No Sustained Effect of Aerobic or Resistance Training on Insulin Sensitivity in Nonobese, Healthy Older Women

Eric D.B. Goulet, Michel O. Mélançon, Isabelle J. Dionne, and Mylène Aubertin Leheudre

It is unclear whether long-term aerobic (AT) or resistance (RT) training can improve insulin sensitivity (IS) beyond the residual effect of the last training bout in older women (54–78 years). Therefore, a group of nonobese, healthy older women underwent 6 months of AT \( (n = 8) \) or RT \( (n = 10) \), and the authors measured IS 4 days after the last training bouts using the hyperinsulinemic-euglycemic clamp technique. Women trained 3 days/week. AT consisted of 25- to 60-min sessions of walking/jogging at 60–95% of maximal heart rate. RT consisted of three sets of nine exercises repeated 10 times at 80% of 1 repetition maximum. AT decreased fat mass, whereas both AT and RT increased fat-free mass. Neither training program, however, improved absolute or relative rates of glucose disposal. The authors therefore concluded that nonobese, healthy older women should perform AT or RT on a daily basis in order to improve IS and maintain the improvement.

Key Words: insulin resistance, elderly, body composition, physiological adaptations

Insulin sensitivity (IS) has been shown to decrease with aging (Ryan, 2000). This might result from a complex set of factors among which physical inactivity (Ryan), oxidative stress (Caranton et al., 1998), and changes in body composition (Ryan), muscle morphology (Coggan et al., 1992), and cellular (Kirwan et al., 2000) and endothelial function (Steinberg et al., 1996) might be involved. A decrease in IS can increase the risk for Type II diabetes, atherosclerosis, hypertension, and cardiovascular disease (Ryan), all of which can substantially decrease the functional capacity and quality of life of older adults. Thus, lifestyle interventions aimed at offsetting the decline in IS with aging have potential public health interest to counteract associated comorbidities.

After physical training, the restoration of glycogen reserves takes approximately 24 hr when a sufficient quantity of carbohydrate is consumed (Ivy, 1991) and is associated with an increase in IS (Bogardus, Finegood, & Ader, 1983; Fell, 1984). The authors are with the Research Centre on Aging, Geriatric Institute of the University of Sherbrooke, Sherbrooke, Québec, Canada, J1H 4C4.
Terblanche, Ivy, Young, & Holloszy, 1982) that is proportional to the magnitude of exercise-induced glycogen depletion (Cartee et al., 1989). It is thus not surprising that aerobic (AT; Clevenger, Parker Jones, Tanaka, Seals, & Desouza, 2002; Cox, Cortright, Dohm, & Houmard, 1999; Hersey et al., 1994; Kirwan, Kohrt, Wojta, Bourey, & Holloszy, 1993; Seals, Hagberg, Allen, et al., 1984; Seals, Hagberg, Hurley, Ehsani, & Holloszy, 1984) and resistance training (RT; Miller et al., 1994; Ryan et al., 2001; Ryan, Pratley, Goldberg, & Elahi, 1996) have been shown to improve IS of older individuals within this specific window of time after training.

Albeit relevant, results of the cited studies do not provide an answer to the following important, pragmatic question: Does the practice of AT or RT at a frequency of 3 times per week, which is representative of the number of times most of older adults train or would be willing to train on a weekly basis, maintain the improvement in IS beyond the first 24 hr after the last training bout? Studies conducted in rats suggest that the improvement in IS after exercise remains until glycogen reserves are completely restored and then declines (Cartee et al., 1989; Garcia-Roves et al., 2003; Kawanaka et al., 1999). Therefore, the capacity of AT or RT to improve IS beyond 24 hr after the last training session would suggest that the chronic physiological adaptations induced by long-term training represent additional regulators of IS that prevail over the presence of large quantities of glycogen inside the muscles.

We think that it is particularly important to determine whether AT or RT can improve IS for more than 24 hr after an exercise session, because it could have significant implications for exercise prescription in older adults. For example, the failure of a given exercise program to increase IS for more than 24 hr would be an indication that individuals would have to train on a daily basis to maintain the improvement in IS. On the other hand, if exercise could improve IS for more than 24 hr, individuals could train at a lower frequency (i.e., 3 days per week) and still maintain the improvement in IS.

In older men, it has been shown that the physiological adaptations induced by 4 months of RT improved IS for up to 7 days after the last training bout (Zachwieja, Toffolo, Cobelli, Bier, & Yarasheski, 1996). The overall results of studies that focused on the effect of AT on IS beyond 24 hr are controversial, however, among older men. Kahn et al. (1990) showed that 6 months of running and cycling improved IS when measured 60 hr postexercise, whereas Short et al. (2003) found no effect of 4 months of cycling when IS was measured 4 days postexercise. In older women, it has been demonstrated that 4–6 months of AT (Short et al.) or RT (Dionne, Brochu, Mélançon, Ades, & Poehlman, 2004) did not improve IS when measured 4 days posttraining. Finally, Tonino (1989) showed that AT combined with calisthenics enhanced IS 7 days after the last training bout in a group composed of both older men (n = 6) and women (n = 5).

Obviously, the paucity of research data, coupled with lack of agreement among some of the observed results, makes it difficult to draw conclusions as to the effect of AT and RT on IS beyond the 24 hr after the last training session in both older men and women. Further studies are therefore necessary to better
understand the effect of both AT and RT on IS past the 24-hr mark after the last training session.

Consequently, the goal of this study was to determine, in nonobese, healthy older women, whether the physiological adaptations induced by 6 months of intense AT or RT could improve IS as long as 4 days after the last training bout. To the best of our knowledge, this is the first study to determine concurrently the effect of AT or RT on IS in older women more than 24 hr after their last training bout.

Methods

PARTICIPANTS

Eighteen older women participated in this study. Inclusion criteria were (a) postmenopausal (as indicated by the absence of menstruation >1 year and levels of follicle-stimulating hormone and luteinizing hormone >40 and >30 U/L, respectively), (b) BMI <30 kg/m$^2$, (c) weight stable (± 2 kg) for the 6 months before the study, (d) no regular participation (<2 times/week) in physical activity for the 6 months before the beginning of the study, and (e) not taking hormone-replacement therapy. Exclusion criteria were (a) glucose intolerance (as indicated by a 2-hr plasma glucose ≥140 and ≤200 mg/dl) or diabetes (plasma glucose ≥200 mg/dl) determined with a 2-hr oral glucose-tolerance test (OGTT), (b) orthopedic limitations, (c) hypertension (>140/90 mmHg), (d) use of medication that could affect metabolism (e.g., β-blockers, hypoglycemic and hypolipidemic agents, oral steroids, etc.), (e) smoking (at the time of the study none of the participants used tobacco), and (f) alcohol consumption >1,500 mg/day). The nature and goals of the project were thoroughly explained to participants, after which they provided written informed consent. The study was approved by the Ethics Committee for Human Research of the University of Vermont. Some of the RT data reported in the present study are a subset of data previously published elsewhere (Dionne et al., 2004).

PRELIMINARY MEASUREMENTS

On their first visit at the laboratory, participants completed a health-history questionnaire, underwent a complete physical examination, had a blood sample drawn (to determine levels of follicle-stimulating hormone and luteinizing hormone), underwent a 2-hr 75-g OGTT, and completed a maximal oxygen-consumption ($VO_{2\text{max}}$) test on a treadmill. Two weeks later, they returned to the laboratory for an overnight stay. Before this visit, participants were asked to follow a standardized diet for 3 days. On the evening of this visit, participants underwent body-composition measurements, and the following morning, after 12 hr of fasting, IS was measured. After metabolic testing, participants were randomly assigned into either the AT or the RT exercise group. An identical posttesting sequence followed the 6 months of training, 4 days after the last training bout of the training programs.
ORAL GLUCOSE-TOLERANCE TEST

The 2-hr 75-g OGTT was performed according to the guidelines of the American Diabetes Association (1997). Participants followed a standardized diet for the 3 days before the test. Insulin and glucose were measured at 0, 60, 90, and 120 min.

STANDARDIZED DIET

Three days before the OGTT and the hyperinsulinemic-euglycemic clamp (pre- and posttesting), participants were instructed to follow a standardized diet comprising ≈58% carbohydrates, 30% fats, and 12% proteins. Meals were prepared and provided to participants by the metabolic kitchen of the general clinical research center.

TRAINING PROGRAMS

The training programs consisted of 6 months of either AT or RT. Participants trained on 3 nonconsecutive days during the week. A personal trainer closely monitored each exercise session in order to measure the compliance rate of participants and ensure that women trained at the intensity and duration initially prescribed. The AT program was divided in two phases and consisted of brisk walking and/or jogging. The first phase lasted 16 weeks. During the first 4 weeks of this phase, participants performed 25 min of walking/jogging at 60% of maximal heart rate (MHR). The intensity of exercise was based on the MHR measured during the VO$_{2\text{max}}$ test. Every 4 weeks thereafter the exercise intensity was increased by 10% of MHR, and the walking/jogging time, by 5 min. Heart rate was monitored during all training sessions using a heart-rate monitor (Polar Electro, Lake Success, NY), and all women were familiarized with the use of this equipment. The second phase (12 weeks) of the AT program consisted of a mix of interval and endurance training. The interval-training sessions were performed once a week and consisted of 4 × 5 min at 95% of MHR, with rest periods of 3 min between intervals. The endurance-training sessions consisted of 45–60 min of walking/jogging at 75–85% of MHR.

The RT program consisted of nine exercises targeting all major muscle groups. These exercises, which were performed on weight-lifting machines at ≈80% of 1-RM, were leg press, chest press, leg extension, shoulder press, sit-ups, seated rows, triceps extensions, arm curls, and leg curls. Participants performed three sets of 10 repetitions with a recovery time between sets of 60–90 s. Strength progression throughout the RT regimen was closely monitored to ensure that participants trained at the expected intensity. To assess strength gains after the study period, participants performed four 1-RM tests (leg press, chest press, shoulder press, and seated rows) before and after the program. In order to not confound the data, it was ensured that the same testing procedures were used throughout the 6-month program and that the participants had the proper lifting technique before undergoing each 1-RM test.
BODY COMPOSITION

Body weight was determined using a metabolic scale showing an accuracy of ±0.1 kg (Scale-Tronix, Wheaton, IL). Dual-energy X-ray absorptiometry (DPX-L densitometer, Lunar Corp., Madison, WI) was used to determine fat-free mass (FFM) and fat mass (FM). In our laboratory, the test–retest coefficients of variation for FFM and FM were 2.0% and 1.2%, respectively. Dual-energy X-ray absorptiometry has been shown to have an accuracy allowing the detection of small changes in body composition after long-term (i.e., 1 year) AT and RT in postmenopausal women (Houtkooper, Going, Sproul, Blew, & Lohman, 2000). Computed tomography (CT; GE High Speed Advantage Scanner, General Electric Medical Systems, Milwaukee, WI) was used to measure visceral and subcutaneous adipose-tissue areas at the L4–L5 intervertebral space. The same individual analyzed all scans, and, in our laboratory, the intra-class correlation for repeated analyses of 10 scans was .99 in 10 women. Specific details regarding the methods used for these measures have been fully described elsewhere (Poehlman, Dvorak, DeNino, Brochu, & Ades, 2000).

DETERMINATION OF MAXIMAL OXYGEN CONSUMPTION
AND MAXIMAL HEART RATE

\(VO_{2max}\) was measured on a treadmill using an incremental test to exhaustion. First, participants performed a light warm-up for 3 min at a self-chosen speed. Immediately after the warm-up, the speed was increased to 5 km/hr, with no inclination on the treadmill. After the first 2 min of walking, the grade was increased by 2.5% every 2 min until participants claimed exhaustion. \(VO_{2max}\) was deemed valid when at least two of the following three criteria were met: a respiratory-exchange ratio \(\geq 1.10\), a heart rate greater than the age-predicted maximum (220 – age), and no further increase in oxygen consumption (<1 ml · kg\(^{-1}\) · min\(^{-1}\)) despite an increase in exercise intensity. \(VO_{2max}\) was defined as the average of the three highest consecutive values of oxygen consumption attained by participants during the test, and MHR was the highest heart-rate value identified.

INSULIN SENSITIVITY

IS was determined using a 2-hr hyperinsulinemic-euglycemic clamp, as previously described by Defronzo, Tobin, and Andres (1979). First, a Teflon® catheter was inserted into the antecubital vein for infusion of insulin and dextrose. Then, another Teflon catheter was inserted retrogradely into the dorsal vein of the contralateral hand for blood drawing. This hand was placed in a hot box and warmed to 50 °C for arterialization of blood. At 0 min, a continuous infusion of insulin was started at a constant rate of 40 mU · m\(^{-2}\) body surface area · min\(^{-1}\). Concomitantly, a variable infusion of 20% dextrose was started to maintain fasting glycemia at ±5%. Samples for blood glucose assessment were obtained at 0 min and then every 5 min throughout the clamp. Insulin was measured at Min –30 and –10 before the clamp and then at Min 0, 30, 60, 70, 90, 105, and 120 during the clamp. The insulin-stimulated glu-
cose disposal rate (M value) was calculated as the average rate of glucose infusion (mg/min) during the last 30 min of the clamp. The M value was calculated during the last 30 min of the clamp because by this time the rate of glucose infusion had attained a constant value. It has been previously shown in older individuals that hepatic glucose production is 96% suppressed with the insulin dose used in the present study (Defronzo, 1979).

ASSAYS

Blood glucose concentrations were determined using the glucose oxidase method with an automated glucose analyzer (YSI, Yellow Springs, OH). Serum insulin was measured by a double antibody RIA (Diagnostics Products, Los Angeles, CA).

STATISTICS

Differences among groups for baseline physical characteristics were determined with independent t tests. A 2 × 2 repeated-measures analysis of variance was performed to detect changes in IS, body weight, BMI, VO$_{2\text{max}}$, FFM, FM, percent body fat, and visceral and subcutaneous adipose tissues with time (pre vs. post) and among treatments (AT vs. RT). The effect of RT on muscle strength was determined using dependent t tests. Pearson product–moment correlation analyses were performed to identify the relationships between variables. Significance was accepted at the p ≤ .05 level. With the exception of IS values, which are reported as M ± standard error (SEM), data are reported as M ± SD. Statistical analyses were performed using SPSS® software (version 9, SPSS Inc., Chicago).

Results

The compliance rate of participants throughout AT or RT was ≥90%. Table 1 shows pre- and posttraining physical characteristics of participants in both exercise programs. Randomization of participants was successful—there were no significant differences between groups for any of the baseline physical characteristics, including IS. Three participants did not consent to VO$_{2\text{max}}$ testing after the 6 months of training (2 with AT and 1 with RT). In addition, technical problems during follow-up testing rendered the interpretation of three CT scans impossible (1 with AT and 2 with RT).

AT had no significant effects on BMI, percent body fat, visceral adipose tissue, and, as shown in Figure 1, absolute (pre, 300.8 ± 34.8 [SEM], and post, 279.2 ± 39.0 [SEM] mg/min) and relative (pre, 7.4 ± 0.8 [SEM], and post, 6.9 ± 1.0 [SEM] mg·kg FFM$^{-1}$·min$^{-1}$) rates of glucose disposal. AT tended to decrease subcutaneous adipose tissue (p = .075). On the other hand, AT significantly reduced body weight and FM and increased FFM and VO$_{2\text{max}}$. There were no significant relationships between AT-induced changes in IS and those of body weight, subcutaneous and visceral adipose tissues, FM, FFM, and VO$_{2\text{max}}$.

RT had no significant effects on body weight, BMI, VO$_{2\text{max}}$, visceral adipose tissue, and, as shown in Figure 1, on absolute (pre, 314.5 ± 26.9 [SEM], and post,
Table 1  Physical Characteristics of Participants Before and After Aerobic or Resistance Training, \( M \pm SD \)

<table>
<thead>
<tr>
<th>Physical characteristic</th>
<th>Aerobic Training ((n = 8))</th>
<th>Pre</th>
<th>Post</th>
<th>Resistance Training ((n = 10))</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td></td>
<td>62.3 ± 4.7</td>
<td>66.3 ± 5.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td></td>
<td>161.9 ± 3.5</td>
<td>161.3 ± 4.1</td>
<td></td>
<td>158.9 ± 5.6</td>
<td>158.5 ± 5.6</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td></td>
<td>71.7 ± 7.1</td>
<td>69.4 ± 5.2*</td>
<td></td>
<td>64.9 ± 8.3</td>
<td>65.1 ± 8.9</td>
</tr>
<tr>
<td>Body-mass index (kg/m(^2))</td>
<td></td>
<td>27.0 ± 2.2</td>
<td>26.7 ± 2.1</td>
<td></td>
<td>25.7 ± 2.8</td>
<td>25.8 ± 3.0</td>
</tr>
<tr>
<td>( V_{O_{2}} \text{max} ) (ml · kg(^{-1}) · min(^{-1}))</td>
<td></td>
<td>21.3 ± 3.0</td>
<td>25.0 ± 1.6*</td>
<td></td>
<td>22.7 ± 3.6</td>
<td>22.8 ± 3.4*</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td></td>
<td>40.1 ± 2.4</td>
<td>40.6 ± 2.5*</td>
<td></td>
<td>37.6 ± 4.2</td>
<td>38.5 ± 4.4*</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td></td>
<td>28.3 ± 4.3</td>
<td>26.1 ± 4.6*</td>
<td></td>
<td>24.3 ± 5.0</td>
<td>23.4 ± 5.9</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td></td>
<td>39.4 ± 2.7</td>
<td>37.4 ± 4.6</td>
<td></td>
<td>37.1 ± 4.3</td>
<td>35.5 ± 5.3*</td>
</tr>
<tr>
<td>Visceral adipose tissue (cm(^2))</td>
<td></td>
<td>152.2 ± 43.5</td>
<td>140.6 ± 50.0*</td>
<td></td>
<td>138.7 ± 39.0</td>
<td>137.0 ± 38.4*</td>
</tr>
<tr>
<td>Subcutaneous adipose tissue (cm(^2))</td>
<td></td>
<td>328.3 ± 70.6</td>
<td>265.5 ± 97.2*</td>
<td></td>
<td>283.2 ± 64.3</td>
<td>259.6 ± 56.2*</td>
</tr>
</tbody>
</table>

*Values significantly different from baseline \((p \leq .05)\).

\( \text{Pre} \) = 6, \( \text{Post} \) = 7, \( \text{Pre} \) = 9, \( \text{Post} \) = 8.

349.5 ± 30.9 \([SEM]\) mg/min and relative \(\text{pre}, 8.5 \pm 0.8 \[SEM]\), and \text{post}, 9.3 ± 1.0 \([SEM]\) mg · kg FFM\(^{-1}\) · min\(^{-1}\)) rates of glucose disposal. Whereas RT only tended to decrease FM \((p = .076)\), it significantly reduced percent body fat and subcutaneous adipose tissue and significantly increased FFM. There were no relationships between the changes in percent body fat, FM, FFM, and subcutaneous and visceral adipose tissues and those of IS. Finally, RT significantly increased muscle strength by 35.5% ± 30.2%, 26.2% ± 8.3%, 25.3% ± 21.4%, and 25.8% ± 6.0% for chest-press, seated-row, shoulder-press, and leg-press exercises, respectively.

**Discussion**

The goal of this investigation was to determine whether the physiological adaptations induced by 6 months of intense AT or RT could improve IS in nonobese, healthy older women beyond the first 24 hr after the last training bout. To address this particular issue, we measured IS 4 days after the last training session had been completed. Our results show that the adaptations induced by AT and RT failed to improve IS in a sustained manner in nonobese, healthy older women.

We found that IS did not improve despite the fact that both training programs increased FFM. An increase in muscle mass enhances the body storage site of glucose, which would be expected, during a clamp procedure, to increase glucose
disposal for a given insulin level, thereby improving IS. It is possible that a greater gain in FFM would have been necessary to improve IS in the current study. On the other hand, despite a greater storage space provided by an increased FFM, it is possible that the lack of increase in glucose disposal could be explained by the muscles being fully loaded with glycogen as a result of the 3 days of rest and diet control before IS measurement. In fact, it has been shown that glucose disposal during a clamp procedure mainly reflects the rate of glucose being stored as glycogen rather than the rate of oxidation (Annuzzi, Riccardi, Capaldo, & Kaijser, 1991; Thiébaud et al., 1982).

It is possible that the accumulation of muscle glycogen that occurred over the 3 days before the clamp masked the potential improvements in IS. Ultimately, the physiological adaptations induced by sustained, long-term AT or RT should combine to increase the translocation of GLUT-4 to the plasma membrane, which
is necessary for the transport of glucose into the cells. Insulin-stimulated glucose transport has been shown to augment in proportion to the increase in GLUT-4 (Kawanaka et al., 1999). Both AT (Short et al., 2003) and RT (Tabata et al., 1999) have been demonstrated to increase GLUT-4 levels. Large increases in muscle glycogen have been shown, however, to substantially reduce the translocation of GLUT-4 to the cell surface, which, in turn, has been demonstrated to reverse the exercise-induced improvement in IS (Garcia-Roves et al., 2003; Kawanaka et al., 1999). This phenomenon could be attributable to a decrease in insulin-stimulated protein kinase B (PKB) phosphorylation of both Ser473 and Thr308 (Kawanaka, Nolte, Han, Hansen, & Holloszy, 2000).

AT significantly decreased FM. Although not statistically significant, the decrease in FM observed with RT approached the threshold for significance. A decrease in FM could increase IS by improving the lipid profile and decreasing the expression of tumor-necrosis-factor alpha (TNF-α) and interleukin-6 (IL-6; Kern, Ranganathan, Li, Wood, & Ranganathan, 2001). The failure to observe an increase in IS despite a loss of FM, however, might have been because the loss of FM was not substantial enough in both training programs to produce such an effect. Altogether, these results indicate that the favorable changes in total FM and FFM induced by 6 months of AT or RT do not improve IS in a sustained manner in older women.

In the present study, neither training program decreased visceral adipose tissue. Hence, some might argue that the absence of change in this fat compartment could explain, at least in part, the absence of improvement in IS, because it has been shown that IS correlates strongly with the amount of visceral fat (Brochu et al., 2000; Cefalu et al., 1995; Gastaldelli et al., 2002). Our results show, however, that there were no significant relationships between the changes in IS and changes in visceral fat. Moreover, DeNino et al. (2001) showed that, in nonobese, healthy older women, the relationship between IS and visceral fat is only modest.

We also found that AT improved \( VO_{2max} \), suggesting that this training regimen probably increased muscle’s capillarization and/or the level and activity of oxidative enzymes. Such modifications could increase the ability of muscle to transport and metabolize glucose, thereby improving IS. It might be possible, in previously sedentary older women, that AT-induced elevation in \( VO_{2max} \) must be substantially higher than the 15% we observed in the current study in order to improve IS in a sustained manner.

Closer inspection of the individual responses indicate that 9 out of 18 women improved IS by an average of 25% (3 with AT and 6 with RT). The remaining 9 participants, however, had an average decrease in IS on the order of 20%. The reasons underlying such variations in IS after AT or RT in participants with similar physical characteristics are unclear and warrant further research.

Using training procedures identical to the ones employed in the present investigation, Poehlman et al. (2000) and Dionne et al. (2004) showed that younger women improved IS 3–4 days after the last AT or RT session. Accordingly, these results suggest that younger but not older women can improve IS in a prolonged manner with exercise training. Similarly, Short et al. (2003) showed that younger
but not older women improved IS in a sustained manner after AT. The reasons underlying such discrepant responses to physical training in older and younger women are not known but could be attributable to the levels of oxidative stress (Jira, Spiteller, & Schramm, 1996), TNF-α (Paolisso et al., 1998), insulin-like growth factor I (IGF-I; Donahue, Hunter, Sherblom, & Rosen, 1990), and GLUT-4 protein (Houmard et al., 1995), which are known to differ between younger and older individuals. The cited results, combined with those found in the present study, suggest that only in younger women do the long-term adaptations induced by AT or RT play a role in improving IS.

It must be acknowledged that our sample was composed of nonobese, healthy older women. Hence, in this specific population of older women who are aging successfully, it could be more difficult to improve IS in a sustained manner with AT or RT than in older women who display health problems. For example, Cuff et al. (2003) recently demonstrated that AT combined with RT improved IS up to 48–72 hr after the last training bout in older obese women with Type II diabetes. It is also possible that a higher frequency and intensity of training combined with some additional months of training would have been required for AT or RT to improve IS in a sustained manner in this population. This will have to be examined in future studies. In the case where these training modifications fail to improve IS, the hypothesis that, because of issues of selective survival, there is not much more that could be done to improve IS in a prolonged manner with training in healthy older women will have to be considered. Nonetheless, although AT or RT could not improve IS in a sustained fashion, the fact that it could potentially slow or halt the decrease in IS observed in normal aging merits attention.

In conclusion, our results indicate that 6 months of intense AT or RT in nonobese, healthy older women do not improve IS 4 days after the last training session. These results were observed despite the fact that both training programs improved several components of body composition (and cardiovascular fitness in the case of AT). These findings thus suggest that the adaptations induced by AT or RT do not maintain the improvement in IS beyond the first 24 hr after the last training bout. Consequently, from a clinical standpoint, we suggest that nonobese, healthy older women perform AT or RT on a daily basis in order to improve and maintain the improvement in IS.

Acknowledgments

We want to thank all the women who participated in this study. We recognize the helpful support provided by the University of Vermont General Clinical Research Center (RR-109) and the Department of U.S. Defense (DE 950226). E.D.B.G., M.A.L., and I.J.D. are financially supported by the Canadian Institutes of Health Research; M.O.M. is supported by the University of Sherbrooke.

References


