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The Effect of Intermittent Hypoxic Exposure plus Sea Level Swimming Training on Anaerobic Swimming Performance

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Abstract

An approach which has been proposed as a time efficient variant of the "live-high train-low" altitude training strategy is that of intermittent hypoxic exposure. The aim of the study was to determine whether 3 weeks of intermittent hypoxic exposure would enhance sea level anaerobic swimming performance. Eight participants (age = 20 ± 2 years; height =1.77 ± 4.80m; mass = 72.0 ± 3.0 kg) took part in the study and were split into two groups: experimental (EXP) and control (CON). For 3 days a week over a 3 week consecutive period, both groups rested for a total of 90 minutes per day in the hypoxic chamber, whilst undergoing their usual training programme. The experimental group rested in a hypoxic chamber at a simulated altitude of 2300m, whilst the control group rested in a hypoxic chamber at sea level conditions. All participants underwent a total of 3 performance tests (100m sprint) 1 week prior to the hypoxic exposure, 2 days post exposure (Post 1) and 9 days post exposure (Post 2). A blood lactate sample was taken at rest, immediately after, 3 and 7 minutes after each time trial. Using a two-way repeated measures ANOVA, the results revealed that there was no significant difference in time for 100m performance ($p = 0.431$), stroke count ($p = 0.824$) or stroke rate ($p= 0.278$), but there was a significant increase over time for blood lactate ($p < 0.01$). This dose of intermittent hypoxic exposure was not sufficient to elicit significant improvements in 100m sprint time in these eight competitive swimmers.

Introduction

The concept of training at altitude in order to improve sea level performance is now well established and is commonly incorporated into many athletes' training programmes (Morton and Cable, 2005). The rationale for altitude training is that the reduction in inspired oxygen is causally related to haematological, hormonal and metabolic adjustments facilitating improvements in sea-level exercise performance (Rodriguez *et al.* 2007).

An increasingly popular approach to altitude training is that of intermittent hypoxic exposure, which has been proposed as a time efficient variant of the "live – high train-low" altitude training strategy. The ultimate goal of this method is to induce sufficient altitude acclimatization and allows beneficial adaptations due to the restricted availability of oxygen to improve both altitude and sea level performance, while maintaining training quality (Hamlin and Hellemans, 2007). A lack of proper training adaptation and / or decreased exercise intensity due to hypoxia can lead to a relative detraining effect in some athletes. Intermittent hypoxic exposure (IHE) allows the athlete to effectively live high, train low, where brief periods of hypoxic exposure are interspersed with prolonged sea level stays and the absolute training stimulus can be maintained (Morton and Cable, 2005).

Previous work by Hamlin and Hellemans (2007) found significant improvements in sea level 1.5km swimming performance following intermittent hypoxic exposure for a total of 90 minutes per day, 5 days per week, over a 3 week period. This is consistent with Hinckson and Hopkins (2005) who found substantial improvements in swimming performance lasting 1-10 minutes following exposure to intermittent hypoxia. While some investigators (Rodriguez *et al.* 1999) have found similar performance enhancements after intermittent hypoxic exposure at rest, others have reported no significant changes in performance parameters (Julian *et al.* 2004). This study looked at highly trained distance runners completing a 4 week regimen, 5:5 minute hypoxia to normoxia ratio for 70 minutes, 5 times per week of intermittent normobaric hypoxia. There were no significant differences in 3000m time trial performance or erythropoietin. It remains possible that intermittent hypoxic methods involving more prolonged episodes of exposure elicit persistent changes in a variety of physiological responses, but that the 5 minute hypoxic bursts, with 5 minutes of ambient breathing between each burst for a total of 70 minutes are simply insufficient or ineffective to initiate and sustain the acclimatization process.

Although previous findings have attributed enhancements following simulated altitude exposure to increases in anaerobic energy system capacity (Gore *et al.* 2001), the effects of altitude training on anaerobic performance have not been reported as much as the effects on aerobic performance (Neubauer, 2001). Evidence reported an increase in muscle buffering capacity following “live-high train-low” altitude exposure. The results revealed that altitude exposure induced a 5-7% increase in skeletal muscle in-vitro buffer capacity without any corresponding elevation in other markers on anaerobic metabolism in a group of 13 male athletes performing an incremental cycle ergometer test. This work can further be supported by Mizuno *et al.* (1990): Saltin *et al.* (1995) with increased muscle buffer capacity in cross country skiers reported after 2 weeks at ‘natural’ altitude.

Furthermore, the results from Roberts *et al.* (2003) indicated that well-trained athletes can use short periods of “live-high train-low” to prepare for the intense demands of competition and they provide additional evidence that improvements in performance after “live-high train-low” exposure may be associated with increased muscle buffer capacity and anaerobic capacity. Given the potential importance of anaerobic metabolism and efficiency to performance even in highly trained endurance athletes, further investigation of possible anaerobic adaptations is clearly warranted (Gore *et al.* 2001).

It has been suggested that altitude exposure and training at sea level may confer an advantage to whole body lactate metabolism and thus performance compared with training at sea level alone (Clark *et al.* 2004). Casas *et al.* (2000) showed an improvement in the lactate threshold and an increase in the ventilatory threshold following intermittent hypoxic exposure. Additionally, Clark *et al.* (2004) revealed that whole body lactate kinetics were altered by hypoxic exposure. Participants who were exposed to the stimulus of "continuous" hypoxic exposure revealed a lower lactate appearance compared with the group who underwent an "intermittent" mode of hypoxic exposure.

The purpose of the present study was to evaluate to what extent intermittent hypoxic exposure in a hypoxic chamber for 1 ½ hours per day, 3 days per week over 3 weeks combined with sea level training can induce physiological changes that enhance anaerobic swimming performance. This investigation will test the hypothesis that short, repeated episodes of hypoxia at rest will improve sea-level anaerobic swimming performance in competitive swimmers.

Methods

Participants

Eight male competitive swimmers from the City of Liverpool swimming club participated in this study. Selection criteria for swimmers were to have recent 100m best times under 1.00 minute. Informed written consent was obtained from each participant before the start of the study, which informed them of the purpose of the study, the extent of their involvement and their right to terminate participation at any time. The study was approved by the Ethics Committee at Liverpool John Moore's University.

All participants were maintaining their sea level training regime throughout the duration of this study and were in similar phases of training and all training together twice daily as a squad. All athletes were healthy, free from injury, lived at sea level and had not been a resident at altitude within the past 6 months.

Study Design

Participants were allocated to one of two groups, hypoxia (H) and normoxia (N) and were ranked based on their 100m time trial performance. The baseline trial was performed one week before the start of the experimental and placebo exposures and the post-exposure time trials were completed 2 and 9 days after the experimental and placebo exposures had finished. The timing of the post tests were determined as this way both the immediate effects of the intermittent hypoxic exposure (IHE), as well as the "off-response" of these effects could be evaluated (Rodriguez *et al*. 2007).

All participants were blind to the study design and all exposure was carried out at the same time of day. The participants were unable to view the percentage of oxygen levels in the chamber and were unable to determine which group they were in when asked at the end of the study.

Hypobaric chamber exposure

A hypobaric chamber located at the Research Institute for Sport and Exercise Science at Liverpool John Moore's University was utilized for this experiment. All participants rested in the chamber for 1 ½ hours per day, 3 days per week over 3 weeks. Under hypoxic conditions, participants rested in a normobaric hypoxic chamber at a simulated altitude of 2300m for 1 ½ hours per day, 3 days per week over 3 weeks. Under normoxic conditions, participants rested in a normobaric hypoxic chamber at a simulated altitude of 0-500m (sea level) for 1 ½ hours per day, 3 days per week over 3 weeks. The rationale for this length of hypoxic exposure was to form a comparison with previous literature, specifically, Rodriguez *et al.* (2007) who performed a placebo-controlled trial to investigate the effects of intermittent hypoxic exposure for 3 hours per day, 5 days per week over 4 weeks.

Evaluation of Performance

Time trials: 100m freestyle: The swimmers performed three 100m performance time trials. They were familiarized with their personal best score pre and post the intermittent hypoxic exposure. All 100m time trials were conducted in a 25-m swimming pool between 5-7pm and were conducted similar to normal swimming events. The participants were tested in the same order for all trials and were asked to perform a warm up session for a one-off 100m sprint. Before each time trial, participants were asked to achieve the best time possible. Starts were made from the starting blocks, using a whistle as the starting signal and finish times were measured in duplicate by a stopwatch. Time was recorded to the nearest 0.1s and stroke count and stroke rate (strokes/min) were recorded every 25m.

Blood Measurements

An ear lobe blood sample was taken at rest, immediately after, 3 and 7 minutes after each time trial while the athletes were seated and analysed using an Arkray Inc lactate prop portable lactate analyser (Kyoto, Japan). The earlobe was wiped with a Mediswab, the first drop of blood discarded and the required amount was then collected.

Evaluation of Training

All participants were asked to maintain their usual training regime during the study. They were also asked to keep a detailed training logbook that included the duration, distance and intensity of each workout in the pool, which the researcher had access to after the intervention was complete. Training intensity was estimated by the participants by giving each training session the qualification of being low, moderate or high intensity.

Statistical Analysis

All statistical analyses were conducted using SPSS for windows (version 15) computer software. A two-way repeated measures ANOVA and training environment was used to examine the changes in physiological and performance variables. In addition, a three way ANOVA was used to assess the effect of IHE on blood lactate and 25m split times. When a significant effect was obtained, post hoc analysis was carried out. All data was expressed as means (± SD) with p values of less than 0.05 assumed to indicate statistical significance.

Results

Training

All participants were able to combine their training during the experimental procedures throughout the duration of the study. During the 1st week of chamber exposure, the swimmers' average training volume was 20 hours per week / 35km per week. It was clear from the training log that one of the swimmers started a progressive taper period during the second week of the intervention phase that continued during the third week of hypoxic exposure. This training period aimed at performance improvement in preparation for a major competition was characterized by a marked decrease in training distance from the average training distance of 35k per week to 10km. The first week of the tapering process consisted of moderate intensity swimming sessions, while the second week during this phase involved low intensity one hour training sessions aimed at increasing speed performance.

Evaluation of Performance

There was no significant difference across time for 100m time trial performance, stroke count or stroke rate (F = 0.768, $p = 0.431$, $p < 0.05$, F = 0.187, $p = 0.824$, $p < 0.05$, F = 1.437, $p = 0.276$, $p < 0.05$, respectively) , nor was there an interaction effect between time and group for all three performance parameters (F = 0.179, $p = 0.724$, $p < 0.05$, F = 0.813, $p = 0.463$, $p < 0.05$, F = 1.279, $p = 0.511$, $p < 0.05$, respectively). Table 1 shows the mean and standard deviation values for the main maximal performance measures for both groups.

Table 1. Performance measures pre intermittent hypoxic exposure and 2 and 9 days post intermittent hypoxic exposure for the experimental (hypoxia) and placebo (normoxia) groups.

	Hypoxia (n = 4)			Normoxia (n =4)		
	Pre IHE	Post 1	Post 2	Pre IHE	Post 1	Post 2
100m	**5**6.95 ± 1.97	57.47 ± 1.06	57.07 ± 1.15	57.56 ± 1.84	57.75 ± 1.33	57.70 ±1.46
S.Count	37.00 ± 4.24	37.25 ± 3.20	37.75 ± 3.77	38.25 ± 0.95	38.25 ± 0.50	8.00 ± 0.81
S.Rate	56.70 ± 6.11	56.05 ± 5.77	56.00 ± 5.77	54.75 ± 3.59	55.00 ±3.19	54.50±3.31
***Blood La**	8.43 ± 0.38	9.08 ± 2.89	7.91 ± 0.99	8.14 ± 0.97	8.20 ± 0.70	8.12 ± 0.81

Values are means ± SD; $n = 4$ for Hypoxia and $n = 4$ for Normoxia. *Significant differences of values between experimental and placebo conditions.

Blood lactate response

There was no significant difference for blood lactate across days or between groups, nor was there an interaction effect between group and day or group and time (F = 0.598, $p = 0.485$, $p < 0.05$, F = 1.513, $p = 0.259$, $p < 0.05$, respectively). Repeated measures showed a significant difference for time. Post hoc analysis identified a significant difference between all times points with the exception of time point 3 and 4 where the results was non-significant ($p = 0.568$, $p < 0.05$). Figure 1 shows an increase in blood lactate over time for both the experimental (hypoxic) and placebo (normoxic) groups pre IHE.

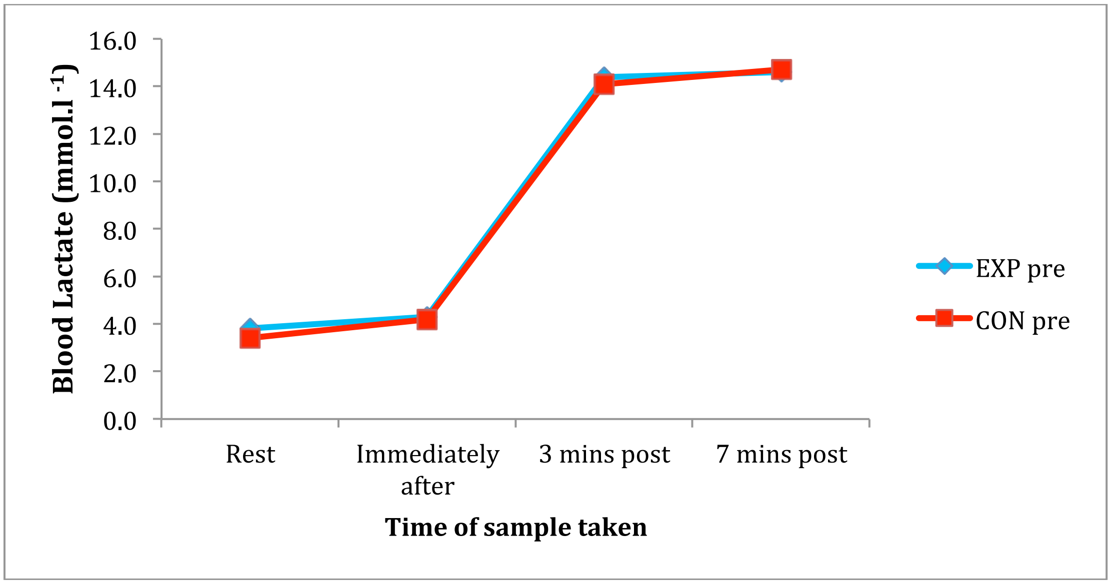

Figure 1: Lactate concentration pre intermittent hypoxic exposure for experimental (hypoxia) and placebo (normoxia) groups.

Figure 2 shows blood lactate values 2 and 9 days post intermittent hypoxic exposure for normoxic and hypoxic groups. Blood lactate levels in the hypoxic group were higher immediately after the 100m swim compared with the normoxia group (9.62 ± 6.92 (SD) versus 4.55 ± 0.59 (SD). Lactate levels were higher in the normoxic group compared with the hypoxic group 3 minutes post exercise (12.95 ± 1.85 (SD) versus 11.42 ± 3.12 (SD)). Blood lactate increased progressively over time for both the hypoxic and normoxic groups 9 days post intermittent hypoxic exposure compared with the hypoxic group (12.87 ± 2.18 (SD) versus 11.17 ± 2.93 (SD)).

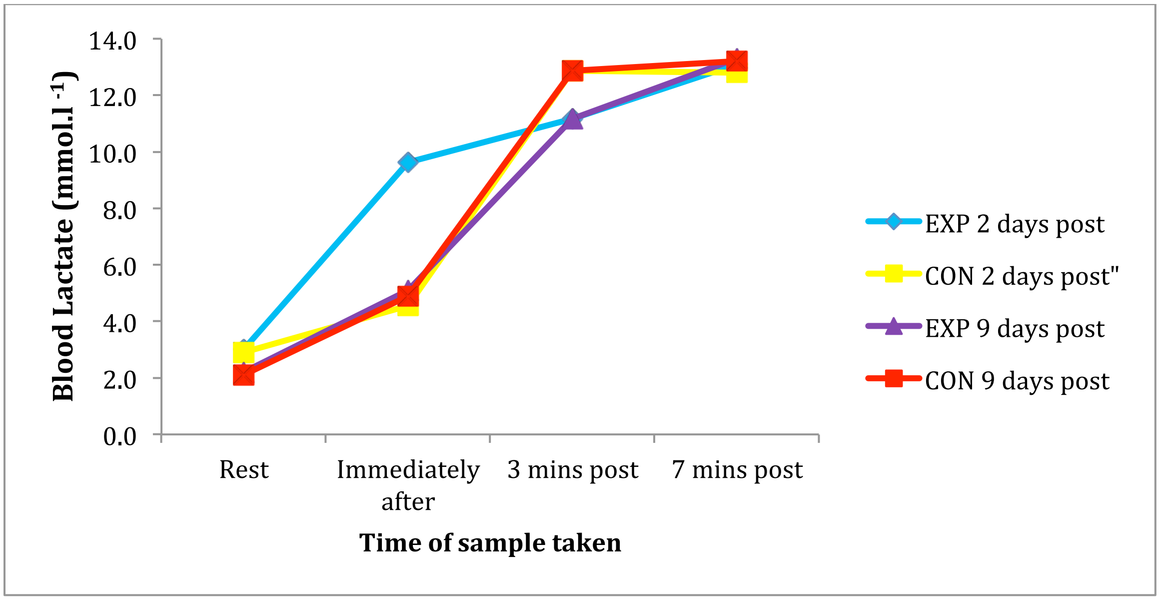

Figure 2: Lactate concentration 2 and 9 days post intermittent hypoxic exposure for experimental (hypoxia) and placebo (normoxia) groups.

Discussion

The aim of the study was to determine whether intermittent hypoxic exposure for 1 ½ hours per day, for 3 days over 3 weeks was sufficient to enhance anaerobic swimming performance. The major finding of this study was that this protocol of IHE did not improve 100m swimming performance in this group of trained athletes nor did it reduce the rate of lactate production. This finding suggests that this dose of IHE was insufficient to have a synergistic effect on performance over sea level training in this group of competitive swimmers.

Previous work with intermittent hypoxic exposure

Previous research in this area has dealt primarily with aerobic responses to altitude training. Hamlin and Hellemans (2007) found that 15 days of IHE at rest substantially improved sea level performance in a group of endurance based athletes. This was the first study to have used a large sample size in a randomized single blind controlled fashion using an intermittent normobaric hypoxic procedure, which contributed to positive and long lasting performance responses. There are only a few studies that have examined the effect of intermittent hypoxic exposure on anaerobic performance (Hendrikson and Meeuwsen, 2002). This study revealed a significant increase in anaerobic mean power among 16 male triathletes, using a cycle ergometer for performance measurement. Furthermore, the study design used by Hendrikson and Meeuwsen (2002) used a stronger hypoxic stimulus, exposing subjects to 105 minutes over a ten consecutive day period whilst training at altitude, compared with the 90 minutes of intermittent and discontinuous hypoxia over 9 days used in the present study.

Bartsch *et al.* (2008) revealed that five of six randomized single blind studies could not show any advantage of IHE for intermittent and endurance based sports over placebo treatment for performance at sea level. Moreover, Tadibidi *et al.* (2007) looked at both aerobic and anaerobic performance in endurance trained men over 15 consecutive day's exposure and found no advantage of IHE over placebo treatment on an incremental cycle ergometer test and the wingate anaerobic test. Consistent with these findings, the results from the present study did not demonstrate improved anaerobic swimming performance following IHE.

Physiological mechanisms of performance improvement with altitude exposure

The mechanisms underlying an improvement in performance at sea level after IHE have been the topic of much debate (Hamlin and Hellemans, 2007). The increased use of anaerobic metabolism at altitude as a substitute for reduced aerobic function is evidenced by the body's alteration in fuel use (Rushall *et al.* 1997). Altitude exposure decreases the reliance on free fatty acids as a fuel, increases the utilization of blood glucose at both rest and in exercise. These changes in fuel use indicate marked alterations in the metabolism underlying both exercise and recovery. Although there are several possible mechanisms for such an effect, some authors have focused on increases in muscle buffering, which would reduce fatigue associated with decreases in pH. Indeed, Mizuno *et al.* (1990) reported an increase in buffering capacity with altitude acclimatization among cross country skiers at a simulated altitude of 2100m continuously over 14 days and related the increase of short term running performance to an improvement in anaerobic capacity. As suggested by the present study, unchanged anaerobic performance suggests that buffer capacity was not influenced by this amount of intermittent hypoxic exposure (Tadibidi, *et al.* 2007).

Blood Parameters

Clark *et al.* (2004) observed no significant difference in blood lactate post-exercise in well-trained cyclists following 20 nights of IHE at a simulated altitude of 2650m, however observed a decrease in peak lactate concentration in participants who were exposed to the stimulus of continuous hypoxic exposure. These results are in agreement with the present study in which the participants were exposed to an altitude of 2300m and revealed no significant difference in blood lactate post IHE. This is in contrast to the findings by Casas *et al.* (2000) who found a significant difference in blood lactate following 17 days of hypoxic exposure at a simulated altitude of 4000m to 5000m for 3 to 5 hours per day with moderate intensity cycle ergometer exercise. It is important to note that the hypoxic stimulus was much greater in the above study compared with that of Clark *et al.* (2004) and of the present study which may explain the non-significant findings for blood lactate post-exercise.

Responders and non-responders to altitude

There has been the suggestion that elite athletes are more sensitive to minor changes to altitude than non-athletes and therefore, will exhibit fuller acclimatization at a lower altitude. Since most swimmers are taken to altitude camps at a relatively low height, the variations and compromises in the acclimatization process may usually be noticeable between individuals. It is reasonable to expect that at moderate to low altitudes some athletes may not react at all whilst for others all of the mechanisms of acclimatization may be exhibitied (Rushall *et al.* 1997).

Limitations of the study

Previous research in this area has been disadvantaged by small sample sizes, which was a limitation in the present study. A cross-over design would have been appropriate but due to the small sample size, this was not possible. It would be unrealistic to exclude the possibility that the amount of training done during the time of the intervention outside of the swimmers training programme could have influenced the outcome of the present study. Furthermore, in the present study, the hypoxic stimulus during each session was 90 minutes. Studies by Terrados *et al.* (1988) and Vogt *et al.* (2001) demonstrated that a hypoxic stimulus of 30 minutes, 3-5 times per week suggest this altitude level was enough to establish significant effects in sea level performance, at least in the muscle level.

Conclusion

To conclude, this dose of intermittent hypoxic exposure was not sufficient to elite significant improvements in 100m performance in competitive swimmers. These observations based on a small sample analysis need to be investigated in a larger group with individuals being exposed to longer exposures of hypoxia before a final judgment on this issue is possible. In addition to intermittent hypoxic exposure, future studies should include training in a hypoxic environment for an improvement for athletes involved in anaerobic events.

References

1. Bartsch, P., C. Dehnert, B. Friedmann-Better and V. Tadibi. Intermittent hypoxia at rest for improvement of athletic performance. *Scand. J. Med. Sci. Sports.* 18 (Supplement 1): 50-56, 2008.
2. Bartsch, P. and B. Saltin. General Introduction to altitude adaptation and mountain sickness. Scand. J. Med. Sci. Sports. 18 (Supplement 1): 1-10, 2008.
3. Casas, M., H. Casas, T. Pages, R. Rama, A. Ricart, L.J. Ventura, J. Ibanez, F.A. Rodgrigues and G. Viscor. Intermittent hypobaric hypoxia includes altitude acclimatization and improves the lactate threshold. Aviat. Space Environ. Med. Feb 71 (2): 125-30, 2000.
4. Clark, A.A., R.J. Aughey, C.J. Gore, A.G. Hahn, N.E. Townsend, T.A. Kinsmann, C.M. Chow, M.J. McKenna and J.A. Hawley. Effects of live high train low hypoxic exposure on lactate metabolism in trained humans. J. Appl. Physiol. 96: 517-525, 2004.
5. Gore, C.J., A.G. Hahn, R.J. Aughey, D.T. Martin, M.J. Ashenden, S.A. Clark, A.P. Garnham, A.D. Roberts, G.J. Slater and M.J. McKenna. Live high: train low increases muscle buffering capacity and sub maximal cycling efficiency. Acta. Physiol. Scand. 173: 275-286, 2001.
6. Hamlin, M.J. and J. Hellemans. Effect of intermittent normobaric hypoxic exposure at rest on haematological, physiological, and performance parameters in multi-sport athletes. J. Sci. Med. Sport. 25 (4): 431-441, 2007.

7. Hendrikson, I.J.M. and T. Meeuwsen. The effect of intermittent hypoxic training in hypobaric hypoxia on sea level exercise: a cross over study. *Eur. J. Appl. Physiol.* 88: 396-403, 2003.
8. Hinckson, E.A. and W.G. Hopkins. Changes in running endurance performance following intermittent altitude exposure simulated with tents. *Eur. J. Sports Sci.* 5 (1): 15-24.
9. Julian, C.G., C.J. Gore, R.L. Wilber, J.T. Daniels, M. Fredericson and J. Stray-Gunderson. Intermittent normobaric hypoxia does not alter performance or erythropoietic markers in highly trained distance runners. *J. Appl. Physiol.* 96: 1800-1807, 2004.
10. Levine, B.D. and J. Stray-Gunderson. Living high-training low: effect of moderate altitude acclimatization with low altitude training on performance. *J. Appl. Physiol.* 83: 102-112, 1997.
11. Mizuno, M., C. Juel, T. Bro-Rasmussen, E. Mygind, B. Schibye, B. Rasmussen and B. Saltin. Limb skeletal muscle adaptation in athletes after training at altitude. *J. Appl. Physiol.* 68: 406-502, 1990.
12. Morton, J.P. and N.T. Cable. The effects of intermittent hypoxic training on aerobic and anaerobic performance. *Ergonomics.* 48: 1535-1546, 2005.
13. Neubauer, J.A. Physiological and Genomic consequences of intermittent hypoxia invited review: physiological and patho-physiological responses to intermittent hypoxia. *J. Appl. Physiol.* 90: 1593-1599, 2001.
14. Rodriguez, F.A., H. Cassas, M. Cassas, T. Pages, R. Rama, A. Ricart, J.L. Ventura, J.Ibanez and G. Viscor. Intermittent hypobaric hypoxia stimulates erythropoiesis and improves aerobic capacity. *Med. Sci. in Sports Exerc.* 31: 264-268, 1999.
15. Rodriguez, F.A., M.J. Truijens, N.E. Townsend, J.S. Gunderson, C.J. Gore and B.D. Levine. Performance of runners and swimmers after four weeks of intermittent hypobaric hypoxic exposure plus sea level training. *J. Appl. Physiol.* 103: 1523-1535, 2007.
16. Roberts, A.D., S.A. Clark, N.E. Townsend, M.E. Anderson, C.J. Gore and A.G. Hahn. Changes in performance, maximal oxygen uptake and maximal accumulated oxygen deficit after 5, 10 and 15 days of live high: train low altitude exposure. *Eur. J. Appl. Physiol.* 88: 390-395, 2003.
17. Rushall, B.S., M.J. Buono, A.A. Sucec and A.D. Roberts. Elite swimmers and altitude training. *Swimming Science Bulletin.* 14: 1-13, 1997.
18. Saltin, B., C.K. Kim, N. Terrados, H. Larsem, J. Svedenhag and C.J. Rolf. Morphology, enzyme activities and buffer capacity in leg muscles of Kenyan and Scandinavian runners. *Scand. J. Med. Sci. Sports.* 5: 222-230, 1995.
19. Tadibidi, V., C. Dehnert, E. Menold and P. Bartsch. Unchanged anaerobic performance. *Med. Sci. in Sports Exerc.* 39: 858-864, 2007.
20. Terrados, N., J. Melichna, C. Sylven, E. Jansson and L. Kaijser. Effects of training at simulated altitude on performance and muscle metabolite capacity in competitive road cyclists. *Eur. J. Appl. Physiol.* 57: 203-209, 1988.
21. Vallier, J.M., P.Chateau and C.Y. Guezennec. Effects of physical training in a hypobaric chamber on the physical performance of competive triathletes. *Eur. J. Appl. Physiol.* 73: 471-478, 1996.
22. Vogt, M., A. Puntschahrt, J. Geiser, C. Zuleger, R. Billeter and H. Hoppeler. Molecular adaptations in human skeletal muscle to endurance training under simulated hypoxic conditions. *J. Appl. Physiol.* 91: 173-182, 2001.
23. Wood, M.R., M.N. Dowsen and W.G. Hopkins. Running performance after adaptation to acutely intermittent hypoxia. *Eur. J. of Sport Sci.* 6: 163-172, 2006.