Muscle glycogen oxidation during prolonged exercise measured with oral $[^{13}C]$glucose: comparison with changes in muscle glycogen content

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Harvey CR, Frew R, Massicotte D, Péronnet F, Rehrer NJ.

Muscle glycogen oxidation during prolonged exercise measured with oral $[^{13}C]$glucose: comparison with changes in muscle glycogen content. J Appl Physiol 102: 1773–1779, 2007. First published February 1, 2007; doi:10.1152/japplphysiol.00690.2006.—Plasma glucose and muscle glycogen oxidation during prolonged exercise [75-min at 48 and 76% maximal O2 uptake ($V_O2_{max}$)] were measured in eight well-trained male subjects [$V_O2_{max} = 4.50$ l/min (SD 0.63)] using a simplified tracer technique in which a small amount of glucose highly enriched in $^{13}$C was ingested: plasma glucose oxidation was computed from $^{13}$C/12C in plasma glucose (which was stable beginning at minute 30 and minute 15 during exercise at 48 and 76% $V_O2_{max}$, respectively) and $^{13}$CO2 production, and muscle glycogen oxidation was estimated by subtracting plasma glucose oxidation from total carbohydrate oxidation. Consistent data from the literature suggest that this small dose of exogenous glucose does not modify muscle glycogen oxidation and has little effect, if any, on plasma glucose oxidation. The percent contributions of plasma glucose and muscle glycogen oxidation to the energy yield at 48% $V_O2_{max}$ (15.1% (SD 3.8) and 45.9% (SD 5.8)) and at 76% $V_O2_{max}$ (15.4% (SD 3.6) and 59.8% (SD 9.2)) were well in line with data previously reported for similar work loads and exercise durations using conventional tracer techniques. The significant reduction in glycogen concentration measured from pre- and postexercise vastus lateralis muscle biopsies paralleled muscle glycogen oxidation calculated using the tracer technique and was larger at 76% than at 48% $V_O2_{max}$. However, the correlation coefficients between these two estimates of muscle glycogen utilization were not different from zero at each of the two work loads. The simplified tracer technique used in the present experiment appears to be a valid alternative approach to the traditional tracer techniques for computing plasma glucose and muscle glycogen oxidation during prolonged exercise.

MUSCLE GLYCOGEN OXIDATION during prolonged exercise can be estimated from the difference between total carbohydrate oxidation, computed by indirect respiratory calorimetry, and plasma glucose oxidation measured with tracer techniques (e.g., 3, 19, 27, 32, 36). In these studies, plasma glucose oxidation is assumed to be equal to its rate of disappearance measured using infusion of $[^{2}H]$glucose (27, 32, 36) or is directly measured, using administration of trace amounts of $[^{14}C]$glucose (3) or $[^{13}C]$glucose (19), from plasma glucose enrichment and labeled CO2 production. When glucose is ingested during exercise, an alternate way to compute plasma, and thus, by difference, muscle glycogen oxidation, without the need for infusing $[^{2}H]$glucose or $[^{2}H]$glucose, is to administer trace amounts of $[^{13}C]$glucose or $[^{14}C]$glucose along with the unlabeled glucose (e.g., 8, 18, 25). We have recently used this method during cold exposure (5–10°C, 90–120 min) in humans, with the ingestion of very small doses of glucose (3–10 g) highly enriched in $^{13}$C (~1,700–4,300‰ $^{13}$CVPDB, where $^{13}$CVPDB is the Vienna-Pee Dee Belemnite standard) (13, 14). Although higher than the amounts infused in most tracer technique studies, these small doses of glucose only contributed ~5–10% to the total plasma glucose flux (13).

In the present experiment, a similar approach was used to describe plasma glucose and muscle glycogen utilization during 75 min of exercise on a cycle ergometer at ~48% and ~76% maximal O2 uptake ($V_O2_{max}$). Muscle glycogen utilization was compared with changes in glycogen content in muscle biopsies from the vastus lateralis. This method has been extensively used to track muscle glycogen utilization during exercise (7, 31); however, in the five studies in which changes in glycogen content in muscle biopsies and muscle glycogen oxidation by tracer technique have been simultaneously measured, these two estimates of glycogen utilization were not closely related (3, 11, 32, 34, 35). On the basis of these observations, we hypothesized that only a weak correlation will be observed between changes in glycogen content in muscle biopsies and muscle glycogen oxidation by tracer technique, both at low and high work loads.

METHODS

Eight healthy, endurance-trained male cyclists volunteered for this study. Their age, height, body mass, and $V_O2_{max}$ (SensorMedics, Anaheim, CA) determined during a preliminary test session on cycle ergometer (Rodby Elektronik) were [mean (SD)], respectively, 23 yr (SD 5), 1.80 m (SD 0.08), 75.4 kg (SD 8.8), and 4.50 l/min (SD 0.63) achieved at 345 W (SD 44). An informed written consent was obtained from all subjects before participation in the study, which was approved by the Otago Ethics Committee.

The subjects performed two 75-min exercise trials at low and high work loads [166 W (SD 22) and 263 W (SD 50), corresponding to 47% (SD 3) and 74% (SD 7) of the maximal work load and 48% (SD 2) and 76% (SD 4) $V_O2_{max}$] separated by 1 wk, with ingestion of small amounts of glucose highly enriched in $^{13}$C. Three days before each exercise session, the subjects performed a 90-min exercise period at ~70% $V_O2_{max}$ to reduce glycogen stores. Thereafter, they rested as much as possible, and ingestion of food from plants with the C4 photosynthetic cycle, which are naturally enriched in $^{13}$C, was avoided to keep a low background $^{13}$C-enrichment of plasma glucose and expired CO2. In addition, 1 wk after the second exercise trial, the subjects also performed a 60-min exercise period (30 min at 48% $V_O2_{max}$ followed by 30 min at 76% $V_O2_{max}$) without ingestion of...
approximately 4.58 V\textsuperscript{\textcircled{O}2} g of glucose/min = 3.23 V\textsuperscript{\textcircled{CO}2} fat (g/min) = 1.70 (V\textsuperscript{\textcircled{O}2} − V\textsuperscript{\textcircled{CO}2})

The amount of energy provided by the oxidation of carbohydrate and fat were computed from their respective energy potential.

The oxidation rate of exogenous glucose (G\text{exo}) was computed as follows (25):

\[ G_{\text{exo}} = V\textsuperscript{\textcircled{CO}2} \left( \frac{R_{\text{exp}} - R_{\text{ref}}}{R_{\text{ref}} - R_{\text{reo}}} \right) + k \]

In this equation, V\textsuperscript{\textcircled{CO}2} (not corrected for protein oxidation) is in liters per minute; R\text{exp}, R\text{ref}, and R\text{reo} are, respectively, the 13C/12C in breath CO\textsubscript{2} in the experimental situation (i.e., when glucose was ingested), in the control situation (with no ingestion of glucose), and in the glucose ingested; and k (0.743 mg/l) is the volume of CO\textsubscript{2} provided by the complete oxidation of glucose. The oxidation of plasma glucose (G\text{plasma}) was computed using a similar equation, but with the 13C-enrichment of plasma glucose (R\text{gpl}) replacing R\text{reo} (20, 25):

\[ G_{\text{plasma}} = V\textsuperscript{\textcircled{CO}2} \left( \frac{R_{\text{exp}} - R_{\text{ref}}}{R_{\text{gpl}} - R_{\text{ref}}} \right) + k \]

Finally, the oxidation of glucose and C\textsubscript{3} products derived from muscle glycogen (G\text{muscle}), either directly or through the lactate shuttle (5), was calculated as the difference between total glucose oxidation (Eq. I) and plasma glucose oxidation (Eq. 4):

\[ G_{\text{muscle}} = G_{\text{total}} - G_{\text{plasma}} \]

These computations are made based on the observation that in response to exercise, 13C\textsuperscript{2}glucose is not irreversibly lost in pools of tricarboxylic acid cycle intermediates (28) and/or bicarbonate (30) and that 13C\textsuperscript{2}CO\textsubscript{2} recovery in expired gases is, thus, complete or almost complete. However, to take into account the delay
between $^{13}$CO$_2$ in tissues and at the mouth (23), as well as the time needed for the $^{13}$C-enrichment in plasma glucose to stabilize (see RESULTS), the computations of exogenous glucose, plasma glucose, and muscle glycogen oxidation were only performed over the last 45 min of exercise, thus allowing for a 30-min equilibration period.

Data presented as means (SD) were compared by two-way ANOVA for repeated measures, and paired $t$-tests used to identify significant differences ($P \leq 0.05$) when the ANOVA yielded a significant $F$ ratio (SPSS-x, SPSS, Chicago, IL). Pearson correlation coefficients were computed using the least squares method between muscle glycogen oxidation and changes in muscle glycogen content in response to each of the two exercise trials.

**RESULTS**

Respiratory gas exchange computed at 15-min intervals was stable over the 75-min exercise period at 48 and 76% VO$_{2\text{max}}$ (Table 1). Substrate oxidation computed from VO$_2$ and VCO$_2$ and urea excretion over the last 45 min of exercise and their respective contribution to the energy yield is displayed in Table 2. Protein oxidation was not significantly different between the two work loads, and its contribution to the energy yield was thus significantly greater at 48 than at 76% VO$_{2\text{max}}$. As expected, the contributions of fat and carbohydrate oxidation to the energy yield were, respectively, higher and lower at 48 than at 76% VO$_{2\text{max}}$.

The isotopic composition of expired CO$_2$ measured at rest before ingestion of the first dose of glucose enriched in $^{13}$C was not significantly different in the two experimental situations (Table 1). In response to exercise without ingestion of glucose, this value slightly, but significantly, increased [from $-23.5 \pm 2.6$ (SD 1.3) at rest to $-22.9$ (SD 1.6) and $-21.6 \pm 2.1$ (SD 0.8) at 48 and 76% VO$_{2\text{max}}$, respectively]. A much larger increase was observed when $^{13}$C-glucose was ingested with significantly greater values at 48 than at 76% VO$_{2\text{max}}$, respectively (Table 1). The $^{13}$C/$^{12}$C in plasma glucose also increased markedly and leveled off beginning at minute 30 and minute 15 at 48 and 76% VO$_{2\text{max}}$, respectively, with the mean value being significantly greater (main effect) at the lower work load (Fig. 1). These values were stable beginning at minute 30 and minute 15, respectively, in response to exercise at 48 and 76% VO$_{2\text{max}}$ (Fig. 1).

As shown in Table 2, the amount of exogenous glucose oxidized over the last 45 min of exercise was small and not significantly different between 48 and 76% VO$_{2\text{max}}$, providing only 1.4–2.6% of the energy yield (significantly greater at 48 than 76% VO$_{2\text{max}}$). The amounts of plasma glucose and muscle glycogen oxidized were both significantly greater at 76 than 48% VO$_{2\text{max}}$ (Table 2). However, although the percent contribution of muscle glycogen oxidation to the energy yield was significantly greater at 76 than 48% VO$_{2\text{max}}$, the contribution of plasma glucose oxidation was not significantly different between the two work loads. The respective contributions of the oxidation of the various substrates (fat, plasma glucose, and muscle glycogen oxidation) to the energy yield significantly increased with time while that of muscle glycogen significantly decreased.

Muscle glycogen content, which was not significantly different immediately before the beginning of exercise in the two experimental situations (Fig. 3), significantly decreased in response to exercise at the two work loads. The reduction observed from the beginning to the end of exercise was significantly greater in response to exercise at 76 than 48% VO$_{2\text{max}}$. Changes in muscle glycogen concentration were related to muscle glycogen oxidation computed by calorimetry and tracer technique across the two work loads ($r = 0.81, P <$

### Table 1. Gas exchanges (not corrected for protein oxidation) and $^{13}$C/$^{12}$C in breath CO$_2$ over the 75-min exercise period at 48% and 76% VO$_{2\text{max}}$

<table>
<thead>
<tr>
<th>Time</th>
<th>VO$_2$, l/min</th>
<th>VCO$_2$, l/min</th>
<th>RER</th>
<th>$^{13}$C/$^{12}$C, %δ $^{13}$CVPDB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting</td>
<td>2.18 (0.28)</td>
<td>1.98 (0.25)</td>
<td>0.91 (0.03)</td>
<td>$-23.3$ (1.7)</td>
</tr>
<tr>
<td>15 min</td>
<td>2.22 (0.27)</td>
<td>2.00 (0.24)</td>
<td>0.90 (0.02)</td>
<td>$-17.1$ (1.5)</td>
</tr>
<tr>
<td>30 min</td>
<td>2.19 (0.25)</td>
<td>1.90 (0.23)</td>
<td>0.91 (0.03)</td>
<td>$-11.8$ (2.8)</td>
</tr>
<tr>
<td>45 min</td>
<td>2.19 (0.21)</td>
<td>1.95 (0.25)</td>
<td>0.89 (0.02)</td>
<td>$-10.2$ (4.0)</td>
</tr>
<tr>
<td>60 min</td>
<td>2.19 (0.29)</td>
<td>1.94 (0.26)</td>
<td>0.88 (0.03)</td>
<td>$-7.9$ (2.7)</td>
</tr>
<tr>
<td>75 min</td>
<td>2.19 (0.29)</td>
<td>1.94 (0.26)</td>
<td>0.88 (0.03)</td>
<td>$-7.9$ (2.7)</td>
</tr>
</tbody>
</table>

Values are means (SD). VO$_2$ and VCO$_2$, oxygen consumption and carbon dioxide production, respectively; VO$_{2\text{max}}$, maximal oxygen uptake; RER, respiratory exchange ratio. $^{13}$C/$^{12}$C was expressed in % difference (δ) by comparison with the Vienna-PDB standard ($^{13}$CVPDB).
0.05), but at neither of the two work loads taken independently was the reduction in muscle glycogen concentration correlated with the amount of muscle glycogen oxidized (Fig. 4).

Plasma glucose concentration was stable over the exercise periods and not significantly different between 48 and 76% \( \dot{V}O_2 \text{max} \) [mean values over the exercise periods: 4.6 (SD 0.4) and 4.8 mmol/l (SD 0.9)]. Plasma insulin concentration was also not significantly different 48 and 76% \( \dot{V}O_2 \text{max} \): 11.8 (SD 6.5) and 13.9 mIU/l (SD 9.3) at rest before exercise, and 3.2 (SD 0.8) and 2.8 mIU/l (SD 0.9) at the termination of exercise, respectively.

**DISCUSSION**

In the present experiment, plasma glucose and muscle glycogen oxidation were computed during prolonged exercise by indirect calorimetry combined with tracer technique using ingestion of several small successive doses of glucose highly enriched in \(^{13}C\). Although the total amount ingested was small (14 g or 0.1 g/min over a 135-min period), exogenous glucose oxidation contributed to a portion of the plasma glucose flux (17% and 9% at low and high work loads, respectively) and could have increased muscle glycogen oxidation and decreased plasma glucose oxidation, respectively. Data obtained using the muscle biopsy technique indicate that carbohydrate ingestion during exercise could spare muscle glycogen stores, although this phenomenon is not regularly reported (see Ref. 31 for review). In contrast, except for the significant but small (3–5%) reduction in muscle glycogen oxidation reported by Campbell et al. (6), studies conducted using tracer technique consistently show that ingestion of carbohydrate over a wide range of rates (0.6–3 g/min) does not modify muscle glycogen oxidation (10, 11, 15, 17, 19, 20, 26, 36). In addition, in these studies, as well as in several other studies of plasma glucose turnover during exercise (e.g., 2, 4, 21), carbohydrate ingestion increased plasma glucose disappearance and oxidation in a dose-dependent manner. With large (1.5 g/min) (36) and very large doses of glucose ingested (∼3 g/min) (19, 20), despite a total or near-total inhibition of glucose release from the liver, plasma glucose oxidation was increased 1.8–2.5 times. With much smaller doses (∼0.6–0.83 g/min), exogenous glucose oxidation was largely (2, 19, 20) or totally (4) compensated by the reduction in liver glucose output, and plasma glucose oxidation only ranged between ∼1 and 1.4 times the value observed without carbohydrate ingestion. These data indicate that the very small rate of glucose ingestion used in the present experiment only minimally modified the metabolic response to exercise and that the oxidation rates of plasma glucose and muscle glycogen computed were good approximations of the values that would have been observed using trace amounts of labeled glucose.

In support of this hypothesis, the contributions of plasma glucose oxidation to the energy yield observed in the present experiment at low work load (15.1% at 48% \( \dot{V}O_2 \text{max} \)) is well in line with consistent results reported at 40–55% \( \dot{V}O_2 \text{max} \) in several studies using conventional tracer techniques (12–18%) (1, 12, 16, 19, 20, 22, 32, 35). The contribution of muscle glycogen oxidation to the energy yield observed in these studies is also well in line with the 12–18% value reported in the present experiment.

**Fig. 1.** Isotopic composition of plasma glucose during exercise at 48% and 76% maximal \( O_2 \) uptake (\( \dot{V}O_2 \text{max} \)). Values are means (SD). *Significantly different from the other values at the same work load (\( P < 0.05 \)).
studies varies more widely with extreme values as low as 25–30% (19, 20, 33) and as high as 66% (22). The contribution computed in the present experiment, which falls in the middle of this range (46%), is in accordance with data from Borghouts et al. (1) (41%), Horton et al. (16) (48%), and Friedlander et al. (12) (56%). Few data concerning the respective contribution of plasma and muscle glycogen oxidation to the energy yield at 70–75% \( \dot{V}O_2 \text{max} \) are available in the literature (3, 11, 32). The values observed in the present experiment at 76% \( \dot{V}O_2 \text{max} \) (15 and 60%, respectively) are well in line with those reported by van Loon et al. (32) during the third 30-min stage of a progressive exercise at 40, 55, and 75% \( \dot{V}O_2 \text{max} \). In the study by Bosch et al. (3), over a 180-min exercise period at 70% \( \dot{V}O_2 \text{max} \), the overall contributions of plasma glucose and muscle glycogen oxidation to the energy yield were 21 and 52%, respectively. However, in this study, the contribution of plasma glucose to the energy yield steadily increased from the beginning to the end of exercise (from 6 to 30%), while that of muscle glycogen steadily decreased (from 76 to 27%). This phenomenon was also observed by van Loon et al. (33, 35) at 60% \( \dot{V}O_2 \text{max} \), as well as in the present experiment at 76% \( \dot{V}O_2 \text{max} \) (Fig. 2). When computed between minute 30 and minute 75 from the data reported by Bosch et al. (3), the contributions of plasma and muscle glycogen oxidation (16 and 62%) were very close to those observed over the same period in the present experiment. Taken together, these comparisons suggest that the simplified tracer technique used in the present experiment to compute plasma and muscle glycogen oxidation during prolonged exercise, with ingestion of successive small doses of glucose highly enriched in \( ^{13} \text{C} \), is a valid alternative approach to the conventional techniques using infusion of labeled glucose.

Muscle glycogen utilization during prolonged exercise can be tracked from changes in muscle glycogen content in muscle biopsies taken before and after exercise, as well as during the exercise period (7, 31). However, there appear to be only five studies in which both changes in muscle glycogen concentra-

![Figure 3](image3.png)

Fig. 3. Glycogen concentrations in muscle biopsy samples taken from the vastus lateralis before and after exercise at 48% and 76% \( \dot{V}O_2 \text{max} \). Values are means (SD). *Significantly lower than the corresponding preexercise value. †Reduction in muscle glycogen concentration significantly different from at 48% \( \dot{V}O_2 \text{max} \) \((P < 0.05)\).

Results from the present experiment are in line with these previous data. The reduction in muscle glycogen concentration and muscle glycogen oxidation computed by tracer technique were both larger in response to exercise at 76% \( \dot{V}O_2 \text{max} \) and muscle glycogen concentration significantly decreased in response to the highest work load only. Similarly in healthy subjects (35) and in Type 2 diabetic patients (34), inhibition of adipose tissue lipolysis during exercise significantly increased muscle glycogen oxidation (as computed by tracer technique) but did not modify changes in glycogen concentration in mixed muscle (35) or in type I and II muscle fibers (34).

The absence of a close relationship between the amount of muscle glycogen oxidized computed by tracer technique, on the one hand, and changes in muscle glycogen concentration, and muscle glycogen oxidation computed by tracer technique have been simultaneously measured (3, 11, 32, 34, 35). In these studies, the correlation coefficients between the two sets of data were not reported, but changes in muscle glycogen concentration and muscle glycogen oxidation by tracer technique were not closely related. In the early study by Bosch et al. (3), the contribution of muscle glycogen oxidation to the energy yield computed by calorimetry and tracer technique was higher in subjects with high initial muscle glycogen stores (carbohydrate loaded) than in control subjects (nonloaded) (62 vs. 52%), but the reduction in muscle glycogen concentration was significantly higher in carbohydrate-loaded subjects over the second hour of the 3-h exercise only. In the study by van Loon et al. (34), muscle glycogen oxidation computed by tracer technique contributed 35, 38, and 58% to the energy yield during the three consecutive 30-min periods of exercise at 40, 55, and 75% \( \dot{V}O_2 \text{max} \), but muscle glycogen concentration significantly decreased in response to the highest work load only.

![Figure 4](image4.png)

Fig. 4. Muscle glycogen oxidation computed by calorimetry and tracer technique, plotted against changes in muscle glycogen concentration over the exercise periods at 48% and 76% \( \dot{V}O_2 \text{max} \). dw, dry weight. NS, not significantly different from 0 \((P < 0.05)\).
on the other hand, is due to the fact that these two approaches do not measure exactly the same phenomenon and that both have technical limitations. Muscle glycogen oxidation computed by difference between total carbohydrate and plasma glucose oxidation includes the amount oxidized through the lactate shuttle (5) but not that oxidized through the Cori and glucose-alanine cycles, which are included in plasma glucose oxidation (29). In contrast, this approach not only measures oxidation of glycogen stores in the working muscles but also in less active or inactive muscles, which can provide lactate to the working muscle (3). This phenomenon cannot be described from changes in muscle glycogen concentration in active muscles. Finally, plasma glucose turnover measured using [2H]- or [3H]glucose, has been shown to adequately reflect plasma glucose oxidation after ~60–90 min of exercise (3, 19). However, data from Bosch et al. (3) showed that over the first 90 min of a 180-min exercise period with and without carbohydrate ingestion, plasma glucose oxidation was much lower than plasma glucose turnover. In this situation, the rate of plasma glucose disappearance will overestimate plasma glucose oxidation and thus underestimate muscle glycogen oxidation. From a technical point of view, these errors can be avoided by using [13C]- or [13C]glucose and by computing plasma glucose oxidation from the enrichment of plasma glucose and labeled CO2 production, as in the present experiment. From a physiological point of view, the data from Bosch et al. (3) indicate that, although glycogen is broken down in active muscle fibers, glycogen synthesis could occur in nonactive muscles and/or in nonactive or less active muscle fibers in the working muscle. This phenomenon could lead to an underestimation of muscle glycogen utilization measured by changes in glycogen concentration in successive biopsies taken from an active muscle. Finally, the muscle fibers in the successive small biopsy samples taken in an active muscle are not necessarily representative of the entire population of fibers of the muscles involved in the exercise, both in terms of active vs. less active or inactive fibers and in terms of differential fiber composition. Taken together, these limitations and these sources of errors explain that muscle biopsy and tracer techniques do not always provide similar data concerning muscle glycogen utilization during prolonged exercise, for which there is unfortunately no gold standard. In this respect, it is worth mentioning that, as reviewed by van Loon et al. (33), because of similar limitations of muscle biopsy and tracer techniques applied to fat metabolism, there are also discrepancies between data concerning intramuscular triglyceride (IMTG) utilization computed, on the one hand, by difference between fat oxidation and the oxidation of plasma free fatty acids measured by calorimetry and tracer technique and, on the other hand, by the change in the IMTG concentration in successive muscle biopsies taken from an active muscle. In addition, van Loon et al. (33) did not observe any correlation between the reduction in IMTG in type I and/or type II fibers in biopsies from the vastus lateralis and triglyceride oxidation measured using [13C]palmitate administration and 13CO2 production.

In conclusion, the simplified tracer technique used in the present experiment, with the ingestion of small amounts of glucose highly enriched in 13C, appears to be a valid alternative approach to the traditional tracer techniques for computing plasma glucose and muscle glycogen oxidation during prolonged exercise. This technique could be useful in situations, or settings in which the infusion of labeled glucose could be a concern for ethical or technical reasons.

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