



Journal of Exercise Physiology **online** (JEP **online**)

Volume 11 Number 4 August 2008

Metabolic Responses to Exercise

Managing Editor

Tommy Boone, Ph.D.

Editor-in-Chief

Jon K. Linderman, Ph.D.

Review Board

Todd Astorino, Ph.D.

Julien Baker, Ph.D.

Tommy Boone, Ph.D.

Lance Dalleck, Ph.D.

Dan Drury, DPE.

Hermann Engals, Ph.D.

Eric Goulet, Ph.D.

Robert Gotshall, Ph.D.

M.Knight-Maloney, Ph.D.

Len Kravitz, Ph.D.

James Laskin, Ph.D.

Derek Marks, Ph.D.

Cristine Mermier, Ph.D.

Daryl Parker, Ph.D.

Robert Robergs, Ph.D.

Brent Ruby, Ph.D.

Jason Siegler, Ph.D.

Greg Tardie, Ph.D.

Chantal Vella, Ph.D.

Lesley White, Ph.D.

Ben Zhou, Ph.D.

Official Research Journal
of The American Society of
Exercise Physiologists
(ASEP)

ISSN 1097-9751

THE METABOLIC OXIDATION OF GLUCOSE: THERMODYNAMIC CONSIDERATIONS FOR ANAEROBIC AND AEROBIC ENERGY EXPENDITURE

CHRISTOPHER SCOTT¹, ZORAN DJURISIC²

¹University of Southern Maine, Gorham, USA

²University of California, Berkeley, USA

ABSTRACT

Scott CB, Djurisic ZM. The Metabolic Oxidation of Glucose: Thermodynamic Considerations for Anaerobic and Aerobic Energy Expenditure. *JEPonline* 2008;11(4):34-43. When estimated per liter of oxygen uptake, the energy expenditure equivalent of fat oxidation is 19.6 kJ, for glucose oxidation 21.1 kJ. The purpose of this manuscript is to answer the question, why the 1.5 kJ (7%) difference? Our answer acknowledges the slight variation in the enthalpy of molecular bond formation that is found as different fuels undergo oxidation. However, this difference usually varies by $\pm 5\%$. When expressed per liter of oxygen consumed, we interpret the slightly larger 7% difference (1.5 kJ) found between the heat produced by fat and glucose oxidation in terms of the inefficiency of the biochemical pathways that break these two substrates down, in addition to the oxidation of the substrates themselves. Fat oxidation is entirely aerobic at 19.6 kJ per liter of oxygen uptake. Complete glucose oxidation has both an anaerobic and aerobic metabolic component. Because both aerobic and anaerobic metabolisms are inefficient (resulting in changes in both heat and entropy), we interpret the 1.5 kJ L⁻¹ O₂ difference between fat and glucose oxidation, in part, as being the result of the anaerobic metabolic component in the degradation of glucose. This separation allows for both an estimate of anaerobic and aerobic energy exchange during exercise and, aerobic energy exchange during recovery (excess post-exercise oxygen consumption, EPOC).

Key Words: Metabolism, Glycolysis, Bioenergetics, Oxygen Uptake, Oxygen Debt.

INTRODUCTION

The stoichiometric relationship of substrate utilization and gas exchange (RER) is well known (1) (Table 1). A question that can be asked is: When expressed per liter of oxygen consumed, why the difference in the 19.6 kJ and 21.1 kJ conversion factors? Through the presented analysis we will attempt to partly dismiss as an explanation the concept of substrate efficiency, as measured by for example less heat lost along with increased VO_2 (oxygen uptake) for fat utilization as compared to glucose. Instead, the difference will be rationalized in terms of thermodynamic concepts of anaerobic and aerobic energy exchange along the metabolic pathways. Human bias is evident in that exercise energy expenditure of all types – both aerobic and anaerobic - has been and often continues to be represented solely by measurements of oxygen uptake. Interestingly enough, our understanding of energy expenditure did not start out this way.

Table 1. Gas exchange (RER) and stoichiometry of substrates

Substrate	RER	Stoichiometry	Heat (kJ/LO_2)
Fat (palmitate)	0.70	16 CO₂: 23 O₂	19.6
Carbohydrate (glucose)	1.00	6 CO₂: 6 O₂	21.1

A Brief History of Energy Expenditure Bias

Two thousand years ago Greek thought held that heat was a prerequisite to life (2). Sixteen hundred years later the Belgian investigator Jean Baptiste van Helmont correctly declared things the other way around, life produced heat. The making of wine, the heat resulting from the fermentation process, was cited as part of his proof (fermentation is of course a non-oxygen related –anaerobic- act of glucose breakdown). A few hundred years later Louis Pasteur directed his talents toward the study of fermentation and recognized this act as being biological (complex) in nature, it was not the simple process of chemical disintegration thought by others. Eduard Buchner received the 1907 Nobel Prize when he demonstrated how the complexity of fermentation, under the appropriate conditions, could take place equally well in both living yeast and the test tube. A. Harden and W.J. Young first recognized the importance of inorganic phosphate (Pi) not as a part of ATP, but as part of the fermentation process (in 1913). By 1940 biochemical history was quite literally made when the Embden-Meyerhoff metabolic pathway was revealed in its entirety. “Indeed, the development of biochemistry and the delineation of this pathway went hand in hand” (p. 349) (3). Biochemistry had anaerobic origins. Yet, as magnificent as these achievements were, the independence of anaerobic glycolytic energy exchange was soon to be displaced.

By the mid-20th century mitochondrial studies and oxygen uptake measurements grew prominent (see 4), quickly overshadowing previous anaerobic discoveries. Animal and human energy exchanges were shown to adhere to proposed thermodynamic laws, biological heat production and oxygen uptake were proportional (under specific conditions) and a measurement of oxygen uptake became the “official” estimate of energy expenditure (5). Succinctly put, anaerobic (glycolytic) energy exchange lost its spotlight and terminology followed suit: anaerobic energy expenditure at the start of exercise was described not as a period of anaerobic metabolic contributions, but instead in the context of an *oxygen deficit* (6); anaerobic energy expenditure throughout exercise was quantified as part of an *oxygen debt* (7).

Table 2. METs levels for selected activities

Activity	METs
Weight training (light)	3.0
Calisthenics	4.0
Gymnastics	4.0
Golf (no cart)	4.5
Outside painting	5.0
Walking (4 mph)	5.0

In the above context, METs represent a multiple of resting metabolic rate; that is, as a relative “measure” of intensity. In other descriptions METs have been translated as 1 kcal per minute so that it can be viewed as also having a duration component (27).

uptake: 1 MET = 1x resting energy expenditure, 2 METs = 2x resting energy expenditure, 3 METs = 3x resting energy expenditure and so on. Upon close examination, does painting outside really have a greater peak energy expenditure than calisthenics (push-ups, sit-ups and the like)? Does golfing increase metabolic rate to a greater extent than gymnastics events (floor exercise, uneven bars, balance beam, vault, etc) or, are semantics to be blamed? If Table 2 also were to include an estimate of anaerobic energy equivalents (Table 3), then the MET values would likely be higher for exercise and activity that contains an anaerobic component (i.e., weight training, calisthenics, gymnastics) It has become apparent that the methodology of measuring oxygen uptake to estimate the energy expenditure of long duration aerobic-type exercise with recovery can not be used to account for the energy expenditure of brief, heavy to severe, anaerobic-type exercise with recovery (8).

Substrate (In-) Efficiency?

In the 19th century it was known that the heat equivalent of oxygen uptake was larger for carbohydrates than for fats; 21.1 kJ/L O₂ for glucose, 19.6 kJ/L O₂ for fat (see 9). It is important to understand however that these equivalents were founded a century before the biochemical pathways of aerobic and anaerobic metabolism were elucidated. Perhaps as a result, explanations of the difference in heat release from fat and carbohydrate oxidation have often been provided solely in terms of “substrate efficiency” (10). Carbohydrates are a more economical fuel as compared to fat when interpreted via an oxygen uptake measurement, having 7% more energy per liter of oxygen consumed:

Equation 1:

$$h = \frac{\Delta_r H(\text{ox., glu cos e}) - \Delta_r H(\text{ox., fat})}{\Delta_r H(\text{ox., glu cos e})} = \frac{21.1 \text{ kJ/L O}_2 - 19.6 \text{ kJ/L O}_2}{21.1 \text{ kJ/L O}_2} = 7\%$$

Where h = efficiency; $\Delta_r H$ = enthalpy of reactant; ox. = oxidation

This explanation of the difference in heat release as substrate efficiency is somewhat though not completely false. As will be demonstrated, per liter of oxygen consumed the difference between fat

Anaerobic substrate phosphorylation (glycolysis) along with Peter Mitchells brilliant Nobel winning explanation of chemo-electrical energy conversion within mitochondria, two very distinct methods of biological energy exchange, continue to be represented as a singular interpretation of energy exchange from 6- (e.g., glucose) and 3-carbon (e.g., pyruvate, lactate) substrates. That is, as one liter of oxygen uptake generating 21.1 kJ (~5 kcal) of heat during the oxidation of glucose or lactate. Clearly however, some forms of exercise (activity) contain both an aerobic and anaerobic energy expenditure component. As Table 1 demonstrates oxygen-only interpretations of energy expenditure can be misleading. MET values portray energy expenditure in multiples of resting metabolic rate, measured as O₂

and glucose metabolism also can be explained by examining the metabolic (biochemical) pathways involved in substrate breakdown and energy exchange by the system, not just the oxidation of the substrate itself.

Aerobic and Anaerobic Energy Exchange

The term “anaerobic” means without air, loosely interpreted as without oxygen. This point needs to be emphasized because the estimation of energy expenditure is now dominated almost exclusively by oxygen uptake measurements, not heat loss. Certainly and importantly, aerobic mitochondrial ATP re-synthesis uses oxygen and results in heat loss because of inefficient energy exchange (5). Yet energy exchange is an imperfect (and irreversible) process for both aerobic and anaerobic metabolisms as dictated by the 2nd law of thermodynamics; whenever energy undergoes exchange, energy is lost to the environment. The energy exchanges of anaerobic glycolytic substrate level ATP re-synthesis are in-efficient, just as they are for mitochondrial ATP re-synthesis (3,11).

Biochemists have long recognized the quantification of Gibbs energy changes (i.e., ΔG) as a monumental achievement. Changes in Gibbs energy identify the potential and the amount of energy exchange for both chemical and biochemical reactions as a composite of enthalpy (ΔH) and entropy (ΔS) changes (in more rigorous format entropy is related to temperature as, $T\Delta S$):

Equation 2:

$$\Delta G = \Delta H - T\Delta S$$

So what “drives” Gibbs energy exchanges during glycolysis? Measurements and calculations of Gibbs energy (ΔG), enthalpy (ΔH) and entropy (ΔS) during the breakdown of glucose reveal that, “...glycolysis is driven mainly by the entropy term [ΔS ; glucose-to-pyruvate] until the last step and the reaction is driven by the enthalpy term [ΔH ; pyruvate-to-lactate]” (11, p. 460). The message here is an important one, changes in either entropy or enthalpy or both can drive a (bio)chemical reaction forward. During the breakdown of glucose-to-pyruvate for example, anaerobic energy exchange is revealed mostly by overall entropy change (ΔS) not heat exchange. Unlike oxygen uptake and heat loss, entropy changes are calculated not measured and as such are generally not considered or applied to the costs of living (e.g., exercise and activity). Because heat is produced only by lactate

Table 3. Hypothetical anaerobic and aerobic MET levels for selected activities

Activity	Aerobic METs	Anaerobic METs	Total
Weight training (light)	3.0	2.0	6.0
Calisthenics	4.0	2.0	6.0
Gymnastics	4.0	3.0	7.0
Golf (no cart)	4.5	0.0	4.5
Outside painting	5.0	0.0	5.0
Walking (4 mph)	5.0	0.0	5.0

Just as the metabolic oxidation of glucose contains an anaerobic energy exchange component, so too does the energy expenditure of brief intense exercise and activity. Like Table 1 the above table concerns peak metabolic elevation not metabolic duration. The addition of an anaerobic component to the traditional all-aerobic MET value serves to identify this increase to metabolic intensity. These anaerobic values are hypothetical and do not represent true measurements

formation the breakdown of glucose-to-lactate is reflected by a measurement of heat (i.e., enthalpy change, ΔH) (11). As will be demonstrated, the acknowledgement and application of both enthalpy (ΔH) and entropy (ΔS) driven changes during glycolytic ATP re-synthesis better defines anaerobic energy exchange, especially when portrayed as a measure of oxygen uptake (12):

Equation 3:

$$\Delta G = \Delta H + \Delta S; \text{ glucose-to-pyruvate (entropy driven)}$$

Equation 4:

$$\Delta G = \Delta H + \Delta S; \text{ pyruvate-to-lactate (enthalpy driven)}$$

Anaerobic and aerobic metabolisms are very different in regard to their respective mechanisms of energy exchange. Energy exchange via anaerobic substrate level phosphorylation (the glycolytic use of phosphate shifts and transfers) has nothing in common with mitochondrial aerobic energy exchange involving the Krebs cycle, electron transport chain, chemiosmotic (H^+) gradients, and membrane bound ATPases (though the Krebs cycle does contain one act of substrate level phosphorylation). As most all biochemistry texts demonstrate, complete glucose oxidation is comprised of both anaerobic and aerobic metabolism; anaerobic glycolysis precedes the aerobic oxidation of pyruvate. Thus, the energy exchange of complete glucose oxidation, when evaluated by an oxygen uptake measurement, is greater than what one may conclude from studying the aerobic part of the process alone (i.e., mitochondrial respiration/oxidation). Again, the earliest measurements of oxygen uptake and carbon dioxide production were not associated with both biochemical and thermodynamic analyses (10) so it went unrecognized, and is sometimes still overlooked, that anaerobic glycolysis provides a “natural energy expenditure extension” to oxygen related energy expenditure. The “anaerobic extension” to the mitochondrial oxidation of pyruvate comes in the form of entropy.

The common conversion of 1 liter of O_2 uptake to the energy release of 21.1 kJ per liter of O_2 uptake during glucose oxidation is due in part to the anaerobic component of glucose degradation. Our working hypothesis acknowledges the anaerobic metabolic component of the reactant glucose whether glycolysis is represented as a change in entropy (ΔS) or a change in enthalpy (ΔH), respectively:

Equation 5:

$$1 \text{ liter of oxygen uptake} = \frac{\text{anaerobic metabolism}}{\text{glucose} \rightarrow \text{pyruvate}} + \frac{\text{aerobic metabolism}}{\text{pyruvate} \rightarrow \text{CO}_2 \text{ and H}_2\text{O}}$$

Thornton's law: Combustion

When expressed per unit of oxygen consumed there should be little variation ($\pm 5\%$) in heat release during the aerobic oxidation of glucose and fat. Combustion is an all-aerobic process and has no anaerobic component. The relationship between oxygen uptake and heat production during combustion (i.e., total aerobic oxidation) was first published in 1917 by William Thornton (13). His results, produced from a variety of combustible gases, depicted a relationship so straight forward it became known as Thornton's Law (or Thornton's rule).

While Thornton's data depicted gas combustion, his rule applies to most combusted liquids and solids. Thornton revealed that the heat loss of many combusted substances is found to be remarkably

correlated with the amount of oxygen consumed (14). Thus, as C-H (carbon-hydrogen) and C-C (carbon-carbon) bonds in fuel are broken and the bonds with oxygen, creating carbon dioxide (CO₂) and water (H₂O) are formed, a similar amount of heat is produced regardless of the source of those electrons; the data is dispersed within $\pm 5\%$. The slight variance that is seen in combustive heat production as bonds are formed with oxygen from different fuel sources is dependent on the *proportion* of the types of bonds within the fuel molecules (e.g., single or double carbon-carbon bonds, carbon-hydrogen bonds, carbon-oxygen bonds). The larger the molecule (e.g., fat) the more consistent the proportion of the different types of bonds tend to be. For smaller molecules (e.g., glucose) the relative impact of a bond to the total chemical energy of a molecule is larger, and thus results in a greater variability in oxygen uptake – heat production relationships. The combustive sciences use Thornton's law to measure oxygen uptake and estimate energy expended as (14):

Equation 6:

$$\Delta H = -450 \pm 23 \text{ kJ per mole of O}_2$$

Thornton's law: Respiration

Antoine Lavoisier suggested over 200 years ago that respiration and combustion were similar processes in regard to heat production and oxygen consumption. He has never been disproven and likely will never be. Based on microcalorimetry of mammalian cell lines, the oxycaloric equivalent for all-aerobic respiration (that contains no anaerobic metabolic component) is slightly higher than combustion (Equation 7) (15-16):

Equation 7:

$$\Delta H = -470 \pm 24 \text{ kJ per mole of O}_2$$

The 20 kJ mol O₂⁻¹ (4%) difference between combustion (at -450 kJ mol O₂⁻¹) and respiration (at -470 kJ mol O₂⁻¹) is the result of non-ATP related "side reactions" within living tissue where noticeable heat production is found (acid-base reactions for example) (15-16). As with combustion, the respiratory oxycaloric equivalent of -470 kJ mol O₂⁻¹ is thought to be remarkably consistent ($\pm 5\%$) for a variety of living tissues undergoing full aerobic respiration.

To use measured oxygen uptake to estimate energy expenditure during biological oxidation (all-aerobic mitochondrial respiration), the oxycaloric equivalent is more convenient when expressed per liter of oxygen. At 0°C and atmospheric pressure a mole of oxygen occupies 22.4 L. Using the ideal gas law the oxycaloric equivalent can thus be converted to Thornton's standard (Equation 8) (17-18):

Equation 8:

$$\Delta H = 19.6 \text{ kJ/L O}_2$$

Most would recognize that the above equivalent is based on fat oxidation. While this is certainly true, it also implies an oxygen to energy expenditure equivalent that does not contain an anaerobic energy exchange component; fat oxidation is all-aerobic.

An estimate of biological energy exchange based on glucose oxidation results from ATP re-synthesis by both aerobic (~32-33 molecules of ATP) and anaerobic (~2-3 molecules of ATP) metabolic components (~ 34-36 total ATP per glucose molecule). Using this information we reason that 1.5 kJ of energy expenditure – as a composite of entropy and enthalpy changes - per liter of oxygen consumed is associated not only from the different proportion of the types of bonds found between oxidized

glucose and fats, but also from the anaerobic metabolic energy exchange of glucose-to-pyruvate (i.e., anaerobic substrate phosphorylation) (Equation 9):

Equation 9:

$$19.6 \text{ kJ aerobic energy exchange} + 1.5 \text{ kJ anaerobic energy exchange} = 21.1 \text{ kJ (total energy exchange per L O}_2\text{)}$$

In Table 2, METs are portrayed not as an estimate of oxygen uptake but rather as a composite of aerobic and anaerobic energy expenditure, anaerobic glycolysis being a “natural extension” to the oxygen uptake of a bout of brief intense exercise (12).

Exercise and Recovery Energy Expenditure

As any historian of the exercise sciences can recall, anaerobic glycolytic energy expenditure has traditionally and incorrectly been portrayed as part of recovery oxygen uptake where $1\text{L O}_2 = 21.1 \text{ kJ}$ (i.e., oxygen debt methodology). Because of the difficulties in accounting for anaerobic energy expenditure, exercise scientists usually resort to measuring only one or two of the three components of total energy expenditure:

- 1. Aerobic exercise energy expenditure** - lacking anaerobic exercise and aerobic recovery measurements.
- 2. Aerobic and anaerobic exercise energy expenditure** - lacking an aerobic recovery measurement.
- 3. Aerobic exercise and aerobic recovery energy expenditure** - lacking an anaerobic exercise measurement.

Aerobic energy expenditure is rather easily estimated as a measure of oxygen uptake but it needs to be kept firmly in mind that heat (energy) (in kJ) is the standard reference, and entropy too is associated with energy exchanges. Using a variety of mammalian cell lines, Gnaiger and Kemp measured a proportional increase in enthalpy that accompanies lactate production (pyruvate-to-lactate is enthalpy driven) (16). Do measurements of lactate account for anaerobic glycolytic energy expenditure? Many contemporary exercise physiologists view *a priori* that lactate concentrations in muscle and blood can not be used to quantify anaerobic glycolytic energy expenditure. This rationale most likely arises from the detailed analyses over the years of an accelerated lactate removal as opposed to lactate production *during* longer duration aerobic-type exercise. Ole Bang demonstrated this some 70 years ago (19-20). Under these conditions – when lactate oxidation exceeds lactate production - it is indeed true that a measurement of blood lactate can not be used to estimate anaerobic glycolytic energy expenditure.

Bang’s analysis went further however. Bang demonstrated that lactate concentrations always peaked in the *recovery* from brief intense exercise. In the 1960s Margaria et al. clearly revealed the usefulness of a measure of recovery blood lactate concentrations in the estimation of anaerobic energy expenditure for brief treadmill exercise where linear relationships were found with blood lactate concentration and work rate (21). The same is true for brief bouts of cycling and swimming (22). Di Prampero and Ferretti (22) may have best expressed the usefulness of lactate peaking in recovery by stating, “Clearly enough, [?blood lactate] is not the energetic equivalent of lactate formation in the working muscles and does not yield any direct information on the stoichiometric relation between lactate formation and ATP re-synthesis. It is nevertheless a very useful quantity allowing us to determine the energy release in the body whenever the blood lactate concentration increases by a given amount...” (22, p. 109). The energy expenditure characteristics of long duration

endurance-related exercise (where lactate oxidation may be equal to or exceed lactate production) do not appear to mimic that of brief strength-related exercise (when lactate production exceeds lactate oxidation). It is of interest to determine how or if EPOC is “different” for bouts of intense weight training as compared to EPOC after lower intensity steady state exercise (23). Whether this is true or not, limitations to the use of a blood lactate measurement to estimate anaerobic glycolytic energy expenditure are profound. The exercise or activity must be brief, intense and probably non-intermittent in nature if lactate concentrations are to be converted into a reasonable estimate of anaerobic energy expenditure.

The oxygen debt hypothesis reasoned that the amount of lactate oxidized in the recovery from exercise can determine anaerobic glycolytic energy expenditure precisely because the 21.1 kJ per liter of O₂ uptake conversion contains a glycolytic (1.5 kJ) component (whether 6- or 3-carbon substrates are oxidized). Obviously enough, the anaerobic glycolytic component can not be accounted for twice: once during exercise (as estimated by blood lactate) and twice as part of recovery oxygen uptake. Our analysis allows for the separation of anaerobic glycolytic energy expenditure (1.5 kJ) from mitochondrial energy expenditure (19.6 kJ per liter of O₂ uptake). Most importantly, the entropy and heat exchanges of rapid anaerobic glycolysis are not reversible in open biological systems; they are not later “taken in” or “taken back” by mitochondria during lactate oxidation and so are not accounted for by recovery energy expenditure or EPOC that contains an anaerobic component at 21.1 kJ per liter of O₂ uptake (24).

How can the anaerobic glycolytic component of exercise energy expenditure as represented by a blood lactate measurement be excluded from the estimation of aerobic energy expenditure (oxygen uptake) during the recovery from exercise? The anaerobic glycolytic component can be dismissed with an all-aerobic conversion (12, 25), that is as 1 liter O₂ = 19.6 kJ when recovery is fueled mostly by the oxidation of fat and lactate but not glucose (26). (Use of the muscles limited stores of ATP and creatine phosphate (CP) does represent a type of oxygen debt because these stores are utilized “anaerobically” during exercise and re-synthesized aerobically during recovery. We acknowledge that the use of stored ATP and CP during exercise is founded within a measurement of oxygen uptake during recovery, but exactly how much is uncertain). Moreover, because the exponential decline in oxygen uptake during recovery is by definition not in steady-state, we refrain from using the ratio of carbon dioxide production and oxygen uptake – the respiratory exchange ratio (RER) – to estimate energy expenditure from fuel utilization within the range of all-fat or all-glucose; that is, 19.6 kJ to 21.1 kJ per liter of O₂ uptake, respectively.

The following, hypothetically, may allow for the reasonable estimation of the three energy expenditure components of at least 1-bout of brief intense exercise with recovery:

- 1. Anaerobic (glycolytic) exercise energy expenditure as:**

$$? \text{blood lactate} \times 3 \text{ ml O}_2 \times \text{weight (kg)} = \text{ml O}_2 \text{ equivalents}$$
- 2. Aerobic exercise energy expenditure as:**

$$1 \text{ liter of exercise O}_2 \text{ uptake} = 21.1 \text{ kJ}$$
- 3. Aerobic (non-glycolytic) recovery energy expenditure as:**

$$1 \text{ liter of recovery O}_2 \text{ uptake} = 19.6 \text{ kJ}$$

Summary

The mechanisms of energy exchange via anaerobic and aerobic metabolism are very different but both result in changes in heat and entropy that comprise exercise and recovery energy expenditure. At 19.6 kJ per liter of oxygen uptake, fat degradation is entirely aerobic. Carbohydrate degradation

has both an aerobic and anaerobic metabolic component at 21.1 kJ per liter of oxygen uptake. We interpret the 1.5 kJ L⁻¹ O₂ difference between fat and carbohydrate breakdown, in part, as being the result of the anaerobic metabolic component in the complete degradation of glucose. In similar fashion, brief intense bouts of physical activity (exercise) can be formatted into a composite of anaerobic and aerobic energy exchange (as total kJ or METs per bout of activity). Blood lactate concentrations can be used as a reasonable estimate of anaerobic energy expenditure but limitations are evident. Recovery energy expenditure does not contain an anaerobic glycolytic energy expenditure component at 19.6 kJ per liter of oxygen uptake.

Address for correspondence: Scott CB, PhD. Department of Exercise, Health and Sport Sciences, University of Southern Maine, Gorham, ME, USA, 04038. Phone (207)780-4566 FAX: (207)780-4745; Email. cscott@usm.maine.edu.

REFERENCES

1. McArdle WD, Katch FI and Katch VL. **Exercise Physiology 5th Ed.: Energy, Nutrition and Human Performance**. Philadelphia: Lippincott Williams & Wilkins, 2001. Pg. 182-183.
2. Mendelsohn E: **Heat and Life: the Development of the Theory of Animal Heat**. Cambridge: Harvard University Press, 1964.
3. Stryer L: **Biochemistry 3rd Ed**. New York: W.H. Freeman & Co., 1988.
4. Bechtel W and Abrahamsen A: In search of mitochondrial mechanisms: interfield excursions between cell biology and biochemistry. **J History Biol** 2007; 40:1-33.
5. Benedict FG and Milner RD: **US Department of Agriculture Bulletin 175**. Experiments on the metabolism of matter and energy in the human body, 1903-1904. Washington DC: US Government Printing Office, 1907.
6. Krogh A and Lindhard J: The regulation of respiration and circulation during the initial stages of muscular hard work. **J Physiol** 1913; 47:112-136.
7. Hill AV and Lupton H: Muscular exercise, lactic acid and the supply and utilization of oxygen. **Q. J. Med** 1923; Jan:135-171.
8. Hale T: History of developments in sport and exercise physiology: A.V. Hill, maximal oxygen uptake, and oxygen debt. **J Sports Sci** 2008; 26: 365-400.
9. Swyer PR. Assumptions used in measurements of energy metabolism. **J Nutr** 1991; 121:1891-1896.
10. Marsh ME and Murlin JR: Muscular efficiency on high carbohydrate and high fat diets. **J Nutr** 1928; 1:105-137.
11. Minakami S and de Verdier C-H: Calorimetric study on human erythrocyte glycolysis: heat production in various metabolic conditions. **Eur J Biochem** 1976; 65:451-460.
12. Scott CB: **Thermodynamics, Bioenergetics and Metabolism: A Primer for the Exercise Physiologist and Sports Nutritionist**. Humana Press-Springer (in press, 2008).
13. Thornton WM: The relation of oxygen to the heat of combustion of organic compounds. **Philos Mag Ser** 1917; 33:196-203.
14. Patel SA and Erickson LE: Estimation of heats of combustion of biomass from elemental analysis using available electron concepts. **Biotech Bioengin** 1981; 23:2051-2067.
15. Kemp RB: Calorimetric studies of heat flux in animal cells. **Thermochim Acta** 1991; 193:253-267.
16. Gnaiger E, Kemp RB: Anaerobic metabolism in aerobic mammalian cells: information from the ratio of calorimetric heat flux and respirometric oxygen flux. **Biochim Biophys Acta** 1990; 1016:328-332.
17. Kleiber M: **The Fire of Life: an Introduction to Animal Energetics**. Robert E. Krieger Publ.:Malabar, FL., 1975, pp. 127.

18. Scott CB: Contribution of anaerobic energy expenditure to whole body thermogenesis. **Nutr Metab** (online). 2005; 2:14.
19. Bang O: The lactate content of the blood during and after muscular exercise in man. **Skand Arch Physiol** 1936;74 (Suppl. 10): 49-82.
20. Brooks GA, Fahey TD and Baldwin KM: **Exercise Physiology: Human Bioenergetics and its Applications**, 4th ed. Boston: McGraw Hill, 2005. Pp. 220-221.
21. Margaria R, Cerretelli P and Mangili F: Balance and kinetics of anaerobic energy release during strenuous exercise in man. **J Appl Physiol** 1964;19: 623-628.
22. di Prampero PE, Ferretti G: The energetics of anaerobic muscle metabolism: a reappraisal of older and recent concepts. **Resp Physiol** 1999;118:103-115.
23. Meirelles C de-M and Gomes PSC: Acute effects of resistance exercise on energy expenditure: revisiting the impact of the training variables. **Rev Bras Med Esporte** 2004; 10:131-138.
24. Scott CB and Kemp RB: Direct and indirect calorimetry of lactate oxidation: implication for whole-body energy expenditure. **J Sports Sci** 2005; 23:15-19.
25. Scott CB: Contribution of blood lactate to the interpretation of total energy expenditure for weight lifting. **J Strength and Cond Res** 2006; 20:21-28.
26. Bahr R: Excess postexercise oxygen consumption – magnitude, mechanisms and practical implications. **Acta Phys Scand** 1992, 144(Suppl 605), 1-70.
27. Nieman DC: **Exercise Testing and Prescription a Health Related Approach**. Boston: McGraw-Hill, 2007. Appendix D. pp. 756-769.