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Exercise for Overweight Children and Diabetes Risk: In Reply

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In Reply:

Dr. Short inquires about the rationale for the intervention duration; whether fatness was adjusted; and the effects of pubertal development in our trial of exercise on diabetes risk in overweight children.

It was important that the stimulus be strong enough to affect outcomes; therefore we elicited vigorous (7.5 MET) physical activity frequently (every day after school) for about 3 months, modeled after prior effective interventions.^{1, 2} It was necessary to repeat the testing and intervention with additional children each semester for several years to power the study; this limited the intervention's duration. We agree with Short that the intervention could be feasible in other settings, and that the time course of improvements was beyond the study's scope. A briefer intervention period would probably result in improved insulin resistance,³ but would result in less fat loss due to lower cumulative energy expenditure. Given the similar effects of 20 min and 40 min of daily exercise, a longer program might result in greater loss of fat with similar effects on fitness and diabetes risk.

Given the longer time course of exercise-induced changes in adiposity than insulin resistance, improved insulin resistance is unlikely to be due to reduced fatness. As specified in the article, neither BMI nor percent body fat were adjusted. Children were randomized to the exercise programs or control condition, and differential effects on outcomes should be attributed to this factor. The children's fatness was reduced because of the exercise programs offered to those groups. Mediation analysis can suggest how those changes may have occurred, based on contemporaneous, collinear data with varying precision. Mediation analyses on these data will be reported in a subsequent publication.

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As expected, puberty (i.e., thelarche/gonadarche stage) was a predictor in the insulin resistance models (insulin area under the curve, $P < .001$; fasting insulin, $P < .001$; Matsuda index, $P = .002$). The adrenarche variable was not significant in any model. We now report the interaction of thelarche/gonadarche stage (classified as Tanner I, II, or III and above) with the group by time interaction in the insulin resistance models, adjusting for cohort, sex, and race. An interaction was detected only for fasting insulin, such that differential change from baseline to post-test between exercise groups and control was detected only in stage III or above. In more developed children, similar decreases in fasting insulin were seen in the low-dose (adjusted mean difference, -18.2 [95% CI, -25.8 to -10.6] $\mu\text{U/mL}$) and high-dose (-15.8 [95% CI, -23.0 to -8.5] $\mu\text{U/mL}$) exercise groups (each $P < .001$ vs the control group). Thus, more developed children might be more responsive to exercise. However, of 3 insulin resistance indicators, only 1 showed evidence of pubertal moderation. These exploratory analyses provide scant support for the hypothesis that pubertal stage affects the benefits of aerobic training in obese children. Conclusions should be tempered by the exploratory nature of these additional analyses.

References

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