

COMMENTS AND RESPONSES

Effects of Exercise Training Intensity on Pancreatic β -Cell Function

Response to Slentz et al.

I read with interest the recent article by Slentz et al. (1). They demonstrated that in middle-aged overweight/obese individuals who were moderately dyslipidemic, a moderate-intensity exercise program (40–55% of aerobic capacity or $\dot{V}O_{2\text{peak}}$) of 8 months long (1,220 kcal/week) improved β -cell function (defined by an increase in the disposition index) three times more than with 1,230–2,020 kcal/week of vigorous intensity exercise (65–80% of $\dot{V}O_{2\text{peak}}$) (1). The authors concluded that moderate-intensity exercise improves β -cell function to a better extent compared with vigorous-intensity exercise.

Their article overlooks the importance of vigorous exercise in clinical studies. First, their article minimized the important findings regarding how vigorous exercise at 2,020 kcal/week for 8 months reduced visceral fat content by 7%, whereas the moderate-intensity exercise group of 1,220 kcal/week did not change visceral fat content. Visceral fat is associated with cardiovascular disease risk (2), and their finding importantly demonstrates that vigorous exercise reduces visceral fat content.

Second, their article downplayed that 2,020 kcal/week of vigorous exercise improved $\dot{V}O_{2\text{peak}}$ 2.5 times more than the 1,220 kcal/week group performing moderate-intensity exercise (1). $\dot{V}O_{2\text{peak}}$ is a

powerful predictor of mortality, more than other established risk factors for cardiovascular disease (3). For every one MET of increased $\dot{V}O_{2\text{peak}}$ (one MET equals the resting metabolic rate, assumed to be $3.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), there is a 12% improvement in survival regardless of whether a person has cardiovascular disease or not (3). Notwithstanding, there is an ~5% decrease in health care cost per one MET increase in $\dot{V}O_{2\text{peak}}$ independent of age and other clinical factors (4).

The amount of physical activity performed per week is correlated to $\dot{V}O_{2\text{peak}}$. Johnson et al. (5) studied the effects of three different 6-month exercise programs on components of the metabolic syndrome: low amount/moderate intensity (jogging 19 km/week at 40–55% of $\dot{V}O_{2\text{peak}}$), low amount/high intensity (jogging 19 km/week at 65–80% of $\dot{V}O_{2\text{peak}}$), or high amount/vigorous intensity (jogging 32 km/week at 65–80% $\dot{V}O_{2\text{peak}}$). The high amount/vigorous intensity group improved the most number of metabolic variables compared with the other two groups and a control group (5). There is a dose response of energy expenditure in reducing the risk factors for the metabolic syndrome (5). This is logical because insulin resistance is negatively related to caloric expenditure from a single bout of exercise.

Vigorous-intensity exercise is necessary to improve $\dot{V}O_{2\text{peak}}$. Increasing weekly energy expenditure to $\geq 2,020$ kcal/week is needed to reduce visceral fat content. These are two critical findings that are arguably more important than the exercise effects on pancreatic β -cell function. The fact that β -cell function was not improved to the same extent as with a moderate-intensity exercise program of low weekly energy expenditure minimizes Slentz et al.'s valuable findings of how vigorous exercise reduces cardiometabolic risk, mortality, and health care costs.

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