Quantifying the Immediate Recovery Energy Expenditure of Resistance Training

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Quantifying the Immediate Recovery Energy Expenditure of Resistance Training

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Abstract

Scott, CB. Quantifying the immediate recovery energy expenditure of resistance training. *J Strength Cond Res* 24(x): 000–000, 2010–As opposed to steady state aerobic-type exercise involving long duration, continuous, rhythmic, large muscle group activities that consume large volumes of oxygen, a resistance training set is brief, intermittent, uses multiple and isolated muscles, and is considered anaerobic in description. Because differences are evident between aerobic- and anaerobic-type exercise, it is proposed that the methods used for estimating resistance training energy expenditure should be different as compared with walking, jogging, cycling, etc. After a single set of weight lifting, for example, oxygen uptake is greater in the recovery from lifting as opposed to during the actual exercise; likewise, the anaerobic energy expenditure contribution to lifting may exceed exercise oxygen uptake. Recovery energy expenditure also does not appear well related to the anaerobic energy expenditure of the previous exercise. Based on this evidence, it is suggested that anaerobic-type exercise should not be based on aerobic-type models. In terms of excess postexercise oxygen consumption, a hypothesis is presented in regard to how non–steady-state energy expenditure in the immediate recovery from intense exercise should be properly quantified (e.g., in-between resistance training sets). The proposed concept is based on possible substrate or fuel use differences during intense exercise and aerobic recovery and the biochemistry and bioenergetics of glucose, lactate, and fat oxidation. It is proposed that immediately after a single weight lifting bout or in-between resistance training sets, as O_2 uptake plummets rapidly back toward pre-exercise levels, a separate energy expenditure conversion is required for recovery that differs from non–steady-state exercise, that is, 1 L of recovery oxygen uptake = 19.6 kJ (4.7 kcal) (not the standard exercise conversion of 1 L of oxygen uptake = 21.1 kJ) (5.0 kcal).

Key Words EPOC, oxygen debt, lactate oxidation, glycolysis

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Introduction

Strength, speed, and power invoke intense muscle contractions that recruit slow- and fast-twitch muscles. In response, metabolic contributions to exercise energy expenditure take place that are not well related to oxygen (O_2) uptake (i.e., aerobic metabolism). Intense contractions "pinch-off" blood flow and "squeeze" blood from working muscles that must subsequently rely on anaerobic metabolism (24). Moreover, intense resistance training as an anaerobic-type exercise is often performed to muscle fatigue, and exercise intensity and fatigue are associated with the volume of O_2 uptake consumed in recovery (1). It should not be much of a surprise then that analysis of and after 1 set of resistance training have shown that anaerobic exercise and recovery aerobic energy expenditures can exceed the aerobic exercise energy expenditure (20,22)–this is unheard of with steady-state aerobic-type exercise. The premise of this manuscript is that the energy expenditure of anaerobic-type exercise should not be based on aerobic models.

Increased O_2 consumption after exercise has been studied for almost 100 years. From elevated levels during exercise, the decline in O_2 uptake back toward rest takes on an exponential pattern of decline where both a fast-curve component (lasting minutes) and a slow-curve component (lasting hours if not days) have been demonstrated (see [3, p. 219]). What causes these 2 components? The fast curve is thought to be completed within a time frame of minutes, where a significant amount of the O_2 consumed is used to replenish the stored adenosine triphosphate (ATP) and creatine phosphate (CP) used during exercise (1,14). The rapid breakdown of glucose and glycogen during intense exercise results in lactate production, and excessive amounts of lactate was once thought to be a leading cause of the long term slow-curve O_2 component, taking several or perhaps many hours to return to resting levels (1,3,16).

From these beginnings, the rational of an *oxygen debt* was born; intense exercise borrows from energy expenditure reserves that are paid back in the form of an extensive O_2 uptake during the recovery from exercise (3,7). Although true for replenishing ATP and CP reserves, the amount of lactate produced during exercise has been shown to have little to do with the amount of O_2 consumed after exercise (7). As a result, it has been suggested that the term oxygen debt

be replaced with excess postexercise oxygen consumption (EPOC) in an attempt to eliminate a causal (lactate production) with effect (recovery O_2 uptake) relationship (3,7).

The long-term slow-curve component of EPOC needs to be studied after a complete workout and requires hours if not days of data collection. Many factors are involved with EPOC's continued presence until a true resting metabolic rate is once again obtained. Yet when pre-exercise metabolic rates are considered and with repeated sets of weight lifting, rest periods often last minutes and so too does EPOC (Table 1). Within this manuscript, an explanation is presented describing why the non–steady-state O_2 uptake found in the immediate recovery from a bout of resistance training requires a standard energy expenditure conversion that is different from that of non–steady-state exercise.

Steady-State and Non–steady-State Oxygen (O_2) Uptake

The respiratory exchange ratio (RER) is calculated as steady-state CO_2 produced divided by steady-state O_2 consumed and is typically defined from values of 0.70 representing total fat oxidation to 1.00 representing total glucose oxidation (6). Oxygen update measurements then undergo conversion to an estimate of energy expenditure based on the RER. The rapid exponential decline of recovery energy expenditure from elevated levels during exercise toward that of rest signifies a non–steady-state, when the RER may exceed 1.00 and exhaled carbon dioxide (CO_2) exceeds O_2 uptake (see Table 1). During and after exercise, RER values above 1.00 are generally thought to be the result of nonrespiratory CO_2 production: The bicarbonate buffering system, for example, involves the removal of hydrogen ions with concomitant CO_2 production and hyperventilation blows off "extra" CO_2. Yet a true measure of the RER is best found only when the system is in a steady state of gas exchange (6).

The plummeting of O_2 uptake in the immediate recovery from exercise, back toward resting levels, along with the rapid increases in exhaled CO_2, are well known after intense exercise and clearly portray a system in nonsteady state. The data in Table 1 reveal elevated RERs after a bout of the bench, wtih EPOC exceeding 1-minute in the very least and typically several minutes in duration. It is doubtful then that recovery energy expenditure measured within the first few minutes of exercise cessation is properly estimated using the O_2 uptake to energy expenditure conversions as dictated by the ratio of CO_2 produced to O_2 consumed. During non–steady-state exercise, energy expenditure is often estimated at a standard conversion of 1 L of O_2 uptake = 21.1 kJ signifying the use of glucose and/or glycogen as substrate, likewise for the recovery O_2 uptake (or O_2 debt or EPOC). Although correct for non–steady-state exercise that is likely dependent on glucose and/or glycogen as a substrate, this may be incorrect for non–steady-state recovery that may not.

Fat and Lactate Oxidation

Rapid glycolysis (as part of anaerobic metabolism) ceases when muscle contraction stops so that recovery is considered to be aerobic in nature ([26]; see [14]). If this is true, both fatty acid and lactate oxidation may play a significant role in fueling the immediate energy expenditure needs of recovery (1,3,8,16). Unfortunately, substrate oxidation immediately postexercise and particularly after anaerobic-type exercise has not been studied well enough to draw specific conclusions (16). Because of this, it must be assumed here that when muscle contraction immediately stops, glycolysis is limited to the point where fat and lactate are the predominantly oxidized fuels.

If so, and as demonstrated by all biochemistry texts, complete glucose oxidation consists of a composite of both anaerobic and aerobic metabolic pathways, accounted for as a measure of O_2 uptake that equals 21.1 $kJ \cdot L^{-1}$. To the contrary, fats are oxidized only via aerobic metabolism at an O_2 uptake of 19.6 $kJ \cdot L^{-1}$; note that there is no anaerobic component to fat oxidation. With rapid anaerobic metabolism, lactate, like fatty acids, is often transported to another part of a muscle cell or via the blood stream to other muscle cells where it too can undergo oxidation via aerobic

Table 1. Excess postexercise oxygen consumption data collection before and after 1 set of the bench press.*†‡

	No. of trials	Rest $\dot{V}O_2$	RER	EPOC time (s)	Range (s)
Lifts to fatigue	78	4.88 ± 0.76	1.46–1.20	327.8 ± 212.3	91–1,156
Nonfatigue lifts	70	4.71 ± 0.41	1.23–1.12	218.4 ± 142.2	71–920
Combined data	148	4.80 ± 0.62	1.35–1.16	276.1 ± 190.0	71–1,156

*EPOC = excess postexercise oxygen consumption; RER = respiratory exchange ratio taken from the first 15-second postexercise to the last 15 seconds of EPOC.

†Values are means ± *SD*.

‡Rest $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$) represents a 5-minute pre-exercise supine rest on the bench with feet on the floor. At the end of the single set, feet were elevated to body level for a completely supine recovery EPOC was recorded until 2 consecutive 15-second measurements fell at or below 5.0 $ml \cdot kg^{-1} \cdot min^{-1}$ (this represents a standing resting $\dot{V}O_2$ value [15]). Lifts to fatigue (22); nonfatigue lifts (20).

metabolism–this type of transport and subsequent oxidation is termed the *lactate shuttle* (2). As with fats, the oxidation of lactate has no anaerobic metabolic component. Unfortunately, the conversion of lactate oxidation to an aerobic energy expenditure estimate is not as straightforward in comparison to fat oxidation, being described by different references as 1 L of O_2 uptake = 19.8 kJ (4), 1 L of O_2 uptake = 21.1 kJ (24, p. E404) and mimicking basal conditions between 19.6 and 21.1 kJ·L^{-1} of O_2 uptake (6, p. 296). Because of this variability, greater precision in the format of a firm standard is required in recognizing a single aerobic-only energy expenditure conversion associated with both fat *and* lactate oxidations.

Biochemistry and Metabolism

The metabolic breakdown of glycogen and glucose has an energy expenditure estimated as 1 L of O_2 uptake = 21.1 kJ; fat oxidation has an energy expenditure estimated as 1 L of O_2 uptake = 19.6 kJ (5). Why the difference? This discrepancy is generally thought to be caused by the efficiency of each fuel; because glucose "produces" more heat per volume of O_2 consumed, it may be considered a more efficient fuel as compared to fat. The reference of Marsh and Murlin in 1928 reveals the idea of substrate efficiency predating the discovery of aerobic and anaerobic biochemistry (in the late 1930s and early 1940s) (13). Although the numerical difference between glucose and fat oxidation–1.5 kJ–is most certainly true, factors other than substrate efficiency are clearly at play and need to be addressed. Chief among these is the acknowledgment of those energy exchanges inherent to the biochemical pathways associated with substrate-level phosphorylation (i.e., glycolysis) and oxidative phosphorylation (i.e., mitochondrial respiration) (9,10,20).

It is the measurement of O_2 uptake (indirect calorimetry) not the measurement of energy (heat) exchange (direct calorimetry) that dictates our current methodology in the estimation of energy expenditure. As an example, the 'biological' breakdown of glucose to CO_2 and water (H_2O) consists of anaerobic and aerobic metabolism as 1 L of O_2 uptake = 21.1 kJ. What portion of the 1 L of O_2 uptake = 21.1 kJ consists of the anaerobic substrate-level phosphorylation component? Consider for the moment that anaerobic and aerobic metabolisms were quantified in terms of the amount of ATP resynthesized in comparison to energy expenditure, not in terms of O_2 uptake. The 1.5-kJ difference (per L of O_2 uptake) between 21.1 and 19.6 kJ at approximately 7% is a similar percentage to the anaerobically resynthesized 2–3 ATP of complete glucose–glycogen breakdown (at 34–36 total ATP). Matching the 7% of total oxidative energy (kJ) loss with the 7% of total ATP resynthesized via substrate-level phosphorylation promotes the hypothesis that for the complete breakdown of 1 glucose molecule, 1.5 kJ of 21.1 kJ represents the anaerobic phase of glucose breakdown (per liter of O_2 uptake) (Figure 1) (20,22). Dismissing the anaerobic metabolic component to substrate breakdown reveals a consistent all-aerobic estimation of energy expenditure at 1 L of O_2 uptake = 19.6 kJ.

Bioenergetics and Energy Exchange

Scientists quantify the approximate internal energy of molecules by burning them, with the resultant heat loss described as the enthalpy change (ΔH). Combustion is

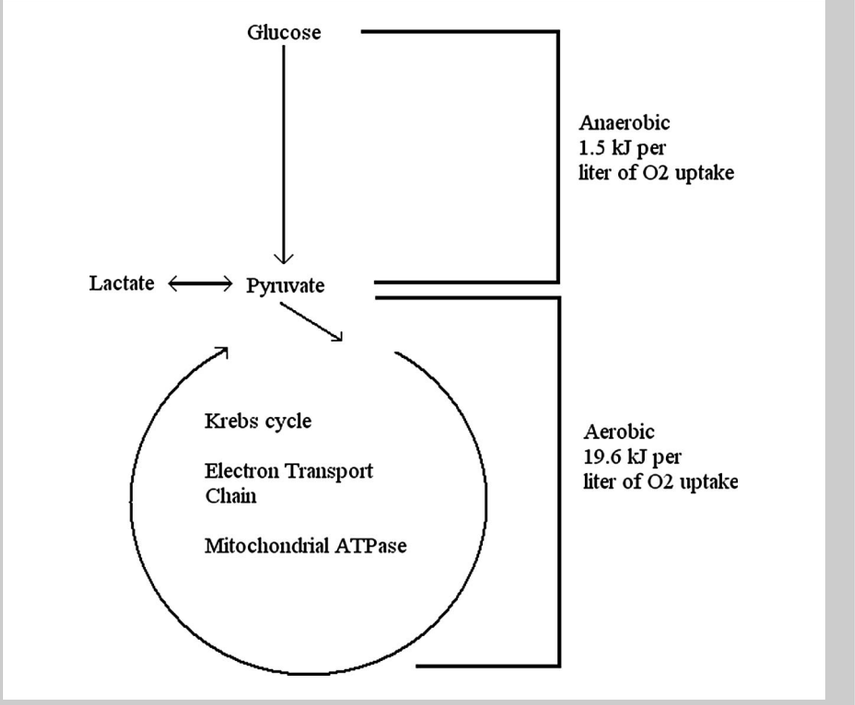

Figure 1. Glucose degradation consists of both anaerobic glycolysis and aerobic respiration where energy expenditure is estimated as a composite measure at 1 L of O_2 uptake = 21.1 kJ. This figure suggests a 1.5 kJ anaerobic component and a 19.6 aerobic component for complete glucose breakdown per liter of O_2 uptake. Fat oxidation is entirely aerobic at 1 L of O_2 uptake = 19.6 kJ. By removing the anaerobic component (at 1.5 kJ) from complete glucose oxidation (at 21.1 kJ), excess postexercise oxygen consumption (EPOC) can be viewed as an entirely aerobic process (at 19.6 kJ) that in no way represents lactate production or removal as the O_2 debt hypothesis can imply.

all-aerobic (as opposed to biological glucose oxidation that contains an anaerobic component) being described by fire scientists in the context of a remarkably consistent relationship between changes in enthalpy during the immediate exchange of electrons from reactants (fuel + O_2) to products (CO_2 + H_2O) (11,18): -111 kJ·mol^{-1} of electrons.

Put another way, heat loss per electron transfer is similar regardless of the fuel undergoing combustive oxidation. This statement is known as Thornton's law and can be rewritten in the context of O_2 uptake as (25): 1 L of O_2 uptake = 19.6 kJ.

It needs to be noted that the above all-aerobic energy expenditure conversion also represents fat oxidation and, unlike fire scientists, exercise physiologists and nutritionists often interpret this conversion as being very different from glucose oxidation at 1 L of O_2 uptake = 21.1 kJ. There certainly is a slight difference in enthalpy between glucose and fat undergoing combustion (as mentioned earlier using the term efficiency), representing only the substrates themselves. Both the substrate and the "mechanism" of metabolic conversion need to be considered as part of energy exchange. But even so, it also must be recognized that anaerobic metabolism too results in energy exchanges as glycolysis (substrate-level phosphorylation) takes place and ATP is resynthesized (9,10,17).

Energy exchange not only results in change from one form to another but also from one place to another. Displacement also comes at a cost, also at less than perfect efficiency (9,10). The measure of how energy is spread out or dispersed within a system (such as a molecule of fuel or along a metabolic pathway) is known as entropy (S) (see [19]). Yet one would be hard pressed to find entropy data (ΔS) within a biochemistry text as glucose or fat undergoes breakdown along the metabolic pathways. Biochemistry rightly tends to focus on Gibbs energy changes (ΔG) because these changes are measurable, and they are paramount in determining what metabolic reactions are and are not spontaneous. Even so, it is important to recognize that changes in enthalpy (ΔH) and entropy (ΔS) also provide valuable information to any act of metabolism that invokes energy exchange. Minikami and de Verdier (17) have provided ΔH, ΔS, and ΔG for reactions along the glycolytic pathway and came to a rather startling revelation: *under aerobic conditions glycolysis is entropy driven* (i.e., associated with ΔS); under anaerobic conditions glycolysis is enthalpy driven (i.e., associated with ΔH). Because differences in fat and glucose oxidation are typically thought of in terms of substrate efficiency or Gibbs energy changes, neither enthalpy nor entropy are, respectively, measured or calculated when using O_2 uptake to estimate anaerobic energy expenditure; in other words, glycolysis, as a mechanism of energy exchange typically does not undergo consideration as being part of a 1 L of O_2 uptake = 21.1 kJ conversion. However, by recognizing both the enthalpy and entropy changes associated with energy exchange (9,10), the anaerobic energy expenditure component to complete glucose oxidation can be hypothesized at 1.5 kJ·L^{-1} of O_2 uptake; 21.1 kJ is again acknowledged here as a composite of anaerobic (1.5 kJ) and aerobic (19.6 kJ) metabolisms per liter of O_2 uptake (Figure 1) (19,21).

Excess Postexercise Oxygen Consumption and the O_2 Debt

The usefulness of a hypothetical estimation and separation of the anaerobic and aerobic metabolic components that are part of an O_2 uptake measurement are readily found when considering the O_2 debt hypothesis where it was implied that O_2 uptake in the recovery from exercise could be used to represent anaerobic energy expenditure associated with lactate production and/or removal. It is in no way too far fetched to consider that if all lactate produced as a result of exercise was removed via recovery cellular oxidation (i.e., conversion to CO_2 and H_2O), then the 1.5-kJ phase of glucose degradation could later be represented within a 1 L of O_2 uptake = 21.1 kJ conversion. That is, if *all* lactate was eventually oxidized. Unfortunately, lactate removal is too complex a subject to be considered only in the context of oxidation (3,7). Although a significant amount of lactate is later oxidized as a fuel, it also can be converted into glucose, glycogen, amino acids, and proteins, in addition to being found in sweat, urine, and saliva. The term EPOC was therefore suggested as a *qualitative* term to dissociate recovery O_2 uptake from lactate production and removal. It is suggested here that complete *quantitative* dissociation only comes by removing the glycolytic component (1.5 kJ) from a measure of EPOC in the immediate recovery from exercise and to do so requires using an all-aerobic conversion where 1 L of O_2 uptake = 19.6 kJ; indeed, this may the most significant rationale for its use.

The limited stores of the high energy phosphates–ATP and CP–provides a vivid example of quantitative-type dissociation, where they are used during intense exercise but resynthesized as part of recovery energy expenditure. Exercise scientists can choose to use the O_2 deficit at the start of exercise or, EPOC in recovery to help estimate the energy expenditure associated with ATP/CP use, but they cannot use both because this would account for ATP/CP use twice. Similarly, blood lactate levels have been used for over 4 decades to help estimate the glycolytic (anaerobic) energy expenditure component of intense exercise (*but only when lactate production exceeds* removal. Exercise scientists adamantly opposed to the use of blood lactate as a means to estimate anaerobic energy expenditure can cite lactate removal exceeding lactate production as proof; this is most certainly true. Yet, it is also another example of aerobic-type exercise serving as an inappropriate model for anaerobically fueled activity, when lactate production does in fact exceed removal (5,12). The 1 L of O_2 uptake = 19.6-kJ conversion dismisses from EPOC the reaccounting of any glycolysis-to-lactate associated ATP that was resynthesized during exercise.

Practical Applications

Exercise scientists can better bridge the gap between science and application by recognizing that aerobic-type and anaerobic-type activities are different from several fundamental standpoints. Because of its intermittent nature, the energy expenditure contributions of weight training are almost always in a nonsteady state. To the contrary, aerobic-type exercise is defined as being steady state. Because of the intense nature of weight lifting, anaerobic and recovery energy expenditures can exceed that of exercise O_2 uptake; they therefore represent an essential component of the total energy expenditure of weight lifting. The energy expenditure of recovery is essentially all-aerobic, lactate and fat appear to be the major fuels oxidized in recovery (neither consists of anaerobic metabolic breakdown), non–steady-state O_2 uptake plummets rapidly, whereas CO_2 production appears to surge immediately in recovery, and EPOC has been suggested to not represent glycolytic ATP resynthesis; all of these concepts are effectively acknowledged by a single recovery energy expenditure conversion of 1 L of O_2 uptake = 19.6 kJ. This conversion can be used as a standard means of quantifying (aerobic) recovery energy expenditure immediately after a single bout of weight lifting or in-between sets of weight lifting.

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