

RESEARCH PAPER

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The effect of 10 days of heat acclimation on exercise performance in acute hypobaric hypoxia (4350 m)

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ABSTRACT

To examine the effect (“cross-tolerance”) of heat acclimation (HA) on exercise performance upon exposure to acute hypobaric hypoxia (4350 m). Eight male cyclists residing at 1600 m performed tests of maximal aerobic capacity (VO_{2max}) at 1600 m and 4350 m, a 16 km time-trial at 4350 m, and a heat tolerance test at 1600 m before and after 10 d HA at 40°C, 20% RH. Resting blood samples were obtained pre-and post- HA to estimate changes in plasma volume (ΔPV). Successful HA was indicated by significantly lower exercise heart rate and rectal temperature on day 10 vs. day 1 of HA and during the heat tolerance tests. Heat acclimation caused a 1.9% ΔPV , however VO_{2max} was not significantly different at 1600 m or 4350 m. Time-trial cycling performance improved 28 sec after HA ($p = 0.07$), suggesting a possible benefit for exercise performance at acute altitude and that cross-tolerance between these variables may exist in humans. These findings do not clearly support the use of HA to improve exercise capacity and performance upon acute hypobaric hypoxia, however they do indicate that HA is not detrimental to either exercise capacity or performance.

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Introduction

There is growing interest in high altitude recreation and endurance events; also, an increasing number of United States troops are being deployed to high altitude terrain. Individuals not properly acclimatized or acclimated to altitudes above 2000 m experience reduced submaximal and maximal exercise performance due to the decline in inspired and arterial oxygen pressure, oxygen saturation of hemoglobin, and arterial oxygen content.¹ During altitude exposure, a specific exercise task requires a greater percentage of maximal oxygen uptake (VO_{2max}) compared to sea level.^{2,3} A 66% decrement in cycle time-trial (TT) performance compared to sea level during acute exposure to 4300 m has been reported.⁴ However, there is a smaller decrease in submaximal endurance performance following 2–3 weeks of high altitude

exposure.^{5,6} Young et al.,⁷ reported that 15 d of altitude exposure at 4300 m did not significantly change VO_{2max} , similar to a report by Faulkner et al.,⁸ showing no change following 6 weeks of training at 2300 m. The improvement of submaximal exercise endurance by acclimation is important because most athletes and military personnel perform prolonged submaximal exercise and many may not have sufficient time or means to properly acclimatize or acclimate to hypoxia. Thus, alternate training and acclimation methods have been explored, these include “live high: train low,” altitude tents, and intermittent altitude exposure.⁴ However, these methods are time consuming and require equipment and facilities that are expensive and may not be available.

It has been suggested that acclimation to one environmental stressor can enhance adaptation to another

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stressor.⁹ This phenomenon, described as “cross-tolerance,” involves the activation of common protective pathways,¹⁰ and has been studied predominantly in small mammals. Hiestand et al.,¹¹ studied mice heat acclimated (HA) to 36–37°C for 10–14 d and found they tolerated a hypoxic environment better than unacclimated mice. Cross-tolerance between cold and hypoxia has also been examined in rats. Those chronically exposed to cold had less tolerance to acute hypoxia than those exposed to hypoxia alone,⁹ demonstrating that cross-tolerance may not always be beneficial. Recent review articles have suggested that HA may lead to molecular and systemic adaptations that benefit individuals exposed to high altitude.^{12–14}

In humans the improvements in sea level exercise performance following HA may result from physiological adaptations, including reduced oxygen uptake and blood lactate at a given submaximal power output,^{15,16} increased $\text{VO}_{2\text{max}}$,^{17,18} muscle glycogen sparing,^{15,19} and plasma volume expansion (ΔPV) that can range from 0–30%.²⁰ Lorenzo et al.,¹⁷ observed increased $\text{VO}_{2\text{max}}$ (5 and 8%), cardiac output (9 and 5%), and improved TT performance (6 and 8%) in cool (13°C) and hot (38°C) environments, respectively, following 10 d of HA. The authors speculated that improvements in performance were likely attributed to the 6.5% increase in plasma volume (PV).

To our knowledge, only one study in humans has examined cross-tolerance, involving the effect of HA on physiological responses during acute altitude exposure in humans. Heled et al.,²¹ reported that 12 d of HA delayed the onset of blood lactate accumulation (OBLA) during a maximal exercise test in normoxia and simulated hypoxia. This supports a previous finding of a right-ward shift in the lactate threshold after HA, which can be beneficial for endurance performance.²² Additionally, the authors reported improved cognitive function and dynamic postural performance during exposure to hypoxia equivalent to 2400 m. Because the main purpose of the Heled et al.,²¹ study was to investigate changes in cognitive function and not exercise performance, it is unclear if HA is beneficial for acute exercise at altitude.

If HA can be shown to improve exercise capacity or endurance during subsequent ascent to altitude, it could encourage the development of HA protocols to maintain and/or enhance exercise performance during these exposures. The purpose of this study was to determine the effects of 10 d of HA on exercise

performance during acute hypobaric hypoxia in trained men. We hypothesized that HA would increase PV which would improve $\text{VO}_{2\text{max}}$, and the 16 km cycling TT at 4350 m.

Material and methods

Subjects

Eight trained men were recruited from the university and local communities. The subjects were cyclists and runners performing an average of 5.9 hr/wk of moderate and 2.6 hr/wk of vigorous exercise throughout the year prior to the study. Subjects were required to have a $\text{VO}_{2\text{max}}$ that met the criteria of ≥ 80 th percentile for males aged 18–44 yr.²³ The mean baseline age, height, weight, and $\text{VO}_{2\text{max}}$ were 28 ± 6 yr, 1.78 ± 0.08 m, 75.7 ± 8.4 kg and 4195 ± 544 mL/min, respectively. All subjects resided at altitudes of 1500–1600 m 6 months prior to the study. Subjects maintained their normal exercise routines over the course of the study, as verified by activity logs. Potential volunteers were excluded if classified as moderate or high risk according to the American College of Sports Medicine.²³ Written informed consent, as approved by the University Human Research Review Committee, was obtained prior to participating in the study.

Experimental design

Subjects reported to the laboratory at the same time of day for each test and were instructed to refrain from strenuous exercise, caffeine and alcohol 24 hr prior to testing. Preliminary testing included determination of $\text{VO}_{2\text{max}}$ and completion of one heat tolerance (HtT) test at ambient pressure (633 mmHg = 1600 m), 2 familiarization 16 km cycle TTs at 1600 m and a 16 km cycle TT and a $\text{VO}_{2\text{max}}$ test at altitude (455 mmHg = 4350 m). The $\text{VO}_{2\text{max}}$ test at 1600 m and HtT test were completed on the same day separated by 1 hr, while all other trials were separated by at least 24 hr. After preliminary testing, subjects began a 10 d HA protocol. Twenty-four hrs following the 10 d HA protocol, the $\text{VO}_{2\text{max}}$ test and TT were repeated at 4350 m, separated by at least 24 hr. Lastly, a final $\text{VO}_{2\text{max}}$ test at 1600 m and HtT test were repeated, separated by 1 hr. Subjects were exposed to acute hypobaric hypoxia for ~ 1.5 hr during all tests at 455 mmHg. Nude body weight was recorded prior to each test by electronic scale (Model 2531, Seca, Danville,

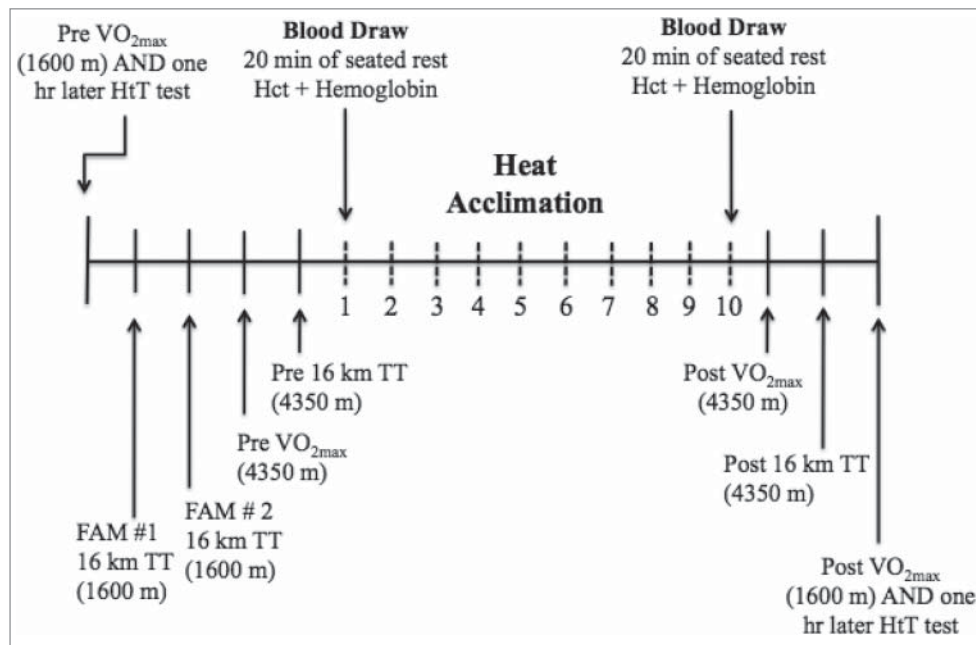


Figure 1. Experimental procedures followed in the present study. VO_{2max} , maximal aerobic capacity; HtT test, heat tolerance test; FAM, familiarization; TT, time-trial; Hct, hematocrit.

VA). The sequence and timing of test procedures are summarized in Figure 1.

Measurement of VO_{2max}

These tests took place in a custom built hypobaric chamber, which is 6.1 m long and 2.4 m in diameter and ventilated with fresh ambient air. All incremental cycle testing was performed on an electronically-braked cycle ergometer (Velotron DynaFit Pro, Racermate, Seattle, WA). Exercise began at 70 W for one minute and work rate increased 35 W every minute until volitional fatigue. Heart rate (HR) was continuously monitored via telemetry (Polar Electro, model FS1, Woodbury, NY), while oxygen saturation (SaO_2), measured on index finger, (GO_2 Pulse Oximeter, Philips Respironics, Andover, MA) and rating of perceived exertion (RPE), using a 6-20 scale, were measured every minute. Breath-by-breath gas exchange was continuously measured using a metabolic cart (ParvoMedics True One 2400, Sandy Utah). The VO_{2max} was recorded as the highest average value over any 15 s period. Prior to each test, the metabolic cart was calibrated to room air and a gas of known concentration (16% O_2 and 4% CO_2). A 3-liter syringe was used to calibrate flow. Per manufacturer's recommendation, the flow rate of the pneumotach was reconstructed for reduced air density for tests at 455 mmHg.

Heat tolerance test

The HtT test was performed in an environmental chamber at 633 mmHg and consisted of cycling (Monark Ergonomic, Model 828E, Varberg, Sweden) for 45 min at a temperature of 40°C and ~20% relative humidity and a power output equal to ~55% of the previously determined VO_{2max} at 1600 m. This test was performed before and after HA to measure the subjects ability to tolerate exercise in the heat. Urine samples were collected before the test to determine hydration status via urine specific gravity (euhydration was classified as ≤ 1.020 g/mL) using a refractometer (REF312ATC, General Tools and Instruments, New York City, NY). If subjects were not euhydrated upon arrival at the laboratory they consumed water, followed 30 min later by a second hydration assessment. Subjects then inserted a rectal thermistor (Model 4TH, Telly Thermometer, Yellow Springs, Ohio) ~10 cm past the anal sphincter. Uncovered skin thermistors (YSI 409B, Thermistor Probe, Dayton, Ohio) were placed on the chest, arm, and thigh to obtain mean skin temperature as: $T_{sk} = 0.43 T_{chest} + 0.25 T_{arm} + 0.32 T_{thigh}$.²⁴ Thermal sensation was assessed using an 8-point scale. Rectal temperature (T_{re}), T_{sk} (Model 44TA, Telly Thermometer, Yellow Springs, Ohio), thermal sensation, HR, and RPE were recorded every 5 min. Urine output was measured after the trial. Upon test completion,

subjects exited the chamber and nude body weight and urine volume during the test were recorded to determine whole body sweat rate.

Familiarization cycle time-trial

Subjects completed a 10 min warm-up at a self-selected workload followed by a 16 km self-paced cycle TT (flat course; Velotron RacerMate 3-D Software, Seattle, WA) at 1600 m. The familiarization tests were performed to minimize a learning effect and were not performed at 4350 m to prevent exposing subjects to high altitude prior to HA. Subjects were informed that selecting a higher gear would allow them to attain higher speeds and were allowed to adjust gears manually throughout the entire trial. Water was provided *ad libitum*. Subjects were informed of distance covered throughout the trial and given verbal encouragement, but were not given any other information. Heart rate, SaO₂ and RPE were measured every 1.6 km and at the end of the TT. The second familiarization trial was performed at least 24 hr after the first TT at the same time of day. Pilot testing in one of our laboratories revealed an intraclass correlation coefficient (0.985) and coefficient of variation (1.2%) across subjects for performance during the 16 km cycle TT at sea level; these values are similar to reports of other trained cyclists.^{25,26}

Cycling time-trial

The 16 km cycle TT was performed in the hypobaric chamber at 455 mmHg (4350 m). Subjects again completed a 10 min warm-up followed by the TT. Subjects were reminded to complete the 16 km TT as fast as possible and water was provided *ad libitum*. Heart rate, SaO₂ and RPE were measured as described above.

Heat acclimation

Subjects completed 10 consecutive days of HA at 1600 m in an environmental chamber at 40°C and ~20% relative humidity. Heat acclimation was achieved using a traditional protocol consisting of 2 50 min cycling bouts on an ergometer (Monark Ergo-med, Model 828E, Varberg, Sweden) with 10 min of seated rest between bouts.^{16,17} In order to prevent a training response by the repeated HA exercise sessions, the exercise intensity for all sessions was determined by subtracting 75 W from the workload that resulted in the ventilatory threshold during the first

VO_{2max} test at 1600 m (Bradley RT, unpublished observation), equating to a VO₂ of ~55% of their VO_{2max} at 1600 m. Core temperature was recorded every 5 min by a rectal thermistor via the Telly Thermometer, along with thermal sensation, HR and RPE. Water intake was provided *ad libitum* during exercise and total volume and urine output were recorded. Body weight loss, water intake and urine volume were recorded to calculate sweat rate. The HA termination criteria included: 1) completion of the 110 min test, 2) T_{re} ≥ 40°C or 3) subject requesting to stop.

Plasma volume change

Prior to HA exercise on day 1 and day 10, subjects were seated for 20 min in a cool room.²⁷ Then 10 mL of venous blood was drawn from an antecubital vein for the determination of hemoglobin (Hb) and hematocrit (Hct). The Hct was determined in triplicate by centrifuging 3 heparinized capillary tubes (Model C-MH30, Unico, Dayton, NJ) at 12,000 rpm for 5 min and Hb was measured by Quest Diagnostics (Albuquerque, NM) using established procedures (Beckman Coulter, LH750). The Hct, corrected by 0.96 for trapped plasma, and Hb values were used to calculate resting ΔPV from day 1 to day 10.²⁸

Statistics

Data are reported as mean ± SD and were obtained using SPSS Version 19.0 (Chicago, IL). Dependent *t*-tests were used to test differences in values between day 1 and day 10 of HA, as well as the HtT test difference in values between pre- and post-HA. The difference in VO_{2max} and the 16 km TT, as well as end-exercise HR, SaO₂, and RPE values in response to HA were also tested using a dependent *t*-test. An *a priori* power analysis using commercially available software (G*Power, Germany) was used to determine the sample size needed to find a change in resting PV between pre- and post-HA. The ΔPV was selected to calculate power because a previous HA study concluded that an increase in PV led to an increase in exercise capacity and ultimately improved TT performance.¹⁷ An α level of 0.05 and power of 0.95 was indicated using 8 subjects.

In addition to traditional hypothesis testing, magnitude-based inferences were utilized to examine the “true” effect of this intervention on the 16 km TT.²⁹ The paired *t*-test *p*-value, mean percent change in

performance, degrees of freedom ($n - 1$), and smallest worthwhile performance benefit (0.3 is the typical within athlete standard deviation; CV for our familiarization TTs at 1600 m was $1.58 \% * 0.3 = 0.5$) were entered into a downloadable spreadsheet (<http://www.sportsci.org/resource/stats/xcl.xls>) to calculate the $\pm 95\%$ confidence limits (CLs) and the percent probability that the HA effect was positive/beneficial ($> 75\%$), negligible/trivial (25-75%), or negative/harmful ($< 25\%$).²⁹

Results

Heat acclimation responses

The responses during day 1 and day 10 of HA and during the HtT test before and after HA are shown in Table 1 and Table 2. The final HtT test was completed an average of 8 days after the 10th day of HA. On day one of the 10 d HA intervention, 5 subjects completed the required 110 min, with 2 terminating at 80 min and one at 90 min. On day 10 of HA, only one subject terminated 5 min short of the required time. In the first HtT test 2 subjects terminated 5 min before the 45 min test and all subjects completed the entire 45 min test during the last HtT test following HA. There was a significant reduction in RPE, thermal sensation, HR and T_{re} values over the course of HA from day 1 to day 10 of HA (Table 1) and also from the pre to post HtT test (Table 2). Measurements of T_{sk} were made only in the HtT tests, where it was also reduced significantly after HA. Sweat rate after HA increased by an average of 10% in both sets of measurements, but was not significant in either. The average ΔPV was 1.9%, increasing in 5 subjects and decreasing in 3 subjects.

Table 1. Values are shown as mean \pm SD for 8 subjects on day 1 and day 10 of heat acclimation.

Parameter	Unit	Day 1	Day 10	<i>p</i>
RPE	—	16 \pm 3	13 \pm 1	0.021
Thermal Sensation	—	7 \pm 1	6 \pm 1	0.002
End HR	(b/min)	162 \pm 18	141 \pm 15	<0.001
End T_{re}	(°C)	39.3 \pm 0.7	38.8 \pm 0.6	0.042
Sweat rate	(ml/hr)	1263 \pm 320	1424 \pm 535	0.33
Hb	(g/dL)	15.8 \pm 1.6	15.7 \pm 0.9	0.69
Hct	(%)	46.2 \pm 3.6	45.8 \pm 1.8	0.63
ΔPV	(%)	—	+1.9 \pm 10.0	—

RPE, rating of perceived exertion; HR, heart rate; T_{re} , rectal temperature; Hb, hemoglobin; Hct, hematocrit; PV, plasma volume.

Table 2. Values are shown as mean \pm SD for 8 subjects during the first and last heat tolerance test.

Parameter	Unit	Pre	Post	<i>p</i>
RPE	—	15 \pm 2	13 \pm 2	0.050
Thermal Sensation	—	8 \pm 1	6 \pm 1	0.010
End HR	(b/min)	166 \pm 16	148 \pm 19	<0.001
End T_{re}	(°C)	38.8 \pm 0.5	38.4 \pm 0.3	0.010
End T_{sk}	(°C)	37.7 \pm 0.3	37.1 \pm 0.6	0.038
Sweat rate	(ml/hr)	1543 \pm 820	1645 \pm 530	0.79

RPE, rating of perceived exertion; HR, heart rate; T_{re} , rectal temperature; T_{sk} , skin temperature.

Effect of heat acclimation on VO_{2max}

The mean values for VO_{2max} and associated measurements at 4350 m and 1600 m before and after HA are shown in Table 3. The average VO_{2max} at 4350 m increased by 2.2%, but was not significant, increasing in 4 and decreasing in 4 subjects. At 1600 m the small decrease of 1.2% was also not significant, decreasing in 5 and increasing in 3 subjects. The other measurements remained relatively unchanged at both altitudes after HA.

Effect of heat acclimation on cycling time-trial

The average time to complete the 16 km TT decreased 28 sec after HA, with an equivalent increase in velocity of 1.6% (Table 4). While not significantly different, there was a trend ($p = 0.07$) for improvement in 16 km TT time and velocity. The 95% CLs for average time was ($-2.5 - 57.7$ sec), while magnitude-based inferences suggested that the effect of HA was 96.5% likely to be beneficial and to improve performance at 4350 m. Individual 16 km data after HA show that 7 of the 8 subjects reduced the time to complete the simulated TT (Fig. 2). End-exercise HR, SaO_2 , and RPE during the TT were not significantly different after 10 d of HA (Table 4).

Discussion

The purpose of the study was to examine whether 10 d of HA would improve exercise capacity and performance during acute hypobaric hypoxia (4350 m) in trained men. Heat acclimation was achieved by our protocol, as supported by the reduced T_{re} , HR, RPE and thermal sensation over the 10 d protocol and by the HtT test (Table 1 and 2). Even though the final HtT test was performed an average of 8 d after the last HA session the measurements indicated subjects were still well acclimated to heat (Table 2). In two recent

Table 3. Values are shown as mean \pm SD for gas exchange and cardiovascular measurements at the end of maximal exercise in response to 10 d of heat acclimation.

Parameter	Unit	455 mmHg (4350 m)			633 mmHg (1600 m)		
		Pre	Post	<i>p</i>	Pre	Post	<i>p</i>
RPE	—	18.5 \pm 1.1	17.9 \pm 1.1	0.18	17.5 \pm 1.7	18.4 \pm 1.2	0.18
VO _{2max}	(mL/min)	3487 \pm 270	3562 \pm 423	0.41	4195 \pm 544	4145 \pm 447	0.57
RER	—	1.22 \pm 0.06	1.23 \pm 0.04	0.93	1.23 \pm 0.06	1.21 \pm 0.04	0.42
SaO ₂	(%)	75.6 \pm 3.8	75.9 \pm 3.7	0.78	90.4 \pm 2.4	90.6 \pm 4.4	0.86
HR	(b/min)	170 \pm 12	170 \pm 9	0.93	173 \pm 13	177 \pm 6	0.21
V _E	(L/min)	175 \pm 33	181 \pm 32	0.22	171 \pm 30	176 \pm 25	0.49
V _f	(b/min)	55.2 \pm 12.1	56.7 \pm 10.9	0.46	54.1 \pm 12.3	54.6 \pm 8.3	0.87
V _T	(L)	3.20 \pm 0.34	3.21 \pm 0.38	0.85	3.22 \pm 0.40	3.23 \pm 0.31	0.89

RPE, rating of perceived exertion; VO_{2max}, maximal aerobic capacity; RER, respiratory exchange ratio; SaO₂, oxygen saturation; HR, heart rate; V_E, ventilation; V_f, ventilation frequency; V_T, tidal volume.

reviews, authors reported that the rate of HA decay is controversial.^{30,31} However, others have reported that adaptations from HA can be retained for up to 3 weeks.³²

Exercise capacity, as indicated by VO_{2max}, was not significantly affected by HA at 4350 m or 1600 m. There was a nearly significant improvement in the 16 km TT performance at 4350 m after HA (Table 4), however magnitude-based inferences suggest that the effect of HA was 96.5% likely to be beneficial and to improve cycling performance at 4350 m. Our results do not fully confirm the reports from animal and human models that exposure to one environmental stressor may improve adaptation to another environment.^{9,11,21} However, the findings do indicate that this HA protocol was not detrimental to exercise capacity or performance upon acute exposure to 4350 m.

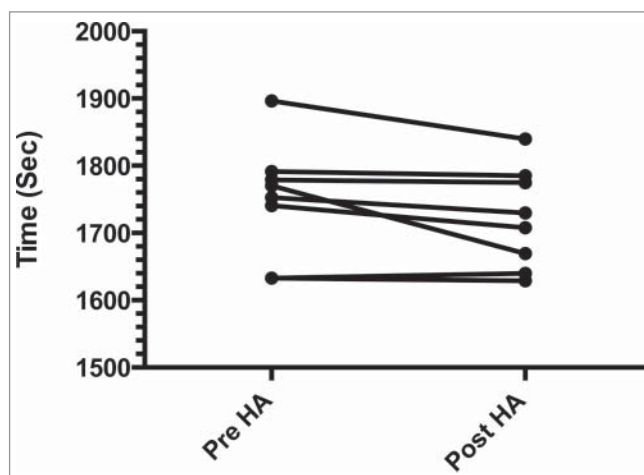
Sawka et al.,¹⁸ employed a 9 d HA protocol with moderately trained subjects (VO_{2max} = 45 mL/kg/min) and found that VO_{2max} was increased by 4% at 21°C. Another study reported a similar increase (5%) in a cool environment (13°C) in highly trained cyclists.¹⁷ Sawka et al.,¹⁸ concluded that the improvement in VO_{2max} must be due to a “training effect” from the HA protocol. However, their subjects

exercised at \sim 50% VO_{2max}, which would not be expected to elicit a training effect in trained individuals.³³ But because their subjects were moderately fit, a training effect following the 9 d HA protocol may still have occurred at \sim 50% VO_{2max}. Lorenzo et al.,¹⁷ attributed their observed VO_{2max} increase to the increase in PV (6.5%) and cardiac output (2.2 L/min). Their improvement in TT performance was likely due to the increased VO_{2max}, which would reduce the relative exercise intensity. However, in the current study VO_{2max} was not altered at 1600 m or 4350 m following HA, which agrees with other findings.^{21,34} Nielsen et al.,³⁴ reported no significant differences in VO_{2max} in response to HA despite increases in PV (+13.1%), cardiac output, and stroke volume. However, they did not explain why there was no change in VO_{2max} in the presence of increased central O₂ delivery and no difference in leg blood flow at 10 min of exercise and at exhaustion.

Table 4. Mean \pm SD of measurements at the end of the 16 km cycle time-trial at 4350 m pre and post heat acclimation.

	Unit	Pre	Post	<i>p</i>
Time	(sec)	1749.5 \pm 86.3	1721.9 \pm 74.7	0.07
Velocity	(km/hr)	33.0 \pm 1.6	33.5 \pm 1.4	0.07
RPE	—	18.8 \pm 1.3	18.4 \pm 1.3	0.28
HR	(b/min)	172 \pm 8	172 \pm 5	1.00
SaO ₂	(%)	76.4 \pm 3.3	76.5 \pm 2.6	0.76

RPE, rating of perceived exertion; HR, heart rate; SaO₂, oxygen saturation.

**Figure 2.** Individual time-trial performance for 8 subjects before and after heat acclimation at 4350 m.

We proposed that HA would elicit an increase in PV in trained subjects, which would increase cardiac output and venous return, ultimately increasing VO_{2max} and improving TT performance at altitude. However, our results do not support a direct relationship between changes in PV and VO_{2max} , as we observed a relatively small ΔPV and a nonsignificant change in VO_{2max} after HA. Because of this unique protocol, there is no prior research that can be compared with our results to support or refute these data. We also speculate that rather than PV expansion, the non-significant improvements in cycling TT performance after HA could be due to attenuated sympathetic input,³⁵ improved recruitment of slow-twitch muscle fibers,¹⁶ glycogen sparing,¹⁵ and improved economy following HA,¹⁸ which would contribute to a reduction in metabolic stress. In addition, cellular adaptations may occur in response to heat stress, which may increase mitochondrial biogenesis,³⁶ resulting in enhanced oxidative capacity, as this is the strongest predictor of performance in highly trained subjects.³⁷ However, these variables were not measured in the present study.

To our knowledge, work done by Heled et al.,²¹ was the first to examine efficacy of a cross-tolerance model in humans. In their study, 12 d of HA improved cognitive function and changes in physiological strain (indicated by the OBLA), but no change in VO_{2max} during acute moderate altitude exposure, suggesting that exposure to heat and not a training effect accounted for their results. However, their altitude stimulus was mild (2400 m) and brief (10 min); thus these findings cannot be extended to real-world settings, as athletes and military personnel sojourn to higher altitude for longer periods of time.

We observed a relatively small change in resting PV (1.9 %) following HA, while others have generally reported sizable increases (5–16%) following HA.^{17,38,39,39} It is important to note that reported changes in PV following HA are highly variable, ranging from 0–30%.²⁰ One study,⁴⁰ examined the effect of PV expansion on exercise capacity and maximal cardiac output in lowlanders acclimatized to altitude for 9 wks at 5260 m and found that PV expansion had no effect on VO_{2max} or maximal cardiac output. However, Robach et al.,⁴¹ studied subjects for 31 d in a hypobaric chamber at altitudes from 4500 m to 8848 m, with incremental VO_{2max} tests performed with and without PV expansion at

sea level, 6000 m, and upon return to sea level. They concluded that improved VO_{2max} by 9% at altitude (6000 m) in acclimated subjects resulted from acute expansion of PV during a maximal exercise test and the resulting increase in venous return, cardiac output and muscle blood flow.

A possible reason why this study did not find a large ΔPV after HA is that the subjects maintained their normal cycle training during the course of the study. The 10 d impact of the additional 100 min of exercise in the heat may not have been a significant perturbation.

One major limitation of the present study was the lack of a control group that performed the daily exercise, but was not exposed to HA. Therefore we cannot completely exclude the potential that our subjects experienced an exercise training effect following 10 d of HA. However, others have implemented a control group and observed no training effect following 10 d of HA in trained cyclists exercising at 50% VO_{2max} .¹⁷ In order to prevent a training effect in the present study we recruited subjects that were classified as trained, with an average VO_{2max} of 4195 mL/min, as well as individualizing the training intensity by having our subjects exercise at a VO_2 below their ventilation threshold. Researchers have reported that exercise intensities greater than ventilation threshold may induce a training effect in trained individuals.⁴² Further, analyses of the power output and VO_2 at the ventilatory threshold and respiratory compensation point after HA were not statistically different, suggesting that a training effect would have been minimal in the present study. Another limitation was the small sample size, which was based on detecting a significant ΔPV following HA. Perhaps a larger sample size may have resulted in significant differences in exercise capacity and performance. Implementing cycling familiarization TTs at 4350 m and repeating multiple TTs before and after HA would probably have reduced the variability of the results. However, this would have elicited additional exposure to high altitude, which could have altered the post-test results. Our subjects also resided at a mild altitude (1600 m) for the last 6 months; therefore, we do not know how sea level residents would have responded.

In conclusion, this study demonstrates that HA is not detrimental to exercise capacity, as measured by VO_{2max} , or exercise performance, as measured by cycling TT in trained male cyclists during acute

altitude exposure (4350 m). These results do not clearly support the cross-tolerance model that exposure to one environmental stressor may improve adaptation to another environment. Additional research is merited to expand and confirm these findings to determine whether cross-tolerance exists in humans between HA and acute altitude exposure and may be beneficial for improvement of altitude performance.

Abbreviations

HA	heat acclimation
Hb	hemoglobin concentration (g/dL)
Hct	hematocrit (%)
HR	heart rate (b/min)
HtT test	heat tolerance test
OBLA	onset of blood lactate accumulation
PV	plasma volume
Δ PV	change in plasma volume (%)
RPE	rating of perceived exertion
SaO ₂	oxygen saturation
T _{re}	rectal temperature (°C)
T _{sk}	skin temperature (°C)
TT	16 km cycle time-trial
VO _{2max}	maximal oxygen uptake (mL/min, L/min or mL/min/kg)

Disclosure of potential conflicts of interest

No potential conflicts of interest are disclosed.

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