The Effect of Age-related Differences in Body Size and Composition on Cardiovascular Determinants of VO₂max

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Background. A reduction in maximal stroke volume (SVₘₐₓ) and total blood volume (TBV) has been hypothesized to contribute to the decline in maximal oxygen uptake (VO₂max) with healthy aging. However, these variables have rarely been collected simultaneously in a broad age range to support or refute this hypothesis. It is also unclear to what extent scaling size-related cardiovascular determinants of VO₂max affects the interpretation of age-related differences.

Methods. A retrospective analysis of VO₂max, maximal cardiac output (Qₐₘₚₙ), TBV, and body composition including fat-free mass (FFM) in 95 (51% M) healthy adults ranging from 19–86 years.

Results. Absolute and indexed VO₂max, Qₐₘₚₙ, and maximal heart rate decreased in both sexes with age (p ≤ .031). SVₘₐₓ declined with age when scaled to total body mass or body surface area (p ≤ .047) but not when expressed in absolute levels (p = .120) or relative to FFM (p = .464). Absolute and indexed TBVs (mL/kg; mL/m²) were not significantly affected by age but increased with age in both sexes when scaled to FFM (p ≤ .013). A lower arteriovenous oxygen difference (aVdO₂) contributed to the reduction in VO₂max with age in treadmill exercisers (p = .004) but not in the entire cohort (p = .128).

Conclusion. These results suggest (a) a reduction in absolute SVₘₐₓ and TBV do not contribute substantially to the age-related reduction in VO₂max, which instead results from a smaller Qₐₘₚₙ due to a lower maximal heart rate, and (b) body composition scaling methods should be used to accurately describe the effect of aging on physical function and cardiovascular variables.

Key Words: Aging—Maximal exercise capacity—Stroke volume—Total blood volume—Body composition.

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MAXIMAL exercise capacity declines with healthy aging as evident by a reduction in maximal oxygen uptake (VO₂max) (1–6). Cross-sectional studies (3,7) have shown that a reduction in maximal cardiac output (Qₐₘₚₙ) significantly contributes to the age-related decline in VO₂max in healthy individuals. Although it is clearly established that maximal heart rate declines with age (3,7,8), previous findings regarding the effect of healthy aging on maximal stroke volume (SVₘₐₓ) are conflicting (3,7,9–12).

Stroke volume during exercise is significantly influenced by total blood volume (TBV) via left ventricular end-diastolic volume and thus the Frank–Starling mechanism (13,14). Small sample studies have reported that TBV is reduced in both sexes with healthy aging as a result of a smaller plasma and erythrocyte volume (15–17). Consequently, it has been hypothesized that a reduction in TBV may contribute to a smaller SVₘₐₓ with advancing age (15,17,18). However, VO₂max, TBV, and SVₘₐₓ have rarely been collected simultaneously in healthy aging individuals to support or refute this hypothesis.

Confounding the interpretation of these data is the fact that cardiovascular anatomic and physiologic variables increase with body size (19,20). Accordingly, SVₘₐₓ and TBV are frequently scaled to total body mass or body surface area (BSA) to compare populations of different body sizes (7,15–17,20,21). However, given that oxygen uptake during exercise is driven by metabolically active tissue (22), scaling cardiovascular determinants of oxygen transport to metabolically active tissue (fat-free mass, FFM) may provide a more precise assessment of cardiovascular capacity in populations of different body sizes and composition.

Therefore, the purpose of this study was twofold: (a) to determine whether the age-related decline in VO₂max is associated with a reduction in SVₘₐₓ and TBV and (b) to examine to what extent scaling size-related cardiovascular determinants of VO₂max affects the interpretation of
age-related differences. Given the age-associated increase in total body mass and adiposity, we hypothesized that $SV_{\text{max}}$ and TBV would be reduced with age when scaled to total body mass and BSA but preserved when scaled to FFM.

**METHODS**

Subjects were recruited within the Dallas-Fort Worth metroplex. All subjects were nonsmokers, were not taking any cardiovascular medications, had a 24-hour ambulatory blood pressure less than 140/90 mmHg, body mass index $\leq 30$ kg/m$^2$, and a normal ECG and exercise stress echocardiogram. No subjects were performing exercise for more than 90 min/wk, with the majority ($n = 92$) performing less than 20 min/d, less than 3x/wk. All subjects signed an informed consent form approved by the Institutional Review Boards of the University of Texas Southwestern Medical Center at Dallas and Presbyterian Hospital.

**Maximal Oxygen Uptake and $Q_{C_{\text{max}}}$**

$VO_{2\text{max}}$ was determined with an incremental cycling ($n = 24$) or treadmill ($n = 71$) exercise test to maximal exertion using the Douglas bag method as previously described elsewhere (23). Gas fractions were analyzed using mass spectrometry and ventilatory volumes by a Tissot spirometer. $VO_{2}$ and $Q_c$ was assessed during seated or standing rest, one level of steady-state submaximal exercise corresponding to approximately $\approx 60\%$ of maximal heart rate, and during maximal exercise. $VO_{2\text{max}}$ was defined as the highest oxygen uptake measured from at least a 30s Douglas bag. $Q_c$ was measured by the $C\text{H}_2$ rebreathing method, which has been previously validated in this laboratory (24). Heart rate was measured via a 12-lead ECG, and $SV_{\text{max}}$ was calculated as $Q_{C_{\text{max}}}/$maximal heart rate. Arteriovenous oxygen difference ($a-vO_2\text{diff}$) was calculated by rearranging the Fick equation ($a-vO_2\text{diff} = VO_2/Q_c$). Typical error of measurement expressed as a coefficient of variation (%) for test–retest reproducibility (25) for $VO_2\text{max}$, $Q_{C_{\text{max}}}$, maximal heart rate, and $SV_{\text{max}}$ for 56 subjects in this cohort were 4.8%, 7.6%, 2.7%, and 7.8%, respectively.

**Body Composition**

Body density and composition were determined by underwater weighing with correction for residual lung volume. Subjects were first weighed for total body mass in air. In the water, subjects were instructed to exhale normally to expel air from their lungs (to functional residual capacity) and then seat themselves in tucked position in an aluminum cage that was suspended in a tank of water (temperature $= 30 \pm 1^\circ$C). Residual lung volume was measured by the nitrogen washout method with a mass spectrometer. Each subject performed at least three adequate measurements defined as a definite plateau in underwater weight, and the mean value was calculated. FFM and fat content were calculated accordingly.

**Total Blood Volume**

On a separate day, TBV was measured using a carbon monoxide rebreathing method modified from that described by Burge and Skinner (26) and has been described in detail elsewhere (27). Typical error of measurement expressed as a coefficient of variation (%) for test–retest reproducibility for hemoglobin mass, which is used to determine the distribution of carbon monoxide, is less than 4% for repeated measures in our laboratory (27).

**Physical Activity Levels**

Daily energy expenditure (kcal) was assessed by a physical activity monitor (Actical, Phillips, USA; RT3; Stayhealthy, USA) worn from 1–7 days.

**Data Analysis**

All statistical analysis was performed using SigmaStat version 11.0 (Systat Software, USA).

T-tests were used to determine sex differences in subject characteristics. Univariate correlations (Pearson’s) were used to determine the relationship between cardiovascular, metabolic and body size, and composition variables. Body size and composition measures, $VO_2\text{max}$, TBV and its compartments (plasma volume [$PV$], erythrocyte volume [$ECV$]), $Q_{C_{\text{max}}}$, and $SV_{\text{max}}$ were regressed on age by least squares linear regression analysis in all subjects and within each sex. Twenty-four subjects performed a maximal cycle test rather than treadmill test. As $VO_2\text{max}$ and $SV_{\text{max}}$ are $5\%–10\%$ lower during maximal cycle compared with treadmill exercise (28), linear regression analysis was performed in 71 subjects (63% F, 55 ± 13 years) who performed treadmill exercise testing only. The value of $p < .05$ was considered statistically significant. Data are presented as mean ± standard deviation (SD) in Tables 1–3.

**RESULTS**

**Subject Characteristics**

The men were younger, heavier, taller, leaner, and had more FFM compared with the women ($p \leq .047$). Absolute and indexed levels (kcal/kg/d) of daily energy expenditure was greater in men versus women ($p \leq .060$), but this result was reversed when scaled to FFM ($p = .002$; Table 1). At rest, absolute and indexed $VO_2$ (mL/kg/min) were greater in men compared with women ($p \leq .008$), but this difference was eliminated when scaled to FFM (mL/kgFFM/min; $p = .672$). Likewise, resting stroke volume was smaller in the women ($p \leq .047$), unless FFM was used as a scaling index ($p = .775$). Heart rate and $a-vO_2\text{diff}$ were not different between the sexes ($p = .544$ and $p = .352$, respectively; Table 2).

$VO_{2\text{max}}$ ranged from 948 to 2,145 mL/min in the women and 1,294 to 3,602 mL/min in the men, with the mean value being greater in the men compared with the women.
Likewise, VO₂max relative to total body mass or FFM was greater in the men compared with the women (p < .001; Table 3). Irrespective of scaling approach, SVₘₐₓ was also greater in the men compared with the women (p ≤ .051). Absolute TBV was larger in the men compared with the women (p < .001; Table 3). Although TBV remained larger in the men when scaled to total body mass and BSA (p < .001), sex differences were eliminated when scaled to FFM (p = .556). There was a positive and significant relationship between metabolic and cardiovascular variables and total body mass, BSA and FFM, but these relationships were negative for body fat percentage (Table 4).

**Table 1. Participants Characteristics**

<table>
<thead>
<tr>
<th>Subjects, n</th>
<th>Men</th>
<th>Women</th>
<th>All Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>46 ± 17</td>
<td>53 ± 15*</td>
<td>50 ± 16</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176 ± 8</td>
<td>164 ± 6*</td>
<td>170 ± 10</td>
</tr>
<tr>
<td>Total body mass, kg</td>
<td>79 ± 11</td>
<td>65 ± 9*</td>
<td>72 ± 12</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.96 ± 0.17</td>
<td>1.72 ± 0.14*</td>
<td>1.85 ± 0.20</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25 ± 3</td>
<td>24 ± 3*</td>
<td>25 ± 3</td>
</tr>
<tr>
<td>Fat free mass, kg</td>
<td>58 ± 7</td>
<td>42 ± 5*</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Fat percent, %</td>
<td>27 ± 7</td>
<td>36 ± 6*</td>
<td>31 ± 8</td>
</tr>
<tr>
<td>Daily energy expenditure, kcal/d</td>
<td>2479 ± 393</td>
<td>1964 ± 372*</td>
<td>2234 ± 454</td>
</tr>
<tr>
<td>Daily energy expenditure, kcal/kg/d</td>
<td>32 ± 6</td>
<td>24 ± 4*</td>
<td>28 ± 6</td>
</tr>
<tr>
<td>Daily energy expenditure, kcal/kgFFM/d</td>
<td>43 ± 6</td>
<td>37 ± 5*</td>
<td>40 ± 6</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>84 ± 11</td>
<td>89 ± 16</td>
<td>87 ± 14</td>
</tr>
<tr>
<td>a-V0₂diff, mL/100 mL</td>
<td>7 ± 1</td>
<td>6 ± 1</td>
<td>6 ± 1</td>
</tr>
</tbody>
</table>

**Table 2. Resting Hemodynamic and Metabolic Parameters**

<table>
<thead>
<tr>
<th>VO₂, mL/min</th>
<th>SV, mL/kg/m²</th>
<th>TBV, mL/kgFFM</th>
<th>a-V0₂diff, mL/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>Women</td>
<td>All Subjects</td>
<td></td>
</tr>
<tr>
<td>2479 ± 458</td>
<td>1539 ± 280*</td>
<td>2014 ± 606</td>
<td></td>
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<tr>
<td>1294–3602</td>
<td>948–2145</td>
<td></td>
<td></td>
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<tr>
<td>32 ± 6</td>
<td>24 ± 4*</td>
<td>28 ± 6</td>
<td></td>
</tr>
<tr>
<td>17–45</td>
<td>14–32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>43 ± 6</td>
<td>37 ± 5*</td>
<td>40 ± 6</td>
<td></td>
</tr>
<tr>
<td>27–60</td>
<td>23–46</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 3. Maximal Hemodynamic and Metabolic Parameters and TBV**

<table>
<thead>
<tr>
<th>VO₂ₘₐₓ, mL/min</th>
<th>SVₘₐₓ, mL/kg</th>
<th>TBV, mL/kg</th>
<th>Maximum a-V0₂.diff, mL/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>Women</td>
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<tr>
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<td>23–46</td>
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**Table 4. Univariate Correlations Between Metabolic, Hemodynamic and Circulating Factors and Body Size and Composition Variables**

**Pooled Data for Men and Women (n = 95)**

<table>
<thead>
<tr>
<th>VO₂ₘₐₓ, L/min</th>
<th>SVₘₐₓ, mL/kg</th>
<th>TBV, mL/kg</th>
<th>Maximum a-V0₂.diff, mL/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total body mass, kg</td>
<td>0.69*</td>
<td>0.63*</td>
<td>0.79*</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>0.74*</td>
<td>0.69*</td>
<td>0.84*</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>0.88*</td>
<td>0.80*</td>
<td>0.86*</td>
</tr>
<tr>
<td>Fat mass, %</td>
<td>−0.54*</td>
<td>−0.49*</td>
<td>−0.37*</td>
</tr>
</tbody>
</table>

**Pooled Data for Treadmill Exercisers (n = 71)**

<table>
<thead>
<tr>
<th>VO₂ₘₐₓ, L/min</th>
<th>SVₘₐₓ, mL/kg</th>
<th>TBV, mL/kg</th>
<th>Maximum a-V0₂.diff, mL/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total body mass, kg</td>
<td>0.76*</td>
<td>0.73*</td>
<td>0.80*</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>0.79*</td>
<td>0.77*</td>
<td>0.84*</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>0.90*</td>
<td>0.80*</td>
<td>0.87*</td>
</tr>
<tr>
<td>Fat mass, %</td>
<td>−0.49*</td>
<td>−0.35*</td>
<td>−0.38*</td>
</tr>
</tbody>
</table>

**Notes.** VO₂ = oxygen uptake; SV = stroke volume; TBV = total blood volume; FFM = free-fat mass.

Values are expressed as mean ± standard deviation. Numbers in parentheses represents range of values.

*p < .05 versus men.
significantly affected by aging (p = .659 and p = .063, respectively); however, fat mass whether expressed in total mass or percentage increased in both sexes with aging (p ≤ .028). For the entire cohort, FFM declined with aging (p = .027); however, this effect was not significant when men and women were analyzed separately (p = .545 and p = .328, respectively).

Resting and Submaximal Steady-State Exercise VO₂ and Hemodynamics With Aging
Irrespective of unit, resting VO₂ declined with advancing age (p ≤ .039). Resting stroke volume scaled to total body mass and size (mL/kg; mL/m²) appeared to decline with aging (p ≤ .006) but not when scaled relative to FFM (p = .334). Heart rate declined (p = .001), whereas a-vO₂diff was not affected by age (p = .259).

Heart rate (% of maximum) during steady-state exercise was not affected by age (p = .454), suggesting a similar relative level of exercise intensity across the ages. Absolute Qc was reduced with age, (p = .008); however, stroke volume was unaffected irrespective of scaling approach (p ≥ .090; Figure 1).

VO₂max and Maximal Exercise Hemodynamics With Aging
Absolute and indexed VO₂max declined with age (p ≤ .020; Figure 2). Likewise, absolute Qc,max declined with age (p < .001), which was independent of body size or composition, as the age-related reduction in Qc,max was still evident relative to total body mass, BSA, and FFM (p ≤ .002; Figure 2). Although the reduction in Qc,max was robust in men regardless of scaling method (p ≤ .019), it only reached statistical significance in women when relative to BSA (p = .015) and total body mass (p = .004), underscoring the influence of fat mass in this calculation (Figure 2). Maximal heart rate declined in both sexes (7 beats/decade in women; 5 beats/decade in men; p < .001). SVc,max declined with age when scaled to total body mass or BSA (p ≤ .047) but not when expressed in absolute levels (p = .120) or relative to FFM (p = .464; Figure 2). Maximal a-vO₂diff was not significantly affected by age (p = .128).

Central Circulatory Factors With Aging
Absolute TBV increased with age in the men (p = .025) but not in the women (p = .452; Figure 3). When scaled to total body mass, the slope of TBV was essentially flat but positive when expressed relative to FFM in both sexes (p ≤ .013; Figure 3), further confirming that TBV is not reduced with age. There was a positive slope with age for PV and ECV in both sexes when indexed to FFM (p < .001; Figure 3).

Treadmill Exercisers Only
Resting and steady-state exercise, VO₂, Qc, and stroke volume were not affected by age. Qc,max declined with age when indexed to total body mass or BSA (p ≤ .003) but not when expressed in absolute levels (p = .272) or indexed to FFM (p = .340), further demonstrating the significant influence of scaling choice on age-related differences. Although SVc,max indexed to total body mass no longer declined with age (p = .396), SVc,max indexed to FFM increased with age (p = .036). Maximal a-vO₂diff declined with age (p = .004). In treadmill exercisers, there was a positive and significant association between FFM and a-vO₂diff (r = 0.47, p < .001), but this relationship was negative for fat mass (r = −0.28, p = .028)

Discussion
This current study demonstrated that despite a decline in absolute and relative VO₂max, absolute levels of SVc,max and TBV are well preserved with healthy aging. Consistent with our hypothesis, the decline in cardiovascular determinants of VO₂max was either attenuated (or surprisingly reversed) when FFM was used as the scaling variable compared with standard body size scaling variables. Therefore, these current results (a) do not support the hypothesis that a reduction in SVc,max and TBV contributes to the age-related decline in VO₂max and (b) underscores the importance of determining the most appropriate scaling variables when investigating changes in cardiovascular determinants of VO₂max in populations of varying body sizes and composition.

The influence of age-related differences in body composition on cardiovascular determinants of VO₂max with aging has been reported previously (18). However, the findings of this previous study are limited by (a) all variables (VO₂max, stroke volume, TBV, FFM) were only collected in 27 subjects (26% of the entire cohort), (b) supine stroke volume and not maximal SV was collected, (c) TBV was only presented relative to total body mass, and (d) the influence of scaling index on age-related differences was not investigated. Therefore, our current study extends previous findings, which are important to delineate the limitations to exercise performance in healthy aging individuals, which in turn may allow for more effective prescription of lifestyle strategies including exercise training (29–31) to increase functional capacity in a globally aging population.

Aging and Cardiovascular Capacity
VO₂max declines 5%–20% per decade in healthy individuals aged 20–65, with greater rates of decline reported in those 70 years and over (1,2,5,6). VO₂max declines at a greater rate with age when scaled to total body mass or BSA rather than FFM (1,2,6), emphasizing the importance of scaling in interpreting these changes. Although an excess of fatness and body mass do not necessarily imply a reduced physiological ability to maximally distribute and utilize oxygen at the tissue level, it may instead limit submaximal and maximal exercise capacity due to a reduced ability to move excess mass through space during exercise (walking, running).
Nevertheless, VO2_max scaled to whole body or appendicular FFM declines with age (1–3), which could reflect either a reduced central component to oxygen delivery and/or a reduced utilization by metabolically active tissue. From a central component, it is clear that maximal heart rate declines (3,7,8); however, SV_max has been reported to decrease (3,9,12), increase (10,11,32), or be unaffected (7,33,34) with age in healthy individuals. Methodological differences including study design (longitudinal versus cross-sectional), sample size, measurement techniques, and exercise mode are likely to contribute to the discrepancies in previous findings. Although rarely investigated, two pieces of evidence suggest that differences in findings regarding SV_max may be heavily influenced by the approach taken to...
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scale to body size measures. First, SV\textsubscript{max} scaled to total body mass was smaller in healthy individuals aged 60 years and older compared with sex-matched young adults (<35 years); however, this effect was no longer statistically significant when SV\textsubscript{max} was scaled to FFM (3). Second, scaling maximal cardiac power (QC\textsubscript{max} × mean arterial pressure) to metabolically active tissue was more precise at eliminating sex-related differences compared with BSA or total body mass in a group of subject ranging in age from 20 to 70 years (35). In this current cohort, the relationship between SV\textsubscript{max} and age was unaffected or negative when scaled to total body mass but positive when scaled to FFM. Such findings highlight a potential confounding effect that may occur when standard scaling variables are used to examine cardiovascular capacity in aging individuals of varying body sizes and composition.

Our finding that TBV and its compartments are preserved (or increased) with age conflicts with previous findings (15–18). However, these previous findings are likely confounded by small sample size (15–17), restrictive age range (15–17), or failure to strictly control for physical activity levels (17). The findings of this current study suggest that irrespective of sex, TBV is reasonably well preserved with aging, which is consistent with previous findings (36,37). Moreover, we found that TBV compartments increased with age in both sexes when scaled to FFM. The only other study that has investigated age-related differences in TBV scaled to FFM reported that ECV and PV were reduced in seven older (mean age 66.1 ± 1.8) men versus total body mass, BSA, and physical activity matched younger (24.7 ± 1.2 years) men (15). The reasons for such divergent findings are unclear but may reflect a larger sample size that includes a broader age range in this current study. Nevertheless, one of the key findings of this current study is that irrespective of scaling approach, TBV is not significantly reduced with healthy aging.

Figure 2. VO\textsubscript{2}max and QC\textsubscript{max} declined with age. For the entire cohort, SV\textsubscript{max} indexed to total body mass declined with age but not when scaled to body surface area or fat-free mass. Men (filled triangles) and women (filled circles). Overall regression line for men (solid line), women (dotted line), and all subjects (solid bold line). *p < .05 regression line against age. The linear regression equation coefficient (r) and p value are for n = 95.
Consistent with previous findings (3), this current study demonstrates an age-related reduction in a-\(\Delta V O_2\) during maximal treadmill exercise. A lower a-\(\Delta V O_2\) with senescence may be multifactorial including a reduced number of mitochondria and associated enzymes, altered Q, distribution, and increased vascular resistance to peripheral blood flow (38–42). However, a large portion of this reduction in a-\(\Delta V O_2\) may be related to physical deconditioning with aging, rather to the aging process itself, as aerobic exercise training increases a-\(\Delta V O_2\) in adults over the age of 60 due to an increased number of mitochondria and enzymes activity (43), increased vasodilator response, and enhanced blood flow redistribution to exercising muscle mass (39).

Scaling Cardiovascular Variables in Populations of Different Body Sizes and Composition

To accurately assess cardiovascular function and capacity in populations of different body sizes and composition, the most appropriate scaling model and scaling variables need to be identified based on physiological relevance. Previous reports (35,44,45) have commented that (a) based on physiologic and mathematical basis, an allometric scaling model is superior compared with the more conventionally used ratiometric model when examining cardiovascular physiologic and anatomic measures in populations of different body size and composition, and (b) based on blood flow distribution and metabolic demand during exercise, FFM would appear to represent a superior variable compared with the conventional scaling body size and mass variables.
In early and late middle-aged adults (30–55 years), allometric modeling was more appropriate at distinguishing differences in arterial structure and function in obese versus nonobese individuals after differences in body size are considered (46), whereas Chantler et al. (35) showed that allometric scaling of maximal cardiac power to metabolically active tissue dramatically reduced gender-related differences compared with conventionally body size variables. The current findings underscore that FFM seems to be the most appropriate scaling index for cardiovascular variables, particularly in women, given the changes in total body mass and size, and more importantly, body composition with aging.

Methodological Considerations

There are several limitations to this current study. It is acknowledged that age-related differences in VO2max may be in fact be nonlinear, with greater rates of decline observed in those older than 70 years compared with younger ages (2). As a result, the statistical analysis performed in this present study may not truly reflect the accelerated rate of decline in VO2max in those 70 years and above. Moreover, it is also acknowledged that there were several results that were close to conventional levels of statistical significance, which likely reflects our sample size and retrospective methodological design. Despite its preferred usage, FFM does have some limitations. FFM is composed of highly metabolically active tissues during exercise such as skeletal and cardiac muscles but also less metabolically active tissues and organs such as the liver and the spleen. Moreover, the usage of FFM neglects adipose tissue blood flow, which increases in active limbs during exercise (47) but still remains much smaller compared with that of skeletal muscle (48). Finally, our subjects were nonobese, normotensive, and were screened for occult cardiovascular disease; therefore, it is unclear whether these current results are applicable to the larger population or to a physically active population.

Conclusions

In summary, this current study demonstrated that while absolute and relative VO2max declines with age, SVmax and central circulatory factors are well preserved with aging in both sexes. A lack of an age-related decline in size-related cardiovascular determinants of VO2max is evident when metabolically active tissue is used as the scaling variable. Collectively, the current findings do not support a hypothesis that a smaller SVmax and TBV contribute to the age-related decline in VO2max. Instead, a reduction in maximal heart rate seems to be more influential in reducing VO2max with advancing age.

Funding

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Conflict of Interest

There are no author conflicts to disclose.

References