Insulin action on heart and skeletal muscle glucose uptake in weight lifters and endurance athletes

TEEMU O. TAKALA,1,2 PIRJO NUUTILA,1 JUHANI KNUUTI,2 MATTI LUOTOLAHTI,3 AND HANNELE YKI-JÄRVINEN4

Departments of 1Medicine and 3Clinical Physiology, 2Turku Positron Emission Tomography Centre, University of Turku, FIN-20521 Turku; and 4Division of Endocrinology and Diabetology, Department of Medicine, University of Helsinki, FIN-00029 HYKS Helsinki, Finland

Takala, Teemu O., Pirjo Nuutila, Juhani Knuutila, Matti Luotolahti, and Hannele Yki-Järvinen. Insulin action on heart and skeletal muscle glucose uptake in weight lifters and endurance athletes. Am. J. Physiol. 276 (Endocrinol. Metab. 39): E706–E711, 1999.—There are no studies comparing myocardial metabolism between endurance- and resistance-trained athletes. We used 2-deoxy-2-[18F]fluoro-o-glucose and positron emission tomography combined with the euglycemic hyperinsulinemic clamp technique to compare the ability of insulin to stimulate myocardial, skeletal muscle, and whole body glucose uptake between weight lifters (n = 8), endurance athletes (n = 8), and sedentary men (n = 9). Maximal aerobic power (ml·kg\(^{-1}\)·min\(^{-1}\)) was higher in the endurance athletes (71 ± 2, P < 0.001) than the weight lifters (42 ± 2) and the sedentary men (42 ± 2). Skeletal muscle glucose uptake (µmol·kg muscle\(^{-1}\)·min\(^{-1}\)) was enhanced in the endurance athletes (125 ± 16, P < 0.01) but was similar in weight lifters (59 ± 12) and sedentary (63 ± 7) men. The rate of glucose uptake per unit mass of myocardium (µmol·kg·min\(^{-1}\)) was similarly decreased in endurance athletes (544 ± 50) and weight lifters (651 ± 45) compared with sedentary men (1,041 ± 78, P < 0.001 vs. endurance athletes and weight lifters). Both groups of athletes had increased left ventricular mass. Consequently, total left ventricular glucose uptake was comparable in all groups. These data demonstrate that aerobic but not resistance training is associated with enhanced insulin sensitivity in skeletal muscle. Despite this, cardiac changes are remarkably similar in weight lifters and endurance athletes and are characterized by an increase in left ventricular mass and diminished insulin-stimulated glucose uptake per heart mass.

BOTH AEROBIC TRAINING AND RESISTANCE TRAINING induce structural changes in the heart, such as an increase in left ventricular mass. With aerobic training, this has been suggested to occur via an increase in venous return (preload), which causes eccentric hypertrophy, and an increase in left ventricular diameter relative to left ventricular wall thickness (1, 14, 17). In contrast, resistance training has been suggested to induce, perhaps via repeated increases in systolic blood pressure (afterload), concentric hypertrophy, which is characterized by thickening of septal and left ventricular walls relative to the left ventricular diastolic diameter (6, 7, 9, 14). These findings, combined with occasional reports of large blood pressure responses to heavy resistance activity, have even raised some concerns about whether resistance training is safe for individuals with hypertension or other cardiovascular diseases (14).

We have previously demonstrated that patients with essential hypertension and endurance-trained athletes exhibit opposite alterations in myocardial glucose uptake under hyperinsulinemic conditions, when glucose is the predominant fuel for the myocardium. In patients with essential hypertension, myocardial glucose uptake, per unit mass of myocardium, is increased in proportion to cardiac work (23), whereas myocardial glucose uptake per unit mass of myocardium is decreased in endurance athletes (18). In the latter group, the lower glucose uptake per unit mass was interpreted to reflect increased use of an alternative fuel or lower oxygen consumption per unit mass of the enlarged left ventricle (18). It is unknown whether resistance training alters heart glucose metabolism similarly in endurance- and resistance-trained athletes. In skeletal muscle, aerobically trained athletes have enhanced insulin sensitivity of glucose uptake, whereas insulin sensitivity in weight lifters is similar to that in sedentary subjects (36). The latter conclusion was based on calculation of muscle glucose uptake with whole body glucose uptake and body composition data (36), rather than on direct measurement. In the present study we compared insulin-stimulated rates of glucose uptake, measured directly in the heart and in skeletal muscle in aerobically trained and resistance-trained subjects using positron emission tomography (PET) and 2-deoxy-2-[18F]fluoro-o-glucose ([18F]FDG). Heart size and dimensions were determined with echocardiography. Our results reveal a clear difference in skeletal muscle insulin sensitivity but a similarly reduced rate of glucose uptake per unit mass of myocardium in both groups of athletes compared with sedentary men.

METHODS

Subjects. Eight male endurance athletes (runners, cross-country skiers, triathletes), eight weight lifters (power lifters), and nine sedentary healthy men participated in the study (Table 1). All athletes were competing at the national level and trained 4–6 times per week. The control group did not exercise regularly. The subjects were healthy as judged by history, physical examination, and routine laboratory tests and were not taking any medications. The nature, purpose, and potential risks of the study were explained to all subjects before written informed consent was obtained. The experimental protocol was reviewed and approved by the Commission of Ethics of Turku University and Turku University Central Hospital.

Study design. All subjects were studied after a 10- to 12-h overnight fast. The subjects had not exercised for 2 days before the study. Two catheters were inserted, one in an antecubital vein for infusion of glucose and insulin and...
Table 1. Characteristics of the study groups

<table>
<thead>
<tr>
<th></th>
<th>Endurance Athletes</th>
<th>Weight Lifters</th>
<th>Sedentary Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>8</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Age, yr</td>
<td>28 ± 1</td>
<td>29 ± 2</td>
<td>29 ± 1</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>76 ± 2</td>
<td>86 ± 5</td>
<td>78 ± 3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>186 ± 1</td>
<td>178 ± 3</td>
<td>182 ± 2</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>22.0 ± 0.3</td>
<td>27.0 ± 0.9</td>
<td>23.4 ± 0.7</td>
</tr>
<tr>
<td>Percent fat, %</td>
<td>9 ± 1*</td>
<td>13 ± 1*</td>
<td>17 ± 1</td>
</tr>
<tr>
<td>V₀₂max, ml·kg⁻¹·min⁻¹</td>
<td>71 ± 2*</td>
<td>42 ± 2</td>
<td>42 ± 2</td>
</tr>
<tr>
<td>Body surface area, m²</td>
<td>1.99 ± 0.03</td>
<td>2.05 ± 0.07</td>
<td>1.98 ± 0.05</td>
</tr>
<tr>
<td>Fasting plasma glucose, mmol/l</td>
<td>5.3 ± 0.1</td>
<td>5.4 ± 0.1</td>
<td>5.2 ± 0.1</td>
</tr>
<tr>
<td>Fasting serum insulin, mU/l</td>
<td>5 ± 1</td>
<td>6 ± 1</td>
<td>7 ± 1</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. *P < 0.05 compared with other groups.

injection of [¹⁸F]FDG and another retrogradely in a heated hand for sampling of arterialized venous blood. Whole body, skeletal, and heart muscle glucose uptake rates were thereafter determined by combining the euglycemic hyperinsulinemic clamp technique with [¹⁸F]FDG and PET, as we will describe in detail.

Whole body glucose uptake. Whole body glucose uptake was quantitated by use of the euglycemic hyperinsulinemic clamp technique (2). Serum insulin was increased for 120 min by a primed-continuous (1 mU·kg⁻¹·min⁻¹) infusion of insulin (Velosulin, Novo Nordisk, Bagsvaerd, Denmark). Normoglycemia was maintained using a variable rate infusion of 20% glucose based on plasma glucose measurements, which were performed every 5 min from arterialized venous blood. Whole body glucose uptake was calculated from the glucose infusion rate during the period of PET scanning (60–120 min). Serum free insulin, plasma lactate, and serum free fatty acid concentrations were measured every 30 min during hyperinsulinemia.

Heart and skeletal muscle glucose uptake. [¹⁸F]FDG was synthesized with an automatic apparatus (10). The [¹⁸F]FDG study was performed as previously described (18). [¹⁸F]FDG (6–7 mCi or 220–260 MBq) was injected intravenously over 2 min. A dynamic scan of the thoracic region was performed for 40 min and continued with a dynamic scan of the femoral region for 25 min. Plasma radioactivity was measured with an automatic gamma counter (Wizard 1480 3 γ-counter, Wallac, Turku, Finland). An eight-ring ECAT 931/08 tomograph (Siemens/CTI, Knoxville, TN) with an axial resolution of 6.7 mm and in-plane resolution of 6.5 mm was used. Before the emission scan, a transmission scan was performed. All data were corrected for deadtime, decay, and measured photon attenuation. Dynamic FDG scans were reconstructed into a 128 × 128 matrix using a Hann filter with a cut-off frequency of 0.5. Final in-plane resolution of the reconstructed images was 8 mm.

For determination of myocardial glucose uptake, three (lateral, anterior, septal) regions of interest (ROIs) were drawn on at least four representative midventricular slices, with care taken to avoid myocardial borders. All myocardial time-activity curves were corrected for partial volume effect and spillover from cavity (28). ROIs within femoral muscles were drawn as previously described (22). The three-compartment model of [¹⁸F]FDG kinetics (29) and graphical analysis according to Patlak and Blasberg (24) were used to quantify the rates of glucose uptake as previously described (22).

Echocardiography. M-mode and two-dimensional echocardiography (model SSD-870, Aloka, Tokyo, Japan) was performed according to the recommendations of the American Society of Echocardiography (26). Left ventricular (LV) mass was calculated according to the Penn convention (3): LV mass = 1.04 · [(end diastolic diameter + posterior wall thickness + septal thickness)² − end diastolic diameter²] − 13.6 g. Maximal aerobic power and body composition. Maximal aerobic power (V₀₂max) was determined using direct measurement of the rate of oxygen consumption and a cycle ergometer (model 800 S, Ergoline, Mijnhardt, The Netherlands) with a continuous incremental protocol. Body fat content was estimated from four skinfolds (subscapular, triceps brachii, biceps brachii, and crista iliaca) as measured with a caliper (4).

Other measurements. Blood pressure and heart rate were determined every 30 min throughout the study using an automatic oscillometric blood pressure analyzer (model HEM-705C, Omron, Tokyo, Japan). Plasma glucose was measured using the glucose oxidase method (GM7 Analysys, Analox Instruments, Hammersmith, London, UK). Serum-free insulin concentrations were measured using a double-antibody radioimmunoassay (Pharmacia Insulin RIA kit, Pharmacia, Uppsala, Sweden) after precipitation with polyethylene glycol. Lactate was measured by enzymatic analysis (16).

Statistics. Group comparisons were performed with ANOVA followed by Tukey’s Studentized range test. Pearson’s correlations were calculated where appropriate. The statistical computation was performed with the SAS statistical program package (SAS Institute, Cary, NC). The results are expressed as means ± SE.

RESULTS

Characteristics of the study groups. Body weight was greater in the weight lifters than the endurance athletes and the sedentary subjects (Table 1). The percent body fat was lower in both groups of athletes than in the sedentary group (Table 1). V₀₂max (ml·kg⁻¹·min⁻¹) was markedly increased in the endurance athletes (71 ± 2) but was similar in the weight lifters (42 ± 2, P < 0.001) and sedentary subjects (42 ± 2, P < 0.001). The absolute left ventricular mass was similarly increased in the endurance athletes (290 ± 15 g) and the weight lifters (264 ± 19 g) compared with the sedentary men (186 ± 16 g, P < 0.01) (Fig. 1). These differences persisted after adjustment for body surface area (Table 2). Left ventricular systolic and diastolic diameters were slightly but not significantly higher in the athletes than in the sedentary men, but the differences did not reach statistical significance (Table 2). The posterior wall of the left ventricle and the interventricular septum were thicker in the endurance athletes and the weight lifters than in the sedentary men (P < 0.01). Resting blood pressure, heart rate, and the rate-pressure product were comparable among the groups (Table 2).

Whole body and skeletal muscle glucose uptake. Plasma glucose (5.0 ± 0.1, 4.8 ± 0.1, and 5.0 ± 0.2 mmol/l in endurance athletes, weight lifters, and sedentary men) and serum-free insulin concentrations (72 ± 12, 56 ± 4, and 64 ± 6 mU/l, respectively) were comparable during hyperinsulinemia. The weight lifters and the sedentary men had comparable rates of insulin-stimulated rates of whole body (38 ± 4 vs. 34 ± 2 µmol·kg⁻¹·min⁻¹, weight lifters vs. sedentary men) and femoral muscle (59 ± 12 vs. 63 ± 7 µmol·kg muscle⁻¹·min⁻¹, respectively) glucose uptake. In con-
The endurance athletes had enhanced insulin-stimulated rates of glucose uptake at the level of the whole body (65 ± 3 µmol·kg⁻¹·min⁻¹, P < 0.01) and in skeletal muscle (125 ± 16 µmol·kg muscle⁻¹·min⁻¹, P < 0.01) compared with each other (Fig. 2). VO₂max and skeletal muscle glucose uptake were positively correlated (r = 0.67, P < 0.001), and skeletal muscle and whole body glucose uptake values were correlated with each other (r = 0.84, P < 0.001). An example of PET images of insulin-stimulated glucose uptake in skeletal muscle is shown in Fig. 3.

Fasting serum FFA concentrations were slightly but not significantly lower in the endurance athletes (390 ± 45 µmol/l) than in the weight lifters (629 ± 98 µmol/l) or the sedentary subjects (560 ± 39 µmol/l). During hyperinsulinemia, serum FFA concentrations were similarly suppressed in the endurance athletes (103 ± 12 µmol/l), the weight lifters (140 ± 9 µmol/l), and the sedentary subjects (111 ± 15 µmol/l). Plasma lactate concentrations were significantly higher in the endurance athletes (1.4 ± 0.1 mmol/l, P < 0.05) than in the weight lifters (1.1 ± 0.1 mmol/l) and sedentary subjects (1.0 ± 0.1 mmol/l) during hyperinsulinemia. Rates of whole body (r = 0.66, P < 0.005) and skeletal muscle (r = 0.58, P < 0.02) glucose uptake were significantly correlated with the plasma lactate concentration during hyperinsulinemia.

Heart glucose uptake. The rate of insulin-stimulated myocardial glucose uptake per gram of myocardium was 48% lower in the endurance athletes (544 ± 50 µmol·kg muscle⁻¹·min⁻¹, P < 0.01) and 37% lower in the weight lifters (651 ± 45 µmol·kg muscle⁻¹·min⁻¹, P < 0.01) than in the sedentary men (1,041 ± 78 µmol·kg muscle⁻¹·min⁻¹). Rates of myocardial glucose uptake per unit mass of myocardium were comparable in the weight lifters and endurance athletes (Fig. 1). Left ventricular glucose uptake (per gram of myocardium) was also significantly inversely correlated with left ventricular mass (r = -0.67, P < 0.001). Examples of PET images of insulin-stimulated glucose uptake in heart muscle in the three groups are shown in Fig. 3. In contrast to skeletal muscle, plasma lactate levels did not correlate with heart glucose uptake, suggesting that the lower rate of glucose uptake in the weight lifters than the sedentary men was not due to increased lactate uptake via lactate mass action.

Table 2. Cardiac dimensions and hemodynamic parameters in weight lifters, endurance athletes, and sedentary men

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Endurance Athletes</th>
<th>Weight Lifters</th>
<th>Sedentary Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular mass index</td>
<td>146 ± 6*</td>
<td>128 ± 7*</td>
<td>93 ± 7</td>
</tr>
<tr>
<td>Left ventricular diameter, mm</td>
<td>36 ± 1</td>
<td>35 ± 1</td>
<td>34 ± 1</td>
</tr>
<tr>
<td>Systole</td>
<td>36 ± 1</td>
<td>35 ± 1</td>
<td>34 ± 1</td>
</tr>
<tr>
<td>Diastole</td>
<td>56 ± 1</td>
<td>55 ± 1</td>
<td>52 ± 2</td>
</tr>
<tr>
<td>Posterior wall, mm</td>
<td>15.5 ± 0.6*</td>
<td>16.4 ± 0.5*</td>
<td>13.0 ± 0.7</td>
</tr>
<tr>
<td>Systole</td>
<td>10.5 ± 0.5*</td>
<td>9.8 ± 0.4*</td>
<td>8.3 ± 0.2</td>
</tr>
<tr>
<td>Diastole</td>
<td>14.6 ± 0.2*</td>
<td>15.5 ± 0.6*</td>
<td>11.4 ± 0.6</td>
</tr>
<tr>
<td>Septum, mm</td>
<td>11.0 ± 0.5*</td>
<td>10.7 ± 0.6*</td>
<td>8.6 ± 0.4</td>
</tr>
<tr>
<td>Blood pressure, mmHg</td>
<td>126 ± 3</td>
<td>113 ± 4</td>
<td>121 ± 4</td>
</tr>
<tr>
<td>Systolic</td>
<td>72 ± 5</td>
<td>73 ± 4</td>
<td>80 ± 3</td>
</tr>
<tr>
<td>Diastolic</td>
<td>61 ± 3</td>
<td>60 ± 4</td>
<td>66 ± 5</td>
</tr>
<tr>
<td>Rate-pressure product, mmHg</td>
<td>7,690 ± 405</td>
<td>6,830 ± 615</td>
<td>8,040 ± 776</td>
</tr>
<tr>
<td>Stroke volume, ml·min⁻¹</td>
<td>102 ± 3</td>
<td>96 ± 6</td>
<td>83 ± 7</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>6.6 ± 0.3</td>
<td>6.1 ± 0.6</td>
<td>5.7 ± 0.5</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. *P < 0.05 vs. sedentary group.
Total left ventricular glucose uptake was calculated by multiplying myocardial glucose uptake by left ventricular mass, and it was comparable in endurance athletes ($161 \pm 13 \, \mu\text{mol/min}$), weight lifters ($172 \pm 18 \, \mu\text{mol/min}$), and sedentary men ($190 \pm 18 \, \mu\text{mol/min}$) (Fig. 1). Rates of left ventricular glucose uptake were also comparable if expressed per body surface area ($81 \pm 7, 83 \pm 7, \text{and} 95 \pm 8 \, \mu\text{mol} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$ for endurance athletes, weight lifters, and sedentary men). Whole left ventricular glucose uptake ($\mu\text{mol/min}$) correlated with cardiac work as determined from the rate-pressure product in endurance athletes ($r = 0.85, \, P < 0.05$), weight lifters ($r = 0.71, \, P = 0.05$), and sedentary men ($r = 0.63, \, P = 0.09$).

**DISCUSSION**

The present study is the first to relate cardiac structural changes to myocardial metabolism in resistance- and aerobically trained subjects. We found the heart to be enlarged in both groups and to utilize less glucose per unit mass than that of sedentary subjects. The rate of glucose uptake was similarly proportional to the degree of left ventricular hypertrophy in both weight lifters and endurance athletes. These similarities in cardiac adaptation were contrasted by a marked difference in the response of skeletal muscle to insulin between the two groups of athletes. In the weight lifters, the rate of insulin-stimulated skeletal muscle glucose uptake was similar to that in the sedentary men, whereas it was markedly enhanced in the endurance-trained athletes.

Both aerobic and resistance training are associated with an increase in left ventricular mass (6, 17, 30). This was also observed in the present study (Fig. 1, Table 2). In keeping with the data of Longhurst and Stebbins (14) and reviews of the literature (5, 9, 14), the absolute left ventricular mass and the left ventricular mass divided by surface area were slightly, albeit not significantly, lower in the weight lifters than in the endurance athletes (Fig. 1, Table 2). Regarding cardiac dimensions, we found, consistent with previous data (5, 9), increases in both posterior and septal wall thicknesses in the endurance athletes and weight lifters compared with the sedentary men. In the meta-analysis of Fagard (6), increases in left ventricular end-diastolic diameter, wall thickness, and left ventricular mass were for endurance athletes 10, 18, and 48%, and for resistance-trained athletes 2.5, 15, and 25%. In our study, respective values were for endurance athletes 8, 27, and 56% and for weight lifters 6, 22, and 42%. Thus, in the present study, the left ventricular end-diastolic diameter in the weight lifters was slightly greater than that predicted from Fagard’s meta-analysis. On the other hand, in the study of Spirito et al. (30), in which morphology of the athlete’s heart was assessed by echocardiography in two groups of elite athletes, long-distance runners and weight lifters, these athletes had similar left ventricular internal diameters (53.3 vs. 53.3 mm) and wall thicknesses (10.5 mm vs. 10.3 mm) (30). In addition, in a recent study of Fisman et al. (8), 29 endurance athletes and 16 weight lifters were found to have similar increases in both left ventricular mass and in septal and posterior wall thicknesses compared with 20 sedentary subjects. One might argue that these inconsistencies are attributable to confounding effects of dynamic components in resistance-training programs. This was unlikely the case for the weight lifters participating in the present study, because rate of maximal oxygen consumption, an accurate estimate of exposure to dynamic exercise compo-
Glucose uptake in weight lifters and endurance athletes

Both the endurance athletes and the weight lifters exhibited a significant decrease in insulin-stimulated myocardial glucose uptake per unit weight of the myocardium. Myocardial work, as estimated from the rate-pressure product, was comparable among the groups. When glucose uptake per unit myocardial mass was multiplied by left ventricular mass, all groups utilized equal amounts of glucose. These data are consistent with our previous data showing that glucose uptake under hyperinsulinenic conditions is proportional to cardiac work, irrespective of skeletal muscle insulin sensitivity. For example, the heart in normal men and women utilizes equal amounts of glucose, although insulin sensitivity of skeletal muscles is enhanced in women compared with equally fit men (19).

In normotensive patients with insulin-dependent diabetes mellitus (21) and non-insulin-dependent diabetes mellitus (33), insulin resistance is observed in skeletal muscles, but cardiac work and insulin-stimulated glucose uptake in the heart during hyperinsulinemia are unaltered. On the other hand, in patients with essential hypertension, cardiac work is increased as is the rate of insulin-stimulated glucose uptake per unit mass of the myocardium (23). Thus, although structural studies have suggested the heart of a resistance-trained athlete to more closely resemble that of a hypertensive heart, the present data demonstrate that the heart of a resistance-trained athlete is metabolically similar to that of an endurance-trained athlete and is characterized by diminished rather than increased glucose uptake per unit heart mass.

Under hyperinsulinenic conditions such as those prevailing in the present study, serum FFA concentrations are almost completely suppressed. Because FFAs are utilized in a concentration-dependent manner in the human heart (20, 22), and because FFA concentrations were similarly suppressed in all of the three groups, it is unlikely that myocardial rates of FFA utilization differed among the groups. Because FFAs and glucose represent the two most important fuels for the human myocardium under resting aerobic conditions (20, 22), it is unlikely that the lower rates of glucose uptake in the trained groups were due to increased utilization of fuels other than glucose. Consistent with this, direct measurement of rates of myocardial FFA utilization under fasting conditions have not revealed differences between endurance athletes and sedentary subjects (32). Regarding plasma lactate concentrations, the athletes exhibited higher levels during hyperinsulinemia than the two other groups. We have previously demonstrated that plasma lactate concentrations under conditions similar to those employed in the present study are determined by the rate of peripheral glucose uptake (35). A significant relationship between whole body and muscle glucose uptake and plasma lactate concentrations was also observed in the present study. The higher plasma lactate concentration in the endurance athletes might have enhanced lactate utilization in the heart (34). On the other hand, an increase in lactate uptake is unlikely to explain the decrease in myocardial glucose uptake, at least not via lactate mass action, in the weight lifters, because plasma lactate concentrations were comparable between weight lifters and sedentary subjects.

Endurance training is associated with increases in insulin sensitivity of glucose uptake in skeletal muscle, in direct proportion to maximal aerobic power (18, 25, 36). The present study confirms, by direct measurement of glucose uptake in skeletal muscle, that resistance training does not increase insulin-stimulated glucose uptake per unit mass of skeletal muscle (36). The failure of resistance training to enhance muscle insulin sensitivity could be due to the inability of resistance training to increase muscle capillary density (11) or to change muscle fiber types toward an insulin-sensitive direction (27, 31). Of note, the lack of favorable effects of resistance training on insulin sensitivity is also the likely explanation for why serum lipids and lipoproteins (12, 37) do not appear to change in an antiatherogenic direction by this type of training.

The study was performed under euglycemic hyperinsulinemic conditions, which are artificial compared with the fed state. However, we were interested to determine how skeletal muscle insulin sensitivity relates to heart insulin sensitivity, and for this purpose the clamp technique is the golden standard, as it allows tissue glucose uptake measurements to be performed under steady-state conditions. Steady state is a requirement not only for accurate use of PET methodology but also for application of the Fick principle to quantify tissue glucose uptake by use of conventional techniques. We were not able to measure FFA or lactate uptake, the rate of glucose oxidation, or the contribution of intracellular substrates such as triglycerides and glycogen to energy metabolism. Separate studies, which use for example the fatty acid tracer [1-14C]fluoro-6-thia-heptadecanoic acid (15) and [15O]O2 (13) to quantify myocardial fatty acid and oxygen uptake, need to be undertaken to clarify the contribution of FFAs to myocardial substrate utilization and the relationship between myocardial glucose uptake and oxygen consumption under hyperinsulinenic conditions.

In conclusion, the present data demonstrate that the heart of a resistance-trained athlete closely resembles that of an endurance-trained athlete with respect to the rate of glucose uptake during hyperinsulinemia. In both groups of athletes, the rate of glucose uptake per unit mass of myocardium was decreased compared with sedentary men. When the rate of glucose uptake per unit mass was multiplied by heart size, glucose uptake was similar in all groups.

We thank the technicians of the Turku PET Centre and Jukka Kapanen, Paavo Nurmi Center, for VO2max determinations.

This work was supported by grants from the Academy of Finland (P. Nuutila, H. Yki-Järvinen), Novo-Nordisk (P. Nuutila, H. Yki-Järvinen), Juseelis (P. Nuutila, H. Yki-Järvinen), Aarne Koskelo (T. O. Takala), and the Finnish Medical Foundation (T. O. Takala).

Address for reprint requests: T. Takala, Turku PET Centre, Turku University Central Hospital, PO Box 52, FIN-20521 Turku, Finland (E-mail: teetaka@utu.fi).

Received 24 August 1998; accepted in final form 4 December 1998.
REFERENCES


