

Short Communication

Can Blood Lactate Concentrations Rise Significantly After Very Short Duration Swimming Bouts?

Hélvio O. Affonso¹, Arilson S. Silva², and Ricardo J. Fernandes^{3*}

¹Pharmaceutical Sciences Graduate Program, University of Vila Velha (UVV), Brazil

²Arilson Silva Swimming coach - Arilson Soares da Silva- EPP, Brazil

³Centre of Research, Education, Innovation and Intervention in Sport (CIF12D), Faculty of Sport and Porto Biomechanics Laboratory (LABIOMEPE), University of Porto, Portugal

***Corresponding author**

Ricardo J. Fernandes, Centre of Research, Education, Innovation and Intervention in Sport (CIF12D), Faculty of Sport and Porto Biomechanics Laboratory (LABIOMEPE), University of Porto, Porto, Portugal

Submitted: 24 December 2018

Accepted: 14 January 2019

Published: 16 January 2019

ISSN: 2379-0571

Copyright

© 2019 Fernandes et al.

OPEN ACCESS**Keywords**

• Lactate; Glycolysis; Swimming; Sprints

Abstract

This study examined the blood lactate concentrations ([La⁻]) after vigorous swimming exercises of very short durations. Three top 10 World ranked male swimmers performed three bouts at maximal intensity in their best swimming technique, one of 10 m (starting without fixed support) and two of 15 m (pushing the wall and the starting block). Blood samples were collected 30 s after the swims and [La⁻] were assessed using a portable analyser. Swimming performances were evaluated using a digital chronometer. The main results were that for maximal intensity efforts lasting approximately 5 to 6 s, independently of swimming front crawl, butterfly or breaststroke, swimmers produced 12 to 22 mmol.l⁻¹ of [La⁻]. Therefore, the idea that creatine phosphate and ATP can power by themselves intense muscle contraction for 5 to 6 s should be questioned. As the current swimmers are 50 m events specialists, it seems logical that their tremendous metabolic power is supported by both high-energy phosphates and anaerobic glycolysis. Elite swimmers are like finely tuned race cars that are trained (or should be) to have a physiological background that match the demands and requirements of their races. In the future, we will try to deeply characterize these type of exertions in very highly trained individuals also looking for non-conventional biomarkers particularly regarding oxidative stress. Practitioners of other sports and of different levels will also be analysed.

ABBREVIATIONS

[La⁻]: Blood Lactate Concentrations; ATP: Adenosine Triphosphate; PCR: Phosphocreatine

INTRODUCTION

For a long time, oxygen uptake and heart rate assessment were seen the best mean of determining swimming endurance capacity and frequently used to characterize swimmers' cardiorespiratory condition. Nowadays, these variables are not considered to be sufficient for the assessment of swimming performance bioenergetics, particularly its anaerobic component. In fact, most competitive swimming events last from ~20 s until ~2 min (the 50, 100 and 200 m distances) where the anaerobic contribution is decisive [1,2]. Therefore, samples for blood lactate concentrations ([La⁻]) determination have been frequently collected in swimming, with the first studies using venous blood samples (e.g. [3]) that are often perceived by swimmers as traumatic. So, capillary blood samples from puncture wound to the fingertip or earlobe are preferentially used (also due to the non-expensive portable battery-operated automated analysers).

However, although [La⁻] determinations are fast, easy and accurate (as the testing procedure is quite sophisticated, technical and scientific), for many years the use and interpretation of lactate levels were based on empirical assumptions. These analyses

lack scientific evidence about their real significance, existing some misconceptions about lactate testing, not only regarding the methodology, but also when it refers to handling the results [4,5]. Lactate is the end product of the anaerobic metabolism, diffusing from the muscle cell into the bloodstream when aerobic and other metabolic pathways are unable to keep up with the removal of pyruvate. The current belief is that glycolysis enters in action after the depletion of the phosphocreatine (CP) energy system, reason why the highest [La⁻] in swimming have been observed at the 100 and 200 m swimming exertions (1 to 2 min of exertion; [6,7]). The current study aims to present [La⁻] data regarding very short maximal swims where it will be shown evidences that glycolysis works at a very high rate much sooner than it is expected.

MATERIALS AND METHODS

Three male elite sprint swimmers (one front crawler, one butterfly and one breaststroker, ranked on the top 10 of the World) volunteered to participate in the current study and signed an informed consent form. Their mean ± SD main body characteristics were: 28.0 ± 2.2 years of age, 80.2 ± 1.72 kg of body mass, 190.0 ± 0.1 cm of body height, 7.5 ± 1.2% of fat mass and 985,1020 ± 970 of FINA Point Score at the 50 m freestyle, butterfly and breaststroke (respectively). The body characteristics assessment was conducted before the beginning

of the experimental protocol, with body mass and fat mass measured using the protocol sum of seven skin folds [8].

In a 50 m swimming pool, each swimmer performed one 10 m clean swimming (starting from the 40 m mark until the wall) and two 15 m bouts (one with block start and another with wall push) at maximal speed in their best swimming technique. In-between bouts it were implemented 10 min rest intervals. Times were assessed using a digital chronometer (Seiko S140, Japan) and blood samples were collected from the fingertip for [La⁻] assessment before and 30 s after every maximal bout using a portable analyser (Lactate Plus, Waltham, MA 02454-9141, EUA). The testing session took place at the competitive period of the training season.

RESULTS AND DISCUSSION

In Table 1 it is described the individual and mean ± SD values of the time of exertions and corresponding [La⁻]. It is possible to observe very high (La⁻) values both at the 10 and 15 m sprints independently of using (or not) block and wall fixed starts (Table).

It is common knowledge that there are three different but closely integrated processes that operate together to satisfy the energy requirements during exercise: the anaerobic energy pathways separated in a lactic and lactic components (referring to the processes involved in the phosphagen splitting and the nonaerobic glycolysis that promotes the breakdown of carbohydrate to lactic acid) and the aerobic energy pathway (the degradation of carbohydrates and fats in the presence of oxygen). When analysing the current literature, it seems consensual that, when exercising, energy is obtained from the three energy-supplying processes, each one contributing sequentially but in an overlapping fashion (e.g. [4,9]). However, maybe because the assessment of anaerobic energy release during exercise is much less precise than the assessment of aerobic energy release [9], it is still believed that ATP and CP storages supports sprint swimming during ~10 s or the first 15 m of a swim race and that only then glycolysis starts contributing significantly (e.g. [10,11]).

In fact, it is known for several years that the glycolytic contribution to maximal efforts lasting 20 to 120 s results in a significant accumulation of muscle and blood lactate [12] and that peak [La⁻] following maximal exercise is positively related to performance in swimming events [13]. However, few publications are available focusing on the fact that ATC-CP is not really a typical energy system but a “temporal” energy buffer that operates at sites of high energy translocation [14] and that anaerobic glycolysis is recruited at the onset of exercise with the glycolytic pathway increasing almost instantaneously [15-17]. This not so “classical” way of seeing Cr and PCr as cellular energy carriers does not turn them less important in skeleton muscle metabolism but highlights the fact that very high [La⁻] might appear in short very intensive workouts due to the fast activation of glycolysis. In fact, after only 6 s, energy derived from this pathway can account for up to 50% of the total energy demand [18] and we believe that in elite athletes the percentage might be even higher.

However, it is important to refer that the 12 to 22 mmol.l⁻¹ [La⁻] observed in the current study for such short and explosive swims might only be possible for subjects with very high levels of muscular strength and power that can make an effective activation of the “fastest” muscle type IIb fibers in response to a very demanding external load. When compared to untrained individuals and/or endurance athletes, practitioners of strength and power sports present a greater neuromuscular activation compared to the same relativized stimulus [19] and individuals who exercise regularly have higher muscle glycogen levels than their sedentary counterparts [20]. This is even more expected in elite swimmers that are (or should be) submitted to optimal sprint swimming sets that result in muscle glycogen depletion followed by specific nutritional programs allowing glycogen super compensation.

CONCLUSION

Given the importance that exercise physiology has in swimming training, coaches and researchers should have a clear understanding about the contribution of glycolysis in short

Table 1: Maximal blood lactate concentrations (before and) after different sprint swimming distances. The stimulus type and corresponding time durations are also displayed.

	Stimulus	Time (s)	[La ⁻] (before) and after (mmol.l ⁻¹)
Front crawler	10 m clean swim	4.54	(0.9) 12.1
	15 m with block start	5.05	(1.2) 17.4
	15 m with wall push	5.51	(1.0) 15.3
Butter flier	10 m clean swim	4.84	(0.6) 16.5
	15 m with block start	5.24	(0.7) 21.9
	15 m with wall push	5.70	(0.9) 18.4
Breaststroker	10 m clean swim	6.10	(1.4) 14.7
	15 m with block start	6.25	(1.2) 22.3
	15 m with wall push	6.65	(1.0) 20.7
Mean ± SD	10 m clean swim	5.16±0.83	(0.97±0.40) 17.4±2.21
	15 m with block start	5.51±0.65	(1.03±0.29) 20.5±2.72
	15 m with wall push	5.95±0.61	(0.97±0.06) 18.2±2.71

[La⁻]: Blood Lactate Concentrations

maximal intensity bouts. We have presented new data on [La] values after 5 to 7 s all out swims in three of the four conventional swimming techniques, evidencing very high rates of anaerobic power in efforts less than 10 s duration. This is original and should have new developments in the future, particularly focusing on other conventional variables (as glycemia and creatine kinase) and non-conventional and oxidative stress biomarkers, checking for indicators of body discomfort in general (muscle soreness and cramps) and gut disturbances in particular (nausea) that are typical of maximal exertions. In fact, previous studies have already indicated increases in both lactate production and oxidative stress in acute exercise [21]. A comparison with elite level swimmers from middle and long distance events will be also welcome.

REFERENCES

1. Zamparo P, Capelli C, Cautero M, Di Nino A. Energy cost of front-crawl swimming at supra-maximal speeds and underwater torque in young swimmers. *Eur J Appl Physiol.* 2000; 83: 487–491.
2. Ribeiro J, Figueiredo P, Sousa A, Monteiro J, Pelarigo J, Vilas-Boas JP. et al. VO₂ kinetics and metabolic contributions during full and upper body extreme swimming intensity. *Eur J Appl Physiol.* 2015; 115: 1117-1124.
3. Holmér I. Physiology of swimming man. *Acta Phys Scand.* 1974; 407: 1-55.
4. Gladen LB. Lactate metabolism: a new paradigm for the third millennium. *J Physiol.* 2004; 558: 5-30.
5. Olbrecht J. Lactate production and metabolism in swimming. In L Seifert, D Chollet, I Mujika (eds.), *World book of swimming: from science to performance.* 2011; 255-275.
6. Olbrecht J. *The science of winning. Planning, periodizing and optimizing swim training.* Luton: Swim shop. 2000.
7. Vescovi JD, Falenchuk O, Wells G. Blood lactate concentration and clearance in elite swimmers during competition. *Int J Sports Physiol Perform.* 2011; 6: 106-117.
8. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. 1978. *Br J Nutr.* 2004; 91: 161-168.
9. Gastin PB. Energy system interaction and relative contribution during maximal exercise. *Sports Med.* 2001; 31: 725-741.
10. *The effect of biomechanics on physiology.* New Zealand: Swim formation Ltd. 2007.
11. Nugent F, Comyns T, Nevill A, Warrington GD. The effects of low-volume, high-intensity training on performance parameters in competitive youth swimmers. *Int J Sports Physiol Perform.* 2018; 24:1-22.
12. Jacobs I. Blood lactate - implications for training and sports performance. *Sports Med.* 1986; 3: 10-25.
13. Bonifazi M, Sardella F, Lupo C. Preparatory versus main competitions: differences in performances, lactate responses and pre-competition plasma cortisol concentrations in elite male swimmers. *Eur J Appl Physiol.* 2000; 82: 368–373.
14. Greenhaff PL. The creatine-phosphocreatine system: there's more than one song in its repertoire. *J Physiol.* 2001; 537: 657.
15. Hultman E, Sjöholm H. Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. *J Physiol.* 1983; 345: 525-532.
16. Ren JM, Semenkovich CF, Holloszy JO. Adaptation of muscle to creatine depletion: effect on GLUT-4 glucose transporter expression. *Am J Physiol.* 1993; 264 (Cell Physiol. 33): 146-150.
17. Holloszy JO, Kohrt WM. Metabolism during and after exercise. *Annu Rev Nutr.* 1996; 16: 121-138.
18. Gaitanos G, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol.* 1993; 75: 712-719.
19. Ahtiainen JP, Hakkinen K. Strength athletes are capable to produce greater muscle activation and neural fatigue during high-intensity resistance exercise than non athletes. *J Strength Cond Res.* 2009; 23: 1129-1134.
20. Putman CT, Jones NL, Hultman E, Hollidge-Horvat MG, Bonen A, McConachie DR et al. Effects of short-term submaximal training in humans on muscle metabolism in exercise. *Am J Physiol.* 1998; 275: 132–139.
21. Groussard C, Rannou-Bekono F, Machefer G, et al. Changes in blood lipid peroxidation markers and antioxidants after a single sprint anaerobic exercise. *Eur J Appl Physiol.* 2003; 89: 14-20.

Cite this article

Affonso HO, Silva AS, Fernandes RJ (2019) Can Blood Lactate Concentrations Rise Significantly After Very Short Duration Swimming Bouts?. *Ann Sports Med Res* 6(1): 1139.