METHODS: Over the course of a 6-year period, eighty-eight active subjects (age = 23.3 + 3.5 yrs, weight = 78.5 + 17.5 kg, VO_{2max} = 42.4 + 5.7 ml/kg/min) completed a graded-exercise test on a cycle ergometer at sea level (SL1), upon acute exposure to 3417 m (ALT1), two weeks following acclimatization at 3417 m (ALT2), and upon return to sea level (SL2). Workloads were increased every two minutes following a two-minute warmup until volitional fatigue. Maximum oxygen consumption was measured using a Parvo TruOne 2400 Metabolic cart. Subject's activity levels were assessed during the 2-week period and were unchanged relative to sea level.
RESULTS: Maximum oxygen consumption significantly declined (P<0.05) from SL1 to ALT 1 (3.48 + .39 l/min vs. 3.04 + .32 l/min). However, by ALT2 VO_2max was not different from SL1(3.48 + .39 l/min vs. 3.31 + .51 l/min). Maximum oxygen consumption was slightly, but not significantly higher upon return to sea level (SL2 = 3.65+ .66 l/min). While body weight changes occurred in some subjects, overall there was no difference in average body weight between any of the testing points.
CONCLUSIONS: These data suggest that exposure to acute altitude results in a reduction in maximum oxygen consumption. However, after two weeks of acclimatization maximum oxygen consumption returns to pre-sea level values in a large multi-year study.

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Does Arterial Oxyhemoglobin Saturation Influence the Hemoglobin Mass-VO2Peak Relationship in Endurance Athletes at Moderate Altitude?

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(No relationships reported)

Total hemoglobin mass (tHb) is a well-established, key predictor of maximal oxygen uptake (VO_2peak) across aerobic fitness levels. Arterial oxyhemoglobin saturation (S_aO_2) has the potential to modify this relationship, especially in populations that experience exercise-induced arterial desaturation.
PURPOSE: To examine how variability in S_aO_2 at VO_2peak modifies the relationship between tHb and VO_2peak at moderate altitude (1625m) in highly trained athletes.
METHODS: 16 male and 17 female competitive, highly trained (>10 hr training per week) cyclists/triathletes took part. On visits one and four tHb was assessed via the optimized carbon monoxide rebreathing method. Visits two and three were identical graded exercise tests (GXT) on a cycle ergometer to determine VO_2peak and S_aO_2 at VO_2peak; the workload began at 4 and 3 W kg^{-1} for men and women respectively, rounded down to the nearest 20 W increment, and power increased 20 W every minute until volitional exhaustion. VO_2 was measured using indirect calorimetry and VO_2peak was calculated as the highest average 30 sec VO_2. S_aO_2 was measured at rest and during exercise using forehead pulse oximetry; S_aO_2 at VO_2peak was calculated as the average S_aO_2 during the same 30 sec used to determine VO_2peak. Duplicate measures of tHb were averaged in order to reduce measurement error. In order to control for the effect of body mass on VO_2peak and tHb, both variables were normalized by body mass prior to analysis.
RESULTS: VO_2peak was significantly higher for the second GXT (+0.06 ± 0.17 L O_2 min^{-1}, p = 0.05) and the difference in VO_2peak was related to the difference in S_aO_2 (r = -0.42, p = 0.02), so results from the second GXT were used. VO_2peak ranged from 62.5 – 83.0 and 44.5 – 67.3 ml kg^{-1} min^{-1} in men and women respectively; tHb ranged from 12.1 – 17.5 and 9.1 – 13.0 g kg^{-1} and S_aO_2 at VO_2peak ranged from 81.7 – 94.0 and 85.7 – 95.0%. tHb explained 32% of the variability in VO_2peak (p = 0.02) in men and 42% in women (p = 0.01), but correcting by end exercise S_aO_2 did not improve either relationship.
CONCLUSION: Across a range of highly trained athletes at moderate altitude, correcting tHb by S_aO_2 at VO_2peak does not appear to explain additional variability in VO_2peak, despite large variability in the levels of observed desaturation.

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Cardiopulmonary Responses, Brain And Muscle Oxygenation During Incremental Exercise On Acute Hypoxia And Hyperoxia

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(No relationships reported)

Changes in inspired oxygen concentration will be affect the peak oxygen uptake compared with normoxia. These underlying mechanism are not fully understood, but peripheral and central mechanisms have been proposed.
PURPOSE: Our study focuses on the effect of acute moderate hypoxia and hyperoxia on cardiopulmonary responses, brain and muscle oxygenation during exercise.
METHODS: Seven healthy male subjects performed on incremental maximal exercise test under normoxia (Norm: 20.9 FIO_2), hypoxia (Hypo: 14.5% FIO_2) and hyperoxia (Hyper: 28.5% FIO_2) conditions. We measured cardiopulmonary measurements (VE, VO_2, HR and Q) and blood gas (PO_2 and PCO_2) on incremental exercise. Near-infrared spectroscopy (NIRS) was also used to monitor concentration (μM) changes of oxy- and deoxyhemoglobin (Δ[O2Hb], Δ[HHb]) in left frontal cortex region of the forehead and ipsilateral vastus lateralis muscle. Changes in total Hb and StO_2 were calculated and used as index of change in regional blood volume. Repeated-measures ANOVA were performed across treatments.
RESULTS: $VO_{2\ peak}$ decreased in Hypo (38.5±3.1 ml/kg/min, p<0.05) and no difference in Hyper (42.6±3.4 ml/kg/min) compered with Norm (42.2±3.9 ml/kg/min). But blood PO_2 at rest and moderate exercise was low in Hypo (57.7±3.1 and 52.2±5.4 mmHg, p<0.05) and high in Hyper (98.6±8.8 and 105.3±9.3 mmHg, p<0.05) compared with Norm (79.3±12.6 and 84.3±4.7 mmHg). Muscle oxygenation dropped progressively during Hypo, and also changes in muscle oxygenation during Hyper were similar to Norm. Interestingly, Brain oxygenation (Δ[O2Hb]) was slightly increased and deoxygenation (Δ[HHb]) was increased during exercise under each three conditions, respectively. Furthermore, changes brain and muscle oxygenation was also greater in Hypo compared with Norm and Hyper (p>0.05).
CONCLUSIONS: Acute hypoxia decrease oxygen uptake with decreased muscle oxygenation and slightly increased brain oxygenation. But it is unlikely that changes in brain and muscle oxygenation was related with oxygen uptake in hyperoxia, despite a similar difference absolute PO_2 in inspired oxygen and blood from hypoxia and/or hyperoxia to normoxia.

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Passive And Active Intermittent Hypoxic Exposure Pre-acclimatization Does Not Alter Heart Rate Variability At Altitude.

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PURPOSE: This study evaluated the impact of passive and active intermittent hypoxic (IH) exposure pre-acclimatization strategies on temporal and spectral power measures of heart rate variability (HRV) in normobaric hypoxia (NH), and natural altitude.

METHODS: Thirty participants (17 male and 13 female, aged 20-62 years), matched by sex, age and maximal aerobic capacity (VO_{2peak}), were randomly allocated to either a control, passive IH or active IH group. Experimental groups completed 10 x 2-h, passive (PIH) or active (AIH), normobaric IH exposures (F_IO_2 = 0.124, ~4,011 m) over the 14-day intervention period (weekends excluded). The control group received no IH exposure. During the intervention period, participants completed 20 minutes daily running training, at an individualised intensity equivalent to 80% heart rate reserve (HRR). Training workload was determined by regressing HR and running speed data from individual VO_{2peak} tests in normal ambient conditions (control and PIH groups) or NH (AIH group, F_IO_2 = 0.124). AIH participants completed the exercise training sessions under supervision, during scheduled IH exposure sessions, while control and PIH groups completed training unsupervised in normal ambient conditions. Within 48 hours of completing pre-acclimatization, participants travelled by air from the UK to Nepal, a journey time of approximately 36 hours. Participants then trekked from 2800 m to 5300 m over 14 days. Temporal (RR, SDNN, RMSSD) and spectral power measures (LFnu, HFnu and LFHF ratio) of HRV were recorded, at rest with spontaneous breathing, in normal ambient conditions (F_IO_2 = 0.209), NH (F_IO_2 = 0.124, ~4011 m) and in hypobaric hypoxia (HH) at 4356 m and 5350 m, during ascent.
RESULTS: Two-way ANOVA (group x condition) with repeated measures revealed neither significant interactions (*P*>0.05), nor between-group (*P*>0.05) nor within-group (*P*>0.05) differences for temporal or power spectral HRV measures between baseline, pre-IH and post-IH. No significant interactions, between-group or within-group changes were noted between post-IH, 4300 m and 5300 m (*P*>0.05) natural altitude.
CONCLUSION: Pre-acclimatization using active and passive intermittent hypoxic exposure did not significantly alter heart rate variability responses during ascent to very high altitude.

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Hematological Response to Uncontrolled Use of Altitude Training by Elite Distance Runners

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Elite endurance athletes typically use “live high - train low” altitude training to enhance sea level performance. Perhaps the most commonly utilized and expected experimental control in altitude training research concerns iron stores and supplementation. Whether elite athletes and coaches independently follow evidence-based best-practice principles regarding iron status and training at altitude, outside of a controlled research setting, is unknown.
PURPOSE: To examine logistical decisions elite U.S. distance runners make regarding altitude training and the hematological outcomes that result from those decisions.
METHODS: Elite U.S. distance runners (n = 58) completed altitude training (living elevation = 2,000 - 2,600m) at their own cost and volition. Total hemoglobin (tHb) mass was measured using CO rebreathing upon arrival and departure from altitude. Questionnaires asked athletes to self-report pre-altitude serum ferritin values, if iron was taken (pill or liquid) at altitude, and workout specifics.
RESULTS: Of the 40 athletes who knew their serum ferritin level at the start of the camp, those with ferritin < 50 $ng \cdot ml^{-1}$ (n = 11) demonstrated a ΔtHb of 0.6 ± 2.0% (ns) and those with ferritin > 50 $ng \cdot ml^{-1}$ (n = 29) significantly increased tHb by 3.7 ± 3.0%. Of those with ferritin levels > 50 $ng \cdot ml^{-1}$, athletes who lived at altitude < 23 days (n = 9) showed a ΔtHb mass of 1.3 ± 1.7% (ns) and those who lived at altitude for > 27 days (n = 20) significantly increased tHb mass by 3.8 ± 2.6%. Of the total cohort, 49 athletes answered questions regarding iron supplementation. Those who supplemented iron in liquid form (n = 27) significantly increased tHb mass by 4.2 ± 3.4%. Those who did not supplement iron (n = 3) or supplemented in pill form (n = 19) showed a ΔtHb mass of 1.5 ± 0.5% (ns) and 1.4 ± 2.7% (ns). Athletes who answered questions regarding training (n = 47) reported completing 8.5 ± 2.5 “higher intensity workouts,” and 3.6 ± 1.1 of those workouts were done at <1,500m. Only 4 of the 47 athletes completed *all* higher intensity sessions at <1,500m.
CONCLUSION: A substantial number of elite U.S. distance runners do not follow what would be considered evidence-based best-practice principles regarding altitude training. Coaches, sport scientists, and clinicians would be prudent to strongly advocate for athletes to follow these principles.

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No Sex Differences in the Cardiac Response to Acute Normobaric Hypoxia

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Acute hypoxia reduces arterial oxygen content, thereby increasing cardiac work to maintain oxygen delivery. Hypoxia may be accompanied by impairments in cardiac function which may be subject to sex differences, although this remains inadequately described.
PURPOSE: Explore sex differences in the cardiac response to acute hypoxia.
METHODS: Thirty healthy participants (15 men, 22±4 yrs, BMI 25.3±3.0 kg/m^2; 15 women, 20±3 yrs, BMI 22.6±1.2 kg/m^2) underwent echocardiographic measures with simultaneous 1-Lead electrocardiogram-gating following ~1.5 hour sham condition (20.0% O_2) and normobaric hypoxic (12.5% O_2) exposure on two separate days, in a randomized order. Systolic function (M-mode, tissue Doppler imaging [TDI]) and diastolic function (mitral filling velocities, TDI) were assessed in triplicate. Systolic function was assessed using fractional shortening (FS), ejection fraction (EF) from 2D Teicholz M-mode (parasternal short-axis), and S-wave velocity from tissue Doppler Imaging (TDI, apical 4-chamber). Diastolic function was assessed using ratios between early (E) and late (A) filling waves assessed from Doppler (E, A) and TDI (E’, A’).
RESULTS: EF, FS, S-velocity (septal) were greater in hypoxia vs sham ($p<0.05$). Markers of diastolic function (E:A, E’:A’) were lower in hypoxia vs sham ($p<0.05$). Men had higher EF vs women across conditions ($p<0.05$). No significant condition by sex interactions were noted.