

The Effect of Exercise Training on Resting Metabolic Rate in Type 2 Diabetes Mellitus

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ABSTRACT

JENNINGS, A. E., ALBERGA, R. J., SIGAL, O. JAY, N. G. BOULÉ, AND G. P. KENNY. The Effect of Exercise Training on Resting Metabolic Rate in Type 2 Diabetes Mellitus. *Med. Sci. Sports Exerc.*, Vol. 41, No. 8, pp. 1558–1565, 2009. **Purpose:** Exercise is a possible means to increase resting energy expenditure, which could offset age-related metabolic declines and facilitate weight management, both of which are particularly important for people who have type 2 diabetes mellitus. We sought to determine the effects of aerobic exercise training and resistance exercise training and the incremental effect of combined aerobic and resistance exercise training on resting metabolic rate (RMR) in previously sedentary individuals with type 2 diabetes. **Methods:** After a 4-wk run-in period, 103 participants (72 male, 31 female, 39–70 yr, mean \pm SD body mass index = $32.9 \pm 5.7 \text{ kg}\cdot\text{m}^{-2}$) were randomly assigned to four groups for 22 wk: aerobic training, resistance training, combined aerobic and resistance exercise training, or waiting-list control. Exercise training was performed three times per week at community-based gym facilities. RMR was measured by indirect calorimetry for 30 min after an overnight fast. Body composition was assessed using bioelectrical impedance. These measurements were taken at baseline, at 3 months, and at 6 months of the intervention. **Results:** RMR did not change significantly in any group after accounting for multiple comparisons despite significant improvements in peak oxygen consumption and muscular strength in the exercising groups. Adjusting RMR for age, sex, fat mass, and fat-free mass in various combinations did not alter these results. **Conclusion:** These results suggest that RMR was not significantly changed after a 6-month exercise program, regardless of modality, in this sample of adults with type 2 diabetes. **Key Words:** AEROBIC EXERCISE, RESISTANCE EXERCISE, PHYSICAL ACTIVITY, ENERGY EXPENDITURE, BODY COMPOSITION, OBESITY

Aging is typically associated with a reduction in resting energy expenditure (21), which is due in large part to the loss of metabolically active muscle tissue (27,34). There is evidence suggesting that regular exercise may offset the decrease in resting metabolism that usually accompanies aging (33). Although many studies have examined the effect of either aerobic or resistance exercise programs on resting metabolic rate (RMR) in healthy adults, the results have been equivocal (32).

Studies of aerobic interventions have generally been successful in demonstrating decreases in body weight and fat mass (9,18,29), but the majority have failed to show increases in fat-free mass and RMR (8,30,38). Conversely, some resistance training exercise programs have been

shown to increase total energy expenditure by increasing RMR in older healthy adults (16). Increases in RMR between 5% and 10% have been demonstrated after resistance training programs (6,16,25), which, in some studies, remained significantly elevated (3%–5%) even after adjustment for fat-free mass (16,26). However, other resistance training studies have shown that RMR did not change significantly in young healthy previously untrained males (35) and females (4).

In people with type 2 diabetes mellitus, pathophysiological differences have been shown to affect RMR (12,15,20,22,24,37). Glycemia is of particular importance and has been shown to have a direct relationship with RMR (12,15,22). To date however, little is known about the effects of regular exercise on resting energy expenditure in type 2 diabetes. Mourier et al. (23) found no training effect on RMR after an 8-wk program of continuous and intermittent aerobic exercise performed at 50–85% of peak maximal oxygen consumption ($\dot{V}O_{2\text{peak}}$) three times per week. In contrast, Araiza et al. (1) reported that a low-intensity 6-wk walking program of 10,000 steps per d, five or more days per week, significantly increased resting energy expenditure in type 2 diabetes. However, because both studies did not measure fat-free mass, it is unclear if

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the observed changes in RMR were induced by changes in body composition. The small sample sizes and the short durations of the exercise training interventions may also explain the different findings in these interventions.

The long-term effect of exercise training programs on RMR in individuals with type 2 diabetes warrants further investigation. Therefore, the purpose of this study was to examine the influence of aerobic exercise training, resistance exercise training, or both on RMR in individuals with type 2 diabetes. To our knowledge, this is the first study to examine the incremental effect of combined aerobic and resistance training exercise in type 2 diabetes versus either type of exercise alone. We hypothesized *a priori* that both resistance exercise training and combined aerobic and resistance exercise training would increase resting energy expenditure to a similar extent, whereas little change would occur in aerobic exercise alone.

METHODS

This project was a substudy of the Diabetes Aerobic and Resistance Exercise (DARE) trial described elsewhere (31). The DARE trial was a single-center, randomized controlled trial with parallel group design that investigated the effects of aerobic and resistance exercise training as well as their combination on glycemic control in type 2 diabetes. Participation in the present substudy was optional, and recruitment to it was closed on August 21, 2002. The Ottawa Hospital Research Ethics Board approved all methods and procedures, and all participants provided written informed consent.

Previously sedentary individuals with type 2 diabetes mellitus ($n = 135$; 93 male, 42 female, 39–70 yr) were recruited through advertising, through physicians, and through word of mouth. Inclusion criteria for the DARE trial included type 2 diabetes for at least 6 months and baseline hemoglobin A_{1c} between 6.6% and 9.9% (normal = 4–6%). Exclusion criteria are described elsewhere (31).

Participants exercised at community-based facilities, supervised by personal trainers. Before randomization, participants entered a 4-wk run-in period to familiarize them with the exercise protocols and assess compliance. Only participants attending at least 10 of the 12 scheduled exercise sessions during the run-in period were eligible for randomization. After randomization, participants exercised three times per week, and training progressed gradually in length and intensity. The combined group did the full aerobic training program plus the full resistance training program. Control participants were asked to revert to their prestudy activity levels. Complete details of the exercise programs are described elsewhere (31).

Outcomes and measurements. The primary outcome in this analysis was absolute RMR ($\text{kJ}\cdot\text{min}^{-1}$). Secondary outcomes included $\dot{V}\text{O}_{2\text{peak}}$, muscular strength, body composition, and RMR adjusted for age, sex, fat-free mass, and fat mass in various combinations.

RMR was measured in the morning after a 12-h fast, a full night sleep (approximately 8 h), and no strenuous physical activity for 48 h. Participants were asked to refrain from taking their blood glucose-lowering medications the day of the test. Participants sat in a semirecumbent position in an isolated semidarkened, temperature-controlled room (mean \pm SD: $21.4 \pm 1.9^\circ\text{C}$, $37\% \pm 12\%$ relative humidity) for 45 min. Participants were asked to refrain from moving, talking, or sleeping but were allowed to read. Oxygen consumption ($\dot{V}\text{O}_2$) was measured continuously during the final 30-min period using an automated gas analysis system (MedGraphics CPX-D Metabolic Cart, St. Paul, MN). The system was calibrated before each test according to manufacturer specifications. $\dot{V}\text{O}_2$, carbon dioxide output ($\dot{V}\text{CO}_2$), ventilation (\dot{V}_E), the respiratory exchange ratio (RER), tidal volume, and respirations were collected every 30 s and averaged over 5-min intervals. The mean from minutes 15 to 25 of the $\dot{V}\text{O}_2$ measurement was used for data analysis because this was found to be the time frame that most consistently reached a steady state, which is defined as a 10-min period during which $\dot{V}\text{O}_2$, \dot{V}_E , and RER did not vary $>10\%$ (14). RMR ($\text{kJ}\cdot\text{min}^{-1}$) was calculated using the following equation:

$$\text{RMR} = 4.1868 \left(\dot{V}\text{O}_2 \left[\left(\frac{\text{RER} - 0.7}{0.3} \right) e_c + \left(\frac{1 - \text{RER}}{0.3} \right) e_f \right] \right) \quad [1]$$

where e_c is the caloric equivalent for the oxidation of carbohydrates ($5.047 \text{ kcal}\cdot\text{LO}_2^{-1}$), e_f is the caloric equivalent for the oxidation of fats ($4.686 \text{ kcal}\cdot\text{LO}_2^{-1}$), RER is the respiratory exchange ratio, and $\dot{V}\text{O}_2$ is the rate of oxygen consumption ($\text{L}\cdot\text{min}^{-1}$).

After the RMR test, muscular strength was assessed for the seated row, bench press, and leg press exercises measured on a multistation gym (Body Solid EXM-2000S, Forrest Park, IL). Muscular strength was measured as the maximum weight the individual could lift eight times determined using an eight repetition max protocol. Proper lifting and breathing techniques were demonstrated by the exercise specialist/researcher before commencing each exercise.

Peak oxygen consumption ($\dot{V}\text{O}_{2\text{peak}}$) was measured on a separate day using a graded treadmill protocol with continuous breath-by-breath analysis using an automated gas analysis system (MedGraphics CPX-D Metabolic Cart) with values averaged over 30 s. Heart activity was monitored throughout using a 12-lead electrocardiogram (v.4.03; Gemarquette Medical Systems Inc., Waukesha, WI). Speed and grade varied between individuals based on their level of conditioning as assessed by the specialist. $\dot{V}\text{O}_{2\text{peak}}$ was measured as the highest minute $\dot{V}\text{O}_{2\text{peak}}$ recorded during the test.

Fat mass and fat-free mass were estimated from bioelectrical impedance (BIA 101A Analyzer, RJL Systems, Clinton, MI) using the equation by Kyle et al. (19). This equation was validated in a subsample of DARE trial participants whose body composition was also measured with underwater weighing using Siri's equations (36 male, 16 female). Pearson correlations between estimated percent

TABLE 1. Baseline physical characteristics for males (M) and females (F) in the combined, resistance, and aerobic training groups and the control group.

		Combined	Resistance	Aerobic	Control
<i>N</i>	M	19	18	13	22
	F	6	7	7	11
Age (yr)	M	54 ± 8	52 ± 7	55 ± 8	56 ± 7
	F	56 ± 7	55 ± 9	56 ± 7	57 ± 7
Height (cm)	M	173 ± 7	175 ± 8	177 ± 6	176 ± 6
	F	162 ± 4	160 ± 8	162 ± 8	161 ± 7
BMI (kg·m ⁻²)	M	31.5 ± 5.0	33.2 ± 6.0	32.2 ± 4.9	32.0 ± 4.8
	F	35.6 ± 6.7	32.4 ± 8.8	35.5 ± 6.1	34.5 ± 6.2
Compliance (%)	M	88.8 (86) ± 12.8	86.1 (85) ± 8.7	87.5 (85) ± 13.3	NA
Mean (median) ± SD	F	87.1 (88) ± 7.3	87.0 (89) ± 10.4	89.8 (91) ± 8.5	NA

Values are presented as means ± SD.

body fat from bioimpedance and underwater weighing were 0.84 in males and 0.75 in females (13).

RMR and body composition measurements were repeated at 3 and 6 months, whereas $\dot{V}O_{2peak}$ and muscular strength were only repeated at 6 months.

Statistical analysis. We used ANOVA to test for baseline differences among all groups for all variables. For the primary analysis, we used a linear mixed-effects model for repeated measures over time, with absolute RMR as the dependent variable, and effects for time, study group, and time-by-group interaction. We used the same procedures for total body mass, fat mass, fat-free mass, $\dot{V}O_{2peak}$, and muscular strength. We ran additional models in which age, sex, fat-free mass, and fat mass were entered as covariates alone and in combinations in addition to time, study group, and time-by-group interaction. Within the mixed models, we estimated 95% confidence intervals (CI) and *P* values for the six intergroup comparisons (combined training vs aerobic training, combined training vs resistance training, aerobic training vs control, resistance training vs control, combined training vs control, and aerobic training vs resistance training)

for change in absolute RMR between baseline and 6 months and over time within each group. Unadjusted 95% CI are presented in the tables. The level of significance was set at an overall alpha level of 0.05. However, we used a Bonferroni correction for multiple comparisons in which *P* values for intergroup comparisons were considered significant when *P* was <0.0083 (0.05 divided by six possible intergroup comparison for each variable). Within-group comparisons were considered significant when *P* was <0.0125 (0.05 divided by four possible within-group changes).

The present analysis is restricted to participants who had a training compliance rate greater than 70% and on whom baseline and 6-month RMR measurements were complete and technically adequate. All analyses were performed using the statistical software package SAS 9.1 (SAS Institute, Cary, NC).

RESULTS

Of the initial 135 participants taking part in the run-in period, 103 (25 of 33 for combined training, 25 of 35 for

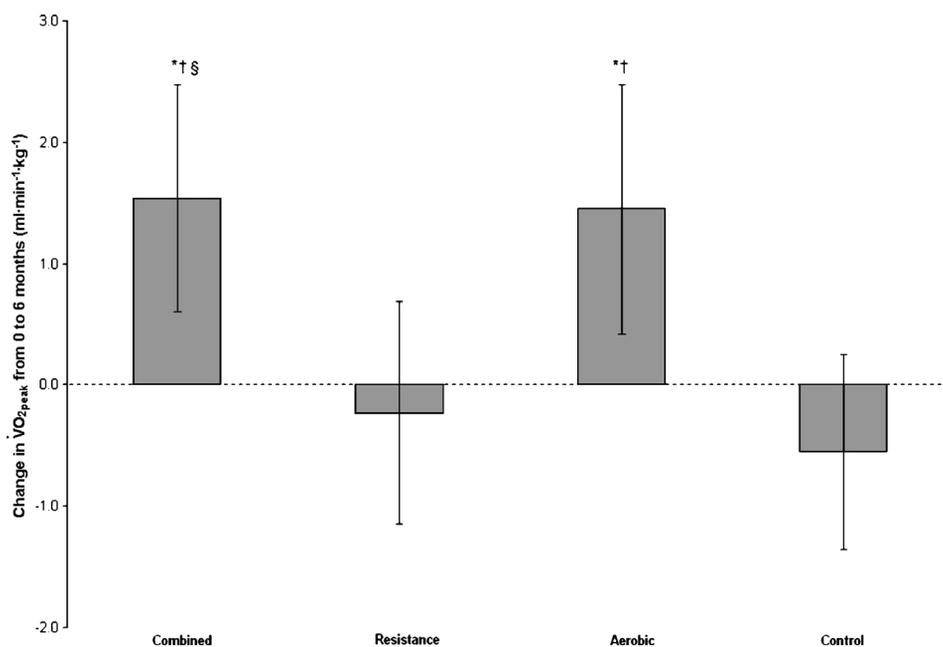


FIGURE 1—Change in peak oxygen consumption ($\dot{V}O_{2peak}$) from 0 to 6 months for combined, resistance, aerobic, and control training groups. Data are presented as means and 95% CI. *Significant change from 0 to 6 months. †Significant difference from control group. §Significant difference from resistance group.

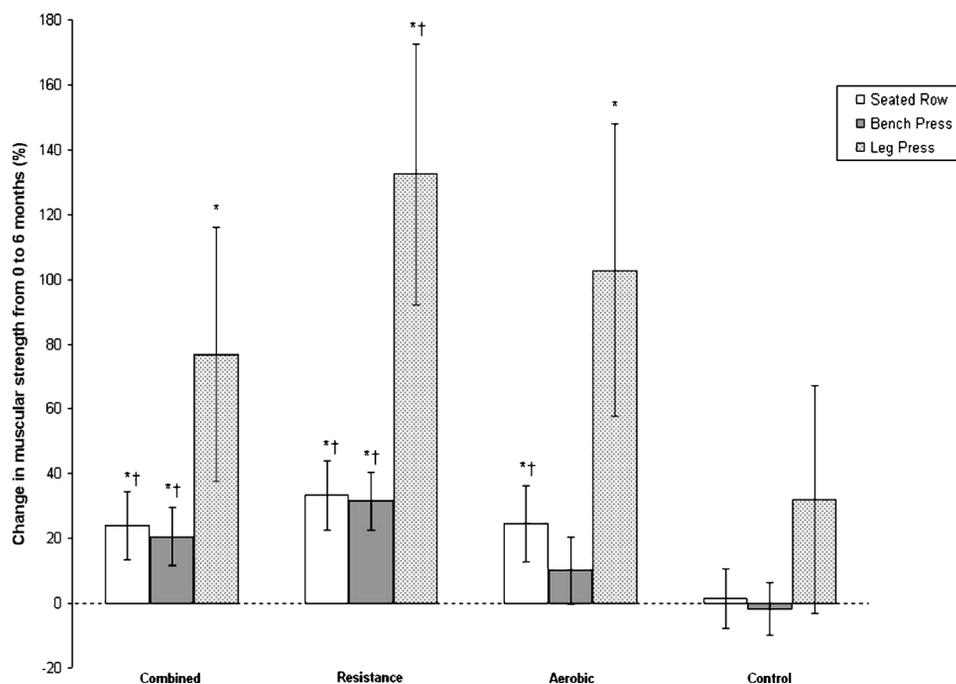


FIGURE 2—Changes in muscular strength from 0 to 6 months for combined, resistance, aerobic, and control training groups. Data are presented as means and 95% CI. *Significant change from 0 to 6 months. †Significant difference from control group.

resistance training, 20 of 33 for aerobic training, and 33 of 34 for control) reached 70% compliance or above after 6 months and had complete data. Preintervention baseline characteristics at 0 month for the 103 participants included in the analyses, and compliance rates for all groups are given in Table 1. There were no significant baseline differences between groups before the run-in period. For the 103 participants reaching 70% compliance or above after 6 months, compliance rates were over 86% in all exercising groups. Out of the 32 participants who were excluded, 4 failed to complete RMR testing at 6 months (2 in resistance training, 1 in aerobic training, and 1 in control). Twenty-eight participants did not reach the compliance cutoff of which half were due to withdrawals for personal (11%; two in resistance training and one in aerobic training), medical (21%; two in combined training and four in aerobic training), or time commitment issues (18%; two in combined training, two in resistance training, and one in aerobic training). The remaining 14 participants were excluded due to low compliance (4 in combined training, 4 in resistance training, and 6 in aerobic training).

General Exercise Outcomes

Aerobic capacity. Changes in $\dot{V}O_{2\text{peak}}$ from 0 to 6 months were significantly different between training groups ($F_{(3,98.6)} = 5.76$, $P = 0.001$). Relative to the control group, changes in $\dot{V}O_{2\text{peak}}$ were greater in the combined training ($P = 0.002$) and aerobic training ($P = 0.006$) groups but not the resistance training group ($P = 0.601$) (Fig. 1). The effect of training group upon changes in $\dot{V}O_{2\text{peak}}$ did not differ

between sexes ($P = 0.518$) and was not influenced by age ($P = 0.473$).

Muscular strength. Change in seated row performance after 6 months was significantly different between groups ($F_{(6,92.5)} = 3.96$, $P = 0.001$; Fig. 2). In comparison to control, seated row performance was significantly improved in combined training ($P = 0.002$) and resistance training ($P < 0.0001$) but not aerobic training ($P = 0.075$). Changes in leg press performance differed significantly among groups ($F_{(6,102)} = 3.58$, $P = 0.003$), but only the resistance training group's change differed significantly from control after accounting for multiple comparisons. Changes in bench press performance differed significantly among groups ($F_{(6,98.8)} = 5.84$, $P < 0.001$). Relative to the control group, significant increases were observed in the combined training ($P < 0.001$) and resistance training ($P < 0.001$) groups but not in the aerobic training group ($P = 0.075$). Although strength was positively and significantly associated with male sex and younger age, the effects of exercise training group did not vary according to age or sex for changes in any of the three strength measures.

Body Composition

Total body mass. Changes in total body mass did not differ significantly among groups ($F_{(6,129)} = 1.60$, $P = 0.152$), although the within-group decrease over time was significant in the combined training group (Table 2). Total body mass was negatively associated with age ($F_{(1,98)} = 11.72$, $P = 0.001$) and positively associated with male sex ($F_{(1,98)} = 5.26$, $P = 0.024$). However, the effects of exercise

TABLE 2. Total body mass, fat-free mass, and fat mass after 0, 3, and 6 months in the combined, resistance, and aerobic training groups and the control group.

Variable	Baseline	3 Months	6 Months	Change from Baseline to 6 Months (95% CI)
Total body mass (kg)				
Combined	94.3 ± 3.7	92.8 ± 3.6	91.7 ± 3.6	-2.6 (-4.1 to -1.1)*
Resistance	96.8 ± 3.7	95.7 ± 3.6	96.2 ± 3.6	-0.6 (-2.1 to 0.9)
Aerobic	98.4 ± 4.1	96.9 ± 4.1	96.5 ± 4.1	-1.9 (-3.6 to -0.2)
Control	96.0 ± 3.2	95.4 ± 3.2	95.7 ± 3.2	-0.3 (-1.6 to 1.0)
Fat-free mass (kg)				
Combined	61.7 ± 2.2	61.1 ± 2.2	60.7 ± 2.2	-0.9 (-2.3 to 0.4)
Resistance	62.2 ± 2.2	61.6 ± 2.2	61.6 ± 2.2	-0.7 (-2.0 to 0.7)
Aerobic	62.0 ± 2.5	61.2 ± 2.4	61.5 ± 2.5	-0.5 (-2.0 to 1.0)
Control	61.6 ± 1.9	61.2 ± 1.9	61.0 ± 1.9	-0.7 (-1.8 to 0.5)
Fat mass (kg)				
Combined	32.8 ± 2.3	31.7 ± 2.2	30.9 ± 2.2	-1.8 (-3.5 to -0.2)
Resistance	34.4 ± 2.3	34.2 ± 2.2	34.6 ± 2.2	0.2 (-1.4 to 1.8)
Aerobic	36.4 ± 2.6	35.6 ± 2.4	35.0 ± 2.5	-1.4 (-3.2 to 0.4)
Control	34.5 ± 2.0	34.2 ± 1.9	34.7 ± 1.9	0.2 (-1.2 to 1.6)

Values are presented as means ± SE. * Significant change from 0 to 6 months. Fat mass and fat-free mass were estimated using bioelectrical impedance.

training group on changes in total body mass over time did not differ according to age or sex.

Fat-free mass. Changes in fat-free mass did not differ significantly among groups ($F_{(6,128)} = 0.26, P = 0.954$). Fat-free mass was negatively associated with age ($F_{(1,98.1)} = 9.09, P = 0.003$) and higher in males than females ($F_{(1,98)} = 73.77, P = 0.001$). However, the effects of exercise training group on changes in fat-free mass over time did not differ according to age ($P = 0.628$) or sex ($P = 0.756$).

Fat mass. Changes in fat mass did not differ significantly among groups ($F_{(6,128)} = 1.34, P = 0.245$). Fat mass was negatively associated with age ($F_{(1,98.1)} = 9.09, P = 0.003$) and higher in males than females ($F_{(1,98)} = 73.77, P = 0.001$). However, the effects of exercise training group on changes in fat mass over time did not differ according to age ($P = 0.658$) or sex ($P = 0.921$).

Resting Metabolic Rate (RMR)

Changes in absolute RMR over time did not differ significantly among groups ($F_{(6,125)} = 0.71, P = 0.640$), and there

were no significant within-group changes after taking into account multiple comparisons (Table 3). RMR was significantly lower with increasing age ($F_{(1,98.4)} = 98.4, P < 0.001$), and males had significantly higher RMR than females ($F_{(1,98.1)} = 18.31, P < 0.001$). There was significant collinearity between sex and fat mass, so we do not present results with these two variables in the same models. However, adjustment for age, sex, fat mass, and fat-free mass, alone or in various combinations, did not significantly influence changes in RMR among groups over time.

DISCUSSION

We examined the effects of aerobic exercise training and resistance exercise training and a combination of aerobic and resistance exercise training on RMR in previously sedentary individuals with type 2 diabetes. The 26-wk exercise training programs failed to demonstrate significant increases in RMR. Although there appeared to be increasing trends in RMR from baseline to 6 months in the exercising groups

TABLE 3. Absolute resting metabolic rate (RMR) and RMR adjusted for covariates after 0, 3, and 6 months in the combined, resistance, and aerobic training groups and the control group.

Variable	Baseline	3 Months	6 Months	Change from Baseline to 6 Months (95% CI)
RMR ($\text{kJ}\cdot\text{min}^{-1}$)				
Combined	5.23 ± 0.25	5.39 ± 0.25	5.44 ± 0.24	0.21 (-0.13 to 0.55)
Resistance	5.29 ± 0.25	5.45 ± 0.25	5.48 ± 0.24	0.19 (-0.15 to 0.53)
Aerobic	5.00 ± 0.27	5.47 ± 0.28	5.38 ± 0.26	0.38 (0.01 to 0.76)
Control	5.42 ± 0.21	5.44 ± 0.22	5.47 ± 0.20	0.05 (-0.24 to 0.34)
RMR adjusted _{fat-free mass} ($\text{kJ}\cdot\text{min}^{-1}$)				
Combined	5.22 ± 0.18	5.42 ± 0.19	5.50 ± 0.17	0.28 (-0.05 to 0.61)
Resistance	5.24 ± 0.18	5.46 ± 0.19	5.47 ± 0.17	0.24 (-0.09 to 0.57)
Aerobic	4.96 ± 0.20	5.50 ± 0.21	5.38 ± 0.19	0.42 (0.05 to 0.79)
Control	5.41 ± 0.16	5.45 ± 0.16	5.51 ± 0.15	0.10 (-0.19 to 0.38)
RMR adjusted _{age, fat mass, fat-free mass} ($\text{kJ}\cdot\text{min}^{-1}$)				
Combined	5.24 ± 0.17	5.45 ± 0.17	5.53 ± 0.17	0.30 (-0.03 to 0.62)
Resistance	5.19 ± 0.17	5.40 ± 0.18	5.42 ± 0.16	0.23 (-0.10 to 0.56)
Aerobic	4.95 ± 0.19	5.49 ± 0.20	5.39 ± 0.18	0.43 (0.07 to 0.80)
Control	5.45 ± 0.15	5.50 ± 0.15	5.54 ± 0.14	0.09 (-0.20 to 0.37)
RMR adjusted _{age, sex, fat-free mass} ($\text{kJ}\cdot\text{min}^{-1}$)				
Combined	5.27 ± 0.18	5.48 ± 0.18	5.55 ± 0.17	0.28 (-0.05 to 0.61)
Resistance	5.23 ± 0.17	5.45 ± 0.19	5.47 ± 0.17	0.24 (-0.09 to 0.57)
Aerobic	5.02 ± 0.19	5.55 ± 0.20	5.44 ± 0.19	0.42 (0.05 to 0.79)
Control	5.50 ± 0.15	5.54 ± 0.16	5.60 ± 0.15	0.10 (-0.19 to 0.38)

Values are presented as means ± SE. Fat mass and fat-free mass were estimated using bioelectrical impedance.

compared with the control group, these were not statistically significant after accounting for multiple comparisons. These findings were observed despite significant increases in $\dot{V}O_{2\text{peak}}$ for the aerobic training groups and in muscular strength for the resistance training groups. As expected, both RMR and fat-free mass were lower in older versus younger participants and in women versus men.

Although this is the first study to comprehensively examine the effect of different exercise training modalities on RMR in type 2 diabetes, similar findings have been reported in nondiabetic populations. In fact, the majority of aerobic exercise training intervention studies in healthy young and middle-aged adults found no effect on RMR. Many of these studies evaluated exercise interventions of the same exercise intensity as the present study (6,9,30), and some interventions used more intense (70%–85% of maximum heart rate) exercise training workloads (29,36,38).

In contrast, resistance exercise training has the ability to increase fat-free mass, which is known to be the primary determinant of changes in RMR (34). This relationship has been demonstrated in several studies (6,16,25). However, because our findings did not show changes in fat-free mass, it is not surprising that RMR did not change as a function of exercise training. Other resistance training interventions have also failed to elicit changes in RMR despite significant increases in fat-free mass (4,35). However, the latter two studies evaluated programs where resistance exercise training was performed only 2 d·wk⁻¹, which may not have been sufficient to initiate changes in RMR.

In terms of other studies evaluating the effect of exercise on RMR in adults with type 2 diabetes, our findings are similar to those of Mourier et al. (23), who found that RMR was not affected by an 8-wk aerobic exercise training program. RMR was not a primary outcome of this study and data were not presented, so it is unclear if there were nonsignificant improvements. Conversely, the findings of Araiza et al. (1) demonstrated a significant effect on resting energy expenditure after a 6-wk walking program in adults (33–69 yr) with type 2 diabetes. The increase in resting energy expenditure was observed despite no change in body composition. The authors suggest this finding may be attributed to the measurement of resting energy expenditure during the post exercise period in which the rate of oxygen consumption is known to remain elevated. Nonetheless, low intensity exercise interventions, such as the Araiza et al. (1) intervention, have been shown to have very small postexercise oxygen consumption (2), and the minimum 12 h after the last exercise event would be sufficient time to dampen this effect. To the best of our knowledge, our study is the only one that has examined the effect of resistance exercise or combined exercise on RMR in individuals with type 2 diabetes.

The lack of change in RMR may be related to fundamental metabolic differences that exist in type 2 diabetes. Pathophysiological abnormalities that affect RMR in type 2 diabetes include elevated endogenous glucose output (37), futile free fatty acid cycling or stimulation of mitochondrial

uncoupling proteins (20,37), and insulin resistance (24). In fact, elevated blood glucose levels have been shown to independently predict higher RMR (12,15,22). Several studies have demonstrated increased RMR in people with poorly controlled type 2 diabetes compared with age- and sex-matched normal controls (10,15). This effect tends to be more of an issue in the early development of type 2 diabetes, (37), and when oral hypoglycemic drug therapy is introduced, resting energy expenditure is reduced by about 5% (10). Gougeon et al. (12) reported that RMR adjusted for body composition was increased when fasting glucose was >10 mmol·L⁻¹ compared with glucose values <10 mmol·L⁻¹. However, other studies that support this finding do not provide enough evidence to suggest a threshold blood glucose value above which RMR is increased (28). In the current study, our participants had relatively good blood glucose control at baseline, but because glycemia was further improved through our intervention especially in the combined training group (31), it is possible that the improved glycemia offset the potential increase in RMR. The explanation for this mechanism warrants further investigation.

Our exercise intervention also failed to demonstrate significant changes in body composition other than decreased within-group total body mass in the combined training group. This is in agreement with a meta-analysis by Boulé et al. (3), which showed that overall body weight was not affected by exercise training interventions. In general, aerobic exercise training interventions with people with type 2 diabetes of moderate intensity without concomitant dietary caloric restriction or behavioral modification tend to produce only minimal weight loss (11). Indeed, the changes in body weight in the exercise groups were small.

Although we expected the small weight reduction observed in the present study to reflect an increase in fat-free mass and decrease in fat mass, this was not found. It should however be noted that our main article (31) showed significant increases in mid thigh muscle cross-sectional area (by computed tomography) in both the aerobic and the resistance exercise training groups compared with the control group after the 6-month training program. The overall lack of change in fat mass and fat-free mass in the present substudy may have been limited by the use of bioelectrical impedance to estimate overall body composition. Bioelectrical impedance has well-documented limitations that include variability due to such factors as hydration and underestimation of percent body fat especially in the obese individual (5).

It is also important to note that our sample size, although larger than that of most other studies, was not sufficient to rule out small but potentially clinically significant changes of a few percent in body composition or RMR. Other potential limitations include the variability in RMR measurements. We did account for sex-related differences, but additional variability such as within-individual day-to-day variability (7), genetic differences, and/or the lack of metabolic uniformity of fat-free mass (17) are more difficult to account for.

In summary, we have demonstrated that aerobic and resistance exercise training as well as combined aerobic and resistance exercise training programs performed over 26 wk did not significantly change RMR in patients with type 2 diabetes.

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Conflict of interest: none.

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