Exposure to a combination of heat and hyperoxia during cycling at submaximal intensity does not alter thermoregulatory responses

AUTHORS: Zinner C[1], Krueger M[2,3], Reed JL[4], Kohl-Bareis M[5], Holmberg H-C[6], Sperlich B[1,6]

[1] Department of Sport Science, University of Würzburg, Judenbühlweg 11, 97082 Würzburg, Germany
[2] Institute of Training Science and Sport Informatics, German Sport University Cologne, Am Sportpark Müngersdorf 6, 50933 Cologne, Germany
[3] The German Research Centre of Elite Sport, German Sport University Cologne, Am Sportpark Müngersdorf 6, 50933 Cologne, Germany
[4] Faculty of Health Sciences, University of Ottawa, ON, Canada
[5] University of Applied Sciences Koblenz, RheinAhrCampus Remagen, Germany
[6] Swedish Winter Sports Research Centre, Department of Health Sciences, Mid Sweden University, Östersund, Sweden

Corresponding author:
Christoph Zinner
Department of Sport Science, University of Würzburg, Judenbühlweg 11, 97082 Würzburg, Germany
E-mail: christoph.zinner@uni-wuerzburg.de

ABSTRACT: In this study, we tested the hypothesis that breathing hyperoxic air ($F_{in}O_2 = 0.40$) while exercising in a hot environment exerts negative effects on the total tissue level of haemoglobin concentration (tHb); core (T_{core}) and skin (T_{skin}) temperatures; muscle activity; heart rate; blood concentration of lactate; pH; partial pressure of oxygen (P_aO_2) and carbon dioxide; arterial oxygen saturation (S_aO_2); and perceptual responses. Ten well-trained male athletes cycled at submaximal intensity at 21°C or 33°C in randomized order: first for 20 min while breathing normal air ($F_{in}O_2 = 0.21$) and then 10 min with $F_{in}O_2 = 0.40$ (HOX). At both temperatures, S_aO_2 and P_aO_2, but not tHb, were increased by HOX. T_{skin} and perception of exertion and thermal discomfort were higher at 33°C than 21°C ($p < 0.01$), but independent of $F_{in}O_2$. T_{core} and muscle activity were the same under all conditions ($p > 0.07$). Blood lactate and heart rate were higher at 33°C than 21°C. In conclusion, during 30 min of submaximal cycling at 21°C or 33°C, T_{core}, T_{skin} and T_{body}, tHb, muscle activity and ratings of perceived exertion and thermal discomfort were the same under normoxic and hyperoxic conditions. Accordingly, breathing hyperoxic air ($F_{in}O_2 = 0.40$) did not affect thermoregulation under these conditions.

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INTRODUCTION

Several investigators have described the effects of either elevated ambient temperature or inspiration of hyperoxic air (with a greater partial pressure of oxygen than ambient air) during exercise on thermoregulatory, neuromuscular, metabolic, and perceptual responses (e.g., [1, 2]). In normoxic, warm environments, vasodilatation of subcutaneous vessels accounts for 80-95% of the elevated blood flow in human skin [3, 4, 5]. In contrast, hyperoxia (HOX) during exercise induces constriction of individual vessels [6], thereby elevating peripheral vascular resistance [7] and reducing blood flow in the forearm [7], calf [8], brain [9], and thigh [10].

To date, two reports on the impact of HOX and heat in combination have appeared. Upon immersing the legs of participants at rest and breathing air containing 21%, 25% or 30% oxygen in hot water (42°C) for 60 min, Yamashita and Tochihara [11] observed that HOX inhibited the elevation of blood flow in the skin by heat. In addition, Tomita and colleagues [12] demonstrated constriction of vascular endothelial cells *in vitro* upon exposure to 100% oxygen, as well as impairment of heat exchange in the skin of resting subjects breathing pure oxygen due to attenuated blood flow in the microvascular network. These findings indicate that HOX-induced cutaneous vasoconstriction reduces heat loss, thereby elevating core temperature. Oxygenation of human skeletal muscle (expressed as the tissue saturation index – TSI) [13], as well as changes in local blood volume (total haemoglobin – tHb) [14], potentially reflecting the balance between oxygen supply and utilization [15, 16], have been assessed by near-infrared spectroscopy (NIRS).

During prolonged submaximal exercise at cool temperatures, core body temperature remains relatively constant. However, humans stop exercising when their core temperature rises above 40.0°C [17], primarily due to attenuation of the central neural drive [18]. At the same time, several researchers have observed enhanced peripheral locomotor muscle activity when the oxygen content of the inspired air ($F_{in}O_2$) is elevated [19, 20]. For example, Tucker and co-workers [19] found that the integrated electromyographic activity (iEMG) of

skeletal muscle during a 20-km time trial of cycling was greater under hyperoxic than normoxic conditions. Similarly, peripheral locomotor muscle activity (i.e., iEMG) during high-intensity exercise at the same workload was higher with hyperoxia than normoxia [20].

Although HOX is used increasingly as an ergogenic aid for performance and recovery [21, 22, 23], to date the impact of combined exposure to HOX and heat during exercise on the thermoregulatory, neuromuscular, metabolic, and perceptual responses of male endurance athletes has not been documented. If during submaximal exercise HOX enhances muscle activation, but reduces vascular blood flow, while heat elevates vascular blood flow, but attenuates muscle activation, what will be the final outcome? Here, we compared the effects of HOX ($F_{in}O_2$ = 0.40) and normoxia ($F_{in}O_2$ = 0.21) during exercise at 33°C and 21°C to test the hypothesis that HOX is more deleterious for thermoregulatory, neuromuscular, metabolic, and perceptual responses in a warm than a cool environment. Our findings should help to improve protocols for sub-maximal exercise and recovery under different environmental conditions.

MATERIALS AND METHODS

The 10 healthy, non-smoking male volunteers (age 28 ± 4 years, body height 184 ± 7 cm, body mass 79.7 ± 7.8 kg; means ± SD) were all well-trained triathletes or cyclists (VO_{2peak}: 62.6 mL·min^{-1}·kg^{-1}), with considerable experience of all the laboratory exercise procedures employed here. They were asked to be adequately hydrated for the tests and to refrain from consuming alcohol for 24 h and food or caffeine for 3 h prior to each test. Prior to participating, all were informed of the protocol and provided their written consent. All procedures were approved by the ethics committee of the German Sport University in Cologne and conducted in accordance with the Declaration of Helsinki.

Procedures

Each participant performed laboratory trials on three separate occasions. During the first visit (pre-test), he received instructions concerning the test procedures, and his appropriate VO_{2peak} as well as the workload for the main testing was determined. In addition, body weight (kg) (Tanita BC 418 MA, Tanita Corp., Tokyo, Japan) and height (cm) were measured.

On the second and third occasions (performance trials), each participant completed one 30-min trial of submaximal cycling at either 21°C or 33°C (both at 30% relative humidity (RH)), in random, counterbalanced order. During these two main trials, each athlete cycled on a stationary ergometer (SRM GmbH, Jülich, Germany) at a fixed power output of approximately 1.5-2.0 W·kg^{-1} (i.e., 157.0 ± 15.0 W), depending on his level of fitness, for 30 minutes (min): the first 20 min under normoxic conditions and thereafter for 10 min while inhaling O_2-enriched air ($F_{in}O_2$ = 0.40) from a Douglas bag (Hans Rudolph Inc, Shawnee, KS, USA) through plastic tubing attached to a mouthpiece. During all testing the participants wore only cycling pants and shoes.

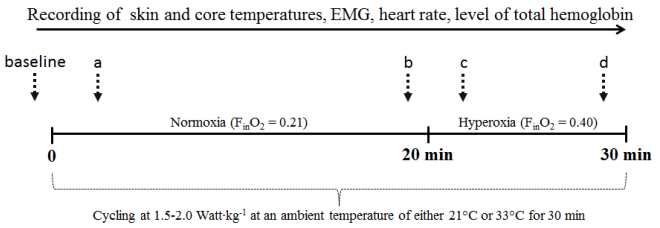

FIG. 1. Illustration of the test protocol.
Note: the two interventions were conducted in randomized, counterbalanced order. Skin and core temperature, EMG (m. *vastus medialis*, *semitendinosus*, and *gastrocnemius medialis*), heart rate and total level of haemoglobin were monitored continuously. The dotted arrows indicate the time points at which perceived exertion and thermal discomfort were rated.

Blood samples were collected from the right earlobe into capillary tubes (Eppendorf AG, Hamburg, Germany) immediately before beginning, and after 5, 18, 23 and 28 min of cycling (see Figure 1). Lactate was analysed by an amperometric-enzymatic procedure (Ebio Plus Eppendorf AG, Hamburg, Germany) and arterial partial pressure of oxygen (P_aO_2) and carbon dioxide (P_aCO_2), arterial oxygen saturation (S_aO_2), hydrogen ion concentration ([H^+]), and pH determined with an automated system (AVL Omni 3 Roche Ltd., Basel, Switzerland). All of these analyses were performed in duplicate, and the mean was used for statistical analyses.

Heart rate was monitored continuously with a telemetric system (Polar Wear Link System and Polar S810i HR Monitor, Polar Electro Oy, Kempele, Finland) and averaged over every 5 seconds (s). Perceived exertion (RPE) and thermal discomfort were rated on Borg's 6-20 scale [24] at the time points shown in Figure 1.

Core body temperature (T_{core}) was recorded with a capsule that measured the surrounding gastrointestinal temperature (Mini Mitter Co. Inc., Bend, OR, USA) and was ingested two hours before beginning to cycle and transmitted once every 60 s to an external receiver (Mini Mitter Co. Inc., Bend, OR) located close to the cyclists' lumbar region [25]. The reliability of this procedure for continuous monitoring of core body temperature has been validated in several sport and occupational studies [25]. Employing adhesive sensors, skin temperature (T_{skin}) could also be recorded telemetrically once every 60 s by the same receiver. Mean skin temperature was then calculated according to the following modification of the equation developed by Hardy and Dubois (1938) [26]: T_{skin} = 0.073·T_{hand} + 0.163·$T_{forearm}$ + 0.203·T_{chest} + 0.193·T_{back} + 0.213·T_{thigh} + 0.153·T_{calf}. Mean body temperature (T_{body}) was calculated using the temperature-weighted equation: T_{body} = 0.65·T_{core} + 0.35·T_{skin} [27]. Alterations in the total tissue level of haemoglobin concentration (tHb) and oxygen saturation (SO_2) were measured at 6 different sites on the right side of the body using the moorVMS-OXY system (Moor Instruments Ltd., Devon, UK), which analyses back-scattered light with a wavelength of 500-650 nm. The sites monitored were (1) above the m. *pectoralis major*, (2) the upper back (m. *trapezius* between the inferior angles of the scapulae), (3) the lower back above

the m. *erector spinae*, (4) the m. *vastus lateralis*, (5) the lower arm (m. *supinator*), and (6) between the thumb and index finger (m. *abductor pollicis brevis*). For measurement of cutaneous levels of haemoglobin, an optical fibre probe was employed.

The neuromuscular activities of the m. *vastus medialis*, *semitendinosus*, and *gastrocnemius medialis* in the right leg were monitored by surface EMG (TeleMyo 2400T; Noraxon Inc., Scottsdale, AZ, USA). For this purpose, pre-gelled bipolar electrodes were placed on the muscle belly in alignment with the underlying fibres, in accordance with international standards [28], and a reference electrode attached to the acromion. Prior to electrode placement, the skin was shaved, abraded slightly and cleaned with alcohol. All EMG signals were amplified (differential amplifier, Biovision, Werheim, Germany), filtered through a hardware band-pass (10–500 Hz at 3 dB), converted to digital units (DAQ 700 A/D card -12 bit, National Instruments, Austin, TX, USA), sampled at 1,000 Hz, and analysed with version 1.06.50 of the MyoResearch software (Noraxon USA Inc., Scottsdale, SZ, USA). Prior to further processing, all of these signals were full-wave rectified. To compare the intensities of activation, the integrated EMG (iEMG) of each muscle was calculated to obtain an indicator of the tension developed and the mean power frequency (MPF). These EMG parameters were determined at four different time points (i.e., after 5, 18, 23 and 30 min of cycling) and averaged across 20 revolutions of the pedals (see Figure 1).

Statistical analyses

In a pilot study on two separate days, measurement of the power output of the ergometer revealed a technical error [%TEM] of 1.7%. Under our laboratory conditions, the coefficient of variation for repeated measurements of blood lactate concentration is routinely 1.2% at 12 mmol·l^{-1}. In the case of PaO_2 and pH, the corresponding coefficients of variation are 3.2% and 3.6%, respectively.

All data analysis involved parametric procedures, and mean values and standard deviations (SD) are presented. All variables demonstrated a normal distribution, so no further transformation was required. Repeated two-way analyses of variance (ANOVA) were performed to look for differences in the parameters under the four different experimental conditions (normoxic or HOX breathing at 21°C or 33°C). If global significance was thereby obtained, Bonferroni post-hoc analysis was applied to identify differences between time points. $P < 0.05$ was considered to be statistically significant, and all analyses were carried out with the Statistica software package for Windows (version 7.1, StatSoft Inc., Tulsa, OK, U.S.A). The effect size Cohen's d (defined as the difference between the means divided by the standard deviation [29]) was calculated and the thresholds for small, moderate, and large effects defined *a priori* as 0.20, 0.50, and 0.80, respectively [29].

RESULTS

Table I documents the mean neuromuscular, metabolic, and perceptual responses of our participants during 30 min of cycling at 21°C

TABLE 1. Neuromuscular, metabolic and perceptual responses of our athletes (means ± SD; n = 10) during cycling for 30 min at 21°C or 33°C while inhaling air containing 0.21 or 0.40 $F_{in}O_2$

Parameter	At rest ($F_{in}O_2$=0.21)		$F_{in}O_2$=0.21				$F_{in}O_2$=0.40			
	Baseline		First measurement ([a])		Second measurement ([b])		First measurement ([c])		Second measurement ([d])	
Temperature	21° C	33° C	21° C	33° C	21° C	33° C	21° C	33° C	21° C	33° C
iEMG [%]										
- m. semitendinosus	n.d.	n.d.	100±0	100±0	96.0±23.8	96.6±40.0	103±25.5	88.4±32.0	97.9±31.1	83.0±29.7
- m. gastrocnemius medialis	n.d.	n.d.	100±0	100±0	86.6±14.5 #	87.6±22.0	89.0±16.0	82.5±32.3	94.7±9.9	89.4±27.9
- m. vastus medialis	n.d.	n.d.	100±0	100±0	94.9±10.1	102±20.8	94.5±9.5	107±15.8	105±17.8	106±12.9
Mean power frequency [Hz]										
- m. semitendinosus	n.d.	n.d.	87.5±18.5	82.4±19.6	88.9±14.6	85.4 ± 15.2 #	89.1±16.6	85.8±16.9	87.3±11.8	88.1±19.5
- m. gastrocnemius medialis	n.d.	n.d.	116±29.9	123±36.3	121±37.4	126±35.1	121±37.4	126±38.4	120±36.7	128±39.7
- m. vastus medialis	n.d.	n.d.	74.4±7.7	74.6±6.4	75.3±5.9	73.0±11.7	76.7±6.1	74.9±10.2	78.3±10.4	79.4±5.8
SaO2 [%]	96.4±0.6	96.0±0.7	95.6±0.5#	95.9±0.6	95.9±0.4	95.8±0.5	99.3±0.3 #	99.1±0.3 #	98.8±0.7 #+	98.8±0.7 #+
PaO2 [mmHg]	85.7±3.4	83.5±4.1	84.4±3.9	81.9±2.7 *	81.4±3.1	83.0±2.3	145±14.3 #	155±17.0 #	136±26.4 #+	136±25.8 #+
Blood lactate concentration [mmol·l^{-1}]	1.0±0.3	0.9±0.2	1.1±0.4	1.1±0.6	1.3±0.7	1.6±0.9 #	1.2±0.4	1.6±1.0 * #	0.9±0.3	1.4±0.9 *
pH	7.39±0.01	7.40±0.02	7.37±0.01 #	7.38±0.02	7.39±0.02	7.40±0.02	7.38±0.02	7.38±0.03	7.38±0.02	7.39±0.02
[H+] [nmol·l^{-1}]	40.3±1.2	40.3±1.8	42.3±1.4 #	41.9±2.4	40.8±1.5	40.2±1.8	41.4±1.5	41.6±2.8	41.3±2.1	40.9±2.0
Heart rate [bpm]	72.9±14.2	69.1±10.0	134±15.8 #	140±12.8 #	137±15.9 #	149±16.0 * #	136 ± 18.1#	151±16.4 * #	138±20.3 #	156±16.8 * #
RPE [6-20 Borg scale]	6.4±0.7	6.5±0.7	11.9±0.7 #	12.7±1.3 #	12.3±0.7 #	13.5±1.4 * #	12.5±0.9 #	13.6±1.4 * #	12.7±1.0 #	14.0±1.7 * #
Thermal discomfort [6-20 scale]	9.9±0.9	12.7±1.3 *	12.4±1.4	14.7±1.3 * #	12.9±1.3	14.9±1.8 * #	13.2±1.4	15.1±1.8 * #	13.1±1.2	15.2±1.7 * #

Note: iEMG = integrated electromyography (defined as 100% at the time of the first measurement with $F_{in}O_2$=0.21); SaO_2 = arterial oxygen saturation; PaO_2; partial pressure of oxygen in the arterial blood; bpm = beats per minute; RPE = rating of perceived exertion; n.d. = not determined. [a] after 5 min of cycling; [b] after 18 min of cycling; [c] after 23 min of cycling; [d] after 28 min of cycling; $^{+}$ $p<0.05$ for 21°C versus 33°C; $^{\#}$ $p<0.05$ for rest versus exercise; $^{+}$ $p<0.05$ for the 2nd measurements at FinO2=0.21 and 0.40

or 33°C while breathing air containing 0.21 or 0.40 $F_{in}O_2$. When HOX was inhaled, the S_aO_2 increased from 95.9 ± 0.4 to 99.3 ± 0.3% at 21°C ($p < 0.01$; effect size = 9.6) and from 95.8 ± 0.5 to 99.1 ± 0.3% at 33°C ($p < 0.01$; effect size = 8.0), respectively. In addition, P_aO_2 increased from 81.4 ± 3.1 to 145 ± 14.3 mm Hg at 21°C and 83.0 ± 2.3 to 155 ± 17.0 mm Hg at 33°C ($p < 0.01$; effect size = 5.9-6.2).

In addition, the lactate concentration in blood, heart rate and perceived exertion and thermal discomfort were significantly higher at 33°C than 21°C ($p < 0.01$; effect size = 1.70). In contrast, at the same time points the MPF, iEMG and blood pH were all the same at both temperatures (lowest $p = 0.07$; effect size = 0.5) and under normoxic and hyperoxic conditions (lowest $p = 0.29$; effect size = 0.4) (Table I). Moreover, there were only small differences in these latter three parameters at rest and during exercise.

As shown in Table II, the mean skin temperature was significantly lower at 21°C than 33°C ($p = 0.001$; effect size = 2.03), but with no significant difference between normoxic and hyperoxic conditions (lowest $p = 0.28$; effect size = 3.5). T_{core} and the calculated T_{body} were independent of both the ambient temperature ($p = 1.00$; effect size = 1.8) and $F_{in}O_2$ ($p = 1.00$; effect size = 1.2). Furthermore, the only significant difference in the total level of haemoglobin at 21°C and 33°C was observed at baseline in the chest ($p = 0.02$; effect size = 1.25), with no effect of $F_{in}O_2$ at any site ($p = 1.00$; effect size = 0.63). In addition, SO_2 at rest and during exercise differed at both temperatures, but was unaffected by $F_{in}O_2$.

TABLE 2. Skin, core and body temperatures and total level of haemoglobin (tHb) in our athletes (means ± SD; n = 10) during cycling for 30 min at 21°c or 33°c while inhaling air containing 0.21 or 0.40 $F_{in}O_2$

Parameter	At rest ($F_{in}O_2$=0.21)		$F_{in}O_2$=0.21				$F_{in}O_2$=0.40			
	Baseline		First measurement (a)		Second measurement (b)		First measurement (c)		Second measurement (d)	
Temperature	21° C	33° C	21° C	33° C	21° C	33° C	21° C	33° C	21° C	33° C
Skin temperature [°C]	31.9±0.6	34.1±1.9 *	31.7±0.6	33.8±2.3*	32.6±0.7	35.4±1.8 *	33.2±0.5	35.6±2.0	33.5±0.4	35.7±1.8 *
Core temperature [°C]	37.3±0.4	37.2±0.4	37.3±0.3	37.3±0.4	37.6±0.5	37.7±0.3 #	37.7±0.5 #	37.9±0.3 #	37.8±0.4 #	38.2±0.3 # +
Body temperature [°C]	35.4±0.4	36.1±0.8	35.3±0.4	36.1±0.9	35.8±0.5	36.9±0.7	36.2±0.4	37.0±0.8	36.3±0.3	37.3±0.7
Total level of hemoglobin [%] in the										
- thigh	21.3±9.2	19.8±8.1	11.9±6.2 #	13.5±5.2	n.d.	n.d.	13.2±6.7	15.1±5.3	11.2±6.0 #	17.6±6.9
- lower arm	18.1±9.1	14.2±7.0	16.4±6.7	17.9±5.6	n.d.	n.d.	17.7±7.4	19.7±4.7	16.1±6.3	20.9±8.8
- hand	22.5±16.0	19.3±11.4	18.5±8.2	21.4±9.7	n.d.	n.d.	20.1±8.8	19.9±7.7	18.6±2.9	22.1±12.3
- lower back	5.6±3.0	6.1±2.1	12.9±6.0	13.4±2.9	n.d.	n.d.	13.2±5.7 #	14.7±4.5 #	13.0±8.4	16.3±5.7 #
- upper back	9.5±5.7	9.8±7.2	13.4±10.1	17.9±4.7	n.d.	n.d.	13.9±4.3	20.5±7.6	14.3±3.6	17.3±9.8
- chest	13.5±4.9	10.9±3.9	22.5 ±12.0	25.8±8.1 #	n.d.	n.d.	20.1±7.4	25.0±8.7 #	16.6±5.5	28.6±12.4 *#
SO2 [%] in the										
- thigh	40.9±13.0	54.5±9.9 *	80.8±12.1 #	86.0±4.6 #	n.d.	n.d.	85.4±5.8 #	88.8±5.2 #	88.1±3.1 #	86.2±3.6 #
- lower arm	48.9±17.1	62.9±18.1*	77.0±11.4 #	86.8±4.3 #	n.d.	n.d.	84.5±5.3 #	89.6±5.2 #	86.8±6.4 #	88.6±5.6 #
- hand	55.3±19.8	61.7±15.5	71.2±11.0 #	77.0±9.3 #	n.d.	n.d.	79.9±10.0 #	86.9±6.1 #	81.4±11.4 #	85.5±8.4 #
- lower back	64.3±14.6	75.8±16.9	85.3±5.9 #	89.6±5.4 #	n.d.	n.d.	88.0±10.1 #	91.8±3.9 #	88.2±9.2 #	91.5±3.8 #
- upper back	67.5±10.4	77.3±11.1	87.5±8.6 #	87.4±6.1	n.d.	n.d.	88.8±6.9 #	91.5±5.6 #	89.5±6.7 #	89.8±3.3 #
- chest	66.2±8.4	78.0±8.2*	79.5±10.6 #	85.2±3.6 #	n.d.	n.d.	86.4±7.5 #	87.6±3.7 #	88.2±6.0 #	85.2±4.9 #

Note: Note: SO_2 = tissue oxygen saturation; n.d. = not determined; [a] after 5 min of cycling; [b] after 18 min of cycling; [c] after 23 min of cycling; [d] after 28 min of cycling; * $p<0.05$ for 21°C versus 33°C; # $p<0.05$ for rest versus exercise; + $p<0.05$ for the first versus the second measurement.

DISCUSSION

The major findings of this first investigation on the impact of combined exposure to HOX and heat on the thermoregulatory, neuromuscular, metabolic, and perceptual responses of male endurance athletes during exercise were as follows: i) the total level of haemoglobin at the sites on the skin monitored was not influenced by the ambient temperature; ii) T_{core} was also unaffected by the ambient temperature; iii) during 30 min of submaximal cycling, muscle activation at 21°C or 33°C and under hyperoxic or normoxic conditions was the same; iv) the concentration of lactate in blood was higher at 33°C, but lower during cycling in hyperoxia; and v) the perceived exertion and thermal discomfort were higher at 33°C than 21°C.

Total level of haemoglobin in the skin

In this study, the mean total levels of haemoglobin (tHb) in the skin of the thigh, lower arm, hand, lower back, upper back, and chest were independent of both ambient temperature and $F_{in}O_2$, i.e., there was no vasoconstriction. As reported previously, local alterations in blood volume reflect changes in blood flow assuming that no alterations in erythrocyte velocity occur [14]. Consequently, oxygen delivery to the muscle can be assessed by NIRS.

In several investigations hyperoxia-induced vasoconstriction at various sites of the body during rest has been observed [7, 8, 9], but to date only two have examined the effects of hyperoxia in a hot environment. Thus, Yamazaki and colleagues (2007) showed that hyperoxia (S_aO_2=99.8%) in combination with hyperthermia induces cutaneous vasoconstriction [30, 31]. Our present findings are in agreement with those of Rousseau [32] and Yamazaki [31] and coworkers in that hyperoxia-induced cutaneous vasoconstriction was observed only in areas of the skin exhibiting elevated basal blood flow. Rousseau and colleagues [32] noted that cutaneous blood flow is regulated primarily by factors other than the supply of oxygen. On the basis of our present observations, we propose that heat-induced vasodilatation counteracts any vasoconstriction evoked by hyperoxia [33].

Core body temperature

We observed only a slight increase in core body temperature during 30 min of submaximal cycling, with no significant difference between normoxic and HOX conditions. However, the effect sizes here were large, which is consistent with the lack of any significant difference in mean skin temperature. At the same time, differences in core body temperature under hyperoxic and normoxic conditions may arise if the duration of exercise is extended [34].

Muscle activation during exposure to both hyperoxia and heat

Several investigators have demonstrated that both the frequency components of EMG signals (i.e., iEMG and MPF) and the conduction velocity of muscle fibres during voluntary isometric contractions (20, 40, 60, and 80% of maximal voluntary contraction) are dependent on skin temperature [35]. However, we could not detect any differences between iEMG and MPF at 21°C or 33°C at any time point.

Alterations in the properties of EMG signals in connection with changes in skin temperature might be influenced by modifications in the properties of fibre membranes [36], which influence the recruitment of muscles for low-level contractions by the alpha motoneurons. In this context, direct modification of the contractile properties of motor units due to changes in fibre membrane conduction induced by temperature has been observed [37].

In this study, hyperoxia did not influence muscle activity at either 21°C or 33°C. During constant high-intensity cycling, Amann and co-workers [20] found a significantly higher iEMG with normoxic ($F_{in}O_2$=0.21) than hypoxic ($F_{in}O_2$=1.00) breathing and concluded that the level of oxygen influences the rate at which locomotor muscle fatigue develops. However, in the present investigation the duration of exercise (30 min) was much longer than that employed by Amann et al. [20] (~5 min) and the intensity lower, suggesting that at lower intensities the availability of oxygen does not limit muscle activation. In agreement, Taylor and colleagues [38] found more extensive activation of a muscle during submaximal cycling at a fixed power output under hypoxic ($F_{in}O_2$=0.12) than normoxic conditions.

Blood concentration of lactate

Under hyperoxic conditions in this study, the lactate concentration in blood was lower at 21°C than 33°C. Improvements in exercise performance (e.g., power output) associated with heating have been shown to be closely related to activation of muscle fibres. For example, muscles containing a higher percentage of type I fibres show more improvement in power output at 44°C than do muscles with a high proportion of type II fibres [39]. If this is the case, due to their higher content of mitochondria and enzymes involved in oxidative phosphorylation, type I fibres probably benefit more from aerobic metabolism, which would explain why lactate clearance in this study was more pronounced under normoxic conditions.

Subjective variables

In the present study, perceived exertion under normoxic and hyperoxic conditions was similar, which is consistent with previous findings. For example, elite kayakers reported no difference in the quality of their recovery after exercise under normoxic or hyperoxic (F = 0.99) conditions [2]. In contrast, Peeling and Andersson reported reduced self-reported feelings of dyspnoea and respiratory exertion and enhanced cognitive function and cerebral oxygenation when a higher concentration of oxygen was provided [2]. However, our present data indicate that hyperoxia probably does not lower the exertion and thermal discomfort felt by individuals exercising at 33°C.

CONCLUSIONS

Our present findings demonstrate that exposure to hyperoxic air ($F_{in}O_2$ = 0.40) during 30 min of submaximal cycling at 21°C or 33°C does not influence the total level of haemoglobin in the skin, core body temperature, muscle activity, or perceived exertion. However, the elevation in blood lactate caused by heat is attenuated by elevated $F_{in}O_2$. Thus, our hypothesis that HOX in a warm environment exerts deleterious effects on thermoregulatory, neuromuscular, metabolic, and perceptual responses was not confirmed. HOX is being used increasingly by athletes as an ergogenic aid for performance and recovery, and our present findings indicate that such use during recovery under hot environmental conditions might enhance metabolite clearance from the blood. Both training and competition may take place under hot conditions, and we conclude that the use of HOX will not influence thermoregulation negatively.

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