Physiological responses to treadmill running with body weight support in hypoxia compared to normoxia

Abstract

Context. Anecdotal reports suggest elite sports clubs combine lower body positive pressure (LBPP) rehabilitation with a hypoxic stimulus in order to maintain or increase physiological and metabolic strain, which are reduced during LBPP. However the effects of hypoxia on cardiovascular and metabolic response during LBPP rehabilitation is unknown.

Objective. Evaluate the use of normobaric hypoxia as a means to increase physiological strain during body weight supported (BWS) running.

Design. Cross over study.

Setting. Controlled laboratory.

Participants. Seven familiarized males (mean ± SD; age, 20 ± 1 years; height, 1.77 ± 0.05 meters; mass, 69.4 ± 5.1 kg; haemoglobin 15.2 ± 0.8 $g \cdot dL^{-1}$). completed a normoxic and hypoxic (F_IO_2 = 0.14) trial, during which they ran at $8km \cdot hr^{-1}$ on an Alter-G^{TM} treadmill with 0, 30 and 60% BWS in a randomised order for 10 minutes interspersed with 5 minutes of recovery.

Main outcome measures. Arterial oxygen saturation, heart rate, oxygen delivery and measurments of metabolic strain via indirect calorimetry.

Results Hypoxic exercise reduced SpO_2 and elevated heart rate at each level of BWS compared to normoxia. However, the reduction in SpO_2 was attenuated at 60% BWS compared to 0% and 30% and consequently oxygen delivery was better maintained at 60% BWS.

Conclusion. Hypoxia is a practically useful means of increasing physiological strain during BWS rehabilitation. In light of the maintenance of SpO_2 and oxygen delivery at increasing levels of BWS, fixed haemoglobin saturations rather than a fixed altitude is recommended in order to maintain an aerobic stimulus.

Introduction

The use of lower body positive pressure (LBPP), or body weight supported (BWS) exercise has become increasingly popular in rehabilitation and injury prevention settings [1-4]. A key benefit of applying LBPP is that cardiovascular fitness and lower limb function can be maintained, while the mechanical strain and subjective pain imposed by ground reaction forces when walking or running without BWS is reduced [4,5]. In athletic groups LBPP permits the use of natural gait patterns during rehabilitation and can also be used for over speed running training [6]. Whereas in aging, obese and stroke patients LBPP can facilitate an aerobic stimuli for individuals that would otherwise have difficulty exercising [7]. However with LBPP the metabolic cost of running is reduced, with increasing BWS leading to greater reductions in both $\dot{V}O_2$ and exercising heart rates [6,8-10]. For example, walking with 75% BWS reduced energy expenditure by 45% compared with a non-supported walk [8]. However the metabolic cost of running at increasing levels of BWS can be offset by increasing treadmill speed [6,8,10]. Indeed both submaximal and maximal $\dot{V}O_2$ achieved with no BWS can be matched by increasing speeds [9]. A conversion table is now available to help inform decision-making regarding matching the metabolic cost of different speeds (6.4 – 16.1km·hr^{-1}) and different levels of BWS of 50, 60, 70, 80 and 90% under normoxic conditions [10].

Increasing treadmill speed in order to increase oxygen consumption and thus the cardiorespiratory response at a given level of BWS may not be appropriate in all

rehabilitation scenarios. An alternative approach to elevate physiological stress at a given level of BWS is to reduce oxygen availability, thus reducing arterial haemoglobin oxygen saturation (SpO_2) and disrupting homeostasis [11]. Anecdotal reports indicate that this method is now used with a view to increasing the aerobic training stimulus when exercising with BWS in rehabilitation settings (e.g. with professional football players). The rationale for this approach is that due to the known reductions in aerobic power associated with exercise at altitude [12-14], a given intensity of work represents a greater relative intensity (higher percentage of $\dot{V}O_2max$) when exercise is performed under hypoxic conditions. Therefore greater physiological and metabolic adjustments are necessary when performing similar tasks at altitude compared to sea level [11]. Key physiological adjustments that attempt to reverse the effects of reduced oxygen availability include hyperventilation and a heart rate mediated increase in cardiac output in order to maintain oxygen delivery to the tissues [11]. Subsequent alterations in substrate utilization due to reduced oxygen availability are also apparent [15-17]. The cardiorespiratory adjustments caused by the imposition of a hypoxic stress therefore represent a potentially useful alternative stimulus, rather than increasing speed, for increasing cardiovascular strain during BWS exercise.

To the best of the authors' knowledge there are no published data available to inform the practice of exposing individuals to reduced amounts of oxygen during BWS. It would of interest to determine whether the increased physiological strain imparted by hypoxia during BWS can match the strain observed during 'normal' non-supported running. Therefore the aim of this study was to compare the physiological response to running at 8 $km \cdot hr^{-1}$ at different levels of BWS whilst breathing hypoxia compared to normoxia. It was hypothesized that cardiorespiratory strain would be increased at each level of BWS in hypoxia compared to similar BWS conditions conducted in normoxia.

Methods

After receiving local ethical approval 9 familiarized healthy males were enrolled on the study. Two participants dropped out after the first experimental visit (reasons not related to the study), therefore data are included for 7 particpants that completed all study visits (mean ± SD; age, 20 ± 1 years; height, 1.77 ± 0.05 meters; mass, 69.4 ± 5.1 kg; haemoglobin 15.2 ± 0.8 $g \cdot dL^{-1}$) regularly conducting at least two structured exercise sessions per week, provided both written and verbal consent before taking part in the study, which was approved by Coventry University Ethics Committee and conducted in accordance with Declaration of Helsinki (1996).

Experimental design

Participants attended the laboratory on 2 separate occasions separated by 7 days and ran on an Alter-G™ treadmill (AlterG® Anti-Gravity Treadmill P200, Freemontm USA) at $8km \cdot hr^{-1}$ at each of 0, 30 and 60% BWS for 10 minutes, whilst breathing normoxia (F_IO_2 = 0.209) or hypoxia (F_IO_2 = 0.142; equivalent to ~3000 m altitude) applied in a counterbalanced, single blinded, cross-over design. Each 10-minute period was interspersed with 5 minutes of standing recovery with no BWS. Hypoxia was generated via a hypoxicator unit (Hypoxico HYP123 Hypoxicator, New York, USA) that was used to fill a 1000L reservoir. Ethylene clear vinyl tubing was used to connect the inspiratory side of the valve to the 1000L Douglas bag. Participants inspired via a mouthpiece attached to a two-way non-rebreathable valve (Harvard Ltd, Eldenbridge, UK). During the normoxic condition the reservoir was filled with ambient air.

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Physiological Measurements

A schematic of the experimental design is shown in Figure 1. Upon arrival to the laboratory subjects were instrumented with a heart rate monitor (Polar RS400, Polar Electro Inc, USA) and finger clip pulse oximeter (WristOx, Nonin Medical Inc, Minnesota, USA) for continual measurements of heart rate (HR) and arterial haemoglobin oxygen saturation (SpO_2) throughout exercise. After a 15 minute standing stabilisation period, a fingertip capillary whole blood sample was obtained for the determination of resting blood lactate (BLa; Biosen C-Line analyser, EKF Diagnostics, Germany).

Indirect calorimetry was used to calculate $\dot{V}E_{BTPS}$, $\dot{V}O_2$, $\dot{V}CO_2$, and RER during exercise. Expired gas was collected into 200 L Douglas bags for 60 seconds during minutes 9 - 10 of every stage. Gas was subsequently analyzed to determine CO_2 and O_2 content, using a calibrated Servomex infrared and paramagnetic gas analyzer respectively (model 1400, Servomex, Crowthorne, U.K.), and gas volume, via a Harvard Dry Gas meter (Cranlea and Company, Birmingham, U.K.). At the end of each exercise period the rating of overall and peripheral (legs) perceived exertion (RPE; 6 to 20 scale) was requested and a finger-tip capillary blood sample was collected for determination of blood lactate. The level of BWS was adjusted at the end of the 5-minute recovery period and the process repeated for the remaining levels of BWS. The order of BWS was randomised for each participant.

Data analysis

All data were checked for normal distribution prior to analysis. Repeated measures were checked for sphericity with Mauchly's sphericity test, and p-values corrected using the Huynh-Feldt method if sphericity was violated. To determine the effects of hypoxia and BWS on cardiorespiratory, blood lactate and perceptual responses, a 2 (normoxia or hypoxia) by 3

(0, 30, 60% BWS) repeated measures linear model was used, with fixed effects for condition and BWS. When interactions or main effects were significant, Tukey post Hoc tests were used to identify differences between the relevant mean data. The delta change in variables between normoxic and hypoxic conditions were assessed using repeated measures ANOVA, with main effects explored as previously stated. Data are presented as means ± SD in text, and mean and individual data in Figures. Effect sizes (partial eta squared; (np^2; small = 0.01, medium = 0.06, large = 0.13) were calculated to analyse the magnitude and trends with data.

Results

No trial x BWS interaction effect was observed for arterial haemoglobin oxygen saturation (SpO_2; f = 1.33, p = 0.28; Figure 2A). SpO_2 was lower in hypoxia compared to normoxia (main effect for trial f = 231, p < 0.0001, η_p^2 = 0.96), and this reduction was similar across levels of BWS (main effect for BWS, f = 0.71, p = 0.49). SpO_2 was maintained throughout normoxia for 0% BWS (98 ± 1%), 30% BWS (97 ± 2%) and 60% BWS (97 ± 1%). In hypoxic conditions SpO_2 was reduced to 80 ± 2%, 80 ± 2% and 83 ± 2% at 0, 30 and 60% BWS respectively (p < 0.001).

Hypoxic exercise led to an 18%, 16% and 14% reduction in SpO_2 at 0, 30 and 60% BWS respectively compared normoxia (f = 9.64, p = 0.0005). Post hoc analysis shows that the absolute change in SpO_2 observed at 60% BWS was not as great as observed at 0% (p < 0.01) or 30% (p < 0.05; Figure 2B).

No interaction effect for heart rate was seen between trial and BWS (f = 0.01, p = 0.98; Figure 2C), and the absolute change in HR during the hypoxic condition in relation to the normoxic condition was similar across 0, 30 and 60% BWS (Figure 2D). Heart rate declined with BWS (main effect for BWS, f = 42.15, p < 0.0001, η_p^2 = 0.63) and was higher in

hypoxia compared to normoxia (main effect for trial $f = 12.23$, $p < 0.0001$, $\eta_p^2 = 0.15$). Specifically, heart rate was 12 bts·min^{-1} higher in hypoxia at 0% BWS (Normoxia 136 ± 9 bts·min^{-1}; Hypoxia 148 ± 17 bts·min^{-1}), 11 bts·min^{-1} higher at 30% BWS (N 114 ± 4bts·min^{-1}; Hypoxia 125 ± 11 bts·min^{-1}) and 11 bts·min^{-1} higher at 60% BWS (Normoxia 99 ± 6 bts·min^{-1}; Hypoxia 110 ± 12 bts·min^{-1}).

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Oxygen delivery ($\dot{D}O_2$), expressed using SpO_2 x HR as an estimation, decreased with increasing levels of BWS (main effect for BWS, $f = 51.94$, $p < 0.001$), and was decreased at each level during hypoxic running compared to normoxia (main effect for trial, $f = 14.97$, $p < 0.001$; Figure 3A). One-way ANOVA returned a main effect for the absolute change in $\dot{D}O_2$ between normoxic and hypoxic conditions ($f = 4.18$, $p = 0.03$), indicating $\dot{D}O_2$ was elevated at 60% BWS compared to 0% BWS when under hypoxic conditions (Figure 3B).

Please insert figure 3 near here

All respiratory data are displayed in Table 1. No interaction effects were observed for any respiratory variables presented. Oxygen consumption declined with BWS (main effect for BWS $f = 67.3$, $p < 0.001$, $\eta_p^2 = 0.79$), and was lower in hypoxia compared to normoxia (main effect for trial, $f = 4.75$, $p = 0.031$, $\eta_p^2 = 0.12$). Respiratory exchange ratio was higher during hypoxia than normoxia (main effect for trial, $f = 36.3$, $p < 0.001$), and no main effects were observed for BWS ($f = 0.67$, $p = 0.51$). No main effects for trial or BWS were noted for minute ventilation or carbon dioxide production. Oxygen pulse ($\dot{V}O_2$ per heart beat) was lower in hypoxic conditions (main effect for trial $f = 18.4$, $p < 0.001$), and also decreased with increasing BWS (main effect for BWS, $f = 21.7$, $p < 0.001$; Figure 3C). Specifically, O_2 pulse decreased at both 30 and 60% BWS compared to 0% ($p < 0.05$; Figure 3D). Blood lactate was higher throughout hypoxia compared to normoxia (main effect for trial $f = 5.63$, p

= 0.02), and was lower during 60% BWS compared to both 30 and 0% (main effect for BWS, f = 4.98, p = 0.01, η_p^2 = 0.17).

Please insert table 1 near here.

Perceptual data are displayed in Table 2. Overall and peripheral RPE did not vary between the environmental conditions (main effect for trial, f = 1.13, p = 0.33). In both normoxic and hypoxic conditions overall and local RPE decreased with increasing levels of body weight support (main for BWS, f = 8.30, p = 0.005, η_p^2 = 0.58).

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Discussion

The aim of the present study was to determine whether the addition of a hypoxic stimulus to BWS running could restore the cardiorespiratory stress to levels similar to running without BWS. It was hypothesised that hypoxia would increase the cardiovascular stress experienced at all levels of BWS studied. Our data show that the addition of moderate hypoxia (3000m asl) during exercise with BWS increased both cardiorespiratory stress, evidenced by the increased exercise heart rate, reduced O_2 pulse, and elevated metabolic stress indicated by the greater respiratory exchange ratio and blood lactate levels. The novel finding in the present study is that as the level of BWS increases, the magnitude change in SpO_2 is reduced, which is indicative of maintained O_2 delivery. This is an important consideration if hypoxia is to be used with greater level of BWS, as its effectiveness at increasing physiological strain appears reduced when BWS is greater than 60%. We suggest that manipulation of the inspired PO_2 to target specific Hb saturations, rather than the use of a fixed altitude, could be used when higher levels of BWS (> 60%) are incorportated into rehabilitation. By doing so practioners could further reduce the use of hypoxia during BSW running as a means to increase cardiovascular strain while also reducting mechanical stress.

The observed reductions in cardiovascular and metabolic strain are in accordance with previous research using similar levels of BWS. By adding 30% and 60% of BWS under normoxic conditions heart rate was reduced by approximately 22 $bts \cdot min^{-1}$ and 37 $bts \cdot min^{-1}$ respectively, relative to 0% BWS, which is similar to values observed by others [18]. Aligned to this oxygen consumption was reduced by approximately 27% with 30% BWS, and 48% with 60% BWS in normoxic conditions. This reduction in $\dot{V}O_2$ with increasing BWS was maintained in hypoxia (30% BWS = 26% decrease in $\dot{V}O_2$; 60% BWS = 50% decrease in $\dot{V}O_2$) relative to 0% BWS. However in absolute terms oxygen consumption tended to be 8-12% lower at all BWS levels studied when work was performed in hypoxic conditions compared to the matched normoxic conditions. Moderate exercise in hypoxia is characterised by slower O_2 uptake kinetics in the early phase of exercise, though ultimatey leads to the same steady state [19]. The slower O_2 uptake kinetics induce a greater O_2 deficit early on in the exercise bout, and implies a greater reliance on anaerobic glycolysis and lactate provision [19]. This is transient, and occurs during the adjustment to a new steady state, alongside a rise in circulating blood lactate, which is inversely related to inspired PO_2 content [20 11]. Thus, if maintaining or minimising the loss of fitness is a goal during BWS rehabilitation, the added cardiovascular strain imparted by hypoxia is desirable.

The use of LBPP and BWS has gained acceptance in rehabilitation settings in populations as diverse at elite sport, recovery from stroke [21] and recovery from knee surgery [22] and those suffering with knee osteoarthritis [4]. For example, Takacs et al (2013) investigated the effects of walking at 5% increments of LBPP (0 – 30% BWS) on subjective knee pain as assessed via visual analogue scale (VAS) scores. These authors found that knee pain during walking rehabilitation was minimised with approximately 12% BWS while walking at 5.0 $km \cdot hr^{-1}$. Although physiological data were not reported, it is likely that participants would have experienced a reduced walking heart rate in the region of < 3 $beats \cdot min^{-1}$ [18]. At low walking

speeds (~5 km.hr^{-1}), the effect of increasing BWS on CV strain is minimal, with Linney et al., (2014) observing a 7 beats·min^{-1} reduction in HR up to 60% BWS. When utilising a slow running gait, the decline in HR was shown to be greater at 30% (117 beats·min^{-1}) and 60% BWS (103 beats·min^{-1}) when compared to 0% BWS (137 beats·min^{-1}) [18]. Although our data show that oxygen consumption was less at all levels of hypoxia, the degree of cardiovascular strain was increased as a result of the reduced exercise SpO_2. Hypoxia induced a rise of ~12 bts·min^{-1} at all levels of body weight support studied, which though not enough to completely restore the cardiovascular strain observed during non-supported running, may be sufficient to match cardiovascular strain up to a level of 15-20% BWS [10,18]. Our perceptual data indicate that the imposition of hypoxia *per se* does not increase overall or localised subjective ratings of perceived exertion, at least at the low running speed studied. Whether increasing levels of hypoxia would negatively impact the subjective sensations of fatigue/difficulty experienced during a rehabilitation exercise bout are yet to be determined.

A common approach used to increase the level of physiological strain and training stimulus is to increase the mechanical load on the body via an increase in running speed at the higher levels of BWS. Indeed, a conversion table is now available in order to guide practitioners in their decision making [10]. The reduction in oxygen consumption at 30% BWS within the present investigation are comparable to those observed by Kline et al., (2015; our data 27%, Kline data = 24% decrease in $\dot{V}O_2$). In order to match the level of oxygen consumption experienced when running at 8km·hr^{-1} at 0% BWS an increase in speed of 48.4% was shown to be necessary [10]. As running speeds are increased with BWS, the magnitude of speed increase required to match oxygen consumption lessens. For example, running with 30% BWS support requires an increase in speed of 45%, 42% and 40% to match $\dot{V}O_2$ achieved without BWS at 9.7, 11.3 and 12.9 km.hr^{-1} [10]. However, this degree of speed increase may not be possible in all scenarios, and with all population groups. The alternative approach

utilised in the present study uses normobaric hypoxia to manipulate oxygen availability thereby allowing a constant external workload to be maintained, with the increase in physiological strain being driven by a reduced oxygen supply.

A key finding in this present study is that increasing BWS support to > 60% allows for a more adequate maintenance of oxygen delivery during conditions of hypoxic stress (Figure 3), and a more variable reduction in SpO_2. This indicates that while a fixed level of hypoxia (e.g. 3000m asl) does increase cardiovascular strain, it may not be the most efficient strategy if the aim is to match the physiological strain encountered during non-BWS running. Instead we recommend practitioners use a targeted level of haemoglobin saturation (for example 80% as observed at 0 and 30% BWS) in order to manipulate the delivery of oxygen and therefore the cardiovascular strain at greater levels of BWS. This approach would allow for personalised adjustments in rehabilitation training load, tailored to match the usual level of strain imparted by normoxic, non-supported running. To optimise the application of combined BWS and hypoxia within rehabilitative training future studies should examine the cardiorespiratory response to different levels of BWS and different degrees of hypoxia at varied speeds within specific populations. Understanding how oxygen delivery is effected by increasing both BWS and velocity would allow for the production of more specific guidelines for use in clinical and rehabilitation settings when maintaining some physiological training response may be desirable.

In conclusion, we show that breathing a gas mixture equivalent to 3000m altitude consistently increased the magnitude of cardiorespiratory stress between 0, 30 and 60% BWS while running at $8km{\cdot}hr^{-1}$ compared to normoxia. The key finding of the present investigation was the observation that increasing BWS > 60% allows greater maintenance of oxygen delivery. Therefore targeting a fixed level haemoglobin saturation via manipulation of the inspired

oxygen fraction is recommended for future studies investigating the utility of hypoxia in BWS rehabilitation settings.

List of abbreviations.

ASL; above sea level. **BTPS;** body temperature pressure saturated. **BWS;** Body weight support. **$\dot{D}O_2$;** oxygen delivery. **F_iO_2;** fraction of inspired oxygen. **HR;** heart rate. **LBPP;** Lower body positive pressure. **RER;** respiratory exchange ratio. **RPE;** Rating of perceived exertion. **SpO_2;** haemoglobin saturation. **STPD;** Standard temperature pressure dry. **$\dot{V}_E$;** minute ventilation. **$\dot{V}CO_2$;** carbon dioxide production. **$\dot{V}O_2$**; oxygen consumption.

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Table 1. Respiratory variables during running at 8km·hr^{-1} with 0, 30 and 60% body weight support (BWS) in normoxic or hypoxic conditions. Data show the mean (95% confidence interval).

BWS	**Experimental condition**	
Oxygen consumption (L·min^{-1})	**Normoxia**	**Hypoxia**
0%	1.97 (1.81 – 2.14)$^{\#}$	1.79 (1.63 – 1.95)
30%	1.44 (1.28 – 1.61)*	1.33 (1.17 – 1.39)
60%	1.03 (0.87 – 1.19)*	0.90 (0.74 – 1.06)
Carbon dioxide production (L·min^{-1})		
0%	1.21 (0.86 – 1.55)	1.30 (0.95 – 1.64)
30%	1.35 (1.01 – 1.70)	1.38 (1.04 – 1.73)
60%	1.23 (0.89 – 1.58)	1.30 (0.96 – 1.64)
Respiratory exchange ratio		
0%	0.83 (0.77 – 0.88) $^{\#}$	0.98 (0.93 – 1.04)
30%	0.86 (0.81 – 0.92) $^{\#}$	0.97 (0.91 – 1.02)
60%	0.86 (0.80 – 0.92) $^{\#}$	1.01 (0.96 – 1.07)
Minute Ventilation STPD (L·min^{-1})		
0%	31.2 (23.9 – 38.3)	33.8 (26.6 – 40.9)
30%	32.1 (24.9 – 39.2)	36.1 (28.9 – 43.2)
60%	29.9 (22.8 – 37.1)	33.1 (25.9 – 40.3)

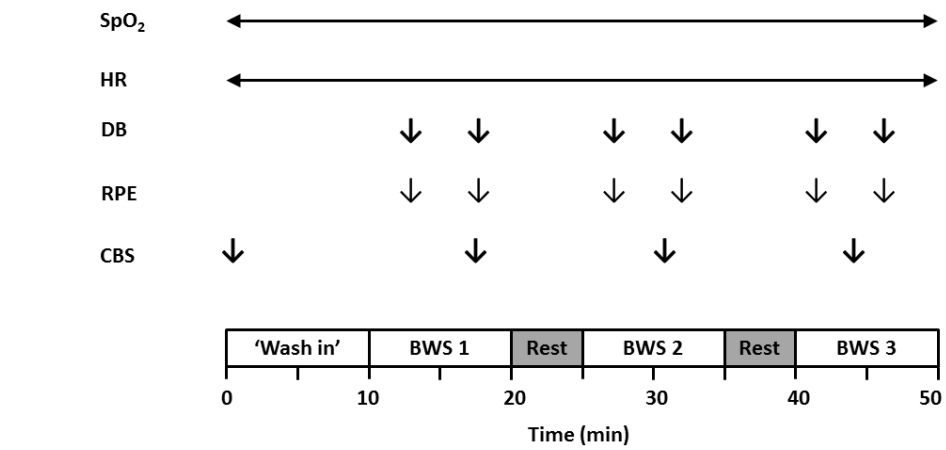
# $p < 0.01$ vs. hypoxia

* $p < 0.05$ vs. hypoxia

Table 2. Overall and peripheral (leg) perceptual responses during running at 8km.hr-[1] with 0, 30 and 60% body weight support (BWS) in normoxic or hypoxic conditions. Data show the mean (95% confidence interval).

	Body Weight Support (%)		
	0	30	60
Normoxia overall	11 (7 – 15)	10 (7 – 13)	8 (6 – 11)*
Hypoxia overall	11 (8 – 15)	10 (7 – 13)	9 (7 – 13)*
Normoxia leg	11 (8 – 15)	9 (7 – 11)*	8 (6 -11)*
Hypoxia leg	12 (7 – 16)	10 (7 – 13)*	10 (6- 13)*

* $p < 0.05$ vs 0% BWS



DB – Douglas bag

CBS- Capillary blood sample

Figure 1. Schematic of the experimental design. Participants completed 2 trial days; 1 in normoxia, and 1 in hypoxia. Each trial consisted of 3 x 10 minute exercise periods at either 0, 30 or 60% BWS interspersed with 5 minutes of rest. The order of BWS was randomized. Heart rate and SpO_2 were collected continuously throughout exercise. Douglas bag measurements (DB) and RPE were collected for 60 seconds during minutes 4-5 and 8-9 of each level of BWS. A capillary blood sample (CBS) was obtained following at the end of each exercise bout.

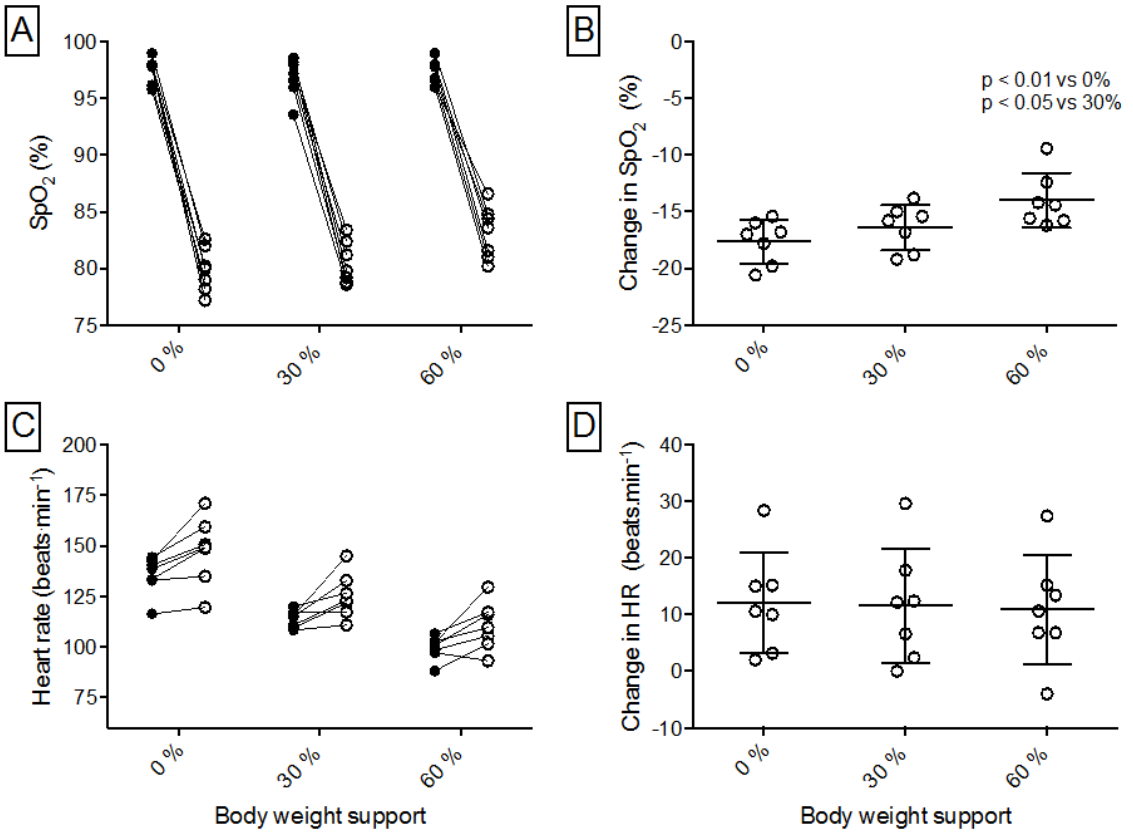


Figure 2. SpO_2 (A) was maintained during the normoxic conditions (closed symbols) and reduced during each hypoxic condition (open symbols). Line graphs present all individual responses to each condition. The absolute change in SpO_2 in hypoxia compared to normoxia (B) indicate that SpO_2 was better maintained with 60% BWS. Heart rate (C) decreased with each level of BWS, and was elevated during each hypoxic condition. The absolute change in HR in hypoxia compared to normoxia was similar at all levels of BWS (D).

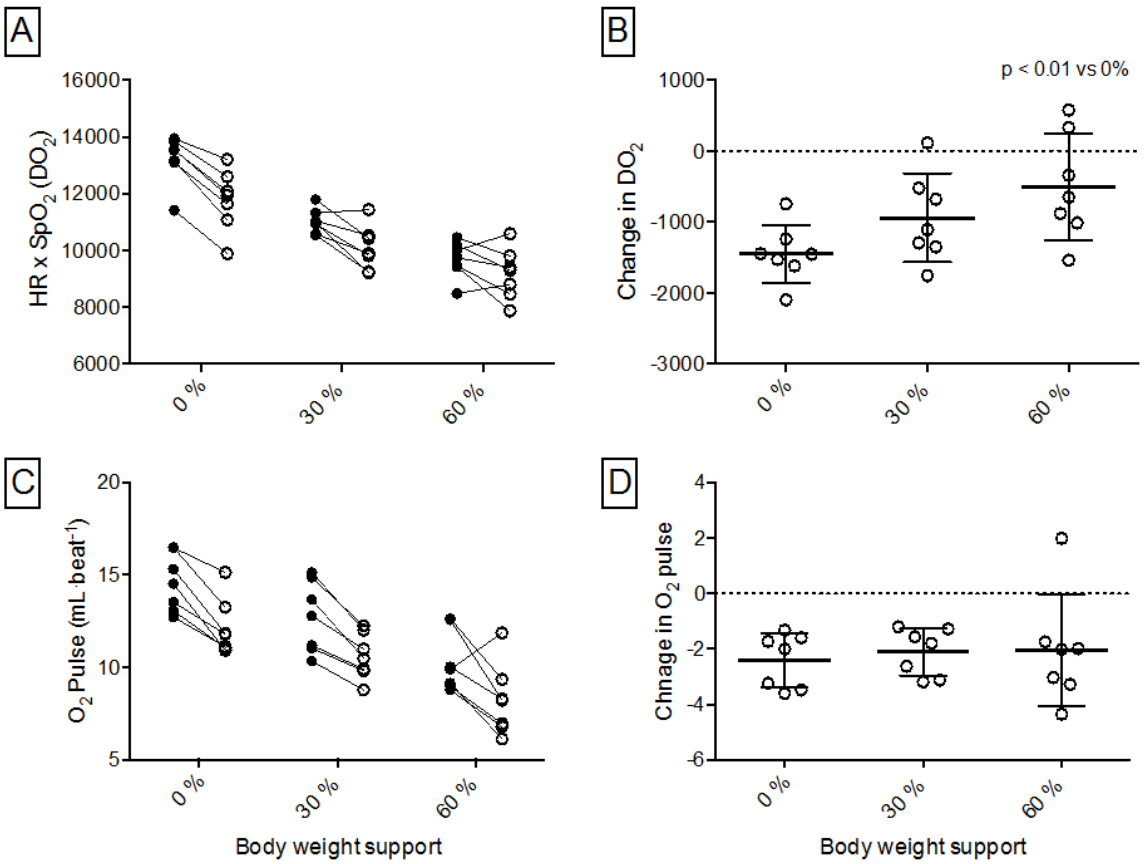


Figure 3. $\dot{D}O_2$ was reduced with increasing levels of BWS (A), and tended to be lower in the hypoxic trial (open symbols) compared to the normoxic trial (closed symbols). $\dot{D}O_2$ was maintained during the 60% BWS hypoxic condition (B). Oxygen pulse (C) decreased with increasing BWS, and was lower during hypoxia compared to normoxia. The absolute change in O2 pulse in hypoxia compared to normoxia was similar at all levels of BWS (D).