

The Effect of Acute Low to Medium Altitude Exposure on Repeated 30-Second Sprint-Performance in Well-Trained Cyclists.

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Abstract: Background: The purpose of this study was to determine the effect of acute hypobaric hypoxia on anaerobic performance during multiple 30-s Wingate tests. **Methods:** Five well trained ($VO_2\text{max} > 72.53 \pm 4.23$ ml/min/kg) completed six 30-s Wingate tests, using a within-subject design, under four hypobaric conditions at approximately sea-level, 1000, 2000 and 3000 meters (1013 mbar, 908 mbar, 781 mbar and 696 mbar) on four separate days. VO_2 kinetics, mean power output (MP), Fatigue index (FI), Peak Power output (PPO), Kalium+, Natrium and pH were measured after each sprint. For each visit the sprints were averaged. **Results:** Compared to sea-level and 1000m MP was significantly lower for 2000m and 3000m. FI was significantly higher at 3000m when compared to sea-level, 1000 m and 2000m. Natrium was found to be significantly higher at 3000m compared to sea-level and 2000 m. pH was significantly lower at 3000 m than at sea-level and 2000 m. No significance was found for PPO, Kalium+ and VO_2 kinetics. **Conclusion:** These results suggests that acute exposure to hypobaric hypoxia impairs 30-s anaerobic performance when altitude $\geq 2000\text{m}$ possibly by reducing pH, the rate of PCr resynthesis and muscle glycogen.

Introduction

Elite athletes usually compete at sea-level in normoxic conditions. However, it is not uncommon for competitions to take place in hypoxic conditions, such as the 1968 Olympic Games, held in Mexico City (2300m) and more recently the 2000 winter Olympics held in Salt Lake City (2003m). Among the group of athletes most often exposed to altitude, are professional cyclists who compete at variable altitude, varying from sea-level to over 2000m of altitude on a regular basis. The effect of acute hypoxia on aerobic performance, has been well described, and has shown a significant decrease in VO₂Max and performance even at mild hypoxia below 1000m (Gore et al. 1997, Ferretti et al. 1997, Benoit et al. 2003, Martin and O’Kroy 1993). Less is known about anaerobic performance under hypoxic conditions. It has been shown that peak and mean power (MP) are unaffected when a single 30s sprint is performed (Oguri et al. 2008) despite greater muscle deoxygenation, lower peak VO₂, and lower SpO₂ that occur in hypoxic conditions when compared to normoxic conditions. It has been suggested that the maintenance of performance is due to increased energy from catabolism of glycogen to lactate. Further that, hypoxia results in alkalosis at rest and elevated pH in the recovery period following a single 30s bout (McLellan et al. 1990), with a similar pH to that seen when supplementing with sodium bicarbonate (Inbar et al. 1983). However, some competitions require athletes to perform multiple anaerobic bouts, such as, soccer, basketball, track cycling, road cycling, mountain biking, cyclo cross etc. with short recovery periods. Better understanding of anaerobic performance at altitude could help athletes optimize performance, by choosing appropriate: pacing, training methodologies and competition strategy. Therefore it is interesting to investigate the effect altitude has on multiple 30s sprints, and where most studies create a hypoxic environment, this study will be conducted in a hypobaric environment. Four altitudes will be tested (0, 1000, 2000 and 3000) on four different occasions, with six successive 30-sprints on each occasion. It is our hypothesis that MP will be affected negatively if multiple sprints are performed.

Methods

Approach and experimental design:

A randomized double-blind experimental design was utilized to assess the effect of normobaric (~1010mbar) and hyperbaric (~908mbar, 781mbar and 696mbar) conditions on repeated 30-s sprint performance (six sprints). Pressures were chosen to approximate; 0, 1000, 2000 and 3000 meters above sea-level. The recovery interval between sprints (2 min), was selected to prevent creatine phosphate (PCr) stores to fully recover between sprints (Bogdanis et al. 1995). Measurements of peak power output (PPO), MP and fatigue index (FI) were recorded to characterize performance changes, and blood samples were analyzed for lactate, pH, SO₂, natrium and kalium+ to elucidate changes in blood parameters.

Participants

Five well-trained male cyclists (VO₂max > 70ml/min/kg), competing at the highest national level (Four A-class and one B-class road cyclists) were recruited for this study (*Table 1*). The subjects were asked to sign a form informing them of the potential risks. None of the subjects had used creatine for more than a year. They were asked to maintain their normal level of physical activity, but to refrain from physical activity the day before each test and were instructed to maintain their normal diet.

Table 1 Physiological subject data

Variables	Mean (n=5)	±SD
Age (years)	29,00	6,80
weight (kg)	76,00	5,86
height (cm)	182,00	3,77
BMI (kg*m ⁻²)	22,71	1,10
VO ₂ Max ml/min/kg	72,53	4,23
VO ₂ Max ml/min	5450,20	193,88

Table notes: data are means

Protocol

Prior to starting the tests, the participants completed a VO₂max-test, and a familiarization test of the Wingate test, by completing a single 30-s all-out sprint, to avoid a learning effect during the trials (Thompson et al. 2013). The VO₂max test was carried out on an electronically-braked constant-power exercise bike (Monark 739E, made by Monark AB in Varberg, Sweden) while monitoring

VO₂ kinetics (Cosmed Quark CPET, made by Cosmed in Rome, Italy). After a 15-minute warm-up at 150 watts, the resistance was increased by 25 watts every min until reaching volitional failure and peak VO₂.

Each subject visited the hypobaric chamber on four occasions (T₀, T₁, T₂ and T₃). They were placed on a bed in the chamber for 15 minutes or until PsO₂ was stable. After the correct pressure had been reached, calibration of gas, air, and flow were each performed. Upon reaching stable peripheral capillary oxygen saturation (SpO₂) (measured with the hypoxico oxi-go-pro pulse oximeter, made by Hypoxico from New York, America), a capillary sample was taken from the subject's middle or ring-finger in the supine position (r_{rest}).

To warm up, the subject cycled for 15 minutes at a constant power output of 150 watts and 100 revolutions per minute (rpm). During the last 5 minutes of the warm-up, a steady state measurement of vo₂ kinetics was done and upon completion of the warm-up another capillary sample was taken (R₀). After a min of rest, the six 30-second sprints were carried out. A break-weight of 9.5% of body weight was chosen to optimize PPO and MP (Patton et al. 1985, Dotan & Bar-Or 1983). They were instructed to ride at 90 rpm and to increase their cadence to 100 rpm during the last 5 seconds of the two-minute recovery period between each sprint. Upon reaching 100 rpm the weight automatically dropped and the subject pedaled maximally for the entire 30s. The subject was told to refrain from standing up and to use his arms minimally and to keep the mask on for the entire duration.

A capillary sample was taken immediately after each sprint (R₁, R₂, R₃, R₄, R₅ and R₆) while the subjects cycled at zero-load intensity. The capillary samples were cooled in a Styrofoam box filled with dry ice and within 5 minutes of completing the last sprint, the samples were analyzed in a blood gas analyzer (ABL90 FLEX analyzer from radiometer, Brønshøj, Denmark)

Statistics

A two-way repeated measures Anova was performed in SPSS to compare the mean differences and to determine whether the samples were significantly different. The bonferroni test was used to compare the means.

Results

A summary of the power, respiratory and capillary measurements can be seen in *Table 2*. When averaging the six sprints (R₁, R₂, R₃, R₄, R₅ and R₆) at each altitude; MP for the 30s wingate-test was significantly lower at 2000m when compared to sea-level (679±84w vs 664 ±94w, p=0.045), significantly lower when comparing 3000m to sea-level (655±89w vs 679 ±84w, p=0.003) and significantly lower when comparing 3000m to 1000m (655±89w vs 679 ±84w, p=0.009). FI was found to be significantly higher at: 3000m than at sea-level (61±13 vs. 57±12, p=0.027), 2000m (61±13 vs. 54±7, p=0.002) and at 1000m (61±13 vs. 56±10, p=0.023). *Figure 2* shows an insignificant decrease in FI from T0-T3, where after there is a significant increase in FI from T3-T4.

Significantly higher natrium⁺ was measured at 3000m compared to sea-level and 2000m (145.83 ±2.79 vs. 144.08 ±1.78, p=0.048 and 144.89 ±0.28, p=0.014). No significant difference in mean power was found between 1000m and sea-level (p=1). Ph-measurements were significantly lower when comparing 3000m to 1000m (7.167 ±0.112 vs. 7.141 ±0.121, p=0.001) and 2000m to 1000m (7.179 ±0.125 vs. 7.141 ±0.121 p=0.003). PPO, Wingate VO₂, Recovery VO₂ and Kalium⁺ were not significantly affected by altitude. No significant effect of altitude was found on any parameter, when comparing corresponding sprints across all altitudes (e.g. T0 R₃ vs. T3 R₃). *Figure 1* shows a tendency towards lower MP with increasing altitude, with a more pronounced difference from for R₂ –R₅.

Table 2 Average performance, respiratory and blood measurements

Variables	T0	T1	T2	T3
Mean Power (watts)	679 (±84)*2;3	679 (±84)*3	664 (±94)*0	655 (±89)*0;1
Peak Power (watts)	965 (±186)	958 (±182)	913 (±201)	945 (±196)
Fatigue index %	57 (±12)*3	56 (±10)*3	54 (±7)*3	61 (±13) *0;1;2
Wingate VO ₂ (ml/min)	2915 (±1137)	3113 (±925)	2742 (±799)	2766 (±784)
Recovery VO ₂ (ml/min)	2446 (±804)	2050 (±458)	2289 (±930)	2178 (±706)
Natrium (mmol/L)	144.08 (±1.78)*3	145.08 (±1.62)	144.89 (±0.28)*3	145.83 (±2.79)*0;2
Kalium (mmol/L)	8.15 (±2.78)	7.34 (±1.33)	6.48 (±0.54)	6.41 (±0.46)
Ph	7.185 (±0.133)	7.141 (±0.121)*2;3	7.179 (±0.125)*1	7.167 (±0.112)*1

Table notes: Data are means (±SD)

*significantly (p<0.05) different from trial(s) (seperated by semicolon)

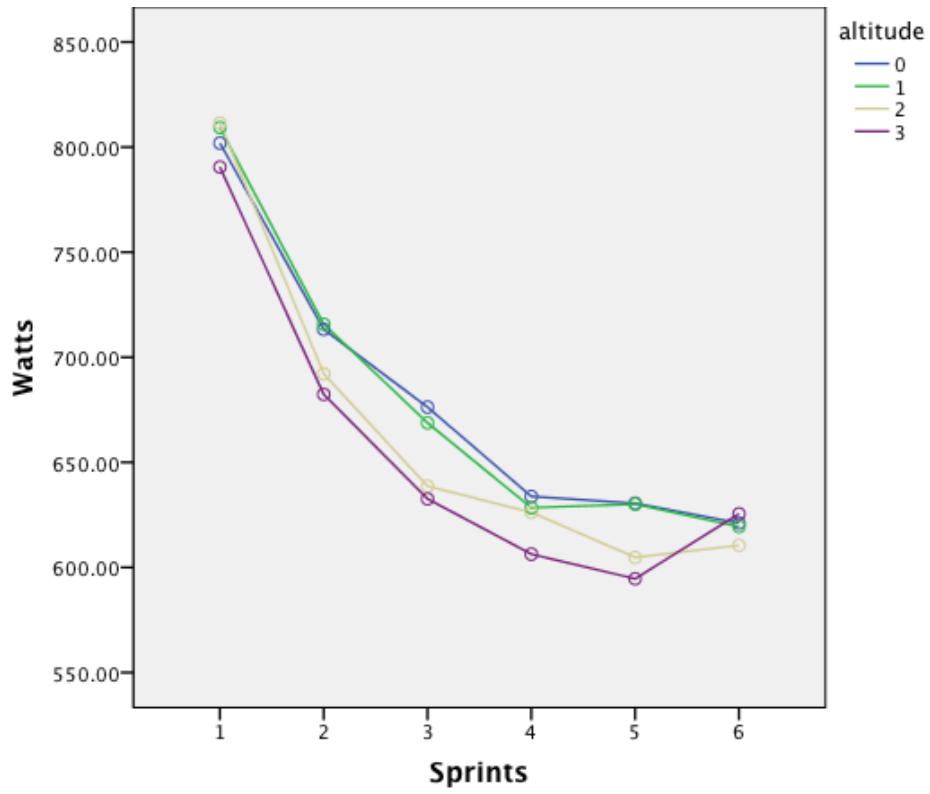


Figure 1 comparing each sprint at different altitudes

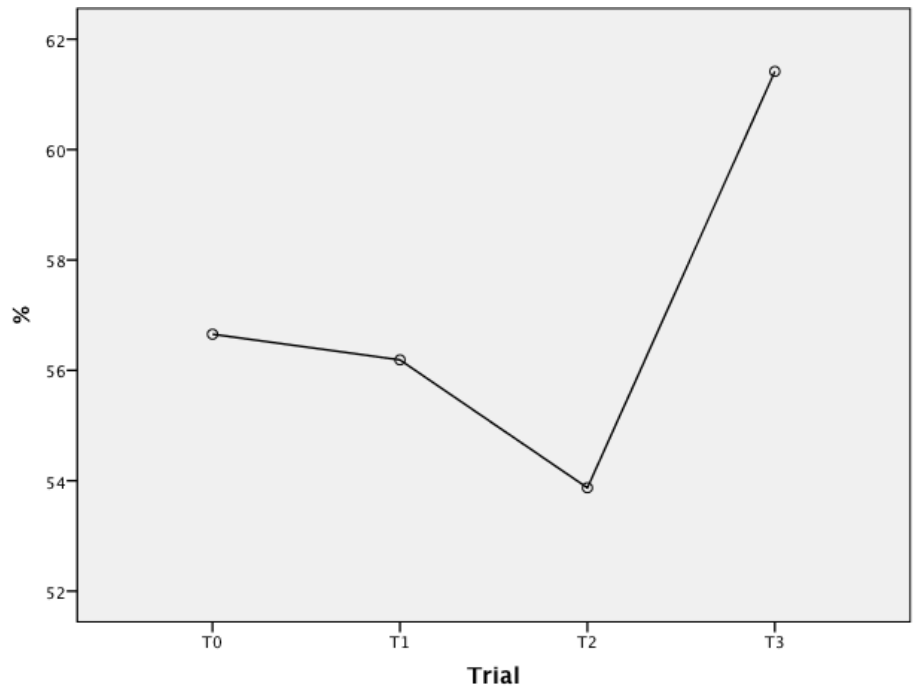


Figure 2 averaged fatigue index

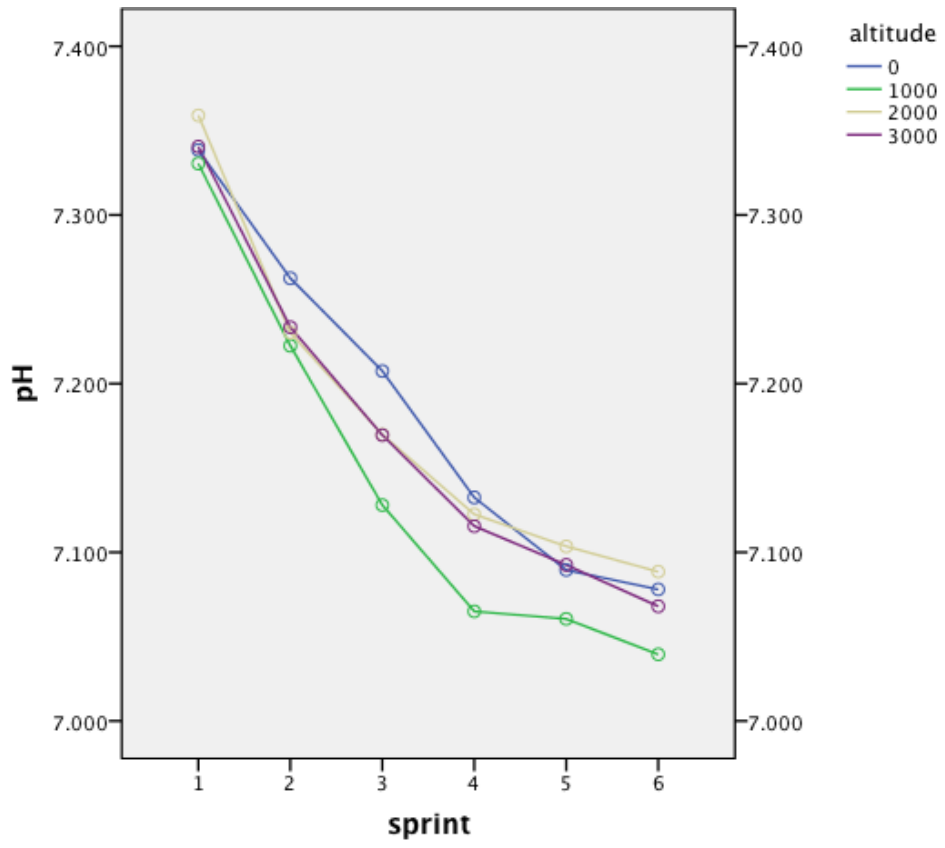


Figure 3 pH level for each altitude

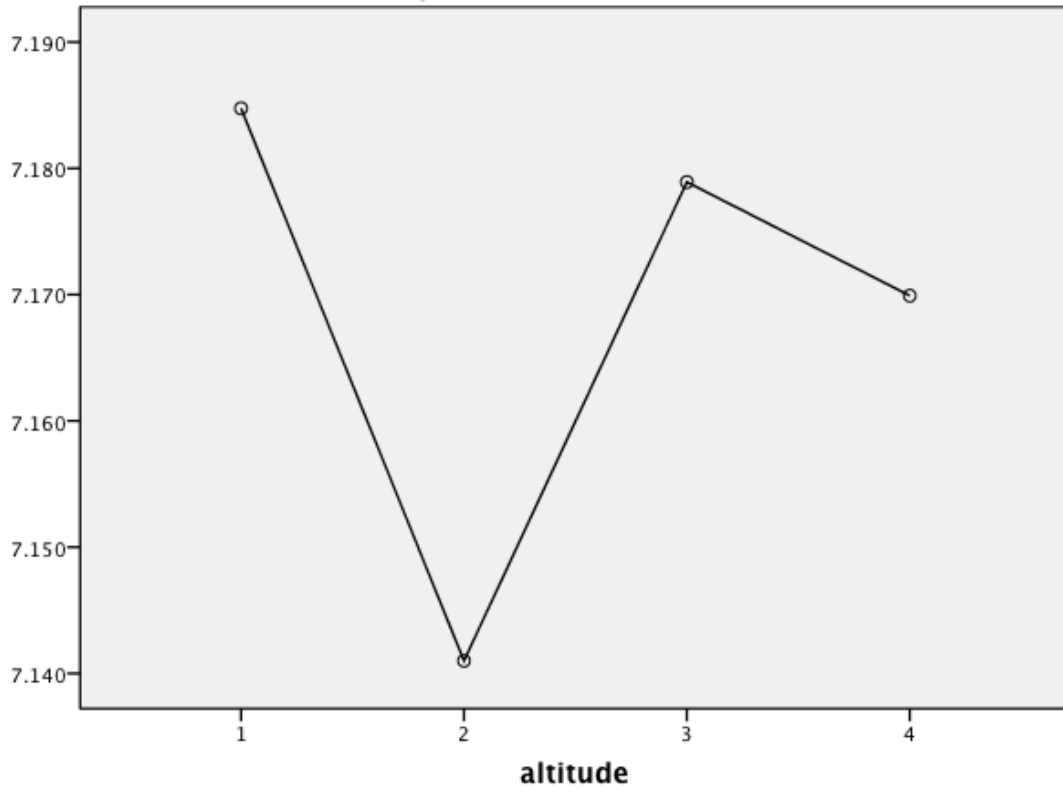


Figure 4 shows the average pH for each altitude

Discussion

We hypothesized in the introduction that MP would be affected negatively by an increase in altitude. The statistical analysis showed that altitude did affect the performance negatively when it comes to MP. The decrease in performance however, was only present at altitudes above ~2000 meters.

Contrary to Gore et al. who found a reduction in VO_2Peak at 580m – Going from about 1000 meters to 2000 meters, had less of an effect than going from 2000 to 3000 meters. As Oguri et al. found, we saw no significant reduction in MP in a single sprint at any of the tested altitudes. This was also the case when looking at individual sprints and comparing it to the same sprint number at a different altitude. It was only when all the sprints from each altitude was averaged that a significant difference was found. This may be due to the small sample size, not yielding enough degrees of freedom to detect the small differences, since Goods P et al. 2014 did find a significant reduction in mean power when testing team sport athletes ($n=10$) for 4 s sprint-performance at simulated altitudes (by lowering oxygen concentration).

One possible explanation for the lower MP could be the significantly reduced pH, it has been shown that supplementing with sodium bicarbonate can significantly increase MP in a 30s sprint (Inbar et al. 1983). T1, T2 and T3 all had significantly lower pH-values than T0. However, T1 yielded the lowest pH-value, even though no significant reduction in MP at T1 was observed. A possible explanation for this could be that pH has been observed to increase at rest at hypoxia (Forster et al. 1975; McLellan et al. 1988; McLellan et al. 1990; Mollard et al. 2007a). Therefore T2 and T3 could have a higher initial pH level than T1 and T0. This may also be the explanation to why MP for a single sprint, is not affected by altitude; along with an increase in catecholamines, which increase more during exposure to hypoxia and in turn, increases utilization of muscle glycogen (Escourrou et al. 1984). Another reason for the lower MP in the repeated sprints could be the and the incomplete resynthesis of PCr (Cottrell et al. 2002).

Strengths and limitations of the study

A problem in the present study, was the difficulty of the protocol. Out of nine recruited subjects only five finished more than three trials. It is possible that the five subjects who completed the study, had lower motivation for the latter trials. However, the subjects were randomized and each subject completed T0, T1, T2 and T3 in random order. Another limitation was the chosen break weight of 9.5% of the body weight of the subject. This weight was chosen based on research done by Patton et al. (1985) and Dotan et al. (1983) to optimize MP and PPO. This research was

done at sea-level which meant, that in the present study, subjects found it hard to complete R₄, R₅ and R₆ at altitudes above 1000 m, higher VO₂ and MP values might have been reached, had the break weight been lower.

Using the finger prick method to collect arterial samples gives a larger likelihood of sample error. Many capillary samples had to be discarded because the sample was inconsistent. The time from completion of a sprint until the capillary sample has been taken is about one minute. This time difference reduces the likelihood of collecting a true sample. Further the diet and training between tests were not controlled.

A strength of the present study is the use of a hypobaric environment. Most studies use a hypoxic environment to assess the effect of altitude on performance (McLellan et al. 1990; Ferretti et al. 1997b; Mollard et al. 2007b; Oguri et al. 2008). Thus this study more closely resembles a real world scenario, with a reduced partial pressure as opposed to a reduced F_IO₂. Another advantage of this study is the training status of the subjects plus the fact that they are all actively competing at the highest domestic level. It has been shown that well trained athletes have a more pronounced performance decline at altitude (Oguri et al. 2008), therefore it is important to use highly trained athletes to be able to generalize the results to world class athletes.

In the future it would be interesting to conduct further tests using a larger sample size. It would also be interesting to have more accurate and continuous blood-measurements by placing an arterial line instead of using finger pricks. The present study only observed a significant effect above 1000m. Therefore future studies could focus on testing subjects above 1000m and testing at smaller increments. Since the significant effect happened at altitudes above 1000m, it might be advantageous for elite athletes seeking to improve their anaerobic performance at altitude, to train at altitudes above 1000m.

Conclusion

These results suggest that acute exposure to hypobaric hypoxia impairs repeated 30-s anaerobic performance when altitude ≥ 2000 m possibly by reducing pH (which was found to be significantly lower), the rate of PCr resynthesis and muscle glycogen.

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