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ORIGINAL RESEARCH

Mathematical simulation of energy expenditure and recovery during sprint cross-country skiing

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Purpose: A cross-country sprint competition relies on maximal effort durations of 3–4 minutes. Significant anaerobic energy contribution is expected. Anaerobic energy contribution has been estimated in different sports to date from the accumulated O_2 deficit. However, the O_2-deficit model can be questioned. We investigate anaerobic energy contribution by applying other methods than the O_2 deficit.

Methods: Theoretical model development.

Results: For sprint cross-country competitions, the anaerobic energy contribution was 20%–25% independent of the employed mathematical model. Recovery times of a minimum 20 minutes were found to be required after sprint races to be sure that the performance in subsequent heats was not influenced.

Conclusion: The O_2-deficit model gave anaerobic energy results in agreement with other models from the literature. Recovery times of a minimum 20 minutes were found to be required after sprint races to be sure that the performance in subsequent heats was not influenced.

Keywords: aerobic, anaerobic, models, endurance sport, recovery

Introduction

In endurance sports, energy expenditure and the ability to utilize metabolic energy to produce external work are the two main performance-determining factors. Energy expenditure is dependent on the body's ability to synthesize (produce) and consume adenosine triphosphate (ATP). Maximal use of ATP in mammalian muscles is around 1.7×10^{-5} mol ATP/g tissue/second. ATP stores in skeletal muscle tissue are around 5×10^{-6} mol/g tissue. Therefore, maximal use can only be applied for around 0.5 seconds without production of ATP.[1] For a muscle mass of, say, 10 kg, this gives 170 mmol/second, which is much more than around 1 mmol/second during rest.[2] Despite the more than 100-fold increase in the rate of ATP consumption from rest to maximal-intensity exercise, the energetic demands of muscles in endurance sports are usually satisfied without depleting intracellular ATP.[3] Three sources are available for ATP production: 1) ATP can be produced aerobically in the mitochondria by oxidative phosphorylation (aerobic energy), 2) ATP can be produced by anaerobic synthesis due to glycolysis/glycogenolysis (lactic energy), and 3) ATP can be produced by phosphocreatine (PCr) breakdown to creatine (Cr) (adenosine diphosphate + PCr gives ATP + Cr in the creatine kinase [CK] reaction) (alactic energy). The maximal amount of anaerobic energy that can be utilized is proportional to the sum of the maximum amount of Cr and lactate that can be accumulated in the body.[1] The equilibrium constant of the CK reaction is around 20.[3] Therefore, the slightest drop in ATP allows the

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reaction to proceed to ATP, and the ATP concentration stays nearly constant until almost all the PCr is exhausted.[3]

The aerobic energy release is the main source in endurance sports with durations above 5 minutes. Anaerobic energy release is the main source with competition times below 1 minute.[1] However, the energy release in so-called middle-distance sports (defined as racing times between 1 and 5 minutes) is determined by a more complex interaction between the various energy sources. When such competition times are performed multiple times within the same day, as in sprint cross-country skiing or some of the speed-skating disciplines, a rapid recovery of the energy systems is required.[4] Although several studies have examined many of these aspects in an isolated fashion through various experiments, mathematical modeling may provide a more integrated understanding.

A crucial metabolic pathway contributing to muscle-ATP regulation in middle-distance sports is glycolysis/glycogenolysis. The end point of glycolysis is pyruvate, which represents a metabolite that can be reduced to form lactate or oxidized to CO_2 or H_2O. The blood lactate concentration is the result of the appearance and disappearance of lactate.[5,6] The working muscles and various tissues produce lactate and release it into the plasma. Both resting and submaximal working of the skeletal muscles, as well as the brain, heart, liver, and kidney, remove lactate from circulation by the use of mitochondria.[7–10] It has been suggested that lactate acts as an intermediary for the transport of carbohydrate from cells and tissue with relatively low oxidative capacity, through the blood lactate pool to cells and tissues with high oxidative capacity.[7–10] Oxidation in the exercising skeletal muscles accounts for around 70%–80%, and the liver (gluconeogenesis in the liver by the Cori cycle) accounts for around 20%–30% of the lactate disappearance during submaximal exercise.[11] Around 20% of the carbohydrate oxidized during exercise passes through the blood lactate pool before being oxidized to carbon dioxide.[11] During mild or moderate exercise (typically below peak fat consumption around 50%–60% of maximal rate of oxygen consumption [VO_{2max}]), the rate of glycolysis increases several-fold, but only leads to a moderate lactate accumulation in the body.[1] During exercise, >60% of VO_{2max} (around peak fat consumption)[2] up to the lactate threshold (LT),[1] (above the LT, no steady state of lactate is developed through time), the steady-state lactate concentration reaches significantly higher values than at rest.[1] Middle-distance sports are performed above the LT, and a progressive accumulation of lactate in muscles and blood develops. In such cases, the capillary blood pH can fall from 7.45 to around 7.05,[12] whereas for skeletal muscles the pH can fall from 7.1 to 6.4.[13] It is not lactate itself that leads to muscle fatigue, but most likely the associated fall in pH.[14,15]

After step changes in work rates for moderate-intensity exercise below the LT, PCr levels, as aerobic power, develop exponentially and reach a steady-state level. Below the LT, a strong similarity has been reported for the time constant for O_2 kinetics response and PCr consumption.[16] Above the LT the anaerobic glycolytic energy supply becomes significant, and the similarities between the rate constants have not been systematically reported.[17] PCr recovery is mainly due to oxidative ATP synthesis. However, even when oxygen is excluded, PCr stores may be rebuilt by anaerobic glycolysis.[18–20] In general PCr is commonly acknowledged as an energy buffer, which supports the transient failure of other metabolic pathways to support ATP. During recovery of PCr levels, the lactate (the pH must be returned) and adenosine diphosphate must be returned to a normal state.

In this paper, the different anaerobic energy contributions by applying different models are studied. It is found how the anaerobic portion of total energy depends on time, and a sprint cross-country skiing scenario is used to study how racing time and recovery depends on the concentration of lactate in the body. Cross-country skiing was chosen because it is a whole-body exercise where both anaerobic and aerobic energy expenditure is important.

Sprint cross-country skiing is a relatively new racing form that demands high aerobic and anaerobic power along with highly developed technical and tactical skills.[21] The heats in sprint skiing (eg, two different semifinals) are performed in sequences after each other, which may lead to individual differences in the duration of breaks before the final. If the duration of breaks between heats is too short, this rationally has an impact on the final sprint performance. This topic was discussed after the Vancouver Olympics in 2010, where the duration of breaks from the two semifinals to the final was around 10 and 15 minutes, respectively.

The main hypotheses of the present study were:

1. The estimated anaerobic energy contribution during sprint racing is dependent on the mathematical models in the literature.
2. Recovery times of a minimum 15–20 minutes are required after cross-country sprint races to be sure that performance in subsequent heats is not influenced.

Methods

Theoretical model development

E_A is the aerobic energy used per unit of mass (that means where the used ATP is synthesized aerobically), E_G is the

anaerobic energy per unit of mass due to glycogenolysis or glycolysis, and E_{CK} is the anaerobic energy per unit of mass due to the CK reaction. Therefore:

$$\underbrace{E}_{\text{total energy}} \overset{mod}{=} \underbrace{E_A}_{\text{aerobic energy}} + \underbrace{E_G}_{\text{anaerobic energy, where ATP synthesized from glycogenolysis/glycolysis}} + \underbrace{E_{CK}}_{\text{anaerobic energy, where ATP synthesized from CK}} \quad (1)$$

where *mod* means a testable model. During the race we assume that the two anaerobic capacities are fully exploited, since the racing time is above 1 minute.[22] Equation 1 can be written as:

$$\frac{E_A}{E} = 1 - \frac{E_G + E_{CK}}{E} \quad (2)$$

Therefore, to calculate the portion of aerobic energy, knowledge of the lactic energy (E_G) and alactic energy (E_{CK}) is needed.

Lactic energy

The relationship between oxygen consumption and blood lactate levels (ie, the lactic energy) can be calculated according to di Prampero and Ferretti[23] and used to estimate the anaerobic energy from glycolysis only. The mL/kg O_2 that corresponds to 1 mmol/L varies from 2.7 to 3.3, due to variation in the relative blood volume versus active muscle mass for an athlete.[23] If this value is set to an average of 3.0 mL/kg O_2, a change in the blood lactate concentration from 0.5 to 15 mmol/L gives:

$$E_G = 14.5 \times 3.0 = 43.5 \text{ mL/kg} \quad (3)$$

Alactic energy

Alactic energy is more complicated, and different models are in use in the literature. One model simply says that alactic power – $Q_{CK}(t)$ – is proportional to the rate of change of aerobic power – $\dot{Q}_A(t)$[17,24,25] – to read:

$$Q_{CK}(t) \overset{mod}{=} \theta \dot{Q}_A(t) \quad (4)$$

where θ is a constant of the proportionality parameter that follows. By time integration, Equation 4 gives:

$$\text{Model 1: } E_{CK} = \theta (Q_A(t) - Q_A(t_0)) \quad (5)$$

Here, the power due to the rate of O_2 consumption (VO_2) is subtracted at the end and start of the exercise, and the number is multiplied by θ. It can be argued that:[25]

$$\theta = (\eta_A / \eta_{CK}) \times \tau_A = \frac{0.6}{0.95} \times 30 \text{ seconds} = 20 \text{ seconds} \quad (6)$$

where η_A, η_{CK} is the efficiency when producing ATP aerobically and from the CK reaction, respectively, and τ_A is a time parameter quantifying the time before the aerobic power reaches a steady state during a steady-state work rate. Typical values for τ_A are 30–36 seconds for moderate intensity exercise.[6,22,23,26–30] For example, di Prampero[22] suggested that τ_A equals 10–24 seconds. Cerretelli et al[29] found that τ_A increases linearly with concentration of lactate up to 36 seconds, and Binzoni et al[30] found 23 seconds for all work rates. We set τ_A to 30 seconds as a compromise.

Another method to calculate the energy due to the CK reaction is a variant of the so called O_2-deficit assumption,[25] here called Model 2. It is assumed that for a given type of exercise, the rate of ATP consumption is the same for the same work rate – *P*.

$$\text{Model 2: } \eta\eta_A E_A + \eta\eta_{CK} E_{CK} + \eta\eta_G E_G = \eta\eta_A \overline{E}_{vir}$$

$$\Rightarrow E_{CK} = \frac{\eta_A}{\eta_{CK}} \left(\underbrace{\overline{E}_{vir} - E_A}_{O_2\text{deficit}} \right) - \frac{\eta_G}{\eta_{CK}} E_G \quad (7)$$

Here, η is the efficiency during muscle contraction, while η_G is the efficiency when producing ATP by anaerobic glycolysis/glycogenolysis. For exercise intensities exceeding the maximal rate of O_2 consumption, the virtual steady state is the steady state that would be attained if it was possible to carry out the exercise under purely aerobic conditions.[23] Obviously, this tentative (or virtual) steady state rate $\dot{\overline{E}}_{vir}$ is never reached, as the increase in O_2 uptake approaches the maximal rate of O_2 consumption. Here, $\overline{E}_{vir}(t) = \int_0^t \dot{\overline{E}}_{vir}(u)du$. It is assumed that for a given type of exercise, $\dot{\overline{E}}_{vir} = a + bP$, where *P* is the work rate. The parameters *a* and *b* are determined by fitting the steady-state rate of O_2 consumption to work rate *P*. The O_2-deficit model applies $\eta_A = \eta_{CK} = \eta_G$. Therefore, $E_{CK} = (\overline{E}_{vir} = E_A) - E_G$. However, $\eta_A = \eta_{CK} = \eta_G$ seems to have no justification in the literature. Now, we set $\eta_A = \eta_G = 0.6, \eta_{CK} = 0.95$ as the baseline.[25]

If $\overline{E}_{vir} - E_A = 76$ mL/kg, this gives:[31]

$$O_2\text{-deficit method: } E_{CK} = \frac{0.6}{0.6} \times 76 - \frac{0.6}{0.6} \times 43.5 = 32.5 \text{ mL/kg}$$

$$\text{Model 1: } E_{CK} = \theta\left(Q_A(t) - Q_A(t_0)\right) = 24.9 \text{ mL/kg}$$

$$\text{Model 2: } E_{CK} = \frac{0.6}{0.95} \times 76 - \frac{0.6}{0.95} \times 43.5 = 20.5 \text{ mL/kg} \quad (8)$$

Obviously, the results will be dependent on the efficiencies.

For comparison, E_{CK} numbers from the O_2-deficit method and the two models can be compared to the number calculated when using the estimate by di Prampero.[22] For exercise leading to exhaustion from 50 seconds to 10 minutes, the sum of the two anaerobic energy sources is essentially constant and equal to 1,400 J/kg =70 mL O_2/kg if it is assumed that 1 mL O_2 corresponds to 20 J. Using Equation 3 and subtracting E_G =43.5 mL/kg gives E_{CK} =26.5 mL/kg. This number is in agreement with the numbers in Equation 8.

The results can be summarized as:

$$\frac{E_A}{E} = 1 - \frac{E_G + E_{CK}}{E}$$

$$\text{Model 1: } E_G + E_{CK} = E_G + \frac{\eta_A}{\eta_{CK}} \tau_A \left(Q_A(t) - Q_A(t_0) \right)$$

$$\text{Model 2: } E_G + E_{CK} = \left(1 - \frac{\eta_G}{\eta_{CK}} \right) E_G + \frac{\eta_A}{\eta_{CK}} \left(\overline{E}_{vir} - E_A \right)$$

$$O_2\text{-deficit method: } E_G + E_{CK} = \overline{E}_{vir} - E_A \quad (9)$$

Results

A skier utilizes the average rate of aerobic power of Q_A. This gives:

$$\frac{E_A}{E} = 1 - \frac{E_G + E_{CK}}{E} = \frac{Q_A t}{Q_A t + E_G + E_{CK}} \quad (10)$$

where t is the time of the race. Ninety percent of the peak rate of oxygen consumption measured in the laboratory is used in the calculations as average aerobic power during a sprint race. This assumption is based on studies showing peak values of around 95% of their maximal rate of oxygen consumption,[32] as well as mean values of around 95% of the peak values during a sprint race.[21]

Figure 1 shows the solution of Equation 10 when Q_A =0.9 × 77.8 mL/kg/minute =70 mL/kg/minute. The mean duration of a cross-country sprint ski heat is 3 minutes 30 seconds, and 3 minutes 10 seconds for men and women, respectively.[4]

An overall mean of 3 minutes 20 seconds is used further. Figure 1 shows the portion of aerobic and anaerobic energy of total energy as a function of time, and in the specific case of 3-minute 20-second duration, the anaerobic contribution is around 20%–25%.

PCr levels quickly recover after a race (after around 30 seconds[3]). However, recovery from anaerobic glycolysis needs longer time periods. Assuming the values of the total energy in a sprint heat for a world-class sprint skier after breaks of different duration following a sprint heat, blood lactate level is calculated. The mean aerobic power was regarded as 70 mL/kg/minute, and the mean peak blood lactate level 15 mmol/L. After 6-, 10-, 15-, and 20-minute breaks after the sprint, the skier would start with 9.5, 6.5, 3.5, and 1.8 mmol/L lactate, respectively.[32] Equation 10 can be reformulated to study the time for a race as a function of the initial blood lactate concentration of the skier. During a race of around 3 minutes 20 seconds, the energy

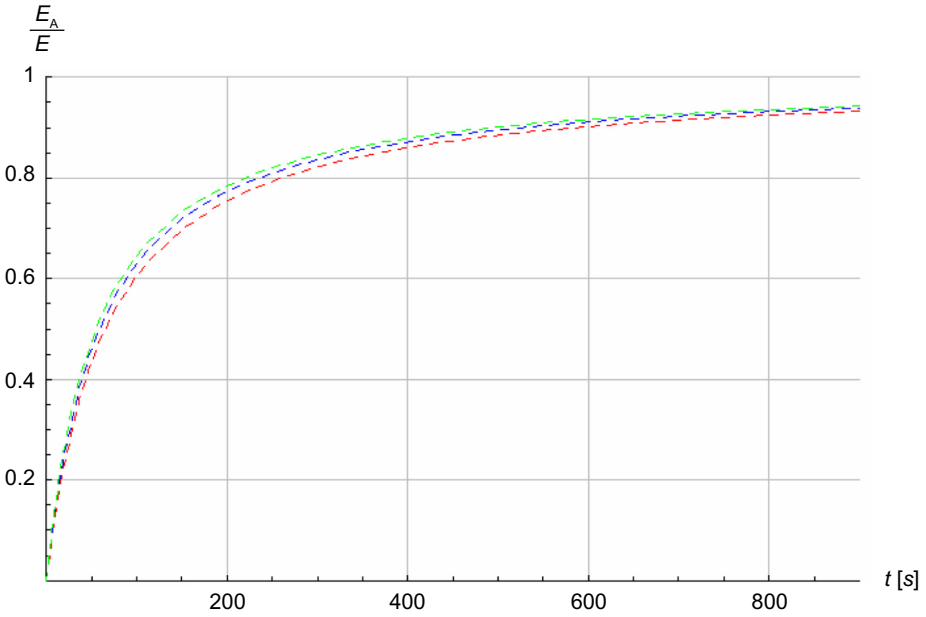

Figure 1 Portion of aerobic energy to total energy as a function of time (t) in seconds (s). Q_A= 70 mL/kg/minute.
Notes: Red, $E_G + E_{CK}$ =76 mL/kg; blue, $E_G + E_{CK}$ =68.4 mL/kg; green, $E_G + E_{CK}$ =64.0 mL/kg.
Abbreviations: E_G, lactic energy; E_{CK}, alactic energy.

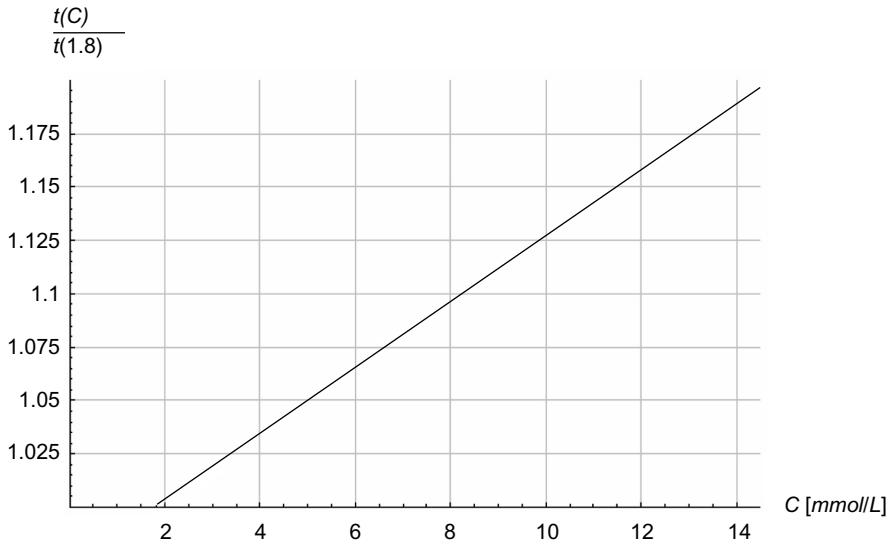

Figure 2 Time (*t*) for a race of 3 minutes 20 seconds relative to time when starting with an initial blood lactate concentration of 1.8 mmol/L as a function of the initial concentration (*C*) of lactate.

used is called E_S, where $_S$ denotes sprint skiing. The energy is then:

$$\frac{E_A}{E_S} = \frac{Q_A t}{Q_A t + E_G + E_{CK}}$$
$$E_S = Q_A t + E_G + E_{CK}$$
$$= 70/60 \times (3 \times 60 + 20) + 68.4 = 302 \text{ mL/kg} \quad (11)$$

Let C denote the blood lactate concentration in millimoles per liter at the start of the race, and let $t = t(C)$ be the time for the race. It can then be written that:

$$t(C) = \left(E_S - E_G\left(C\right) - E_{CK}\right)/Q_A$$
$$E_G\left(C\right) = \left(15 \text{ mmol/L} - C\right) \times \lambda,$$
$$\lambda = 3 \text{ mL/kg}/\left(\text{nmol/L}\right) \quad (12)$$

This gives the time relative to the time when the initial concentration is 1.8 mmol/L (corresponding to 20 minutes' recovery) as:

$$\frac{t\left(C\right)}{t\left(1.8\right)} = \frac{E_S - E_G\left(C\right) - E_{CK}}{E_S - E_G\left(1.8\right) - E_{CK}}$$
$$\approx 1 - \frac{E_G\left(C\right)}{E_S} + \frac{E_G\left(1.8\right)}{E_S}$$
$$= 1 + \lambda \frac{C - 1.8 \text{ mmol/L}}{E_S} \quad (13)$$

As an example, using the approximation above and an initial concentration of 6.5 mmol/L (corresponding to 10 minutes' recovery) gives $t(6.5)/t(1.8) = 1 + 3(6.5 - 1.8)/302 = 1.05$. Therefore, the difference between 20 minutes' break and 10 minutes' break is around 5%. The second term of Equation 13 can be plotted to study the effect more carefully. Figure 2 shows the result. The exact result was found to be closer to 7.5%.

The current findings reveal differences of around 6%, 3%, and 2% in racing time between breaks of duration 10 versus 20 minutes, 10 versus 15 minutes, and 15 versus 20 minutes, respectively. This means that the duration of breaks may impact performance in the subsequent heat when the breaks are below 20 minutes between the heats. With 20-minute breaks between heats, world-class skiers show blood lactate levels around 2 mmol/L, which is closer to the resting levels of blood lactate concentration – around 0.5 mmol/L. However, these effects only concern low-altitude competitions.

Conclusion

Anaerobic energy in sprint cross-country skiing was investigated by using two mathematical models that have been recently published in the literature. The models were used to study the anaerobic portion of energy as a function of time. These models have been used to study sprint-skiing racing scenarios in more detail. It was shown that the anaerobic versus aerobic portion of energy should be considered when choosing the duration of a sprint race; today's races contain between 25% and 30% anaerobic and 75%–80% aerobic energy. Also, the duration of breaks between different racing heats in sprint skiing is of importance: based on literature values for blood lactate during recovery, the simulations showed that recovery times of 20 minutes between heats of world-class skiers are necessary to ensure that the breaks do

not impact performance in subsequent heats. Only hypothesis 2 was accepted.

The simulations showed recovery times of 20 minutes between heats of world-class skiers are necessary to ensure that the breaks do not impact performance in subsequent heats. At higher altitudes, such as in the Olympic Games in Sochi in 2014, at 1,500 m above sea level, the resting time probably requires even longer durations.

Only mean values of energy during the last 3 minutes 20 seconds of a race were calculated, despite the possible variable work rates and intensities during the sprint heats. How the latter aspects of sprint skiing, as well as the differences in tactics within the heats, may impact the results were not considered.

It is also assumed that aerobic power remains constant across differences in pH (here indicated by blood lactate concentration). This assumption is not obviously correct, because beta oxidation in the mitochondria is shown to be affected by pH. Earlier studies of sprint skiing showed no significant differences in mean or peak oxygen uptake across heats.[21,33,34] Finally, other factors affect recovery, such as muscular and neuromuscular fatigue, but were not considered in these analyses. Overall, this paper provides novel knowledge about energy demands and recovery in cross-country sprint skiing.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Johnson AT. *Biomechanics and Exercise Physiology*. New York: John Wiley and Sons; 1991.
2. Shephard RJ, Åstrand PO. *Endurance in Sport*. Vol 2. Oxford: Blackwell Science; 2000.
3. McMahon TA. *Muscles, Reflexes, and Locomotion*. Princeton (NJ): Princeton University Press; 1984.
4. International Ski Federation. Results FIS World Cup. 2014. Available from: http://data.fis-ski.com/global-links/all-fis-results.html?place_search=&seasoncode_search=2014&sector_search=&date_search=&gender_search=&category_search=WC&codex_search=&nation_search=&disciplinecode_search=&date_from=today&search=Search&limit=50. Accessed April 6, 2014.
5. Moxnes J, Hausken K. A mathematical model for training impulse and lactate influx and outflux during exercise. *Int J Mod Phys C*. 2009;20:147–177.
6. Moxnes J, Sandbakk Ø. The kinetics of lactate production and removal during whole body exercise. *Theor Biol Med Model*. 2012;9:7.
7. Brooks GA. Lactate production under fully aerobic conditions: the lactate shuttle during rest and exercise. *Fed Proc*. 1985;45:2924–2929.
8. Brooks GA. Current concepts in lactate exchange. *Med Sci Sports Exerc*. 1991;23:895–906.
9. Brooks GA. Lactate shuttles in nature. *Biochem Soc Trans*. 2002;30:258–264.
10. Brooks GA. Link between glycolytic and oxidative metabolism. *Sports Med*. 2007;37:341–343.
11. Stanley WC, Wisneski JA, Gertz EW, Neese RA, Brooks GA. Glucose and lactate interrelations during moderate-intensity exercise in humans. *Metabolism*. 1988;37:850–858.
12. Hermansen L, Saltin B. Blood lactate concentration during exercise at acute exposure to altitude. In: Margaria R, editor. *Exercise at Altitude*. Amsterdam: Exerpta Medica Foundation; 1967:48–53.
13. Hermansen L, Osnes JB. Blood and muscle pH after maximal exercise in man. *J Appl Physiol*. 1972;32:304–308.
14. Myers J. Dangerous curves. A perspective on exercise, lactate, and the anaerobic threshold. *Chest*. 1997;111:787–795.
15. Juel C. Lactate-proton cotransport in skeletal muscle. *Physiol Rev*. 1997;77:321–358.
16. Rossiter HB, Ward SA, Doyle VL, Howe FA, Griffiths JR, Whipp BJ. Interference from pulmonary O_2 uptake with respect to intramuscular [phosphocreatine] kinetics during moderate exercise in humans. *J Physiol*. 1999;518:921–932.
17. Meyer RA. A linear model of muscle respiration explains monoexponential phosphocreatine changes. *J Appl Physiol*. 1988;75:648–656.
18. Jubrias SA, Esselman PC, Price LB, Cress ME, Conley KE. Large energetic adaptations of elderly muscle to resistance and endurance training. *J Appl Physiol*. 2001;90:1663–1670.
19. Crowther GJ, Kemper WF, Carey MF, Conley KE. Control of glycolysis in contracting skeletal muscle. II. Turning it off. *Am J Physiol Endocrinol Metab*. 2002;282:E74–E79.
20. Lanza IR, Wigmore DM, Befroy DE, Kent-Braun JA. In vivo ATP production during free-flow and ischaemic muscle contractions in humans. *J Physiol*. 2006;577:353–367.
21. Stöggl T, Lindinger S, Müller E. Analysis of a simulated sprint competition in classical cross country skiing. *Scand J Med Sci Sports*. 2007;17:362–372.
22. di Prampero PE. Factors limiting maximal performance in humans. *Eur J Appl Physiol*. 2003;90:420–429.
23. di Prampero PE, Ferretti G. The energetics of anaerobic muscle metabolism: a reappraisal of older and recent concepts. *Respir Physiol*. 1999;118:103–115.
24. Mahler M. First order kinetics of muscle oxygen consumption, and an equivalent proportionality between Q_{O_2} and phosphorylcreatine level. *J Gen Physiol*. 1985;86:135–165.
25. Moxnes JF, Hausken K, Sandbakk Ø. On the kinetics of anaerobic power. *Theor Biol Med Model*. 2012;9:29.
26. Losnegard T, Myklebust H, Hallén J. Anaerobic capacity as a determinant of performance in elite skiers. *Med Sci Sports Exerc*. 2012;44:673–681.
27. Bangsbo J, Krustrup P, González-Alonso J, Saltin B. ATP production and efficiency of human skeletal muscle during intense exercise: effects of previous exercise. *Am J Physiol Endocrinol Metabol*. 2001;280:E956–E964.
28. Bertuzzi RC, Franchini E, Ugrinowitsch C, et al. Predicting MAOD using only a supramaximal exhaustive test. *Int J Sports Med*. 2010;31:477–481.
29. Cerretelli P, Pendergast DR, Paganelli WC, Rennie DW. Effects of specific muscle training on VO_2-on response and early blood lactate. *J Appl Physiol*. 1979;47:761–769.
30. Binzoni T, Ferretti G, Sechenker K, Cerretelli P. Phosphocreatine hydrolysis in 31P-NMR at the onset of constant-load exercise in humans. *J Appl Physiol*. 1992;73:1644–1649.

31. Losnegard T, Myklebust H, Spencer M, Hallén J. Seasonal variation in VO_{2max}, O_2-cost, O_2-deficit, and performance in elite cross-country skiers. *J Strength Cond Res*. 2013;27:1780–1790.
32. Sandbakk Ø, Holmberg HC, Leirdal S, Ettema G. The physiology of world-class sprint skiers. *Scand J Med Sci Sports*. 2011;21:e9–e16.
33. Mikkola J, Laaksonen M, Holmberg HC, Vesterinen V, Nummela A. Determinants of a simulated cross-country skiing sprint competition using V2 skating technique on roller skis. *J Strength Cond Res*. 2010;24:920–928.
34. Vesterinen V, Mikkola J, Nummela A, Hynynen E, Häkkinen K. Fatigue in a simulated cross-country skiing spring competition. *J Sports Sci*. 2009;27:1069–1077.