

An alternative to oxygen deficit as a way to quantify anaerobic contributions in running

DAVID WILFRED HILL¹ , ANDREA ELIZABETH RIOJAS¹, BRIAN KEITH MCFARLIN^{1,2}, JAKOB LANGBERG VINGREN^{1,2}

¹Department of Kinesiology, Health Promotion, and Recreation, University of North Texas, United States of America

²Department of Biological Sciences, University of North Texas, United States of America

ABSTRACT

The purpose of this study was to determine if the sum of estimates of the phosphocreatine contribution and the glycolytic contribution (we refer to this sum as *PCr+glycolysis*) provides an alternative to oxygen deficit as a way to quantify the anaerobic contribution in running. Thirty university students performed three treadmill tests, each test at one speed individually selected for each participant; one test was terminated after 3 min, one after 7 min, and one at exhaustion (mean \pm SD, 10.3 \pm 0.4 min). Oxygen deficit was calculated by subtraction of the accumulated oxygen uptake from the total oxygen cost. Phosphocreatine and glycolysis contributions were determined from post-exercise VO_2 responses and blood lactate concentrations, respectively. The mean values for *PCr+glycolysis* were \sim 3 mL \cdot kg⁻¹ lower ($p < 0.05$) than oxygen deficit across three exercise durations, but well correlated ($r \geq 0.80$, $p < 0.05$) at each. These results confirm the validity of *PCr+glycolysis* as an alternative to oxygen deficit to quantify the anaerobic contribution in running exercise.

Keywords: Anaerobic capacity; Exercise; Glycolysis; Phosphocreatine; Severe; Sports performance.

Cite this article as:

Hill, D.W., Riojas, A.E., McFarlin, B.K., & Vingren, J.L. (2019). An alternative to oxygen deficit as a way to quantify anaerobic contributions in running. *Journal of Human Sport and Exercise, in press*. doi:<https://doi.org/10.14198/jhse.2020.154.11>

 **Corresponding author.** Department of Kinesiology, Health Promotion, and Recreation, University of North Texas, United States of America.

E-mail: David.Hill@unt.edu

Submitted for publication August 2019

Accepted for publication October 2019

Published *in press* November 2019

JOURNAL OF HUMAN SPORT & EXERCISE ISSN 1988-5202

© Faculty of Education. University of Alicante

doi:10.14198/jhse.2020.154.11

INTRODUCTION

Oxygen deficit, which represents the difference between the estimated total oxygen cost of exercise and the measured oxygen uptake, is an accepted measure of anaerobic contribution (Noordhof et al., 2010). In 2010, Bertuzzi and colleagues challenged the feasibility of using oxygen deficit, especially in sport testing, arguing that the methods are too laborious and require too great a commitment of time and resources by athletes. The time-consuming element of the calculation of oxygen deficit to which they referred is procedure of estimating oxygen deficit from the results of a series of submaximal efforts performed during a number of laboratory visits, which is also the most contentious part of the calculation (e.g., Green and Dawson, 1996). However, with the results of only a single severe intensity exercise test, using the fast phase of the post-exercise VO_2 response profile to estimate the energy produced from available phosphagens, adenosine triphosphate (ATP) and phosphocreatine (PCr) (Henry, 1951; Margaria et al., 1933) and using peak post-exercise blood lactate concentration to estimate the energy from glycolysis (Margaria et al., 1933), Bertuzzi and colleagues (2010) generated a measure of total anaerobic contribution. Studies have shown that this *PCr+glycolysis* measure and oxygen deficit did not differ and were highly correlated for cycling (Bertuzzi et al., 2010; Miyagi et al., 2017; Urso et al., 2016) The method has been validated for running in one study (Zagatto et al., 2016).

Because of the widespread use of the *PCr+glycolysis* measure, including its application to a variety of exercise modes, such as rock climbing (Bertuzzi et al., 2007), a variety of martial arts, such as taekwondo (e.g., Lopes-Silva et al., 2018), table tennis (Zagatto and Gobatto, 2012), and rowing (de Campos Mello et al., 2009), we felt it important to confirm its validity. In running, metabolic demand is a function of body mass and it is customary to report measures of aerobic and anaerobic fitness (e.g., $\text{VO}_{2\text{max}}$ and maximal accumulated oxygen deficit) relative to mass. Therefore, the purpose of this study was to evaluate *PCr+glycolysis* as an alternative to oxygen deficit in running. The hypothesis was that *PCr+glycolysis* and oxygen deficit, expressed relative to body mass, would provide similar values for anaerobic contribution in running.

METHODS

Participants

This study was approved by the Institutional Review Board for the Protection of Human Subjects at the university and conducted in accordance with the ethical standards of the latest *Declaration of Helsinki* (World Health Organization, 2013). Participants were recruited from kinesiology majors' courses at a large university. After the procedures, risks, and benefits of the study were carefully explained to the potential participant, he or she provided written informed consent. A total of 30 students participated: there were 13 women, of mean (\pm SD) age, 22 ± 2 yr., height, 1.66 ± 0.09 m, and mass 67.2 ± 9.0 kg, and 17 men, 23 ± 3 yr., 1.80 ± 0.05 m, 81.6 ± 9.0 kg, participated. Participants were involved in recreational sport or fitness activities, but not organized sport activities. As we instructed, they did not alter their usual exercise, diet, or sleep habits over the course of the study.

Measures

Four measures were obtained. The first, oxygen deficit, is a measure of anaerobic contribution during a bout of exercise; in the case of exhaustive severe intensity exercise, the oxygen deficit value is maximal (often called the maximal accumulated oxygen deficit, or MAOD) and, in this case, provides a measure of anaerobic capacity. The second and third measures were the anaerobic contribution from available phosphagens (ATP

and PCr) and the anaerobic contribution from glycolysis, respectively. These two were summed to obtain the fourth measure, *PCr+ glycolysis*.

Procedures

To eliminate confounding effects of circadian rhythmicity on oxygen deficit (Hill, 2014), each participant's sessions were scheduled at the same time of day (± 1 h) in controlled environmental conditions (20 °C to 22 °C; ~50% relative humidity), with no distractions. In the first session, after consent was obtained, the participant was screened using the Physical Activity Readiness Questionnaire (PAR-Q; Thomas et al., 1992) and a brief medical history, and familiarized with testing procedures. In the second session, the participant performed an exhaustive incremental test, which was used to determine VO_{2max} and to provide information to use in identifying a running speed that could be sustained ~10 min. In the remaining sessions, the participant exercised at this speed. Sessions were separated by at least 48 hours and were completed within a 21-day period.

The exhaustive incremental treadmill test began with 4 min of standing, to allow the participant to accommodate to the mouthpiece and nose clip, then 2-min stages at 135 $m \cdot min^{-1}$, 145 $m \cdot min^{-1}$, 155 $m \cdot min^{-1}$, and 165 $m \cdot min^{-1}$; subsequent stages were 1 min each (+10 $m \cdot min^{-1}$). VO_2 and other cardio-respiratory measures were collected throughout the test.

For constant-speed tests, the participant ran (for 3 min, for 7 min, or to exhaustion, randomized order) at the speed that had been identified to be sustainable for ~10 min. This speed was selected based on previous results in our lab (Hill et al., 1997), which showed that 92% of the peak speed in an incremental test could be sustained ~10 min; in the present study, 92% of the speed sustained for at least 30 s in the incremental test was calculated as a starting point for selecting the participant's speed. This was adjusted slightly up or down by the primary investigator, using his experience and expertise, based on how long the participant sustained the speed in the last stage that was attempted. Upon termination, the participant stood for seven minutes. VO_2 and other cardio-respiratory measures were collected throughout the test and during the 7-min recovery. Blood samples were obtained during recovery.

The reason for using three different exercise durations in this study was to determine if the *PCr+glycolysis* measure was robust, that is to ensure that the relationship between oxygen deficit and *PCr+glycolysis* was not affected by duration (3 min versus 7 min) or by relative intensity (submaximal versus exhaustive).

During each test, expired gases were analysed on a breath-by-breath basis using a MedGraphics (St. Paul, MN USA) $CardiO_2$ metabolic system, which was calibrated before each test. Baseline VO_2 was the lowest 30-s average of breath-by-breath values during the 4-min pre-exercise period; steady-state VO_2 was the 30-s average at the end of each stage; and VO_{2max} in the incremental tests (VO_{2peak} in the constant-speed tests) was the highest 30-s average in the test.

For each test, breath-by-breath VO_2 data were reduced to rolling 5-breath averages and fitted to a two-component model (equation 1) using nonlinear regression on KaleidaGraph 4.50 (Reading, PA, USA):

$$VO_2(t) = VO_{2baseline} + A_{primary} \times (1 - e^{-(t - TD_{primary}) / \tau_{primary}}) + A_{slow} \times (1 - e^{-(t - TD_{slow}) / \tau_{slow}})$$

In this model, $VO_{2baseline}$ is the measured steady state VO_2 . $A_{primary}$ and A_{slow} are the projected increases in VO_2 from the primary and slow components. $TD_{primary}$ and TD_{slow} are the time delays preceding these responses. The time constants of the responses, $\tau_{primary}$ and τ_{slow} , represent the rate at which the VO_2 is

increasing in each phase and are equal to the time required for the VO_2 to increase 63% of the projected amplitude. The actual increase in VO_2 due to the slow component, A'_{slow} , was calculated using equation 2:

$$A'_{slow} = A_{slow} \times (1 - e^{-(\text{exercise duration} - TD_{slow}) / \tau_{slow}})$$

The area under the VO_2 response curve and the area under the fast phase of the curve were calculated, and the difference between these two areas (i.e., the excess oxygen uptake attributed to the slow component) was determined by subtraction. In most 3-min tests, data did not fit a two-component model well. Therefore, for 3-min tests, analysis was restricted to a single-component model (i.e., fast phase only).

The oxygen demand ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) in the constant speed tests was calculated using equation 3, assuming that the relationship between oxygen demand and speed is upwardly curvilinear (Hill, 1999; Hill and Vingren, 2011, 2013; Hill and Lupton 1923; Li, Niessen, Chen, & Hartmann, 2015). Using validated methods (Hill and Vingren, 2011, 2012, 2013; Hill, 2014), oxygen demand ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) at $100 \text{ m} \cdot \text{min}^{-1}$ was estimated by interpolation of the measured baseline VO_2 value and the VO_2 at $135 \text{ m} \cdot \text{min}^{-1}$, the value for the parameter a in equation 3 was determined by regression of the speed and steady state VO_2 data from the incremental tests, and oxygen demand in the constant-speed tests was estimated by extrapolation (equation 3):

$$\text{oxygen demand} = \text{oxygen demand at } 100 \text{ m} \cdot \text{min}^{-1} + a \times (\text{speed} - 100 \text{ m} \cdot \text{min}^{-1})^{1.05}$$

The total oxygen cost ($\text{mL} \cdot \text{kg}^{-1}$) for each test was calculated as the product of oxygen demand and exercise duration plus the excess oxygen cost, which reflects decreasing efficiency over the course of the exercise (Noordhof et al., 2015) and is equal to the excess oxygen uptake attributed to the slow component. Oxygen deficit ($\text{mL} \cdot \text{kg}^{-1}$) was calculated by subtracting the accumulated oxygen uptake from the total oxygen demand.

The post-exercise VO_2 response profile were described using nonlinear regression on KaleidaGraph, by fitting 5-breath averages to a two-component model (equation 4):

$$VO_2(t) = A_{baseline} + (A_{fast} \times) (e^{-(t-TD) / \tau_{fast}}) + (A_{slow} \times) (e^{-(t-TD) / \tau_{slow}})$$

$VO_2(t)$ is the value for VO_2 at time = t . $A_{baseline}$ is the baseline VO_2 , towards which the VO_2 is returning. A_{fast} and A_{slow} are the amplitudes for the two exponential terms, in this case, the amount that the VO_2 is decreasing as the contribution from each phase reduces to zero. The time constants of the responses, τ_{fast} and τ_{slow} , represent the rate at which the VO_2 is decreasing in each phase and are equal to the time required for the VO_2 to decrease 63% of the projected amplitude, as the contribution from each phase reduces to zero. TD is the common time delay, which is the time between the end of exercise before the VO_2 begins to drop precipitously as a function of the fast and slow components of the post-exercise response. The area under the fast curve of the fast phase of the post-exercise exercise VO_2 response represents the PCr contribution.

Exactly 4 min, 5 min, and 6 min after each exercise test, two fingertip blood samples were obtained and analysed using identical Nova Biomedical Lactate Plus lactate analyser (Hawthorne, New York, USA). This technology has been validated against bench chemistry reference methods (Fell et al., 1998). The highest value was recorded as the peak post-exercise blood lactate concentration. Resting blood lactate concentrations, from the familiarization visit, were $1.0 \pm 0.1 \text{ mM}$ (range, 0.8 mM to 1.2 mM). The glycolysis contribution was calculated according to Margaria and colleagues (1933), assuming a resting blood lactate concentration of 1.0 mM (equation 5):

$$\text{glycolysis} = 3.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{mM}^{-1} \times (\text{peak blood lactate concentration} - \text{resting concentration})$$

Assuming a resting value of 1.0 mM, rather than individually sampling prior to each run, may introduce an error no greater than 0.6 mL·kg⁻¹ in any particular estimate of glycolytic contribution and would have no effect on the overall mean.

Statistical analysis

The statistical analyses were performed using SPSS v22 (IBM, Armonk, NY, USA). Values of the measures of anaerobic contribution were compared using a two-way analysis of variance (ANOVA), with repeated measures across method (*PCr+glycolysis* versus oxygen deficit) and test duration (3 min versus 7 min versus exhaustive). Significance was set at $p < 0.05$. Initially, we used a three-way (method \times duration \times sex) ANOVA. As there was no significant interaction effect involving sex, the data were collapsed across sex. Correlations between *PCr+glycolysis* values and the criterion oxygen deficit values were calculated. A Bland-Altman plot (Bland and Altman, 1986) of the differences between *PCr+glycolysis* and oxygen deficit values versus the criterion measure of anaerobic contribution (Krouwer, 2008) was constructed to evaluate the similarity of the two measures. Throughout this paper, values are presented as means \pm SD and, where appropriate, 95% confidence intervals for means and effect sizes (Cohen's d) for differences are provided.

RESULTS

Mean $\text{VO}_{2\text{max}}$ from the incremental tests was $44.4 \pm 4.9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Mean speed for constant speed tests was $201 \pm 19 \text{ m} \cdot \text{min}^{-1}$; two tests had fixed durations (3 min or 7 min) and, in the third test, time to exhaustion was $617 \pm 23 \text{ s}$ ($10.3 \pm 0.4 \text{ min}$). The exercise data that were used to calculate oxygen deficit are in Table 1. The post-exercise data that were used to calculate *PCr+glycolysis* are in Table 2.

Table 1. VO_2 responses during the exercise bouts which were used in the calculation of oxygen deficit.

	3 min	7 min	Exhaustive
$\text{VO}_{2\text{baseline}}$ ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) (95% C.I.)	4.9 ± 0.1 (4.9, 5.0)	5.0 ± 0.1 (4.9, 5.0)	5.0 ± 0.1 (5.0, 5.0)
TD_{fast} (s) (95% C.I.)	$4 \pm 2^*$ (3, 5)	12 ± 4 (10, 13)	11 ± 5 (9, 13)
τ_{fast} (s) (95% C.I.)	$33 \pm 6^*$ (31, 35)	25 ± 4 (23, 26)	25 ± 3 (24, 26)
A'_{fast} ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) (95% C.I.)	$36.9 \pm 4.6^*$ (35.1, 38.6)	33.7 ± 4.2 (32.1, 35.3)	34.1 ± 4.5 (32.4, 35.8)
TD_{slow} (s) (95% C.I.)	— **	110 ± 9 (105, 115)	121 ± 11 (117, 125)
A'_{slow} ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) (95% C.I.)	— **	5.4 ± 3.8	5.3 ± 3.7
$\text{VO}_{2\text{peak}}$ ($\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) (95% C.I.)	41.8 ± 4.0 (40.3, 43.3)	44.1 ± 4.3 (42.5, 45.7)	44.4 ± 4.6 (42.6, 46.1)

Oxygen demand (mL·kg ⁻¹ ·min ⁻¹) (95% C.I.)	47.6 ± 3.1 (46.4, 48.8)	47.6 ± 3.1 (46.4, 48.8)	47.6 ± 3.1 (46.4, 48.8)
Total oxygen cost (mL·kg ⁻¹) ^a (95% C.I.)	142.8 ± 9.3 (139.3, 146.3)	333.2 ± 21.7 (324.9, 341.5)	489.3 ± 31.9 (477.2, 501.4)
Excess oxygen uptake (mL·kg ⁻¹) (95% C.I.)	— **	18.3 ± 2.6 (17.3, 19.3)	31.5 ± 6.8 (28.9, 34.1)
True total oxygen cost (mL·kg ⁻¹) ^b (95% C.I.)	142.8 ± 9.3 (139.3, 146.3)	351.5 ± 22.2 (343.1, 359.9)	520.8 ± 32.9 (508.3, 533.3)
Accumulated uptake (mL·kg ⁻¹) (95% C.I.)	102.6 ± 9.9 (98.8, 106.4)	285.9 ± 19.3 (278.6, 293.2)	427.0 ± 33.0 (414.5, 439.6)
Oxygen deficit (mL·kg ⁻¹) ^c (95% C.I.)	40.2 ± 5.2 (38.3, 42.1)	65.6 ± 6.1 (63.3, 67.9)	93.8 ± 8.5 (90.6, 97.0)

* Data from the single-component model (no slow component).

** While inspection of residuals from the single-component modelling of the VO₂ response in the 3-min tests suggested the presence of a slow component, we could not fit these data to a two-component model. Therefore, excess oxygen uptake could not be quantified in the 3-min tests. We estimate that the oxygen deficit may have been underestimated by ~2 mL·kg⁻¹.

^a total oxygen cost = oxygen demand x exercise duration.

^b true total oxygen cost = total oxygen cost + excess oxygen uptake.

^c oxygen deficit = true total oxygen cost – accumulated oxygen uptake.

Table 2. VO₂ responses and blood lactate concentrations during recovery after the exercise bout, used in the calculation of PCr+glycolysis.

	3 min	7 min	Exhaustive
VO _{2peak} (mL·kg ⁻¹ ·min ⁻¹) (95% C.I.)	41.8 ± 4.0 (40.3, 43.3)	44.1 ± 4.3 (42.5, 45.7)	44.4 ± 4.6 (42.6, 46.1)
TD (s) (95% C.I.)	3 ± 2 (2, 4)	3 ± 2 (2, 4)	1 ± 1 (1, 1)
tau _{fast} (s) (95% C.I.)	51 ± 5 (49, 53)	50 ± 6 (48, 52)	53 ± 6 (51, 55)
A _{fast} (mL·kg ⁻¹ ·min ⁻¹) (95% C.I.)	28.1 ± 3.9 (26.6, 29.6)	31.1 ± 4.2 (29.5, 32.7)	34.2 ± 4.3 (32.6, 35.8)
TD (s) (95% C.I.)	3 ± 2 (2, 4)	3 ± 2 (2, 4)	1 ± 1 (1, 1)
Tau _{slow} (s) (95% C.I.)	349 ± 33 (336, 361)	441 ± 42 (325, 357)	765 ± 59 (743, 788)
A _{slow} (mL·kg ⁻¹ ·min ⁻¹) (95% C.I.)	8.8 ± 2.9 (7.7, 9.9)	8.0 ± 2.2 (7.2, 8.8)	5.2 ± 3.7 (3.8, 6.6)
VO _{2baseline} (mL·kg ⁻¹ ·min ⁻¹) (95% C.I.)	4.9 ± 0.1 (4.9, 5.0)	5.0 ± 0.1 (4.9, 5.0)	5.0 ± 0.1 (5.0, 5.0)
PCr (mL·kg ⁻¹)	25.3 ± 2.6	27.5 ± 4.0	30.7 ± 4.1

(95% C.I.)	(24.3, 26.3)	(26.0, 29.0)	(29.2, 32.8)
blood lactate (mM)	5.1 ± 0.6	11.7 ± 2.2	18.7 ± 3.2
(95% C.I.)	(4.8, 5.3)	(10.9, 12.4)	(17.6, 19.9)
glycolysis (mL·kg ⁻¹)	13.4 ± 2.0	35.2 ± 7.1	58.6 ± 10.5
(95% C.I.)	(12.7, 14.2)	(32.7, 37.8)	(54.8, 62.3)
<i>PCr</i> +glycolysis (mL·kg ⁻¹)	38.7 ± 3.1	62.7 ± 6.7	89.3 ± 8.7
(95% C.I.)	(37.6, 39.8)	(60.3, 65.1)	(86.2, 92.4)

Results of the two-way ANOVA revealed a significant effect of method ($p < 0.001$) and a significant effect of duration ($p < 0.001$). However, there was not a significant interaction, which would have been necessary to justify individual comparisons of *PCr*+glycolysis and oxygen deficit values from each of the three tests (3-min, 7-min, exhaustive). Nevertheless, effect sizes for the difference between the *PCr*+glycolysis and oxygen deficit were calculated individually, separately using results from each of the three tests; effect sizes were 0.32, 0.36, and 0.52, in the 3-min, 7-min, and exhaustive tests, respectively. Correlations between the two variables were 0.80 ($p < 0.01$), 0.83 ($p < 0.01$), and 0.94 ($p < 0.01$), in the three tests, respectively. When values were collapsed across the three durations, the correlation between the two estimates of anaerobic contribution was 0.99 ($p = 0.001$; $n = 90$). The Bland-Altman plot (Bland and Altman, 1986) of the differences between *PCr*+glycolysis and oxygen deficit values versus criterion measure, oxygen deficit, which is in Figure 1, demonstrated the similarity of the two measures. The mean values for the oxygen deficit and *PCr*+glycolysis are presented in the last line of Tables 1 and 2, respectively.

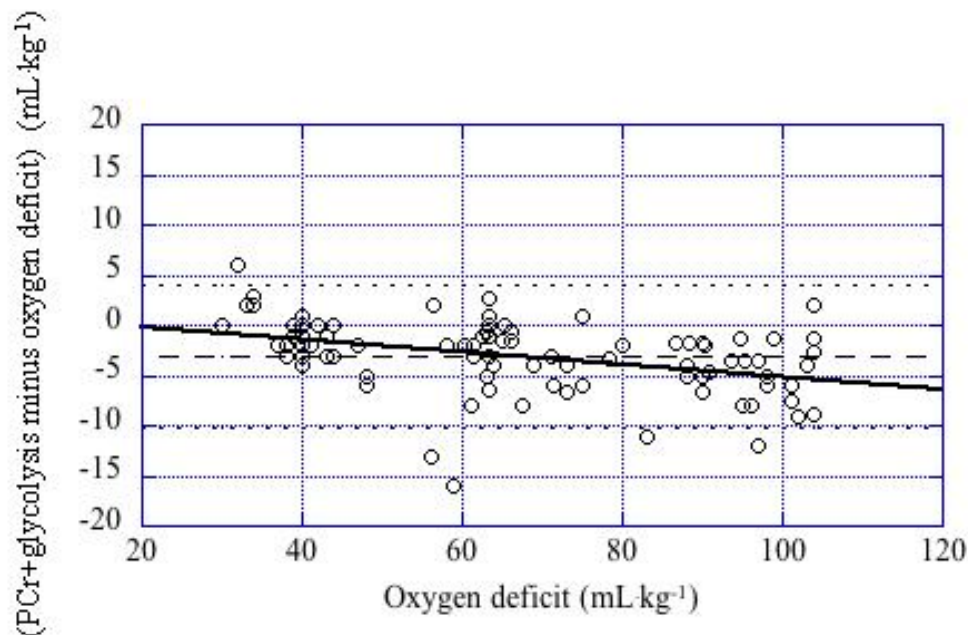


Figure 1. Bland-Altman plot of differences (*PCr*+glycolysis minus oxygen deficit) versus oxygen deficits, the criterion measure of anaerobic contribution in the 3-min, 7-min, and exhaustive exercise tests. The dashed line represents the mean difference (-3.0 ± 3.6 mL·kg⁻¹) and dotted lines represent the 95% confidence interval ($-10.1, 4.1$). The solid line represents the relationship between differences and oxygen deficits (difference = $1.1 - 0.063 \times$ oxygen deficit), which was not statistically significant.

DISCUSSION

The important result in the present study is that *PCr+glycolysis* provides a valid alternative to oxygen deficit as an estimate of anaerobic contribution, in non-exhaustive and exhaustive bouts of severe intensity running exercise. Similar values for *PCr+glycolysis* and oxygen deficit were obtained from tests performed for 3 min, for 7 min, or to exhaustion (~10.3 min). *PCr+glycolysis* provides an attractive alternative to oxygen deficit because it does not require any tests or assumptions about oxygen demand and it gives information about the two components of the anaerobic contribution.

Although there were earlier reports that the PCr and glycolytic contributions could be concurrently and independently quantified (e.g., Beneke et al., 2002; Bertuzzi et al., 2007), it was the work of Bertuzzi and colleagues (2010), that stimulated its adoption. In the first of several validation studies, Bertuzzi and colleagues (2010) reported that oxygen deficit in cycling tests (mean, 39 ml·kg⁻¹) and the sum of PCr (mean, 9 ml·kg⁻¹) and glycolysis measures (mean, 30 ml·kg⁻¹) did not differ and were highly correlated, although, notably, when expressed in units of ml O₂ and not when expressed relative to body weight. Urso and colleagues (2013) also had nine men perform exhaustive cycle ergometer tests, and they reported a significant relationship between the measure of glycolytic contribution and the difference between oxygen deficit and the measure of PCr contribution, which suggests that there was a meaningful relationship between *PCr+glycolysis* and oxygen deficit. Miyagi and colleagues (2017) calculated *PCr+glycolysis* and oxygen deficit values for men performing exhaustive cycle ergometer exercise at a range of work rates. At 115% of the work rate associated with VO_{2max}, which caused exhaustion after ~3 min, the values were significantly related if expressed in absolute terms ($r = 0.68$, $p < 0.01$) or relative to lower leg lean mass ($r = 0.55$, $p < 0.05$), but not when expressed relative to body mass ($r = 0.27$) or lean mass ($r = 0.45$). This pattern was repeated in the tests at work rates that resulted in exhaustion after 2½ min to 5½ min.

Using running exercise, Zagatto and colleagues (2016) demonstrated the robust nature of the *PCr+glycolysis* measure, reporting that it was unaffected across a range of speeds (120%, 115%, 110%, 105%, and 100% of the speed associated with VO_{2max}), speeds which elicited exhaustion in ~1 min to ~5 min. In addition, when values were expressed relative to body mass, correlations between *PCr+glycolysis* and oxygen deficit were 0.57 ($p < 0.05$) in ~5-min tests (100% of speed at VO_{2max}) and 0.77 ($p < 0.01$) in ~2½-min tests (115% of speed at VO_{2max}); however, there were not significant correlations in tests at 120%, 110%, or 105% of speed at VO_{2max}. Using results at (only) 115% and 100% of the speed at VO_{2max}, they concluded that *PCr+glycolysis* provides a valid alternative to oxygen deficit in running. Recently, Panissi and colleagues (2017) applied the *PCr+glycolysis* method to a session of high intensity interval training (running) and found no difference between mean values of *PCr+glycolysis* and oxygen deficit. In the present study, we reported values on a per-kg basis, and we found strong relationships ($r \geq 0.80$) for each exercise duration (3 min, 7 min, and exhaustion); thus we provide even stronger evidence regarding the validity of *PCr+glycolysis* in running than this prior validation study (Zagatto et al., 2016), in which there were strong relationships ($r = 0.56$ and $r = 0.77$) for only two of five different test durations. In addition, we included women as participants. Analyses revealed no interaction effect involving sex, suggesting that the *PCr+glycolysis* method is equally valid for women as for men.

Conceptually, our procedures were the same as in previous validation studies (Bertuzzi et al., 2010; Zagatto et al., 2016; Miyagi et al., 2017), and our methods differed only very slightly. First, we used a validated method of determining oxygen deficit (Hill and Vingren, 2011, 2012, 2013; Hill, 2014) that takes into account the increase in oxygen demand during constant speed severe intensity exercise (Noordoof et al., 2015) making the assumption that the slow component is explained by an increase in oxygen demand or loss of skeletal

muscle contractile efficiency (Jones et al., 2011). Second, in determining the glycolytic contribution, we used the conversion factor of $3.3 \text{ mL O}_2 \cdot \text{kg}^{-1}$ per mM increase in blood lactate concentration (Margaria et al., 1933), rather than $3.0 \text{ mL O}_2 \cdot \text{kg}^{-1}$ per mM. In addition, the lactate concentration values obtained in the present study likely underestimate values that would have been measured using a Yellow Springs (YSI Inc, Yellow Springs, OH, USA) (Franchini et al., 2004). Third, while these other researchers reduced oxygen deficit estimates to account for the use of stored oxygen, we did not. Of note, their 10% reduction would be $\sim 9 \text{ mL O}_2 \cdot \text{kg}^{-1}$ in our exhaustive tests, which is considerably higher than the $2.3 \text{ mL O}_2 \cdot \text{kg}^{-1}$ calculated by Barstow and colleagues (1990). To recapitulate, the small methodological differences between this and previous validation studies (Bertuzzi et al., 2010; Urso et al., 2013; Zagatto et al., 2016; Miyagi et al., 2017) resulted in slightly larger estimates for both *PCr+glycolysis* and oxygen deficit in the present study.

To validate the *PCr+glycolysis* measure requires the ability to measure oxygen deficit, which is dependent upon the ability to estimate the energy demand or oxygen demand. While the estimation of oxygen demand of heavy or severe intensity exercise has long been contentious and a source of controversy (e.g., Green and Dawson, 1996; Noordhof et al., 2010) and continues to be the focus of research study, it can be accomplished in activities like running, cycling, and rowing, by extrapolation from steady state responses in moderate intensity exercise. However, this method cannot be applied to activities like rock climbing, martial arts, and surfing, or to most team sports. Quantifying the anaerobic contribution in these sports or measuring the anaerobic capacities of athletes in these sports can be a very useful tool for coaches, athletes, and sport scientists. Thus, the importance of a measure like *PCr+glycolysis* is obvious, and the importance of carefully validating this method against oxygen deficit is clear.

Despite difficulties in validating the *PCr+glycolysis* measure, it has been used in a variety of activities, including rock climbing (Bertuzzi et al., 2007), martial arts (Lopes-Silva et al., 2018), and table tennis test (Zagatto and Gobatto, 2012). However, it has been validated for only one of these activities. Zagatto and Gobatto (2012) reported that *PCr+glycolysis* in table tennis tests lasting ~ 2 min, ~ 3 min, ~ 5 min, and ~ 8 min averaged $60 \text{ mL} \cdot \text{kg}^{-1}$, which was not different from the oxygen deficit in the exhaustive ~ 5 -min test (mean, $60 \text{ mL} \cdot \text{kg}^{-1}$). Across the four tests in this latter study, *PCr* estimates averaged $43 \text{ mL} \cdot \text{kg}^{-1}$, which is far higher than reported in other studies (e.g., Beneke et al., 2002; Zagatto et al., 2016; Miyagi et al., 2017; the present study) and the glycolysis contribution averaged only $18 \text{ mL} \cdot \text{kg}^{-1}$, suggesting that both oxygen deficit and the *PCr+glycolysis* methods must be evaluated further prior to use in activities other than large muscle activities like cycling and running. In the present study, we have not simply confirmed that *PCr+glycolysis* provides a valid replacement for MAOD, we have demonstrated a stronger relationship than in previous studies. We attribute this to the methods used in calculating *PCr+glycolysis* and in calculating MAOD.

Not only is the estimation of exercise efficiency a contentious element in the calculation of oxygen deficit (e.g., Green and Dawson, 1996; Noordhof et al., 2010), but it is also, in and of itself, an important factor related to sport performance (Joyner and Coyle, 2008). While one attraction of the *PCr+glycolysis* method is that it does not require measurement of, or assumptions about, exercise efficiency and oxygen demand, we believe that the method can also be used to generate a measure of exercise efficiency. If we add our *PCr+glycolysis* value to the accumulated oxygen uptake to obtain an estimate of total oxygen cost, divide this by the exercise duration to obtain an estimate of the average oxygen demand, and then divide this average oxygen demand by the work rate (divide by speed, for running), we obtain a meaningful measure of the oxygen cost of exercise (i.e., the mathematical inverse of 'efficiency'), with units of $\text{mL} \cdot \text{min}^{-1}$ per W (or $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ per $\text{m} \cdot \text{min}^{-1}$, for running). For other activities, the efficiency can simply be described by the oxygen demand, for example, the oxygen demand (in $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) of a simulated elite taekwondo competition or the oxygen demand of a Class 5.10 Grade II climb. Thus, using the *PCr+glycolysis* concept,

a measure of efficiency can be determined without the need for performing the series of submaximal steady state trials. For continuous severe intensity activities, these efficiency values are somewhat intensity-specific, as the slow component contribution to the true total oxygen cost is greater with lower intensities and longer durations of exercise. However, the impact will be small, as the (larger) total oxygen cost is divided by (the longer) exercise duration when generating the measure of average oxygen demand.

CONCLUSION

It has become common practice in several laboratories to replace the cumbersome and somewhat contentious oxygen deficit measure with the sum of the phosphocreatine contribution, obtained from kinetics of the post-exercise VO_2 response curve, and the glycolytic contribution, obtained from peak post-exercise blood lactate concentration. Results of the present study provide strong support for prior studies validating this *PCr+glycolysis* measure against oxygen deficit (Bertuzzi et al., 2010; Urso et al., 2013; Zagatto et al., 2016; Miyagi et al., 2017) or using the measure as an alternative to oxygen deficit (e.g., Bertuzzi et al., 2007; de Campos Mello et al., 2009; Zagatto and Gobatto, 2012; Ferreira et al., 2014 Lopes-Silva et al., 2018). It is concluded that the *PCr+glycolysis* measure provides a valid estimate of anaerobic contribution in running exercise, in women as well as in men.

DISCLOSURE OF INTEREST

The authors report no conflict of interest.

REFERENCES

- Barstow, T. J., Lamarra, N., Whipp, B. J. (1990). Modulation of muscle and pulmonary O₂ uptakes by circulatory dynamics during exercise. *Journal of Applied Physiology* 68: 979–989. <https://doi.org/10.1152/jappl.1990.68.3.979>
- Beneke, R., Pollman, C., Bleif, I., Leithäuser, R. M., Hüttler, M. (2002). How anaerobic is the Wingate Anaerobic Test for humans? *European Journal of Applied Physiology* 87: 388–392. <https://doi.org/10.1007/s00421-002-0622-4>
- Bertuzzi, R. C. M., Franchini, E., Kokubun, E., Kiss, M. A. (2007). Energy system contributions in indoor rock climbing. *European Journal of Applied Physiology* 101: 293–300. <https://doi.org/10.1007/s00421-007-0501-0>
- Bertuzzi, R. C. M., Franchini, E., Ugrinowitsch, C., Kokubun, E., Lima-Silva, A. E., Pires, F. O., Nakamura, F. Y., Kiss, M. A. P. D. M. (2010). Predicting MAOD using only a supramaximal exhaustive test. *International Journal of Sports Medicine* 31(7): 477–481. <https://doi.org/10.1055/s-0030-1253375>
- Bland, J. M., Altman, D. G. (1986). Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* i(8476): 307–310. [https://doi.org/10.1016/s0140-6736\(86\)90837-8](https://doi.org/10.1016/s0140-6736(86)90837-8)
- de Campos Mello, F., de Moraes Bertuzzi, R. C., Grangeiro, P. M., Franchini, E. (2009). Energy systems contribution in 2,000 m race simulation: a comparison among rowing ergometers and water. *European Journal of Applied Physiology* 2009 615–619. <https://doi.org/10.1007/s00421-009-1172-9>
- Fell J. W., Rayfield, J. M., Gulbin, J. P., Gaffney, P. T. (1998). Evaluation of the Accusport lactate analyser. *International Journal of Sports Medicine* 19: 199–20. <https://doi.org/10.1055/s-2007-971904>
- Ferreira, M. I., Barbosa, T. M., Costa, M. J., Neo, D. A. Neiva, H. P., Vilaça, J., Marinho, D. A. (2014). Effects of swim training on energetic and performance in women masters' swimmers. *Journal of Human Sport and Exercise* 11(1): 99–106. <https://doi.org/10.14198/jhse.2016.111.08>

- Franchini, E., Matsuchigue, K. A., Colantonio, E., Kiss, M. A. P. D. (2004). Comparação dos analisadores de lactato Accusport e Yellow Springs. *Brazilian Journal of Science and Movement* 12: 39–44. <https://doi.org/10.18511/rcbm.v12i1.540>
- Green, S., Dawson, B. T. (1996). Methodological effects on the VO₂–power regression and the accumulated O₂ deficit. *Medicine and Science in Sports and Exercise* 28: 392–397. <https://doi.org/10.1249/00005768-199603000-00016>
- Henry, F. M. (1951). Aerobic oxygen consumption and alactic debt in muscular work. *Journal of Applied Physiology* 3: 427–438. <https://doi.org/10.1152/jappl.1951.3.7.427>
- Hill, A. V., Lupton, H. (1923). Muscular exercise, lactic acid, and the supply utilization of oxygen. *Quarterly Journal of Medicine* 16: 135–171. <https://doi.org/10.1093/qjmed/os-16.62.135>
- Hill, D. W. (1999). Determination of the accumulated O₂ deficit in exhaustive short-duration exercise. *Canadian Journal of Applied Physiology*, 21, 63–74.
- Hill, D. W. (2014). Morning–evening differences in response to exhaustive severe-intensity exercise. *Applied Physiology, Nutrition, and Metabolism*, 39, 248–254. <https://doi.org/10.1139/apnm-2013-0140>
- Hill, D. W., Vingren, J. L. (2011). Maximal accumulated oxygen deficit in running and cycling. *Applied Physiology, Nutrition, and Metabolism*, 36, 831–838. <https://doi.org/10.1139/h11-108>
- Hill, D. W., Vingren, J. L. (2012). The effect of pedalling cadence on maximal accumulated oxygen deficit. *European Journal of Applied Physiology*, 112, 2637–2643. <https://doi.org/10.1007/s00421-011-2240-5>
- Hill, D. W., Vingren, J. L. (2013). Effects of exercise mode and participant sex on measures of anaerobic capacity. *Journal of Sports Medicine and Physical Fitness*, 53, 255–263.
- Hill, D. W., Williams, C. S., Burt, S. E. (1997). Responses to exercise at 92% and 100% of the velocity associated with VO_{2max}. *International Journal of Sports Medicine* 18: 325–329. <https://doi.org/10.1055/s-2007-972641>
- Jones, A. M., Grassi, B., Christensen, P. M., Krstrup, P., Bangsbo, J., Poole, D. C. (2011). Slow component of VO₂ kinetics: mechanistic bases and practical applications *Medicine and Science in Sports and Exercise* 43(11) 2046–2062. <https://doi.org/10.1249/mss.0b013e31821fcfc1>
- Joyner, M. J., Coyle, E. F. (2008). Endurance exercise performance: the physiology of champions. *Journal of Physiology* 586: 35–44. <https://doi.org/10.1113/jphysiol.2007.143834>
- Krouwer, J. S. (2007). Why Bland–Altman plots should use X, not (Y+X)/2 when X is a reference method. *Statistics in Medicine* 27(5): 778–780. <https://doi.org/10.1002/sim.3086>
- Li, Y., Niessen, M., Chen, X., Hartmann, U. (2015). Overestimate of relative aerobic contribution with maximal accumulated oxygen deficit: a review. *Journal of Sports Medicine and Physical Fitness* 55: 377–382.
- Lopes-Silva, J. P., Da Silva Santos, J. F., Artiola, G. G., Loturco, I., Abbiss, C., Franchini, E. (2018). Sodium bicarbonate ingestion increases glycolytic contribution and improves performance during simulated taekwondo combat. *European Journal of Sport Science* 18(3): 431–440. <https://doi.org/10.1080/17461391.2018.1424942>
- Margaria, R., Edwards, H. T., Dill, D. B. (1933). The possible mechanism of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. *American Journal of Physiology* 106: 689–714. <https://doi.org/10.1152/ajplegacy.1933.106.3.689>
- Miyagi, W. E., de Poli, R. A. B., Papoti, M., Bertuzzi, R., Zagatto, A. M. (2017). Anaerobic capacity estimated in a single supramaximal test in cycling: validity and reliability analysis. *Science Reports* 7: article number 42485. <https://doi.org/10.1038/srep42485>

- Noordhof, D. A., de Koning, J. J., Foster, C. (2010). The maximal accumulated oxygen deficit method: A valid and reliable measure of anaerobic capacity? *Sports Medicine* 40(4): 285–302. <https://doi.org/10.2165/11530390-000000000-00000>
- Noordhof, D. A., Mulder, R. C. M., Malterer, K. R., Foster, C., de Koning, J. J. (2015). The decline in gross efficiency in relation to cycling time trial length. *International Journal of Sports Physiology and Performance* 10: 64–70. <https://doi.org/10.1123/ijspp.2014-0034>
- Panissa, V. L. G., Fukuda, D. H., Caldeira, R. S., Gerosa-Neto, J., Lira, F. S., Zagatto, A. M., Franchini, E. (2018). Is oxygen uptake measurement enough to estimate energy expenditure during high-intensity intermittent exercise? Quantification of anaerobic contribution by different methods. *Frontiers of Physiology* 9: 868. <https://doi.org/10.3389/fphys.2018.00868>
- Thomas, S., Reading, J., Shephard, R. J. (1992). Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Canadian Journal of Sport Sciences* 17: 338–345.
- Urso, R. R., Silva-Cavalcante, M. D., Correia-Oliveira, C. F., Bueno, S., Damasceno, M. V., Lima-Silva, A. E., Bertuzzi, R. (2013). Determinação dos metabolismos láctico e alático da capacidade anaeróbia por meio do consumo de oxigênio (Determination of lactic and alactic metabolisms of the anaerobic capacity using oxygen uptake). *Revista Brasileira de Cineantropometria e Desempenho Humano* 15: 616–627. <https://doi.org/10.5007/1980-0037.2013v15n5p616>
- World Health Organization. (2013). Declaration of Helsinki: Ethical principles for medical research involving human subjects. *Journal of the American Medical Association* 310: 2191–2194. <https://doi.org/10.1001/jama.2013.281053>
- Zagatto, A. M., Bertuzzi, R., Miyagi, W. E., Padulo, J., Papoti, M. (2016). MAOD determined in a single supramaximal test: a study on the reliability and effects of supramaximal intensities. *International Journal of Sports Medicine* 37: 700–707. <https://doi.org/10.1055/s-0042-104413>
- Zagatto, A. M., Gobatto, C. A. (2012). Relationship between anaerobic parameters provided from MAOD and critical power model in specific table tennis set. *International Journal of Sports Medicine* 33: 613–620. <https://doi.org/10.1055/s-0032-1304648>



This work is licensed under a [Attribution-NonCommercial-NoDerivatives 4.0 International](https://creativecommons.org/licenses/by-nc-nd/4.0/) (CC BY-NC-ND 4.0).