids stimulate mixed muscle protein synthesis in elderly appendicular muscle mass (kg)

<table>
<thead>
<tr>
<th>Muscle mass and covariates</th>
<th>Slope ± SE</th>
<th>Age (kg/y)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appendicular muscle mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>−0.119 ± 0.039</td>
<td>−0.43</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Height</td>
<td>−0.102 ± 0.038</td>
<td>−0.38</td>
<td>0.01</td>
</tr>
<tr>
<td>Height and physical activity</td>
<td>−0.120 ± 0.052</td>
<td>−0.34</td>
<td>0.03</td>
</tr>
<tr>
<td>Height and protein intake</td>
<td>−0.127 ± 0.045</td>
<td>−0.41</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Arm muscle mass (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>−0.052 ± 0.013</td>
<td>−0.52</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Height</td>
<td>−0.051 ± 0.014</td>
<td>−0.50</td>
<td>0.01</td>
</tr>
<tr>
<td>Height and physical activity</td>
<td>−0.035 ± 0.018</td>
<td>−0.28</td>
<td>0.06</td>
</tr>
<tr>
<td>Height and protein intake</td>
<td>−0.051 ± 0.016</td>
<td>−0.44</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Leg muscle mass (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>−0.068 ± 0.030</td>
<td>−0.33</td>
<td>0.03</td>
</tr>
<tr>
<td>Height</td>
<td>−0.051 ± 0.029</td>
<td>−0.27</td>
<td>0.08</td>
</tr>
<tr>
<td>Height and physical activity</td>
<td>−0.085 ± 0.038</td>
<td>−0.33</td>
<td>0.03</td>
</tr>
<tr>
<td>Height and protein intake</td>
<td>−0.075 ± 0.033</td>
<td>−0.34</td>
<td>0.03</td>
</tr>
</tbody>
</table>
men (4), whereas others report no effect of higher-protein meals on myofibrillar protein synthesis (6). When men in the present study with a protein intake <0.80 g·kg\(^{-1}·d^{-1}\) were excluded, there was no relation between appendicular skeletal muscle mass and protein intake adjusted for body size and physical activity, suggesting that protein intake above the RDA is not linked to preservation of muscle mass in older men. Although our statistical associations cannot prove causality, these results provide preliminary data suggesting that higher protein intake may not completely offset the age-related decline in appendicular skeletal muscle mass. These data need to be confirmed in a longitudinal study of both older men and women.

We also examined the association between serum albumin concentration and total appendicular skeletal muscle mass. Serum albumin is synthesized in the liver and is a marker of nutritional status. Data suggest that low serum albumin is associated with low appendicular skeletal muscle mass in elderly women and men (11). Although there is no obvious direct mechanism to link low serum albumin with reduced muscle mass, Baumgartner et al (11) hypothesized that reduced protein metabolism with aging may occur concurrently in the liver and muscle causing similar decrements in both serum albumin and muscle mass. Our data show that serum albumin was not associated with appendicular skeletal muscle mass in older men after physical activity and protein intake were controlled for (\(r = 0.20, P = 0.20\)). The lack of association suggests that low serum albumin may not be a predictor of reduced muscle mass; however, future studies are needed with more subjects, other measures of muscle protein metabolism, and a larger range of serum albumin concentrations to elucidate the direct mechanism linking low serum albumin to age-related muscle mass loss.

**Study limitations**

The current cross-sectional data suggest that maintaining physical activity levels and protein intake may not attenuate the age-related loss of appendicular skeletal muscle mass in older men. Nevertheless, longitudinal data are needed to confirm this rate of muscle mass loss. Second, a uniaxial accelerometer may provide a reasonable proxy measure of physical activity (29, 30), although the inability of this device to measure physical activity in all planes of motion warrants the use of other more accurate measures of activity, such as doubly labeled water, in future studies. Third, quantification of appendicular skeletal muscle mass from DXA is a reasonable proxy measure of muscle mass (10); however, this method is not a true gold standard and assumes that the contribution of skin, underlying connective tissue, and interstitial fat to total appendicular skeletal muscle mass is negligible. Finally, the health of our older population limits the generalizability of these results to other more frail populations.

**Summary**

Our cross-sectional data suggest that healthy, older white men may lose \(\pm 1.2\) kg appendicular skeletal muscle mass per decade. Maintaining physical activity and dietary protein intake may not completely attenuate the loss of appendicular skeletal muscle mass in older men. Prospective studies are needed to confirm these results and to determine whether different modes of physical activity that have an anabolic effect (e.g., strength training) can attenuate the age-related loss of appendicular skeletal muscle mass in the elderly.

**REFERENCES**


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