RESEARCH ARTICLE

MONITORING BLOOD GLUCOSE DURING MAXIMAL INTENSITY RACING AT NATIONAL SWIMMING COMPETITIONS

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ARTICLE INFO	ABSTRACT

Introduction: Glucose flux is known to be affected by exercise and many articles have been written concerning glucose rate of appearance, rate of disappearance, and of oxidation. Little is published on the response of blood glucose during maximal competition however. An investigation was undertaken to monitor Blood glucose during competition swimming and to see if there was any relationship to blood lactate and duration of effort.
Methods: One hundred and four (104) swimmers from New Zealand world championship trials and South African Olympic trials (National events). Male (n=62) and Female (n=42) subjects were aged between 16 and 24 years. Peak in glucose and lactate levels was measured. The data were pooled for each event (50m to 1500m).
Results: Representing race periods from 22 seconds to 18 minutes. The highest values were seen in the 800 and 1500m swims, with values between 8.5 and 11.2 mmol/L respectively.
Findings: There appears to be a critical balance point around 45 to 50 seconds where the blood glucose value will remain at or around the resting value although this does not mean that there is not considerable glucose flux at this point. Across different time lines, at maximal effort, blood glucose appears to respond in a predictable way. This may prove useful for the identification of parameters for performance and for training also.

INTRODUCTION

Measurement of glucose flux has been widely used to monitor changes in metabolism (Friedlander *et al.,* 1997). The vast majority of these experiments however have been at lower intensities (Coggan *et al.,* 1992; Hargreaves and Proietto, 1994). Even in these days of portable analysers, it is still difficult to measure during actual competition performances, where the response may be very different to that seen in a simulation. Previously, lactate has been used as a marker of relative effort (Pyne *et al.,* 2001) and was assumed (wrongly) to be an accurate predictor of competition outcome (Rushall 1991). Glucose flux is known to be affected by exercise (Hargreaves and Proietto, 1994; Hargreaves, 1997) and many articles have been written concerning glucose rate of appearance (Ra), rate of disappearance (Rd), and of oxidation (Rox). Much of the research carried out to date has looked at either lower intensity exercise (50-65% MVO2) or what is deemed high intensity exercise (approx. 80 to 85% MVO2) (Brooks, 1997; Coggan *et al.,* 1995), and the metabolic events that occur at these intensities.

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Although some authors have looked at maximal intensity exercise over shorter bursts (Bangsbo *et al.,* 1992; Medbo *et al.,* 1988), only one study appears to have investigated the changes in blood glucose at these intensities (Swanwick and Matthews, 2017), with the authors identifying a glucose turn point, that may be used as a marker of exercise intensity, and subsequent continued rise towards maximal intensities. At lower intensities, liver glycogenolysis and the flux of blood glucose can provide the carbohydrate for the required energy demand (Maughan, 2009). There has been much conjecture regarding what occurs at moderately high intensity exercise (approx. 80 to 85% MVO2) (Brooks, 1997; Coggan *et al.,* 1992) in terms of the metabolic events at this level of effort. Unfortunately, much of this area is fraught with mis-understanding. This relative intensity is based on or around the "Anaerobic threshold". Such a term is influenced by a wide range of responses depending upon the level of fitness of the subjects used for assessment (trained/untrained) (Parker *et al.,* 1997; Rushall and King, 1994). Additionally, where the subjects are deemed to be "trained", additional variability occurs depending upon the subjects' event type (endurance/power/speed) and specialization of the athlete (strength/power/agility). This range of different populations probably contributes to the range of different profiles seen over
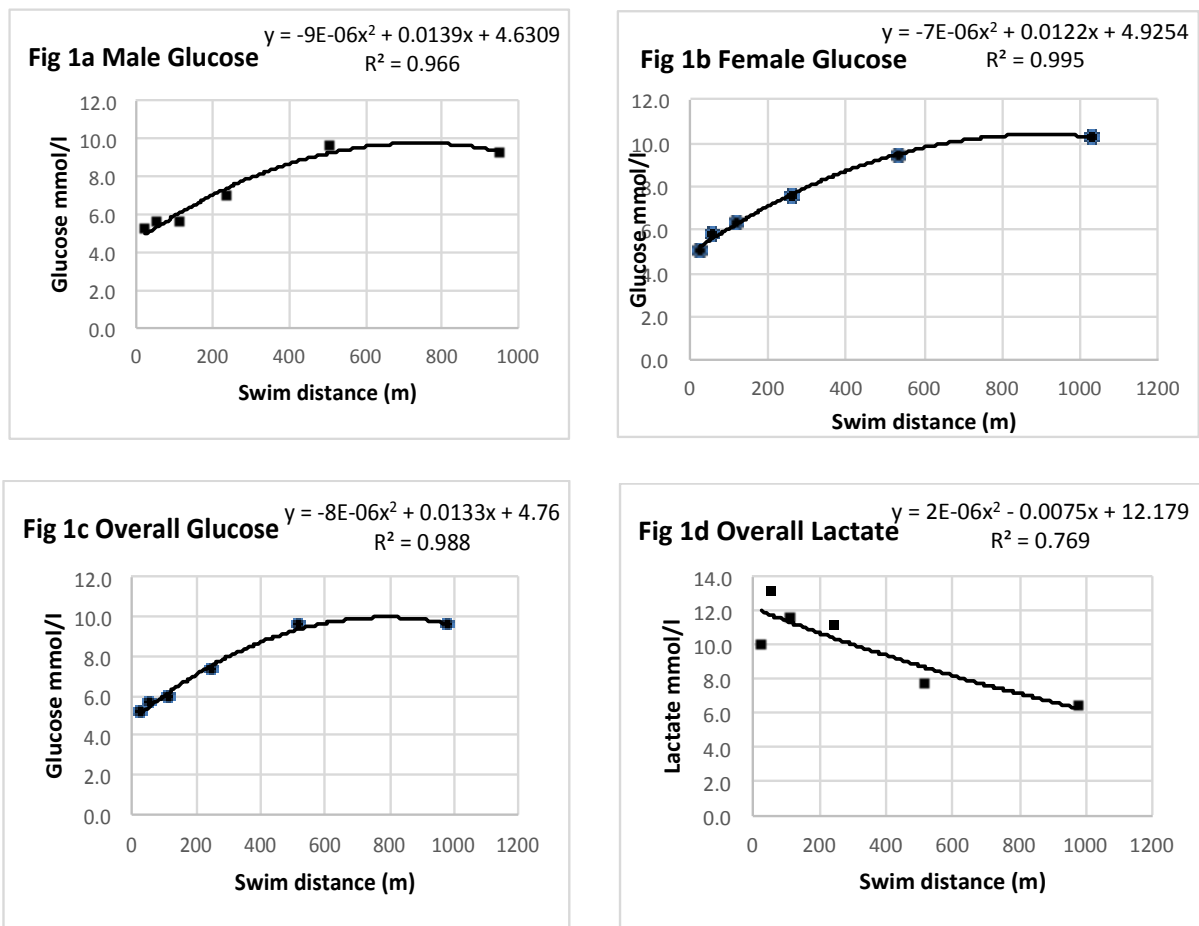
Fig 1a Male Glucose — $y = -9\text{E-}06x^2 + 0.0139x + 4.6309$, $R^2 = 0.966$

Fig 1b Female Glucose — $y = -7\text{E-}06x^2 + 0.0122x + 4.9254$, $R^2 = 0.995$

Fig 1c Overall Glucose — $y = -8\text{E-}06x^2 + 0.0133x + 4.76$, $R^2 = 0.988$

Fig 1d Overall Lactate — $y = 2\text{E-}06x^2 - 0.0075x + 12.179$, $R^2 = 0.769$

Figure 1a,b,c,d. relationship of blood glucose and lactate to swim distance in males, females and overall

the varying timelines of different protocols used (Zacca *et al.,* 2010). In such circumstances, the relative contribution of fat, protein, and the source of carbohydrate can also be altered. At these higher intensities, the recruitment pattern of slow and fast twitch muscle fibres, dependent upon the demanded power output, will also influence the amount and rate of energy required and its path in metabolism (Dudley *et al.,* 1982). Along with this issue, there is also the potential risk of nutritional state bias, dependent on pre, or post prandial, etc (Jeukendrup and Gleeson, 2010). The training state of the subject can create a wide range of changes. Changes in the efficiency of the system to meet the demand, changing muscle fibre recruitment patterns, increased or decreased mitochondrial density, capillary density, enzymic activity, blood flow, and oxygen extraction capability etc.,can all have an influence (Friedlander *et al.,* 1997). To date, there appears to be little research into blood glucose levels during maximal efforts such as those seen in the majority of Olympic sports and high level sporting competition. The purpose of this paper was to identify the relationship between blood glucose and a range of maximal efforts over a range of time lines in competitive swimmers.

METHODS

At two national swimming championships, data were gathered on the responses in swimmers to maximal intensity efforts in swimming of different durations.

Subjects: One hundred and four (104) swimmers from New Zealand world championship trials and South African Olympic trials (National events).

Male (n=62) and Female (n=42) subjects were aged between 16 and 24 years. All swimmers (and parents, where required) were given information on the type of testing and the purpose of the testing to be carried out. Subjects were made fully aware that, if at any time they felt uncomfortable or wished to withdraw from the testing, they could do so. Each of the subjects who took part in the study gave their informed consent to provide samples for the study. The study had prior approval from the Ethics Committee of the Christchurch College of Education, New Zealand.

Study design

Each swim began with a dive start, started with the official starting signal. End time of the swim for each swimmer in each event was taken from the official results. Where possible, stroke count was recorded for each 50-meter lap to allow the calculation of the stroke characteristics (distance per stroke and the stroke rate). As study data were collected during race conditions, heart rate was assumed to be maximal for each subject at the end of each swim. Therefore, the swimmers were asked to provide their maximal heart rate, where known. Following the completion of a race, swimmers immediately came to the testing bay. Capillary blood (25 μl) was collected from ear lobe puncture two minutes after the completion of each swim and until a peak in glucose and lactate levels was found. Lactate concentration was analyzed by a Lactate Pro Lactate meter (Axon Labs., Austria). Glucose was analyzed using Accu-chek Abbiba (Roche, USA). The data were pooled for each event (50m to 1500m), representing race periods from 22 seconds to 18 minutes. The swim pace was controlled by the swimmer. Relative intensities equate to an approximate

MVO$_2$ of 96% in 1500m and 170% in 50m events. Dietary intake, 2 hours prior to competing was noted for each swimmer and testing was conducted in accordance with the protocols of the Australian National Swimming Team for in competition testing (19). Glucose, lactate and time data were matched and plotted separately for males, females and combined. Mean values, correlation statistics, standard error, and confidence limits were calculated for the data.

RESULTS

The groups used were of a high calibre, as represented in the mean times of both males and females. Male and female responses were plotted separately as well as being analysed as a combined group. Single gender and total group means, standard deviation, and confidence limits are shown in table 1. The means of each group of swims (50s, 100s, etc) were then plotted and a 2nd order polynomial line of best fit created for each gender and the overall group. To further analyse the relationship between the distances, we calculated Rsq values for each distance and also carried out 2-way t-test for each comparative distance. The results are shown in table 2.

swimming racing, the blood glucose response produced a distinct profile against race time. At shorter times (below 30 seconds) the blood glucose either fell below the resting value or stayed close to it. As performance time extended, there was a clearly identifiable rise in the post swim blood glucose level noted for the competition effort. The highest values were seen in the 800 and 1500m swims, with values between 8.5 and 11.2 mmol/L. There appears to be a critical balance point around 45 to 50 seconds where the blood glucose value will remain at or around the resting value although this does not mean that there is not considerable glucose flux at this point. The profile of the blood glucose against time also had a higher average correlation than lactate. The profile for males and females appears to be different, however there is no statistical difference between them. Although there was no significant difference between the values gained for the 100 and 200 events there was also no relationship between the results of each distance. This non-significant difference is more likely caused by the larger standard deviation between these two events. For the 800 and the 1500m events, there was no significant difference between the glucose values gathered, even though the time of each event are significantly different.

Table 1. Descriptive Statistics for each distance swum

	50 free		100		200		400		800		1500	
	time (sec-1)	glucose (mmol/l)	time (sec-1)	glucose (mmol/l)	time (sec-1)	glucose (mmol/l)	time (sec-1)	glucose (mmol/l)	time (sec-1)	glucose (mmol/l)	time (sec-1)	glucose (mmol/l)
Mean Male	23.92	5.19	52.11	5.61	113.17	5.67	236.00	6.96	505.50	9.68	950.75	9.28
St Dev Male	0.64	0.82	0.98	0.67	1.68	0.78	11.82	0.58	15.42	1.68	8.41	0.65
Mean Female	27.59	5.03	58.32	5.84	121.93	6.35	260.00	7.62	532.67	9.47	1027.50	10.30
St Dev Female	0.61	0.35	1.26	0.48	1.49	1.26	3.65	2.74	7.04	1.48	35.93	0.92
Mean Overall	25.14	5.14	53.59	5.70	116.56	5.93	252.06	7.35	517.50	9.48	964.86	9.49
St Dev Overall	1.84	0.70	2.77	0.65	4.56	1.05	11.40	1.75	17.03	1.62	45.80	0.94
Conf Overall	0.79	0.30	1.11	0.26	1.61	0.37	6.74	1.04	12.61	1.20	36.64	0.75
SEE Overall	0.72		0.66		1.03		1.87		1.65		0.77	

Table 2. Correlation and t-test matrix

			R squared value			
	50	100	200	400	800	1500
50	1	0.058	0.000	0.084	0.013	0.044
100	0.05	1	0.128	0.020	0.032	0.226
200	0.00	0.25	1	0.077	0.001	0.396
400	0.00	0.00	0.04	1	0.077	0.195
800	0.00	0.00	0.00	0.00	1	0.021
1500	0.00	0.00	0.00	0.00	0.90	1
			2 way test			

A comparison of the glucose response between the different distances showed there was a significant difference between each distance with the exception of between the 100m and 200m and the 800m and 1500m. Glucose values were still marginally higher in the 200 than the 100, but not significantly. In the longer distances (800 & 1500), there was a plateau seen after the 8 to 9 minute time line (800m) with no significant difference between the 800 and 1500 glucose responses. Differences between males and female averages are shown in table 1 and figure 1abcd. There was no significant difference between the two groups. The female polynomial fit is marginally better than the males.

DISCUSSION

Our results showing a relationship between exercise intensity and blood glucose. During maximal efforts in competition

At lower intensities, it has been observed that blood glucose will drop with the extension of exercise time (Carter and Rennie, 2001), and that this represents the difference between the use of muscle glycogen and the ability of hepatic glycogen and gluconeogenesis to meet the difference in energy requirement (Coggan *et al.,* 1995). In this present study however, the efforts were all maximal, even though the relative intensity difference between a 50m and a 1500m swim are significantly different (170% MVO$_2$ vs 98% MVO$_2$) (Faina *et al.,* 1997; Maglischo, 2003). Where, in shorter events there may be a drop in blood glucose, representing the inability of the system to keep up with the massive demands made under such conditions (Bogdanis *et al.,* 1996), in the longer events the higher blood glucose represents a vastly different scenario. Blood glucose measurements are changeable in relative terms to improved performance (Friedlander *et al.,* 1997). Swimmers tested here may have been at different relative training states,

and this would to some degree account for the differences in intra individual results seen over the same time line. Moreover, glucogon is responsible for 60% of the hepatic glucose output during exercise and is even more pronounced when there is a corresponding fall in insulin (Zinker *et al.,* 1993). Knowing this, the likelihood of higher insulin due to prior feeding would dampen the liver glucose output in subjects who had consumed either solid or liquid quantities in the lead up to the race. This would probably have a profound effect on the end glucose profile that they produced. A number of authors (Gollnick *et al.,* 1972; Hargreaves, 1997) have demonstrated that prior feeding can affect the source of glucose used, the effect of glucagon and insulin, all of which may have a marked effect of the blood glucose profile, yet if the ingestion is below 100g of carbohydrate then exercise performance is not affected. While other authors have suggested that exercise after feeding does not produce exercise induced hypoglycaemia (Jeukendrup and Gleeson, 2010), none have looked at the effects on very high intensity exercise such as that used in this research. It is possible therefore that prior feeding also played a part in some of the variation seen at each of the different exercise time lines produced by the events. Hepatic glucose output is also based on a feed forward system (Kjaer, 1995) during intense exercise. This difference created by the higher intensity, as compared to the feedback loop seen in lower intensity exercise, may also add to the differences seen between the responses to these competitive events compared to many of those seen in previous literature that has used substantially lower intensities on which to draw their conclusions.

Glucose production is apparently relatively insensitive to minor decreases in plasma glucose (Kjaer, 1995) however, the rise in motor activity because of the rise in exercise intensity does set the level of glucose production during physical activity (Kjaer, 1995; Parker *et al.,* 1997), the relative efficiency of different swimmers (or swim speeds at different distances) would therefore also contribute to possible changes seen in a glucose profile of different individuals, particularly where the skill of one individual is greater than others. The level of catecholamine is also known to influence hepatic glucose output (Garceau *et al.,* 1984), and the levels of catecholamine is greatly increased with maximal efforts over that seen at lower intensities. Muscle glycogen use creates a high glucose-6-phosphate level in the cell which inhibits the action of hexokinase. By so doing, the uptake of plasma glucose into the cell is greatly diminished (Gollnick, 1972). In such a situation, where the hepatic glucose output continues to rise due to high motor unit activity, the plasma glucose level would be expected to also rise. Glucose uptake is only increased when the number of glycogen empty fibres increase (Gollnick, 1972). Recruitment pattern and recruitment number of motor units will also be affected by the absolute intensity of an event (50m compared to 1500m), which would also influence plasma glucose uptake. In the shorter events, where the recruitment of motor units would be at its highest (Gibson, 2004), the uptake of plasma glucose as well as the use of muscle glycogen would both be at a very high level. Additionally, the level of catecholamines would also be high (Kjaer *et al.,* 1987; Tesch *et al.,* 1989), whereas in the longer races (800m to 1500m - 8 mins to 18 mins) the recruitment of motor units is likely to be less at any one time, and greater cycling of motor unit activity is likely to take place. This would also result in lower catecholamine levels (Kiens *et al.,* 1993). Combined with high glucogon activity and a lower insulin activity with prolonged exercise (Berger *et al.,* 1976),

may maintain a higher hepatic glucose output. The working fibres are less likely to take it up, leading to a diminished uptake of plasma glucose, primarily by resting fibres or motor units. The result of this being a higher plasma glucose being observed. Swimmers with greater skill can also move with greater efficiency. This means that the relative cost of the performance will be lower than a less skilful performer. This is likely to also affect the source of glucose used and its relative effect on the blood glucose profile in much the same way that it has been showed the changes in glucose flux with training (Friedlander *et al.,* 1997). Although in this data set, there was a range of blood glucose at each distance (time of the event swum), the normative value within each distance group showed a consistent rise as the time line got longer. The degree of rise got less as the distance swum reached the maximum for pool events. It is speculated that from the data in this pool we could predict the point at which blood glucose would begin to reduce again and the distance that would be associated with this. Having the ability to train toward this level of glucose control and, being able to predict the level required for a particular event, would greatly enhance the ability to ensure that training was indeed being effective in the preparation of athletes for events. Additionally, where athletes are diabetic, being able to predict the level at which blood glucose needs to be at, to ensure good performance outcomes, would help greatly in allowing this population to compete more effectively and safely.

Conclusion

Across different time lines, at maximal effort, blood glucose appears to respond in a predictable way. This may prove useful for the identification of parameters for performance and for training also. Although it is likely that the profile will be affected by a number of conditions such as (1) Prior feeding; (2) State of training; (3) Relative vs absolute intensity; (4) Fibre Recruitment pattern, it is expected that by setting up more stringent testing conditions the variation seen due to prior feeding could be greatly reduced.

Practical Applications

Measurement of blood glucose in sporting situations is simple and non-time consuming. When testing is carried out using standardised protocols around food consumption, the results can give a good insight of what is happening in terms of energy metabolism during strenuous exercise. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation, and statement that results of the present study do not constitute endorsement by the publishing body.

REFERENCES

Bangsbo, J., Graham, T.E., Kiens, B., saltin, B. 1992. "Elevated muscle glycogen and anaerobic energy production during exhaustive exercise in man." *J. Physiol.,* (451): 205-227.

Berger, M., Hagg, S.A., Goodman, M.N., Ruderman, N.B. 1976. Glucose metabolism in perfused skeletal muscle. Effects of starvation, diabetes, fatty acids, acetoacetate, insulin and exercise on glucose uptake and disposition, *Biochemical Journal,* 158(2)191-202;

Bogdanis, G. C., Nevill, M. E. Boobis, L. H. Lakomy, H.K. 1996. Contribution of phosphocreatine and aerobic

metabolism to energy supply during repeated sprint exercise. *Journal of Applied Physiology,* Vol. 80, no. 3, 876-884

Brooks, G.A. 1997. Importance of the crossover concept in exercise metabolism. *Clin Exp Pharmacol Physiol.,* (11): 889-895.

Carter, S. L. C. Rennie, M. A. 2001. TarnopolskySubstrate utilization during endurance exercise in men and women after endurance training. *American Journal of Physiology - Endocrinology and Metabolism Published,* June Vol. 280 no. 6, E898-E907.

Coggan, A.R., Kohrt, W.M., Spina, R.J., Kirwan, J.P., Bier, D.M., Holloszy, J.O. 1992. Plasma glucose kinetics during exercise in subjects with high and low lactate thresholds, *J Appl Physiol.,* 73(5):1873-1880.

Coggan, A.R., Raguso, C.A., Williams, B.D., Sidossis, L.S., Gastaldelli, A., 1995. "Glucose kinetics during high intensity exercise in endurance trained and untrained humans." *J. Appl Physiol.,* 78 (3):1203-1207.

Dudley, G.A., W.M. Abraham, and R.I. Terjung, 1982. Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle. *Journal of Applied Physiology, Respiratory, Environmental, and Exercise Physiology,* 53(4): 844-850.

Faina, M. Billat, V. Squadrone, R. De Angelis, M. Koralsztein J.P. Dal Monte, 1997. Anaerobic contribution to the time to exhaustion at the minimal exercise intensity at which maximal oxygen uptake occurs in elite cyclists, kayakists and swimmers, *European Journal of Applied Physiology and Occupational Physiology,* Volume 76, Issue 1, pp 13–20, 354.

Friedlander, A.L., Gretchen, A., Casazza, M.A., Hording, M.J., Brooks, G.A. 1997. Training induced alterations of glucose flux in men. *J Appl Physiol.,* 82(4):1360-1369.

Garceau, D. Yamaguchi, N. Goyer, R. Guitard, F. 1984. Correlation between endogenous noradrenaline and glucose released from the liver upon hepatic sympathetic nerve stimulation in anesthetized dogs *Canadian Journal of Physiology and Pharmacology,* 62(9): 1086-1091, 10.1139/y84-181.

Gibson, A St Clair and T D Noakes Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in human Br J Sports Med 2004; 38:797–806. doi: 10.1136/ bjsm.2003. 009852

Gollnick, PD, RB Armstrong, CW SaubertT IV, K. Piehl, and B. Saltin. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. *J. Appl. Physiol.,* 1972; 33 (3): 3 12-3 19.

Hargreaves, M., Interactions between muscle glycogen and blood glucose during exercise. *Exerc Sport Sci Rev.,* 1997; 25: 21-39.

Hargreaves, M., Proietto, J. 1994. Glucose kinetics during exercise in trained men. *Acta Physiol Scand,* 154(2):221-5.

Jeukendrup, A., Gleeson, M. 2010. Sport nutrition: an introduction to energy production and performance, No. Ed.2

Kiens, B., Essen-Gustavsson, B., Christensen, N J., and Saltin, B. 1993. Skeletal muscle substrate utilization during submaximal exercise in man: effect of endurance training. *J Physiol.,* 469: 459–478.

Kjaer M, Secher NH, Galbo H. 1987. Physical stress and catecholamine release. Bailliere's clinical endocrinology and metabolism, 1; 1(2):279-98.

Kjaer, M. 1995. Hepatic fuel metabolism during exercise. Exercise metabolism. M. Hargreaves. Champaign IL., Human Kinetics, 73-97.

Maglischo E.W. 2003. Swimming Fastest. Human Kinetics, PP342. ISBN-13: 9780736031806

Maughan R. 2009. Carbohydrate metabolism. Surgery-Oxford International Edition, 1;27(1):6-10.

Medbo, J. I., Mohn, A. C., Tabata, I., Bahr, R., Vaage, O., Sejersted, O. M. 1988. Anaerobic capacity determined by maximal accumulated O2 deficit. *Journal of Applied Physiology Published 1 January,* Vol. 64 no. 1, 50-60 DOI:

Parker, D., Robergs, R.A., Quintana, R., Frankel, C.C., Dallam, G. 1997. Heart rate threshold is not a valid estimation of the lactate threshold. *Med. Sci. Sports. Exerc.,* 29(5): supplement abstract 1344.

Pyne, D. B., Lee, H. and Swanwick, K.M. 2001. Monitoring the lactate threshold in world-ranked swimmers. *Med. Sci. Sports Exerc.,* Vol. 33, No. 2, pp. 291–297.

Rushall. B.S. King, H.A. 1994. The value of physiological testing with an elite group of swimmers. *Australian Journal of science and medicine in sport,* 26(1/2): 14-21.

Swanwick, E., Matthews, M. 2017. Glucose Turn Point as a Marker of Exercise Intensity in Elite Swimmers. *Int J Sports Exerc Med.,* 3: 067.

Tesch, P. A., Thorsson, A., Fujitsuka, N. 1989. Creatine phosphate in fiber types of skeletal muscle before and after exhaustive exercise, *Journal of Applied Physiology,* 1 April Vol. 66 no. 4, 1756-1759

Vissing, J. Iwamoto, G. A. Fuchs, I. E. Galbo, H. Mitchell, J. H. 1994. Reflex control of glucoregulatory exercise responses by group III and IV muscle afferents. American Journal of Physiology - Regulatory, *Integrative and Comparative Physiology Published,* Vol. 266 no. 3, R824-R830

Zacca R, Wenzel BM, Piccin JS, Marcilio NR, Lopes AL, de Souza Castro FA. 2010. Critical velocity, anaerobic distance capacity, maximal instantaneous velocity and aerobic inertia in sprint and endurance young swimmers. *European journal of applied physiology,* 1; 110(1):121-31.

Zinker BA, Lacy DB, Bracy DP, Wasserman DH. 1993. Role of glucose and insulin loads to the exercising limb in increasing glucose uptake and metabolism. *Journal of Applied Physiology,* 1; 74(6):2915-21.

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