Prior exercise increases net hepatic glucose uptake during a glucose load

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The aim of the present study was to determine whether prior exercise enhances net hepatic glucose uptake (NHGU) in response to glucose load. Animals and surgical procedures. Twelve mongrel dogs of either gender (mean weight, 24 ± 2 kg) were studied. Animals were housed in a facility that met American Association for the Accreditation of Laboratory Animals Care guidelines and were fed a standard diet of meat and chow (34% protein, 14.5% fat, 46% carbohydrate, and 5.5% fiber based on dry weight). Experimental protocols were approved by the Vanderbilt University School of Medicine Animal Care and Use Committee. At least 16 days before each experiment, a laparotomy was performed under general anesthesia. Two Silastic catheters (0.03 mm ID) were inserted in the inferior vena cava for tracer and indocyanine green (ICG) infusion. Two more Silastic catheters (0.03 mm ID) were inserted in a jejunal and in a splenic vein (advanced so that the tips were in the portal vein) for intraportal infusion of glucose. Silastic catheters (0.04 mm ID) were also inserted in the portal vein

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EXERCISE markedly increases the metabolic demands of the organism, mostly due to increased needs of the contracting muscle (17, 29). Net muscle glucose uptake is greatly enhanced during exercise and remains elevated in the postexercise state to replenish muscle glycogen stores that were depleted during muscle contraction. Increased insulin sensitivity and glucose effectiveness facilitate muscle glucose uptake and glycogen synthesis in the postexercise state (27–29). This process is markedly accelerated if exogenous glucose is administered after the cessation of exercise (15, 26). In fact, the excess whole body glucose uptake measured during glucose infusion after exercise has been ascribed solely to skeletal muscle glucose disposal (17, 29). The liver, on the other hand, even if its glycogen stores are also depleted by prior exercise, remains a net producer of glucose in the immediate postexercise state in fasted animals (32). The liver can become a net consumer of glucose if exogenous glucose is administered after exercise (8). Nevertheless, neither net hepatic glucose uptake (NHGU) nor net hepatic glycogen deposition appears to be different from that measured in the absence of prior exercise (8). In another study, Matsuhisa et al. (20) observed that prior contraction of the rabbit hindlimb caused an increase in the rate of liver deposition of the glucose analog 3-fluoro-3-deoxy-D-glucose (3FDG) during a glucose load. In the aforementioned studies, however, direct hepatic effects of prior exercise were difficult to ascertain, because one or more of the determinants of hepatic glucose uptake [glucose load, arterial-portal venous (a-pv) glucose gradient, pancreatic hormone concentrations] were uncontrolled.

The aim of the present study was to determine whether prior exercise enhances NHGU in response to controlled changes in arterial glucose, insulin, glucagon, hepatic glucose load, and a-pv gradient. To address this aim, isotopic and arteriovenous-difference techniques were used in the chronically catheterized, conscious dog model after either a prolonged treadmill exercise period or an equivalent period of rest.

MATERIALS AND METHODS

Animals and surgical procedures. Twelve mongrel dogs of either gender (mean weight, 24 ± 2 kg) were studied. Animals were housed in a facility that met American Association for the Accreditation of Laboratory Animals Care guidelines and were fed a standard diet of meat and chow (34% protein, 14.5% fat, 46% carbohydrate, and 5.5% fiber based on dry weight). Experimental protocols were approved by the Vanderbilt University School of Medicine Animal Care and Use Committee. At least 16 days before each experiment, a laparotomy was performed under general anesthesia. Two Silastic catheters (0.03 mm ID) were inserted in the inferior vena cava for tracer and indocyanine green (ICG) infusion. Two more Silastic catheters (0.03 mm ID) were inserted in a jejunal and in a splenic vein (advanced so that the tips were in the portal vein) for intraportal infusion of glucose. Silastic catheters (0.04 mm ID) were also inserted in the portal vein...
Experimental procedures. Animals were either exercised at a moderate intensity (100 m/min, 12% grade) on a motorized treadmill (n = 6) or remained sedentary (n = 6) from t = −180 to −30 min (Fig. 1). The exercise duration and intensity used in these experiments have been shown previously to result in a twofold increase in heart rate and an increase in O$_2$ uptake to 50% of maximum (22). A period of exercise recovery or continued rest followed (−30 to 100 min). At time = −70 min, a primer of [3-$^3$H]glucose (30 µCi) was given, followed by venous infusions of [3-$^3$H]glucose (0.3 µCi/min) and ICG (0.1 mg/min), which were continued for the duration of the study. ICG was used as a backup for the Doppler method of flow measurement. After a 10-min transition period (t = −30 to −20 min), from t = −20 to 0 min (basal period), three blood samples were drawn for assessment of basal levels of metabolic variables. From t = 0−100 min (experimental period), glucose was given via a constant intraportal infusion (3.5 mg·kg$^{-1}$·min$^{-1}$) and by a variable infusion into the inferior vena cava to clamp the arterial blood glucose at 130 mg/dl. From t = 0−100 min, endogenous pancreatic hormone secretion was also suppressed via a continuous somatostatin infusion into the inferior vena cava (0.8 µg·kg$^{-1}$·min$^{-1}$). Insulin and glucagon were replaced via intraportal infusions of 1.2 µmol·kg$^{-1}$·min$^{-1}$ (4-fold basal) and 0.5 ng·kg$^{-1}$·min$^{-1}$ (basal), respectively. Arterial samples were drawn at 5-min intervals from t = −20 to 100 min. Portal, hepatic, and common iliac venous samples were drawn at t = −20, −10, 0, 60, 70, 80, 90, and 100 min. Portal vein, hepatic artery, and external iliac artery blood flows were recorded continuously from the frequency shifts of the pulse sound signal emitted from the Doppler flow probes (9, 10).

Processing of blood samples. Plasma and deproteinized blood samples that were not analyzed the day of the study were stored at −70°C after the completion of the experiment. Plasma glucose levels were determined during experiments by the glucose oxidase method with a glucose analyzer (Beckman Instruments, Fullerton, CA). For the determination of plasma [3-$^3$H]glucose radioactivity, samples were deproteinized with barium hydroxide and zinc sulfate, the supernatant was evaporated, and the residue was dissolved in 1 ml of water and 10 ml Ecolite$^-$ (ICN Biomedicals, Irvine, CA). Radioactivity was then determined by liquid scintillation counting with a Beckman LS 5000TD counter. Whole blood (samples deproteinized by 1:3 dilution in 4% peridolic acid) lactate, glycerol, alanine, glucose, and plasma free fatty acids were measured by enzymatic methods (18) on a Technicon autoanalyzer (Tarrytown, NY) or on a Monarch 2000 centrifugal analyzer (Instrumentation Laboratories, Lexington, MA).

Immunoactive insulin was measured with a double antibody system (interassay coefficient of variation (CV) of 10%;...
Ref. 21]. Immunoreactive glucagon was measured in plasma samples containing 50 µl of 500 Kallikrein-inhibitor units/ml Trasylol (FBA Pharmaceuticals, NY) with a double antibody system (CV of 7%) modified from the method developed by Morgan and Lazarow (21) for insulin. Insulin and glucagon antisera, standard glucagon and insulin, and the 125I-labeled insulin were obtained from Linco Research (St. Charles, MO).

Calculations. The tracer-determined total rate of glucose appearance (Ra) was determined by steady-state equations for isotope ([3-3H]glucose) dilution (3). Endogenous glucose Ra was calculated by subtracting the glucose infusion rate (portal vein + vena cava) from the total glucose Ra.

Net hepatic balances of lactate, glucose, alanine, FFA, and glycerol were determined by the following formula: HAF \times ([H] - [A]) + PVF \times ([H] - [P]), where [A], [P], and [H] are the arterial, portal vein, and hepatic vein blood or plasma substrate concentrations, respectively, and HAF and PVF are the hepatic artery and portal vein blood or plasma flows, respectively, determined with Doppler flow probes. The load of a substrate reaching the liver was calculated as follows: [P] \times PVF + [A] \times HAF. Net hepatic substrate fractional extraction was calculated as the ratio of net hepatic balance to hepatic load.

Net limb balances were calculated as follows: LF \times ([A] - [I]). LF is limb blood flow through the external iliac artery, and [I] is the substrate level in the common iliac vein. Limb fractional substrate extraction was calculated as the limb substrate uptake divided by the limb substrate load (LF \times [A]). Blood levels and flows were used for the calculation of all hepatic and limb balances, with the exception of FFA balances for which plasma concentrations and flows were used. The ratio of blood to plasma glucose was calculated for the basal period and the glucose infusion period for each dog at each of the four sampling sites. Plasma glucose values were then multiplied by their corresponding ratio (i.e., blood glucose to plasma glucose) to convert to blood glucose concentrations. The advantage of plasma glucose measurements is that a large number of replicates can be obtained quickly and at little added cost.

RESULTS

Arterial blood glucose, a-pv glucose gradient, and pancreatic hormone levels. Arterial blood glucose was similar between the two groups at baseline and rose by ~80% during the experimental period in both groups (Fig. 2). The a-pv glucose gradient was positive in both groups at baseline (3.1 ± 0.4 in sedentary and 4.3 ± 0.4 mg/dl in exercised dogs) and became markedly negative during the experimental period (−17.9 ± 1.8 in sedentary and −19.8 ± 1.7 in exercised).

Fig. 2. Arterial plasma glucose, insulin, and glucagon in 42-h-fasted dogs after either rest or 150 min of moderate exercise, at baseline and during intraportal glucose infusion good mixing of glucose was assessed using Doppler flow probes. The load of a substrate reaching the liver was calculated as follows: [P] \times PVF + [A] \times HAF. Net hepatic substrate fractional extraction was calculated as the ratio of net hepatic balance to hepatic load.

The arterial plasma glucose, insulin, and glucagon levels were measured during the experimental period (Fig. 2). The a-pv glucose gradient was positive in both groups at baseline (3.1 ± 0.4 mg/dl in sedentary and 4.3 ± 0.4 mg/dl in exercised dogs) and became markedly negative during the experimental period (−17.9 ± 1.8 in sedentary and −19.8 ± 1.7 in exercised dogs).
tary and $-19.3 \pm 3.4\ \text{mg/dl in exercised dogs}$. There were no differences in the a-pv glucose gradient between the two groups.

As expected, arterial plasma insulin at baseline was slightly lower in the exercised than in the sedentary dogs. Insulin levels rose by about threefold during the experimental period and were similar in the two groups. Arterial plasma glucagon was higher at baseline in exercised compared with sedentary dogs. During the experimental period, the pancreatic clamp equalized glucagon levels in the two groups. The presence of a metabolic steady state was reflected by the stability of the measurements described previously during the last 40 min of the experimental period.

Tracer-determined total and endogenous glucose $R_a$. $R_a$ was not significantly higher in the exercised compared with the sedentary animals at baseline ($4.0 \pm 0.5$ vs. $2.8 \pm 0.3\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$). Total $R_a$ (endogenous + exogenous) became significantly greater in the exercised animals during the experimental period ($11.1 \pm 0.8$ vs. $7.0 \pm 0.5\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$). Endogenous glucose $R_a$ was completely suppressed during the experimental period in both groups.

Hepatic glucose metabolism. The basal hepatic glucose load was $23 \pm 1\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in the sedentary dogs and $25 \pm 1\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in the exercised dogs (Fig. 3). It increased to $36 \pm 2\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in sedentary and to $40 \pm 1\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in exercised dogs during the experimental period. Exercised dogs had a significantly higher basal net hepatic glucose output compared with sedentary animals ($4.2 \pm 0.3$ vs. $1.9 \pm 0.2\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$, $P < 0.01$). Animals in both groups shifted to NHGU during the experimental period. There was significantly more net glucose uptake in exercised than in sedentary dogs ($-3.2 \pm 0.4$ vs. $-1.8 \pm 0.2\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$, $P < 0.05$). The total change in net hepatic glucose balance from baseline to the experimental period was therefore $3.7 \pm 0.5\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in sedentary dogs and $7.3 \pm 0.7\ \text{mg \cdot kg}^{-1} \cdot \text{min}^{-1}$ in exercised animals ($P < 0.001$ vs. sedentary). The net hepatic fractional extraction of glucose during the experimental period was greater in the exercised than in the sedentary dogs ($0.8 \pm 0.01$ vs. $0.05 \pm 0.01$, $P < 0.02$).

Hindlimb glucose metabolism. The basal net hindlimb glucose uptake was $4.0 \pm 0.9\ \text{mg/min in the sedentary dogs}$ and $8.4 \pm 0.9\ \text{mg/min in the exercised dogs}$ ($P < 0.05$ vs. sedentary). During the experimental period, it rose to $13.2 \pm 1.4\ \text{mg/min in sedentary}$ and to $32.8 \pm 3.5\ \text{mg/min in exercised dogs}$ ($P < 0.01$ vs. sedentary). Net hindlimb glucose fractional extraction paralleled the response of net hindlimb glucose uptake ($2.6 \pm 0.5\%$ in sedentary dogs and $4.6 \pm 0.4\%$ in exercised dogs at baseline, $P < 0.05$; $4.7 \pm 0.5\%$ in sedentary dogs and $13.4 \pm 1.0\%$ in exercised dogs during the experimental period, $P < 0.05$).

Blood levels and hepatic and hindlimb balances of gluconeogenic precursors. Arterial lactate concentrations were similar at baseline and increased by $-80\%$ in both groups (Fig. 4). Exercised animals displayed significantly more basal net hepatic lactate uptake ($-12.5 \pm 1.2$ vs. $-7.3 \pm 1.7\ \text{mmol \cdot kg}^{-1} \cdot \text{min}^{-1}$, $P < 0.05$). During the experimental period, all animals shifted to net hepatic lactate output, with no significant difference between the two groups. The basal net hindlimb lactate output was $-21.8 \pm 4.0$ and $-13.8 \pm 4.7\ \text{mmol/min}$ ($P > 0.05$; nonsignificant) in exercised and sedentary dogs, respectively. During the experimental period, net hindlimb lactate output was suppressed in both groups.

Net hepatic alanine and glycerol uptakes were higher at baseline in exercised dogs; similar values of hepatic
uptake of both metabolites were detected during the experimental period (data not shown).

Blood flows. Although basal hepatic artery blood flow was higher in the exercised dogs than in the sedentary dogs \( (P < 0.05) \), total splanchnic blood flow was similar between the two groups throughout the study (Table 1). The external iliac artery blood flow was similar between groups throughout the study.

**DISCUSSION**

It is well established that prior exercise increases insulin sensitivity and glucose effectiveness \( (12) \), leading to enhanced rates of net muscle glucose uptake and glycogen synthesis \( (7, 8, 14) \). By comparison, far less is known about how prior exercise affects NHGU. In the present study, the influence of prior exercise on the net hepatic uptake of an intraportal venous glucose load was investigated in conscious dogs in which circulating glucose, insulin, glucagon, a-pv glucose gradient, and hepatic glucose load were carefully controlled. Prior exercise led to a 74\% greater NHGU and a 58\% greater net hepatic glucose fractional extraction compared with sedentary controls. The total change in net hepatic glucose balance from the basal period to the glucose infusion period was 3.7 and 7.3 mg·kg\(^{-1}\)·min\(^{-1}\) in sedentary and exercised dogs, respectively. Whole body glucose uptake was 57\% greater in exercised compared with sedentary animals. It could be calculated that 26 and 28\% of the whole body glucose uptake could be accounted for by NHGU in the sedentary and exercised dogs, respectively. Although the exercised dogs consumed more glucose, the percent contribution of hepatic and nonhepatic tissues to whole body glucose uptake was similar.

The hypothesis that liver glucose uptake could be stimulated by prior exercise is consistent with previous studies in humans \( (16) \) and rabbits \( (20) \). Kawamori et al. \( (16) \) showed that the splanchnic disposal of an oral glucose load during a euglycemic-hyperinsulinemic clamp was about twice as high after exercise as after rest in non-insulin-dependent diabetes mellitus patients. In this study, glucagon levels were not controlled or even measured, and only an indirect estimate of the NHGU was made. Matsuhisa et al. \( (20) \) measured the accumulation of phosphorylated 3FDG in the liver of anesthetized rabbits after 30 min of electrical stimulation of the hindlimb muscles. The accumulation of phosphorylated 3FDG in the liver was approximately twofold greater after muscle contraction compared with nonelectrically stimulated control rabbits. The effect of prior exercise directly on the liver was difficult to assess because the animals that underwent muscle contraction had higher circulating insulin. Although the stud-

**Table 1.** External iliac artery, hepatic artery, portal vein, and total splanchnic blood flows

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Experimental Period</th>
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<tbody>
<tr>
<td>External iliac artery, ml/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedentary</td>
<td>209±18</td>
<td>200±12</td>
</tr>
<tr>
<td>Exercised</td>
<td>241±21</td>
<td>185±21</td>
</tr>
<tr>
<td>Hepatic artery, ml·kg(^{-1})·min(^{-1})</td>
<td></td>
<td></td>
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<tr>
<td>Sedentary</td>
<td>6.7±0.4</td>
<td>8.1±0.5</td>
</tr>
<tr>
<td>Exercised</td>
<td>10.4±0.7*</td>
<td>10.1±1.2</td>
</tr>
<tr>
<td>Portal vein, ml·kg(^{-1})·min(^{-1})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedentary</td>
<td>22.1±1.8</td>
<td>16.7±1.4</td>
</tr>
<tr>
<td>Exercised</td>
<td>22.4±2.7</td>
<td>17.8±1.4</td>
</tr>
<tr>
<td>Total splanchnic, ml·kg(^{-1})·min(^{-1})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedentary</td>
<td>28.9±1.8</td>
<td>24.8±1.3</td>
</tr>
<tr>
<td>Exercised</td>
<td>32.8±2.4</td>
<td>27.9±1.0</td>
</tr>
</tbody>
</table>

Data are means ± SE; \( n = 6 \) group. Dogs were 42 h fasted and were studied after 150 min of either rest or moderate exercise. Baseline value is average of 3 measurements \( (−20 \text{ min} − 0 \text{ min}) \), and experimental period value is mean of 5 steady-state measurements \( (60−100 \text{ min}) \). *P < 0.05, exercised vs. sedentary.
ies previously cited provide indirect support for the hypothesis that prior exercise stimulates NHGU, in
none were the factors that are known to influence NHGU controlled nor was NHGU measured directly.
Hamilton et al. (8) showed that rates of NHGU were similar in exercised dogs and in sedentary controls,
when glucose was infused intraduodenally at 8 mg·kg\(^{-1}\)·min\(^{-1}\). Because a greater suppression of endog-
ogenous R\(_{s}\) was seen in exercised animals at a time when
rates of NHGU were similar to sedentary controls,
unidirectional hepatic glucose uptake must also have
been reduced by prior exercise. Endogenous pancreatic
hormone secretion was not suppressed in the study by
Hamilton et al., and circulating glucagon levels were
greater in the exercised dogs. Because glucagon can
antagonize NHGU (13), the higher glucagon levels may
have masked a NHGU response in the exercised dogs.
Maehlum et al. (19) have reported that a greater
proportion of an oral glucose load escapes hepatic
retention in exercised than in sedentary humans. Al-
though this finding may appear contrary to the findings
from the present study, it should be noted that in the
work by Maehlum et al. the experimental setting was
significantly different from ours. The human subjects
in their study were administered glucose orally in a
bolus. In this situation, the glucose load reaching the
intestine is the same in the two experimental groups,
but the load reaching the liver is greater in the exer-
cised subjects, as exercise enhances the ability of the
gut to absorb ingested glucose (5, 8). Because this
exercise-induced increase in hepatic glucose load is
greater than the increase in NHGU, a greater amount
of glucose leaves the splanchnic region in response to
oral glucose after exercise.

All factors known to stimulate NHGU were con-
trolled in the present study. It could be argued, how-
ever, that other extrahepatic determinants such as
catecholamines, cortisol, and growth hormone, which
were not measured, may also have influenced liver
metabolism. Arterial catecholamine levels have been
previously demonstrated to increase two- to threefold
during an exercise bout comparable with that used in
this study (2). Nevertheless, the experimental period
started 30 min after the cessation of exercise, at which
time catecholamine levels have been shown to return to
basal levels (2). Further, catecholamines mainly affect
liver glucose metabolism by increasing the availability of
 gluconeogenic precursors from the periphery. In this
study, the net liver uptake of glyceral and alanine and
the net liver output of lactate were similar in exercised
and sedentary dogs. Cortisol and growth hormone
levels are also increased during exercise; their influ-
ences on hepatic glucose metabolism, nevertheless,
have not to date been clearly defined. It should be
noted, however, that the increased secretion of the
aforementioned counter regulatory hormones favors net
glucose output by the liver, not NHGU. Controlling the
levels of these hormones should, if anything, have
enhanced the effect of prior exercise on NHGU.

A strong positive correlation exists between NHGU
and both insulin concentration and hepatic glucose load
(23, 24). Increased sensitivity to one of these factors, or
both, may cause the increase in NHGU after exercise. A
positive correlation also exists at rest between NHGU
and the magnitude of a negative a-pv glucose gradient
until values of approximately –20 mg/dl, above which
no further enhancement of NHGU was observed (25).
The a-pv glucose gradients measured in our study (–18
and –19 mg/dl) were very close to the value that
maximally stimulates NHGU (25). It is possible, how-
ever, that prior exercise increases the sensitivity or
responsiveness of NHGU to the a-pv glucose gradient.
At the cellular level, glucose transport across the
hepatic portal system is primarily mediated by the
GLUT-2 isoform of the glucose transporter family. Once
in the cytoplasm, glucose is phosphorylated through a
reaction catalyzed by the enzyme glucokinase (GK).
GLUT-2 is not believed to play a limiting role in
the movement of glucose from the extracellular to the
intracellular space of the hepatocyte. GK activity, on
the other hand, is thought to be rate limiting for glucose
uptake in the liver and can be regulated in response to
different stimuli, such as glucose, fructose, and gluca-
gon concentrations (1). An increase in GK activity
occurs because of the translocation of GK from the
nucleus of the hepatocyte to the cytosol and the loss of
the inhibitory binding with its regulatory protein.
Although the effect of exercise on GK activity has not
been completely elucidated, a significant increase in
GK activity has been reported after exercise in rats (4).
Hamilton et al. (8) observed that GK activity was not
significantly different in exercised and sedentary dogs
after an intraduodenal glucose load. In these previous
studies, GK analysis was performed on crude liver
tissue homogenates, and enzymatic movement from
the nuclear membrane into the cytosol could not be
detected. In addition, other determinants of liver glu-
cose uptake were not controlled.

Because liver glycogen content and synthetic rates
were not measured in the present study, it is impossible
to specify the intrahepatic fate of the excess glucose
taken up by the liver in response to a glucose load in the
postexercise state. The net hepatic balances of the
 gluconeogenic precursors lactate, alanine, and glyceral,
on the other hand, were similar in sedentary and
exercised animals. This suggests that glycogen storage
was probably the preferential pathway through which
the liver of exercised dogs directed the excess glucose
taken up compared with sedentary animals.

Prior exercise induced greater net hindlimb glucose
uptake during glucose infusion, despite similar hind-
limb glucose and insulin loads in exercised and seden-
tary animals. This is consistent with numerous previ-
ous studies in which prior exercise led to greater net
skeletal muscle glucose fractional extraction due to
enhanced muscle insulin sensitivity (28, 30) as well as
increased insulin-independent glucose uptake (6, 29).
The greater net glucose uptake by the skeletal muscle
has been previously shown to result in greater rates of
intramuscular glycogen synthesis (19, 29). Although
muscle glycogen was not measured in the present
study, these previous reports are consistent with our
observation that net muscle lactate output was similar in sedentary and exercised animals.

As stated previously, the contribution of the liver to whole body glucose uptake is quantitatively smaller than that of the skeletal muscle (in the present study the liver accounted for 26–28% of whole body glucose uptake, whereas the large majority of the remainder could be accounted for by total whole body glucose uptake). The importance of the liver as a contributor to glucose uptake could be accounted for by total whole body glucose uptake, whereas the large majority of the remainder was accounted for by the liver. The observation that net muscle lactate output was similar in sedentary and exercised animals.

RESULTS


