

SWACSM Abstract

Repeated, Short Cold-Water Immersions are Sufficient to Habituate to the Cold, but Do Not Lead to Adaptations During Exercise in Normobaric Hypoxia

GEOFFREY DORSETT, TREVOR GILLUM, FACSM

Department of Kinesiology; California Baptist University; Riverside, CA

Category: Undergraduate

Advisor / Mentor: Gillum, Trevor (tgillum@calbaptist.edu)

ABSTRACT

Cold and hypoxia naturally exist together, yet it is unknown if habituation to the cold can lead to improved exercise in hypoxia. **PURPOSE:** To assess the effects of repeated cold-water immersions (CWI) on pulmonary, metabolic, and sympathoadrenal responses to graded exercise in hypoxia. **METHODS:** 16 (2 female) participants (age: 21.2 ± 1.3 years; body fat: 12.3 ± 7.7 %; body surface area 1.87 ± 0.16 m², VO_{2peak} : 48.7 ± 7.9 mL/kg/min) underwent 6 CWI in $12.0 \pm 1.2^\circ$ C. Each CWI was 5 minutes, twice daily, separated by ≥ 4 hours, for 3 consecutive days, during which metabolic data were collected. The day before CWIs began, and the day after they ended, participants ran in normobaric hypoxia ($F_{IO_2} = 0.135$) for 4 minutes at: 25%, 40%, 60%, and 75% of sea level VO_{2peak} . **RESULTS:** CWI had no change in VO_2 ($p > 0.05$), but reduced the V_E (CWI #1 – 27.1 ± 17.8 vs CWI #6 – 19.9 ± 12.1 L/min) ($p < 0.05$), V_T (CWI #1 – 1.3 ± 0.4 vs CWI #6 – 1.1 ± 0.4 L) ($p < 0.05$), and $V_E:VO_2$ (CWI #1 – 53.5 ± 24.1 vs CWI #6 – 41.6 ± 20.5) ($p < 0.05$) during subsequent CWI. Further, post exercise plasma epinephrine was lower after CWI compared to before (73.4 ± 34.6 , 103.3 ± 43.1 pg/ml) ($p < 0.05$), with no change in pre exercising values (75.4 ± 30.7 , 72.5 ± 25.9 pg/ml). However, there was no change in pulmonary (V_E , V_T , V_E/VO_2) or metabolic (VO_2 , SmO_2 , SpO_2) variables across all workloads during hypoxic exercise pre-CWI compared to post-CWI. **CONCLUSION:** CWI habituated participants to cold water, but this did not lead to adaptations during exercise in normobaric hypoxia.

INTRODUCTION - Exercise responses to different types of extreme environmental conditions have been of interest for many years. The effects of acclimation to one stressor on another, termed cross-adaptation, has received significantly less attention. Of particular interest is the interaction of cold and hypoxia adaptations, as the two tend to accompany each other naturally. Adaptations to cold can be achieved through short, repeated cold water immersions (CWI), and serve to reduce ventilation (V_E), tidal volume (V_T), heart rate (HR), and catecholamines [1-3].

Given that the classical response to hypoxia include increased ventilation, energy expenditure, and preferential use of glycogen [4], the pulmonary and sympathetic adaptations elicited by cold exposure may prove beneficially upon acute exposure to hypoxia. Indeed, Lunt *et al* [3] demonstrated a reduced HR, VO_2 , RER, catecholamines, and V_E during exercise in hypoxia after six CWI over three days. Currently, these are the only data to examine exercise in hypoxia after cold habituation. In that study, a brief, low intensity exercise (100 W, 10 min) was observed, and thus further work is needed to better understand this phenomenon across a wider range of exercise intensities and participant characteristics.

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Therefore, the purpose of our study was to assess the pulmonary, metabolic, and sympathoadrenal response to acute, graded exercise in hypoxia after CWI. Our hypothesis was twofold: 1) that CWI would reduce the pulmonary response to the cold, and 2) CWI adaptations would lower pulmonary, metabolic, and epinephrine responses to acute exercise in hypoxia.

METHODS AND MATERIALS - 16 (2 female) recreationally active participants (age: 21.2 ± 1.3 years; body fat: 12.3 ± 7.7 %, VO_{2peak} : 48.7 ± 7.9 mL/kg/min) completed six CWI over three consecutive days (Tuesday, Wednesday, and Thursday). Immersions were performed twice daily, for five minutes in $12.0 \pm 1.2^\circ$ C water, with frequent stirring, and separated by ≥ 4 hours. Exercise in normobaric hypoxia ($F_{I}O_2 = 0.135$, simulating ~3500 meters) was performed one

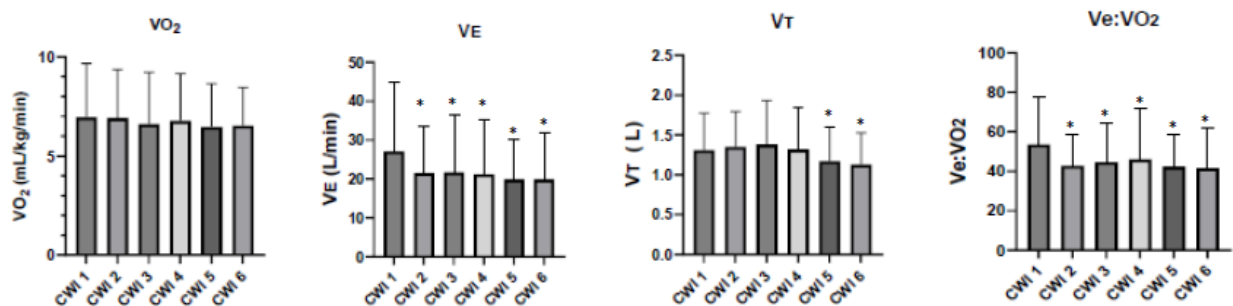
day prior- (Monday), and one day after (Friday) the CWI. The hypoxic exercise bouts took place during the same time of day to avoid diurnal variation. Exercise consisted of treadmill running at workloads of 25%, 40%, 60%, and 75% of sea level VO_{2peak} in normobaric hypoxia ($F_{iO_2} = 0.135$). Participants exercised for four minutes/stage. Data from the last 90 seconds of each workload was manually screened to ensure VO_2 did not change by ≥ 250 ml/min and HR did not change by ≥ 5 beats/min. Blood was drawn immediately before and after exercise in hypoxia.

STATISTICAL ANALYSIS - During exercise in hypoxia, metabolic and pulmonary data, along with SmO_2 and SpO_2 were analyzed with a two factor (time x intensity) repeated measures ANOVA. The last 90 seconds of data provided by the metabolic cart were averaged and used for each workload. RPE was analyzed with a two-tailed Wilcoxon matched-pairs signed-rank test. Metabolic data collected during cold water immersion were analyzed using a one way repeated measures ANOVA to determine habituation. Where a main effect was observed, Tukey's post hoc test was performed to isolate differences. All data are reported as mean \pm standard deviation (SD). A power analysis [3] suggested 12 participants were needed.

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RESULTS - Cold Water Immersions - Repeated CWI resulted in habituation to the cold. Specifically, V_E ($p < 0.00$), V_T ($p < 0.00$), and $V_E:VO_2$ ($p < 0.00$) were lower in subsequent CWI. VO_2 was unchanged during the CWI ($p = 0.08$) (Fig 1).

Fig 1. The metabolic and pulmonary response to successive CWI.



* $p < 0.05$ compared to CWI 1.

Exercise in Hypoxia - Despite the habituation to the cold, CWI had no effect over a wide range of exercise intensities on $\dot{V}O_2$ ($p=0.53$; interaction $p=0.67$), V_E ($p=0.67$; interaction $p=0.86$), V_T ($p=0.16$; interaction $p=0.54$), or $V_E:\dot{V}O_2$ ($p=0.75$; interaction $p=0.37$) during exercise in normobaric hypoxia (Fig 2). Further, heart rate ($p=0.07$; interaction $p=0.87$), SpO_2 ($p=0.73$; interaction $p=0.73$), and SmO_2 ($p=0.19$; interaction $p=0.21$) were similar between conditions (Table 1). Finally, RPE at 25% ($p=0.27$), 40% ($p=0.51$), 60% ($p=0.98$), and 75% ($p=0.45$) were not different between conditions (Table 1).

Fig 2. The metabolic and pulmonary response to exercise in hypoxia.

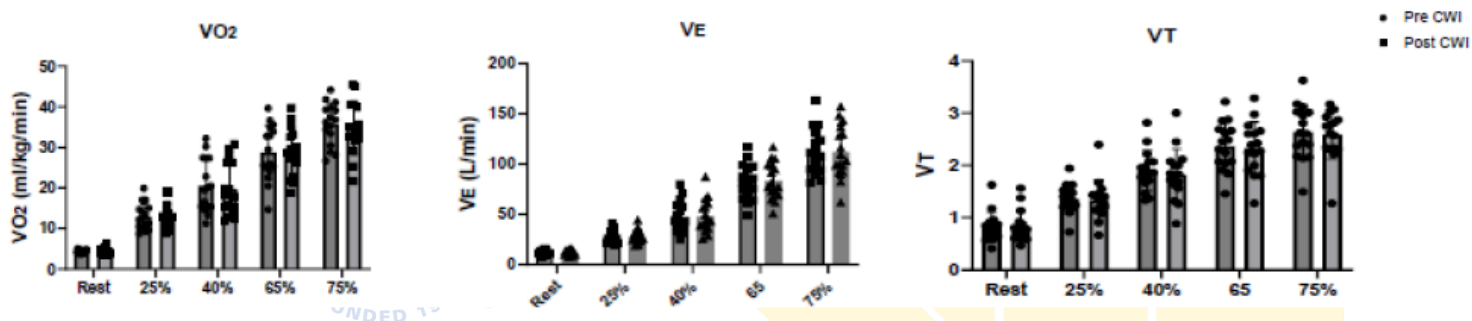


Table 1. Hypoxic exercise before and after CWI.

<i>Exercise in hypoxia prior to CWI</i>					<i>Exercise in hypoxia post CWI</i>				
	25%	40%	65%	75%		25%	40%	65%	75%
HR (bpm)	108±19	137±20	161±13	171±14	HR (bpm)	108±17	144±14	167±12	178±6
RPE	7.0±1.3	9.6±2.5	12.8±3.3	16.1±4.3	RPE	6.6±0.8	9.3±2.1	13.0±3.4	15.9±4.3
SmO₂ (%)	47.9±7.8	41.1±11.7	26.3±8.5	20.6±9.1	SmO₂ (%)	51.6±12.1	41.8±17.2	30.8±12.4	24.2±13.1
SpO₂ (%)	81.6±3.4	80.1±5.1	80±4.7	77.4±3.3	SpO₂ (%)	81.4±3.0	80.7±3.8	80.9±4.4	79.5±4.7

CWI did not change hematocrit (Hct) ($p=0.24$) or hemoglobin (Hb) ($p=0.44$). However, CWI lowered post exercise plasma epinephrine ($p=0.01$), despite there being no change to pre exercise concentrations ($p=0.93$) (Table 2).

Table 2. Blood markers before and after hypoxic exercise, before and after six CWI.

	Pre CWI Pre Ex	Pre CWI Post Ex	Post CWI Pre Ex	Post CWI Post Ex
Hct (%)	47.2±3.5	48.6±3.3	46.1±3.5	48.4±3.6
Hb (g/dL)	15.2±1.3	15.9±1.4	15.3±1.4	16.0±1.4
Epi (pg/ml)	75±73	105±105	72±71	73±67*

* $p<0.05$ from Pre CWI post exercise.

DISCUSSION - The main finding of the current investigation is that six CWI of five minutes each, twice daily for the three consecutive days resulted in habituation to the cold. This was evidenced by a decrease in V_E , V_T , and $V_E:VO_2$ during successive CWI. In addition, these adaptations lowered post hypoxic exercise epinephrine concentration. However, CWI did not realize any meaningful pulmonary, metabolic, or perceptual change during exercise in acute hypoxia, over a wide range of exercise intensities.

Cold Water Immersions - The current data show a clear reduction in the initial cold shock response as V_E , V_T , and $V_E:VO_2$ were reduced during successive CWI. Keatinge *et al* [5] were the first to demonstrate that repeated immersions in 15°C reduced ventilation and heart rate. This finding has been supported through the years [1, 2, 6], and yet a novel piece of the current data is the short duration of CWI realizing changes in the initial cold shock response. Indeed, we are aware of only one other group to show such a finding [1]. Thus, our data continue to support the observation that brief, repetitive CWI, that do not alter core temperature, are sufficient to alter the initial cold shock response.

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A reduction in the metabolic rate in response to the cold is often associated with a larger reduction in core temperature [1]. These data are congruent with the current data which demonstrated similar VO_2 values across CWI. Mean VO_2 was reduced ~8% in the current study, which is similar to the reduction in VO_2 observed after five CWI of five min in Tipton *et al* [1], but neither reduction crossed the *a priori* line of statistical significance. Taken together, our data demonstrate that CWI were sufficient to reduce the initial cold shock response, however, VO_2 was similar throughout the CWI and the metabolic response was unaltered. Thus, we accept our first hypothesis that brief bouts of CWI is capable of reducing V_E and the initial cold shock response.

Hypoxic Exercise – Hypoxic exposure increases ventilation, energy expenditure, and preferential use of glycogen [4], thus, ventilatory and sympathetic adaptations elicited by CWI may prove beneficial in acute hypoxic exposure. The current study observed a reduction in post exercise epinephrine concentration after CWI. A reduction in sympathetic activity after cold exposure has been observed in the animal literature [7] and in male open water swimmers [8]. However, the metabolic and pulmonary implications of reduced epinephrine remain in question as the current data demonstrated no difference in any variable, despite a lowering of circulating epinephrine. Thus, we reject our second hypothesis that adaptations to CWI would lower $\dot{V}O_2$, V_E , and perceptual responses to exercise in hypoxia.

Despite an identical CWI protocol, our data are largely in contrast to Lunt *et al.* While our data showed a reduced post hypoxic exercise epinephrine concentration after CWI, no change was observed in pulmonary, metabolic, perceptual, or hematological variables after CWI. In addition, we report no changes after CWI at rest in hypoxia, and at workloads much higher than analyzed in Lunt *et al.* This could be due to differences in participant characteristics ($\dot{V}O_{2peak}$, % body fat) as both effect heat storage and dissipation [9]. Our data agree with animal work demonstrating no benefit of cold exposure upon subsequent hypoxic exposure [10-12].

In conclusion, six CWI of five minutes, twice daily for the three consecutive days resulted in habituation to the cold. This was evidenced by a decrease in V_E , V_T , and $V_E:\dot{V}O_2$ during successive CWI. In addition, these adaptations lowered post hypoxic exercise epinephrine concentration. However, CWI did not realize any meaningful pulmonary, metabolic, or perceptual changes during exercise in acute hypoxia, over a wide range of exercise intensities.

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