Effect of the volume and intensity of exercise training on insulin sensitivity

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Houmard, Joseph A., Charles J. Tanner, Cris A. Slentz, Brian D. Duscha, Jennifer S. McCartney, and William E. Kraus. Effect of the volume and intensity of exercise training on insulin sensitivity. J Appl Physiol 96: 101–106, 2004. First published September 12, 2003; 10.1152/japplphysiol.00707.2003.—Physical activity enhances insulin action in obese/overweight individuals. However, the exercise prescription required for the optimal enhancement is not known. The purpose of this study was to test the hypothesis that exercise training consisting of vigorous-intensity activity would enhance insulin sensitivity more substantially than moderate-intensity activity. Sedentary, overweight/obese subjects (n = 154) were randomly assigned to either control or an exercise group for 6 mo: 1) low-volume/moderate-intensity group (~12 miles walking/wk at 40–55% peak O2 consumption (Vo2peak)), 2) low-volume/high-intensity group (~12 miles jogging/wk at 65–80% Vo2peak), and 3) high-volume/high-intensity group (~20 miles jogging/wk at 65–80% Vo2peak). Training volume (miles/wk) was achieved by exercising ~115 min/wk (low-volume/high-intensity group) or ~170 min/wk (low-volume/moderate-intensity and high-volume/high-intensity groups). Insulin action was measured with an insulin sensitivity index (S1) from an intravenous glucose tolerance test. In the control group, there was a decrement (P < 0.05) in S1. In contrast, all the exercise groups significantly (P < 0.05) increased S1; the relative increment in the low-volume/moderate-intensity and high-volume/high-intensity groups (~85%) were greater than in the low-volume/high-intensity group (~40%). In conclusion, physical activity encompassing a wide range of intensity and volume minimizes the insulin resistance that develops with a sedentary lifestyle. However, an exercise prescription that incorporated ~170 min of exercise/wk improved insulin sensitivity more substantially than a program utilizing ~115 min of exercise/wk, regardless of exercise intensity and volume. Total exercise duration should thus be considered when designing training programs with the intent of improving insulin action.

MATERIALS AND METHODS

Study design. These findings are part of the Studies of Targeted Risk Reduction Interventions Through Defined Exercise (13, 14). Subjects were stratified for race, age, and gender and were randomly assigned to one of three exercise training groups or a nonexercising control group for 6 mo. The research protocol was approved by the relevant institutional review boards, and all subjects provided written, informed consent. Initially, 228 subjects were randomized; data are presented from the 154 subjects who completed the training/testing (randomized/completed: control group, 48/40; low-volume/moderate-intensity group, 67/41; low-volume/high-intensity group, 41/30; high-volume/high-intensity group, 72/43).

Subjects. Inclusion criteria were sedentary (not participating in a regular exercise program); overweight/obese [body mass index (BMI), >25 to ≤35 kg/m2]; dyslipidemia (either an LDL cholesterol concentration of 130–190 mg/dl or an HDL cholesterol concentration below 40 mg/dl for men or below 45 mg/dl for women); and postmenopausal status. Exclusion criteria included no medications that could alter carbohydrate metabolism and evidence for diabetes, orthopedic conditions prohibiting exercise, hypertension, and heart disease.

Exercise training. The exercise training programs were as follows (volume, intensity): 1) low-volume/moderate-intensity group = ca-

INSULIN RESISTANCE APPEARS to be an integral component in the development of cardiovascular disease (CVD) (5). Regular endurance-oriented exercise training enhances insulin action (8, 10); accordingly, physically active individuals are at a lower risk for CVD (4, 22, 23). It is not evident, however, whether exercise training programs differing in exercise intensity [percentage of peak O2 consumption (Vo2peak)] also differ in their relative ability to alter insulin action. Some data suggest that only vigorous, intense (i.e., ≥70% Vo2peak) exercise can enhance insulin sensitivity (12, 24), whereas other findings indicate that insulin sensitivity can improve with mild-to-moderate-intensity physical activity (18, 21). Similar discrepancies are evident when the effect of physical activity on other indexes of health are examined. Epidemiological data suggest that vigorous-intensity physical activity is needed for a longer life span (17) and to reduce risk for CVD (4, 22, 23). However, other studies suggest that simply increasing energy expenditure, whether from light- or moderate-intensity exercise, reduces the risk for CVD (4, 23, 26).

These findings indicate that a discrete exercise prescription for optimally improving insulin action is lacking. The purpose of the present study was to utilize a prospective, randomized design examining a relatively large number of subjects to directly compare exercise training programs of differing intensities and weekly training volumes (energy expenditure per week on exercise) in an attempt to discern an effective exercise prescription for improving insulin action. It was our hypothesis that vigorous-intensity physical activity would improve insulin action to a greater degree than moderate-intensity exercise training. To test this hypothesis, we compared changes in insulin action in three exercise training programs differing in exercise intensity and/or volume in individuals at risk for CVD (obese/overweight, sedentary, middle-aged).
loric equivalent of ~12 miles/wk, 1,200 kcal/wk at 40–55% \( \dot{V}O_2_{\text{peak}} \); 2) low-volume/high-intensity group = caloric equivalent of ~12 miles/wk, 1,200 kcal/wk at 65–80% \( \dot{V}O_2_{\text{peak}} \); and 3) high-volume/high-intensity group = caloric equivalent of ~20 miles/wk, 2,000 kcal/wk at 65–80% \( \dot{V}O_2_{\text{peak}} \). The specific training regimens were chosen to compare different volumes/doses of exercise (1,200 and 2,000 kcal/wk) in groups exposed to the same intensities of training (65–80% \( \dot{V}O_2_{\text{peak}} \)) and different intensities (40–55 and 65–80% \( \dot{V}O_2_{\text{peak}} \)) in groups with the same exercise volume (1,200 kcal/wk). The exercise volumes (1,200 and 2,000 kcal/wk) were consistent with recommendations of the Surgeon General’s report (4) and the Harvard Alumni Study (17). The 65–80% \( \dot{V}O_2_{\text{peak}} \) level was chosen because this is the traditional intensity of exercise prescribed for cardiovascular fitness benefit (1). The lower exercise training intensity (40–55% \( \dot{V}O_2_{\text{peak}} \)) was chosen because it approximates brisk walking or moderate-intensity exercise levels advised in current health guidelines (1, 4, 20, 23). For the high-volume/high-intensity group, the goal was to expend 23 kcal/kg of body mass \(^{-1}\text{wk}^{-1}\) on exercise, which is equivalent to a volume of ~20 miles/wk of walking or jogging for a 90-kg person (range: 19.2–20.6 miles/wk for a 70- to 110-kg person) (13, 14). The exercise prescription was 14 kcal/kg \(^{-1}\text{wk}^{-1}\) for the two low-volume groups, which is the caloric equivalent of walking/jogging 12 miles/wk. Although the volume of exercise is expressed in terms of walking or jogging, the actual exercise modes included cycle ergometer, treadmill, and elliptical trainers to enhance variety and adherence. Once the exercise volume (kcal·kg \(^{-1}\text{wk}^{-1}\)) was calculated, subjects selected, with the assistance of an exercise physiologist, an appropriate exercise frequency and duration to achieve their weekly dose. All exercise sessions were verified by direct supervision and/or use of a heart rate monitor that provided recorded data (Polar Electro, Woodbury, NY). To minimize musculoskeletal injury there was an initial ramp period of 2–3 mo followed by 6 mo at the appropriate exercise prescription. Subjects were counseled to maintain baseline body weight and not reduce dietary intake. Body mass was measured weekly, and the subject was notified to increase energy intake if body mass declined by >3%.

Insulin sensitivity. Insulin sensitivity was determined with a 3-h intravenous glucose tolerance test (IVGTT) (2), as described previously (11). The IVGTT was performed ~24 h after the final exercise bout. After fasting samples were obtained, glucose (50%) was injected into a catheter placed in an antecubital vein at a dose of 0.3 g/kg body mass. Insulin, at a dose of 0.025 U/kg body mass, was injected at minute 20. Blood samples were obtained at minutes 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 25, 30, 40, 50, 60, 70, 80, 90, 100, 120, 140, 160, and 180 and the samples were centrifuged, and plasma frozen at ~80°C for the subsequent determination of insulin and glucose. Insulin was determined with immunoassay (Access Immunoassay System, Beckman Coulter, Fullerton, CA) and glucose was determined with an oxidation reaction (YSI model 2300 Stat Plus, Yellow Springs Instruments, Yellow Springs, OH). An insulin sensitivity index (SI) was calculated by using the minimal model (2); a higher SI indicates enhanced insulin sensitivity. Acute-phase insulin secretion (AIRG) and glucose effectiveness (S\(e\)) were also determined from the IVGTT (2).

Statistics. Variables were compared with a group \(\times\) time repeated-measures ANOVA and post hoc testing performed with contrast-contrast analyses. Relative changes (%) (after training/before training) were compared between groups with ANOVA and analyses of covariance (ANCOVA); post hoc analyses was performed with a Fisher’s least squares differences test. Correlations (Pearson product) were performed between relative changes for selected variables. Statistical significance was denoted at the \(P \leq 0.05\) level. Data are presented as means ± SE. There were no differences in insulin action according to gender; data for men and women were thus combined.

RESULTS

Subjects. Approximately 30% (32%) of the subjects did not complete the 6 mo of training and/or elect to perform the posttraining testing, consistent with our a priori estimation of a 30% dropout rate from a review of other similar studies (14). Characteristics for subjects that completed the exercise training (\(n = 154\)) are presented in Table 1. There were no significant differences between the groups in age, stature, or BMI (Table 1). Each of the groups consisted of ~50% women and 14–30% ethnic minorities (Table 1).

Exercise training. Characteristics of the exercise programs are presented in Table 2. Adherence was calculated as a percentage by dividing the time spent exercising by the time needed to meet the defined exercise prescription. The rate of adherence was significantly (\(P < 0.05\)) lower in the high-volume/high-intensity group compared with the low-volume/high-intensity group. As expected, to achieve the calculated exercise volumes, the low-volume/moderate-intensity and high-volume/high-intensity groups exercised for significantly greater durations per week than the low-volume/high-intensity group. The exercise frequency chosen to attain the assigned exercise dosage was significantly (\(P < 0.05\)) lower in the low-volume/high-intensity group.

Anthropometrics. As presented in Table 3, despite recommendations to the contrary, there were slight reductions in body mass in the exercise groups. The control group increased body mass (~1 kg), whereas the low-volume/moderate-inten-

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Table 1. Baseline characteristics of the subjects who completed posttraining testing

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control ((n = 40))</th>
<th>Low Volume/Moderate-Intensity ((n = 41))</th>
<th>Low-Volume/High-Intensity ((n = 30))</th>
<th>High-Volume/High-Intensity ((n = 43))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>51.4±1.2</td>
<td>53.1±0.9</td>
<td>52.6±1.3</td>
<td>51.4±0.9</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.71±0.02</td>
<td>1.73±0.02</td>
<td>1.70±0.01</td>
<td>1.72±0.01</td>
</tr>
<tr>
<td>BMI, kg/m(^2)</td>
<td>29.8±0.6</td>
<td>30.0±0.5</td>
<td>29.9±0.6</td>
<td>29.2±0.4</td>
</tr>
<tr>
<td>Race, (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>28(70)</td>
<td>34(83)</td>
<td>23(77)</td>
<td>37(86)</td>
</tr>
<tr>
<td>Black</td>
<td>10(25)</td>
<td>7(17)</td>
<td>6(20)</td>
<td>5(12)</td>
</tr>
<tr>
<td>Other</td>
<td>2(5)</td>
<td>0</td>
<td>1(3)</td>
<td>1(2)</td>
</tr>
<tr>
<td>Gender, (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>17(43)</td>
<td>17(41)</td>
<td>15(50)</td>
<td>20(47)</td>
</tr>
<tr>
<td>Male</td>
<td>23(57)</td>
<td>24(59)</td>
<td>15(50)</td>
<td>23(53)</td>
</tr>
</tbody>
</table>

Values are means ± SE; \(n\), no. of subjects. Nos. in parentheses are percentages for each group. BMI, body mass index.
Adherence % 86.0

Prescribed volume, *miles/wk 12 12 20

0.0001) group

of the change in S I , relative changes (after training/before increased significantly (P < 0.01) in the control group and decreased in the low-volume/moderate-intensity and high-volume/high-intensity groups. There were no significant changes over time in fasting plasma glucose.

Insulin sensitivity. Venous access could not be obtained during the entire IVGTT in all of the subjects. IVGTT data are as follows: control, n = 32; low-volume/moderate-intensity, n = 37; low-volume/high-intensity, n = 28; high-volume/high-intensity, n = 36. As presented in Table 3, there were no initial differences between groups in S I . There was a significant (P < 0.0001) group × time interaction; post hoc comparison indicated that S I in the control group significantly (P < 0.05) declined over time, whereas S I in each of the exercise groups increased significantly (P < 0.001). To compare the magnitude of the change in S I , relative changes (after training/before training) between the groups were compared (Fig. 1). There was a significant interaction, with the relative decline of S I in the control group (−4.0 ± 7.0%) being significantly (P ≤ 0.05) different from the increases observed in all the exercise groups (low-volume/moderate-intensity group 88.0 ± 18.7% increase; low-volume/high-intensity group, 37.6 ± 8.9% increase; high-volume/high-intensity group, 82.7 ± 15.3% increase). The relative increases in S I in both the low-volume/ moderate-intensity and the high-volume/high-intensity groups were significantly (P < 0.05) greater than the relative increase in the low-volume/high-intensity group.

When adjusting for exercise frequency, adherence (Table 2), and change in body mass (Table 3) with ANCOVA, there was still a greater relative increase (P < 0.05) in S I in the low-volume/moderate-intensity and high-volume/high-intensity compared with the low-volume/high-intensity group. Results similar to Fig. 1 were also obtained when the change in body mass was made equivalent across all the exercise groups, by deleting subjects with >6% loss in body mass, and when subjects with low adherence (<50%) were excluded from data analyses. In the exercise groups, there were no statistically significant relationships between the changes in BMI, body mass, and S I . There was a significant negative relationship (r = −0.36, P < 0.01) between initial S I and the change in S I with training. However, ANCOVA using initial S I as the covariant produced results identical to Fig. 1.

Insulin secretion (AIRG) and glucose effectiveness (S G ) did not change significantly with exercise training (Table 3).

**DISCUSSION**

The main finding of the present study was that exercise programs consisting of a training duration of ~170 min/wk, regardless of variations in exercise intensity (range, 40–80% $V_{O2\text{peak}}$) and weekly training volume (12–20 miles/wk) (Table 2), improved insulin sensitivity to a similar degree (Fig. 1). Our findings (Table 3, Fig. 1) thus suggest that, within the exercise training paradigms used in the present study, exercise duration is one of the primary factors that controls the response of insulin action to exercise training. The conclusion that weekly

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Low-Volume/ Moderate-Intensity</th>
<th>Low-Volume/ High-Intensity</th>
<th>High-Volume/ High-Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin action</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$S_a$, mU/l·min$^{-1}$</td>
<td>3.3 ± 0.3</td>
<td>2.9 ± 0.3*</td>
<td>2.6 ± 0.3</td>
<td>4.3 ± 0.6*</td>
</tr>
<tr>
<td>AIRG, mU/mM</td>
<td>4.9 ± 0.9</td>
<td>5.4 ± 0.9</td>
<td>5.0 ± 0.6</td>
<td>5.0 ± 0.6</td>
</tr>
<tr>
<td>$S_c$, 10²/min</td>
<td>2.2 ± 0.2</td>
<td>1.9 ± 0.1</td>
<td>1.9 ± 0.2</td>
<td>2.1 ± 0.2</td>
</tr>
<tr>
<td>Insulin, mU/l</td>
<td>7.6 ± 0.6</td>
<td>8.7 ± 0.7*</td>
<td>11.3 ± 1.4</td>
<td>8.1 ± 1.0*</td>
</tr>
<tr>
<td>Glucose, mg/dl</td>
<td>90.5 ± 1.8</td>
<td>93.6 ± 1.6</td>
<td>94.0 ± 1.3</td>
<td>93.0 ± 1.5</td>
</tr>
<tr>
<td>Body mass</td>
<td>29.8 ± 0.6</td>
<td>30.1 ± 0.6*</td>
<td>30.0 ± 0.5</td>
<td>29.7 ± 0.5*</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>87.2 ± 2.5</td>
<td>88.1 ± 2.6*</td>
<td>90.4 ± 2.4</td>
<td>89.6 ± 2.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. $S_a$, insulin sensitivity index from the intravenous glucose tolerance test (IVGTT); AIRG, insulin secretion from the IVGTT; $S_c$, glucose effectiveness from the IVGTT; insulin and glucose, fasting insulin and glucose. *Significantly different from before training within that experimental group, P ≤ 0.05.
exercise duration appeared to be an important variable influencing changes in insulin sensitivity was reached by integrating several pieces of information. First, weekly exercise frequency (~3 sessions/wk) and volume (~12 miles/wk) were similar between the low-volume/moderate-intensity and low-volume/high-intensity groups (Table 2). However, SI was enhanced to a greater extent (Fig. 1) with the exercise training encompassing the longer weekly training duration. Second, both of the exercise training groups with longest weekly training durations (low-volume/moderate-intensity and high-volume/high-intensity groups, ~170 min/wk) enhanced insulin sensitivity to a similar degree (Fig. 1), independent of exercise intensity and weekly training volume (Table 2). Third, the increment in insulin action in the longer duration vs. the shorter duration groups remained evident after statistically adjusting for differences in the change in body mass (Table 3) and exercise adherence and frequency (Table 2) between the groups (see RESULTS). These findings suggest that exercise duration should be considered when designing training programs with the intent of improving insulin action and reducing the risk for CVD.

It is important to consider that the subjects examined were middle aged, overweight/obese, previously sedentary and that they exhibited evidence of dyslipidemia. The improvements in insulin sensitivity observed in all of the exercise programs (Fig. 1, Table 3) are thus functionally applicable in terms of recommending physical activity to the sedentary overweight individuals that comprise a large segment of the population of the United States (23). In contrast, there was a decrement in insulin action in subjects that remained sedentary (Fig. 1, Table 3). In a cross-sectional study, our laboratory (11) reported that the yearly decline in SI over the lifespan approximated 0.1 (mU·l⁻¹·min⁻¹); the reduction in the present study (Table 3) was more pronounced. However, we assumed a linear relationship in our laboratory’s cross-sectional study (11), whereas insulin resistance has been reported to develop more profoundly during middle age in association with weight gain and inactivity (3, 5). It is thus not clear whether the rate of decline in our control group is indicative of what truly occurs in middle-aged, overweight individuals leading a sedentary lifestyle. Despite this caveat, in addition to the exercise duration effect discussed above, the present data also indicate that 1) a lack of regular exercise for as little as 6 mo can result in a significant increase in insulin resistance and 2) regularly performed physical activity encompassing a relatively wide range of intensity and dosage can prevent the worsening of insulin resistance that develops with a sedentary lifestyle. It should also be considered that there was a relatively high dropout rate in the present study (see RESULTS); such a lack of adherence and/or cessation of exercise training is common in long-term exercise training studies and/or when recommending a physically active lifestyle in general (4, 14, 22, 23). The present data must thus be interpreted within the context that, although exercise training is an effective method for improving/main- taining insulin action, it may not be a clinical intervention with a high rate of adherence in some individuals.

Information directly comparing the impact of exercise training programs differing in intensity/duration/volume with minimal weight loss on insulin action is sparse. Oshida et al. (21) reported that training 30–40 min/day, 4 days/wk, at 40% maximal O₂ consumption improved insulin sensitivity (glucose clamp, glucose metabolism-to-plasma insulin index) by approximately threefold despite no change in body mass or aerobic capacity. In a large cross-sectional study that utilized the minimal model technique to measure insulin sensitivity and questionnaires for physical activity, Mayer-Davis et al. (18) concluded that increased participation in nonvigorous as well as vigorous physical activity was associated with a significantly higher SI.

On the contrary, findings from other experiments suggest that low- to moderate-intensity training does not improve insulin sensitivity. Seals et al. (24) examined the impact of 6 mo of low/moderate-intensity training (walking, 3–4 times/wk for ~30 min/walk) that was subsequently followed by 6 mo of high-intensity training (jogging 30–45 min/day, 3–4 days/wk) on oral glucose tolerance test (OGTT) responses; the OGTT was performed ~14 h after the final exercise bout. With the low-intensity training, there were no changes indicative of enhanced insulin sensitivity. Conversely, after the high-intensity training, insulin area under the curve during the OGTT was reduced by ~30%. Similar findings were obtained by Kang et al. (12), who studied obese men before and after 7 consecutive days of exercise training at either 50% V₂O₂ peak (70 min/day) or 70% V₂O₂ peak (50 min/day). There were no changes in insulin sensitivity with the moderate-intensity training (OGTT, 24 h after the final training bout), but there was a significant improvement of ~30% after training at 70% V₂O₂ peak.

A possible explanation for our observation of an improvement in insulin action with relatively moderate-intensity exercise (low-volume/moderate-intensity group) vs. no changes in other studies (12, 24) may involve the methods used to determine insulin sensitivity. Studies that did not report improved insulin action with low- to moderate-intensity training (12, 24) utilized an OGTT. The OGTT is not designed to quantitatively measure insulin sensitivity (25). In contrast, studies that reported improvements with mild- to moderate-intensity training utilized either an IVGTT (present study; Ref. 18) or euglyce- mic clamp (21), procedures that are specifically designed to measure insulin sensitivity (25). Our data thus agree with those of others (18, 21) that relatively moderate-intensity physical activity can enhance insulin sensitivity, which is a relevant finding in relation to clinical recommendations for intervention/prevention. The present study, however, supplies addi-
tional information (18, 20) in that we examined distinct exercise programs encompassed by current recommendations (1, 21–23) where exercise intensity, frequency, volume, and duration were directly measured and changes in insulin action directly compared in a relatively large number of subjects at risk for developing CVD.

Insulin sensitivity can be improved by exercise, weight loss, or weight loss in conjunction with exercise training (6, 16). To examine the effect of exercise training alone, we attempted to minimize weight changes by monitoring body mass on a weekly basis. Despite these efforts, there were slight declines in body mass in both the low-volume/moderate-intensity (−0.8 kg) and high-volume/high-intensity (−1.8 kg) groups but not the low-volume/high-intensity group (Table 3). The lack of a reduction in body mass may explain why the low-volume/high-intensity exercise group did not exhibit the same degree of improvement in insulin action (Fig. 1). The differences in insulin action according to exercise duration persisted, however, after statistically adjusting for changes in body mass by performing ANCOVA and by equalizing weight loss across the exercise groups by omitting subjects with >6% weight loss (see RESULTS). Also, within the exercise groups, there was no relationship between changes in insulin sensitivity and body mass. These data suggest that the minimal weight loss (1−2 kg) seen in the longer duration exercise training groups in the present study were not the primary factor responsible for the observed improvement in insulin action.

Although exercise training has been reported to reduce glucose-stimulated insulin secretion (3, 7, 19), we observed no significant mean alterations in AIGR in the present study (Table 3). A possible explanation may involve factors inherent with the study of obese individuals. Krotkiewski et al. (15) reported that obese individuals with high or normal insulin secretion decreased glucose-stimulated insulin secretion after physical training; however, obese subjects with low initial secretion levels increased insulin secretion with training. In the present study, we had a wide range of AIGR (Table 3) and a negative relationship (r = −0.22, P < 0.05) between initial AIGR and the relative change (after training/before training) in AIGR; this variation may have minimized mean changes between the groups with exercise training. Dietary variation may also play a role as Helge (9) reported that both insulin action (homeostasis model assessment) and insulin secretion did not change with physical training in obese individuals consuming a fat-rich diet, as opposed to an improvement in insulin action in obese individuals consuming a carbohydrate-rich diet. These findings (9, 15) indicate that the response of glucose-stimulated insulin secretion to physical activity can be influenced by several variables, in contrast to the rather consistent observation of an increase in insulin action/sensitivity with physical activity (Fig. 1; Refs. 3, 7, 9, 15).

In summary, physical activity encompassing a wide range of intensity and volume prevents increases in the insulin resistance that develops progressively with a sedentary lifestyle. However, an exercise prescription that incorporated an exercise duration of ~170 min of exercise/wk with a frequency of three to four sessions per week improved insulin sensitivity significantly more than a program utilizing ~115 min of exercise/wk and a frequency of three sessions per week, regardless of exercise intensity and volume. Exercise duration should thus be considered when designing training programs with the intent of improving insulin sensitivity.

GRANTS

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