

Is it the resistance training itself or the combined associated weight loss that improves the metabolic syndrome-related phenotypes in postmenopausal women?

Ozlem Soyuk¹
Gulistan Bahat²

¹Division of Endocrinology and Metabolism, Department of Internal Medicine, Istanbul Medical School, Istanbul University, Capa, Istanbul, Turkey; ²Division of Geriatrics, Department of Internal Medicine, Istanbul Medical School, Istanbul University, Capa, Istanbul, Turkey

Dear editor

We read the article entitled “Resistance training improves isokinetic strength and metabolic syndrome-related phenotypes in postmenopausal women” by Oliveira et al¹ with great interest. In the study, the authors examined the effects of 12 weeks of resistance training (RT) on metabolic syndrome-related phenotypes in postmenopausal women. They reported that total cholesterol, low-density lipoprotein cholesterol levels, total cholesterol/high-density lipoprotein cholesterol ratio, blood glucose, basal insulin, and homeostatic model assessment of insulin resistance were all significantly reduced with RT ($P < 0.01$). Accordingly, they concluded that a 12-week progressive RT program induces beneficial alterations on metabolic syndrome-related phenotypes in postmenopausal women.

While we appreciate this detailed study,¹ we have some comments on the results and subsequent conclusions. In their study, Oliveira et al reported that, in addition to the aforementioned parameters (indices), body mass (68.9 ± 13.3 kg before RT vs 68.1 ± 13.2 kg after 12 weeks' RT), body mass index (BMI) (28.4 ± 5.0 kg/m² before RT vs 27.9 ± 4.8 kg/m² after 12 weeks' RT), and waist circumference (WC) (98.3 ± 13.2 cm before RT vs 94.6 ± 12.6 cm after 12 weeks' RT) were also significantly decreased after the RT period.¹

Increased body weight is a very well-known major risk factor for the metabolic syndrome documented both in cross-sectional and longitudinal studies.^{2,3} Obesity, particularly abdominal obesity, is associated with insulin resistance. Furthermore, the importance of weight management in preventing progression of metabolic syndrome components has also already been illustrated.⁴ Hence, we suggest that the reported association of RT with beneficial alterations on metabolic syndrome-related phenotypes may be due to the combined weight, BMI, and WC reduction, rather than the effect of RT itself.

In line with our comment, in the study aimed at addressing whether RT alone improves cardiometabolic health in overweight and obese adults, Bateman et al⁵ concluded that RT was not effective at improving the metabolic syndrome score; however, aerobic training (AT) was effective. Combined AT and RT was similarly effective but not different from AT alone. Importantly, in this study, body mass was significantly decreased with AT and combined AT-RT, but was not changed with RT alone.⁵ In a similar study aiming to clarify effects of exercise modality on insulin resistance, Davidson et al⁶ also reported that insulin resistance improved compared with controls

Correspondence: Gulistan Bahat
Division of Geriatrics, Department of Internal Medicine, Istanbul Medical School, Istanbul University, Capa, 34390, Istanbul, Turkey
Tel +90 212 414 2000 ext 33204
Fax +90 212 532 4208
Email gbahatozturk@yahoo.com

with aerobic exercise alone and in the combined exercise groups, but not in the resistance exercise group. Again, in this later study, body weight was not significantly reduced in the resistance exercise group.⁶ On the other hand, Dunstan et al⁷ reported that high-intensity progressive RT was effective in improving glycemic control in older patients with type 2 diabetes when combined with moderate weight loss.

The results of these studies^{5–7} suggest that RT seems not to affect the metabolic syndrome parameters if not accompanied with weight, BMI, or WC reduction. Therefore, we suggest that in the Oliveira et al¹ study, RT and weight loss should be analyzed with regression analysis for their relation to metabolic syndrome parameters in order to clarify whether RT itself is independently associated with metabolic syndrome.

Disclosure

The authors report no conflicts of interest in this communication.

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Author's reply

Ricardo Moreno Lima

André Bonadiaz Gadelha

Lauro C Vianna

College of Physical Education, University of Brasília, Brasília,
Federal District, Brazil

Correspondence: Ricardo Moreno Lima
Universidade de Brasília (UnB), Campus Universitário Darcy Ribeiro,
Faculdade de Educação Física, Brasília, Distrito Federal, Brazil
Tel +55 61 8109 9444
Fax +55 61 3107 2500
Email ricardomoreno@unb.br

Dear editor

We are enthusiastic that our study has earned the attention of others, and we are pleased to have the opportunity to further debate the merits of our findings. Soyluk and Bahat's letter to the editor¹ raises some interesting insights regarding the interpretation of our results, in particular, whether or not resistance training (RT) independently ameliorates metabolic syndrome-related phenotypes in postmenopausal women. We would like to address the perceptions of these authors and are confident that such discussion will be valuable for health professionals designing clinical interventions for older adults.

In our study,² we demonstrated that a 12-week RT program induces beneficial alterations on metabolic syndrome-related phenotypes in postmenopausal women. The benefits were related to waist circumference (WC) reduction, positive alterations on lipid profile, and improvements in glycemic control. The letter to the editor¹ suggests that the observed alterations on metabolic profile may be due to weight, body mass index (BMI), and WC decreases, rather than to the effect of RT itself. In this regard, it should be noted that although body weight and BMI reached borderline significance, these reductions were modest (from 68.9 ± 13.3 kg to 68.1 ± 13.2 kg, and from 28.4 ± 5.0 kg/m² to 27.9 ± 4.8 kg/m², respectively), and with small effect sizes (0.05 and 0.09, respectively), while the reduction in WC was more compelling ($P < 0.001$) and with larger effect size (from 98.3 ± 13.2 cm to 94.6 ± 12.6 cm; effect size = 0.30). WC is consistently related to cardiometabolic risk³ and is a pivotal criterion for metabolic syndrome definition.^{4,5} Thus, the RT protocol induced a decrease in WC, which per se is a metabolic syndrome-related phenotype. This observation is in agreement with previous RT studies in older individuals.⁶⁻⁸

The points raised in the letter, however, lead to an important question: were changes observed for metabolic profile related to changes in WC? Furthermore, Soyluk and Bahat¹

suggested regression analyses to address this question, and we are thankful for that suggestion. As such, we performed the regression analyses, and the results did not show significance, with only a trend for the relationship between ΔWC and Δ total cholesterol ($r = 0.31$; $r^2 = 0.09$; $P = 0.08$) evident. Certainly, these results do not rule out the possibility of RT-induced visceral fat decrease to partially explain metabolic improvements.

Despite this result, the positive metabolic effects of exercise in general, and RT in particular, should also be considered in light of other underlying physiological mechanisms. A consistent body of evidence^{9,10} indicates that physical inactivity is causative in the development of metabolic diseases. In animal models, cessation of voluntary physical activity precipitated a rapid (just 24 hours) decline in insulin-mediated glucose uptake that was paralleled by a reduction in molecular signaling pathways related to the insulin receptor and its activation.¹¹ These observations confirm the rapid decline in insulin sensitivity when physically active humans discontinue regular exercise.¹² Conversely, only 7 days of exercise training were sufficient to significantly improve glycemic control in sedentary, middle-aged individuals.¹³ In regard to resistance exercises, Singhal et al¹⁴ noted that different training intensities acutely induce reductions in postprandial lipemia, lessening exposure to elevated atherogenic low-density lipoprotein cholesterol levels. These findings highlight how metabolic variables can be rapidly affected by both physical activity and inactivity, without alterations in body fat. In support of this idea, Poehlman et al¹⁵ demonstrated metabolic improvements as a result of RT that occurred without alterations in total body fat, subcutaneous tissue, or visceral adipose tissue. Importantly, these findings have been recapitulated by other groups.^{16,17} Taken together, the results of these studies provide evidence that, in addition to weight loss, other mechanisms might contribute to metabolic improvements following RT.

In summary, the results of our study² show that RT improves metabolic syndrome-related phenotypes in postmenopausal women, and thus, these results have important clinical implications. It was not in the scope of the study to examine the mechanisms underlying such alterations. Soyluk and Bahat's letter to the editor¹ raised the interesting question of whether RT per se, or its combination with weight loss, explained metabolic improvements. They suggested regression analyses to examine such a question, which we performed, but the results did not reveal significant association. A plethora of studies indicate that although weight loss is important, other mechanisms are likely to contribute

to RT-induced metabolic improvements. Nevertheless, it is reasonable to suggest that the positive effects of RT on metabolic traits would be extended if associated with reduction in body weight, particularly visceral fat. In any case, the “take-home” message from our study remains the same: RT should be considered in the prevention of metabolic syndrome, and our results² reinforce the idea that this mode of exercise is an important component of physical activity programs for elderly people.

Disclosure

The authors report no conflicts of interest in this communication.

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