# ORIGINAL ARTICLE

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# Reduced performance of male and female athletes at 580 m altitude

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**Abstract** This study examined the effect of mild hypobaria (MH) on the peak oxygen consumption ( $\widehat{V}{O}_{2\text{peak}}$ )  $\frac{V_{\text{O}}}{V_{\text{peak}}}$ <br>
(SEM); trained<br>  $\cdot$  min<sup>-1</sup>].<br>
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b and performance of ten trained male athletes [ $\bar{x}$  (SEM);  $\bar{x}$  (SEM);<br>
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2 min- $\overline{VO}_{2\text{peak}}$  = 72.4 (2.2) ml·kg<sup>-1</sup>·min<sup>-1</sup>] and ten trained  $\dot{V}O_{2\text{peak}}$  = 72.4 (2.2) ml·kg<sup>-1</sup>·min<sup>-1</sup>] and ten trained<br>
cemale athletes [ $\dot{V}O_{2\text{peak}}$  = 60.8 (2.1) ml·kg<sup>-1</sup>·min<sup>-1</sup>].<br>
Subjects performed 5-min maximal work tests on a cycle<br>
ergometer within a hypobaric cham female athletes  $[\dot{V}\text{O}_{2\text{peak}} = 60.8 \text{ (2.1) } \text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}].$  $\dot{V}\dot{O}_{2peak} = 60.8$  (2.1) ml·kg<sup>-1</sup>·min<sup>-1</sup>].<br>d 5-min maximal work tests on a cycle<br>a hypobaric chamber at both normo-<br>Pa) and at MH (92.66 kPa), using a<br>design. MH was equivalent to 580 m<br>at MH decreased significantly c Subjects performed 5-min maximal work tests on a cycle ergometer within a hypobaric chamber at both normobaria (N, 99.33 kPa) and at MH (92.66 kPa), using a counter-balanced design. MH was equivalent to 580 m altitude.  $VO_{2\text{peak}}$  at MH decreased significantly com-VO<sub>2peak</sub> at MH decreased significantly com-<br>h N in both men [- 5.9 (0.9)%] and women<br>))%]. Performance (total kJ) at MH was also<br>ignificantly in men [- 3.6 (0.8)%] and women<br>2)%]. Arterial oxyhaemoglobin saturation<br> $\dot{V$ pared with N in both men  $[-5.9 \ (0.9)\%]$  and women  $[-3.7 \ (1.0)\%]$ . Performance (total kJ) at MH was also reduced significantly in men  $[-3.6 (0.8)\%]$  and women [– 3.8 (1.2)%]. Arterial oxyhaemoglobin saturation  $(S_aO_2)$  at  $VO_{2peak}$  was significantly lower at MH com- $VO_{2peak}$  was significantly lower at MH com-<br>
1 N in both men [90.1 (0.6)% versus 92.0<br>
d women [89.7 (3.1)% versus 92.1 (3.0)%].<br>
2 at  $\dot{VO}_{2peak}$  was not different between men<br>
n, it was concluded that relative, rather tha pared with N in both men  $[90.1 \ (0.6)\%$  versus 92.0  $(0.6)$ %] and women [89.7 (3.1)% versus 92.1 (3.0)%]. While  $S_aO_2$  at  $VO_{2peak}$  was not different between men  $VO_{\text{2peak}}$  was not different between men<br>
was concluded that relative, rather than<br>  $_{\text{ak}}$  may be a more appropriate predictor<br>  $_{\text{stralia}}$ <br>
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y Unit, The Universi and women, it was concluded that relative, rather than absolute,  $\dot{V}O_{2\text{peak}}$  may be a more appropriate predictor

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of exercise-induced hypoxaemia. For men and women, it was calculated that 67–76% of the decrease in  $\dot{V}O_{2\text{peak}}$ could be accounted for by a decrease in  $O_2$  delivery, which indicates that reduced  $O_2$  tension at mild altitude (580 m) leads to impairment of exercise performance in a maximal work bout lasting  $\approx$  5 min.

Hypobaria · Women

## Introduction

 $\dot{V}$ O<sub>2peak</sub><br>lelivery,<br>altitude<br>nce in a<br><br>mean a<br><br>factor<br>healthy<br>oglobin<br>les even<br>n 1960;<br>50% of<br>xaemia<br>arterial **Key words** Performance · Hypoxaemia ·  $VO_{2peak}$  · Hypobaria · Women<br> **Introduction**<br>
Pulmonary gas exchange is not considered a limiting peak oxygen consumption  $(\dot{VO}_{2peak})$  in 1 untrained individuals because arterial oxyhae Pulmonary gas exchange is not considered a factor limiting peak oxygen consumption  $(\dot{V}O_{2\text{peak}})$  in healthy  $VO_{2peak})$  in healthy<br>al oxyhaemoglobin<br>resting values even<br>and Nielsen 1960;<br>oroximately 50% of<br>duced hypoxaemia<br> $2\%$  and an arterial<br>elow resting levels<br>maximal exercise at<br>set al. 1992, 1993).<br>concluded that an<br>urable re untrained individuals because arterial oxyhaemoglobin saturation  $(S_aO_2)$  is maintained near resting values even during maximal exercise (Asmussen and Nielsen 1960; Lawler et al. 1988). However, in approximately 50% of endurance-trained men, exercise-induced hypoxaemia (EIH), characterized by an  $S_aO_2 \leq 92\%$  and an arterial oxygen tension  $(P_aO_2)$  2.4 kPa below resting levels (Powers et al. 1992), occurs during maximal exercise at sea level (Dempsey et al. 1984; Powers et al. 1992, 1993). Furthermore, Powers et al. (1989) concluded that an  $S_aO_2$  below  $\approx$  92–93% caused a measurable reduction of maximal oxygen consumption  $(\dot{V}O_{2\text{max}})$  with an ap- $VO_{2\text{max}}$ ) with an ap-<br>for each 1% decrease<br>al. 1992; Lawler et al.<br>bwers et al. 1992) have<br>hat decrease inspired<br>et altitudes of approxi-<br>te EIH and reduce<br>at 580 m altitude and<br>antly  $(-6.8\%)$ . These<br>the aerobic power o proximate 1% decrease in  $VO_{2\text{max}}$  for each 1% decrease  $VO_{2\text{max}}$  for each 1% decrease<br>nson et al. 1992; Lawler et al.<br>993; Powers et al. 1992) have<br>itions that decrease inspired<br>imulate altitudes of approxi-<br>centuate EIH and reduce<br>ore et al. (1996) reported that<br>greater at in *S*aO2. Other studies (Johnson et al. 1992; Lawler et al. 1988; Martin and O′Kroy 1993; Powers et al. 1992) have shown that hypoxic conditions that decrease inspired oxygen tension  $(P_1O_2)$  to simulate altitudes of approximately 2000–3500 m accentuate EIH and reduce  $\dot{V}O_{2\text{max}}$ . More recently, Gore et al. (1996) reported that  $VO_{2\text{max}}$ . More recently, Gore et al. (1996) reported that EIH was also significantly greater at 580 m altitude and that  $\dot{VO}_{2\text{max}}$  was reduced significantly (– 6.8%). These results support the concept that the aerobi EIH was also significantly greater at 580 m altitude and that  $\dot{V}\text{O}_{2\text{max}}$  was reduced significantly (– 6.8%). These  $VO_{2\text{max}}$  was reduced significantly (– 6.8%). These<br>s support the concept that the aerobic power of<br>d individuals decreases progressively as one begins<br>zend from sea level (Squires and Buskirk 1982).<br>ven the important co results support the concept that the aerobic power of trained individuals decreases progressively as one begins to ascend from sea level (Squires and Buskirk 1982).

Given the important contribution of maximal aerobic power to cycling performance (Craig et al. 1993; Olds

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et al. 1995), a small decrease in  $\overline{VO}_{2\text{max}}$ , as a con- $VO_{2max}$ , as a conduced  $O_2$  delivery,<br>performance. How-<br>r of performance in<br>f runners (Noakes<br>. 1992) and cyclists<br>uggested that mea-<br>ork output ( $W_{peak}$ )<br>than is  $VO_{2max}$ . In<br>d the performance<br>d McKenzie (1994)<br>, but not sequence of decreased  $S_aO_2$  and reduced  $O_2$  delivery, would be expected to compromise performance. However,  $\dot{V}\text{O}_{2\text{max}}$  is not the best predictor of performance in  $VO_{2max}$  is not the best predictor of performance in<br>ance-trained athletes. Studies of runners (Noakes<br>1990), swimmers (Hawley et al. 1992) and cyclists<br>ey and Noakes 1992) have suggested that mea-<br>ents of peak speed and p endurance-trained athletes. Studies of runners (Noakes et al. 1990), swimmers (Hawley et al. 1992) and cyclists (Hawley and Noakes 1992) have suggested that measurements of peak speed and peak work output  $(W_{peak})$ are better predictors of performance than is  $\hat{V}\text{O}_{2\text{max}}$ . In  $\dot{W}O_{2\text{max}}$ . In<br>riformance<br>nzie (1994)<br>t  $\dot{V}O_{2\text{peak}}$ ,<br>endurance-<br>ypoxaemia<br>doubt as to<br>loubt as to<br>nsie perfor-<br>test would<br>core et al.<br>nd perfor-<br>test would<br>core tal.<br>in this phe-<br>Lusker and this phe-<br>Lusker and the only study which has explored the performance consequences of EIH, Koskolou and McKenzie (1994) found a decrease in total work (kJ), but not  $VO_{2\text{peak}}$ ,  $VO_{2peak}$ ,<br>durance-<br>oxaemia<br>ubt as to<br>perfor-<br>did not ased on<br>exact and perfor-<br>it would<br>f EIH in<br>rs et al.<br>his phe-<br>ker and<br> $\dot{V}O_{2peak}$ <br>status<br>g status<br>the high during a 5-min performance test when endurancetrained men experienced moderate hypoxaemia  $(S_a O_2 \leq 87\%)$ . However, there remains some doubt as to the minimum level of EIH that will decrease performance, since Koskolou and McKenzie (1994) did not measure  $S_aO_2$  directly from arterial blood. Based on previous observations at 580 m altitude (Gore et al. 1996) we hypothesized that both  $\dot{V}O_{2\text{peak}}$  and performance (total work) in a 5-min maximal cycle test would be reduced.

 $VO_{2peak}$  and performal cycle test would<br>orevalence of EIH in<br>1984; Powers et al.<br>examined this phe-<br>ales (McCusker and<br>owever, the  $\dot{VO}_{2peak}$ <br>d the training status<br>dest. Given the high<br>of endurance-trained<br>duced haemoglobi Although data investigating the prevalence of EIH in men are extensive (Dempsey et al. 1984; Powers et al. 1992, 1993), only two studies have examined this phenomenon in endurance-trained females (McCusker and Brilla 1992; Pedersen et al. 1992). However, the  $\dot{V}O_{2\text{peak}}$  $V$ O<sub>2peak</sub><br>g status<br>he high<br>trained<br>oglobin<br>we hy-<br>female<br>O2), the<br>be even<br>e aim of<br>rude re-<br>ale envalues (57 and 52 ml·kg<sup>-1</sup>·min<sup>-1</sup>) and the training status of their subjects were relatively modest. Given the high relative  $\dot{V}O_{2\text{max}}$  and cardiac output of endurance-trained  $VO_{2\text{max}}$  and cardiac output of endurance-trained<br>thletes, combined with reduced haemoglobin<br>ation ([Hb]) (Kjellberg et al. 1949), we hy-<br>ed that EIH may be more prevalent in female<br>than in male athletes. Moreover, in re female athletes, combined with reduced haemoglobin concentration ([Hb]) (Kjellberg et al. 1949), we hypothesized that EIH may be more prevalent in female athletes than in male athletes. Moreover, in response to increased altitude and lower oxygen tension  $(P_1O_2)$ , the  $\dot{V}O_{2\text{peak}}$ ,  $S_aO_2$  and performance of women may be even more susceptible than that of men. Therefore, the aim of this study was to determine whether 580 m altitude reduced 5-min cycling performance in male or female endurance-trained athletes.

#### Methods

#### Subjects

VO<sub>2peak</sub>, *S*<sub>a</sub>O<sub>2</sub> and performance of women may be even<br>nore susceptible than that of men. Therefore, the aim of<br>his study was to determine whether 580 m altitude re-<br>duced 5-min cycling performance in male or female en Ten men and ten women (Table 1) gave written, informed consent to participate in the study. All subjects were either cyclists or triathletes and were life-time non-smokers with no history of asthma. The International Union Against Tuberculosis Questionnaire (Burney and Chinn 1987) was completed by all subjects to obtain a respiratory history, and spirometry was conducted using a Vitalograph (Med. Industr., Hamburg, Germany), with the subjects performing a minimum of three trials whilst standing and wearing a nose clip. Stretch height and mass [Toledo Digital scales, 120 (0.02)kg capacity] were measured with the subject in light clothing and barefoot. Normal lung function was confirmed because the mean forced vital capacity (FVC) and forced expiratory volume in one second (FEV<sub>1</sub>) were  $\geq 100\%$  of Australian normative data (Gore et al. 1995) predicted for appropriate sex, age and height.

**Table 1** Physical characteristics of the men  $(n = 10)$  and women  $(n = 10)$  at normobaria. (FVC Forced vital capacity,  $FEV<sub>1</sub>$  forced expiratory volume in 1s). Values are means (SEM)

	Males	Females
Age (years)	23.8(1.9)	26.7(1.7)
Height (cm)	177.7(1.3)	166.3(2.0)
Mass (kg)	72.38(2.13)	$*58.56(1.35)$
FVC(1)	6.08(0.25)	1,53(0.23)
$FEV_1$ (1)	4.66(0.24)	$*3.65(0.19)$
	5.21(0.13)	$*3.56(0.13)$
$VO_{2\text{peak}}$ (1 min <sup>-1</sup> ) $VO_{2\text{peak}}$ (ml·kg <sup>-1</sup> min <sup>-1</sup> )	72.3(2.0)	$*60.8(2.0)$

\* Significant difference from men, *P* < 0.05

#### Test protocol

The research techniques and protocol were approved by the Australian Defence Medical Ethics Committee. Each subject completed two 5-min maximal work tests on a geared, air-braked cycle ergometer (Hayes, Adelaide, Australia) with a minimum of 24 h between tests. One test was conducted at normobaria ( $N = 99.33$ ) kPa) and the other at mild hypobaria (MH =  $92.66$  kPa), with the order of testing counter-balanced and double-blinded. The 6.67kPa difference between tests is equivalent to 580 m altitude (International Civil Aviation Organisation 1954). All subjects completed a 5-min maximal work test at sea level, as a habituation trial, 1 week prior to the test protocol. Prior to the 5-min performance test, all subjects warmed up for 5 min with standardized work rates of 200 and 125 W for men and women, respectively. Total work (kJ) and power output  $(W)$  during each test was assessed using SRM cranks (Ingenieurbüro Schoberer, Jülich, Germany) with information recorded on a data logger and analyzed using SRM software. The accuracy of the SRM cranks was verified before the study commenced using a first principles calibration rig (Stanef 1988). Cardiac frequency  $(f_c)$  was recorded with a Polar PE4000 receiver (Polar Electro, Kempele, Finland) and transmitter belt.  $\frac{VQ_{2peak}}{VQ_{2peak}}$  (l · min<sup>-1</sup>) 5.21 (0.13)  $\frac{VQ_{2peak}}{6}$ <br>
Significant difference from men,  $P < 0.05$ <br>
Test protocol<br>
The research techniques and protocol were approved<br>
The research techniques and protocol were approve  $\sqrt{VO_{2peak}}$  (ml·kg<sup>-1</sup>·min<sup>-1</sup>) 72.3 (2.0) \*\*<br>
\*Significant difference from men,  $P < 0.05$ <br>
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baric chambe All tests were conducted in a hypobaric chamber (Thompsons, Castlemaine, Australia) located at the Royal Australian Air Force (RAAF) Base Edinburgh, Adelaide, Australia. The chamber dimensions, operating conditions, altimeter calibration and ascent/ descent rates replicated those of our previous study (Gore et al. 1996). Normobaria of 99.3 kPa rather than 101.3 kPa was chosen since the chamber did not have hyperbaric capability and since 99.3 kPa represents the lowest barometric pressure that might occur at sea level in Adelaide. Temperature ( $\overline{C}$ ) and relative humidity (%) within the chamber were monitored with a Solomat MPM 200 Environmental Analyser (UK), calibrated by Telstra Products and Services to National Association of Testing Authorities standards. Mean (SEM) chamber temperature and relative humidity during normobaria tests were  $25.0$  (0.4)<sup>o</sup>C and 48.6 (1.8)% respectively. Mean chamber temperature and relatively humidity during the mild hypobaric trials were  $25.1$   $(0.3)$ °C and  $43.7$   $(1.1)$ %, respectively.

#### Oxygen consumption

Oxygen consumption  $(\dot{V}O_2)$  was measured every 30 s with a cus- $VO<sub>2</sub>$ ) was measured every 30 s with a cus-<br>ndirect calorimetry system interfaced to an<br>e inspired volume of air was measured with<br>(PK Morgan, Rainham, Kent, UK) con-<br>o-way respiratory valve (Hans Rudolph,<br>SA). The ve tom-built open-circuit indirect calorimetry system interfaced to an Apple IIe computer. The inspired volume of air was measured with a Morgan ventilometer (PK Morgan, Rainham, Kent, UK) connected to a R2700 two-way respiratory valve (Hans Rudolph, Kansas City, Miss., USA). The ventilometer was calibrated according to the manufacturer's specifications with 10 l of air administered with a 1.0-l calibration syringe (PK Morgan). This was then verified by administrations of 60, 120, 180  $1 \cdot \text{min}^{-1}$  with a 3.0-1 calibration syringe (Hans Rudolph). The precision and accuracy of Morgan ventilometers have been established previously (Hart et al.

1994). Expired gases passed through 1 m of Clean-bor tubing (Vacumed, Ventura, Calif., USA) to a 2.6-l mixing chamber (Sportech, ACT, Australia), with the total system dead space being ≈ 3.7 l. Gas samples from the mixing chamber passed through a 15 cm CaCl<sub>2</sub> drying tube before reaching  $O_2$  (Ametek S-3 AI) and  $CO_2$ (Ametek CD-3A) gas analysers. The metabolic gas analysers were calibrated immediately before and after each test with three alphastandard gas mixtures (BOC Gases, Australia), with  $O_2$  and  $CO_2$ concentrations spanning the physiological range. All components of the open-circuit calorimetry system were calibrated when chamber pressure was confirmed as stable. Post-test checks of both the ventilometer and gas analysers were completed before chamber pressure was returned to ambient conditions.  $\dot{V}\text{O}_{2\text{peak}}$  was calculated as the mean of the two highest consecutive  $30\text{-}s\ \dot{V}\text{O}_2$  samples during exercise.

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(unpublished) tests<br>  $VO_{2peak}$   $VO_2$  samples<br>ical error of<br>indirect ca-<br>on a similar<br>or an incre-<br>roth total lished in<br>stablished in<br>blished in starting 5-<br>significantly<br>ined during<br>DD  $\times$  32 mm<br>h the tip up-<br>tized locally Prior to the experiments, the precision or technical error of measurement [TEM, (Denegar and Ball 1993)] of the indirect calorimetry system was assessed using duplicate tests on a similar group of ten well-trained male cyclists. The TEM for an incremental cycling  $VO_{2\text{max}}$  was 2.6%. A TEM of 1.3% for both total work and average power output for a 5-min test was established in five male high-performance cyclists. Preliminary (unpublished) tests on 16 male cyclists also demonstrated that the  $\dot{V}O_{2\text{peak}}$  during 5min maximal work tests ( $\bar{x} = 5.23$  1 min<sup>-1</sup>) was not significantly different ( $t = -1.66$ ,  $P = 0.12$ ) from the  $\dot{V}O_{2\text{max}}$  obtained during incremental tests ( $\bar{x}$  = 5.16 l·min<sup>-1</sup>).

#### Arterial blood sampling

VO<sub>2max</sub> was 2.6%. A TEM of 1.3% for both total<br>ge power output for a 5-min test was established in<br>erformance cyclists. Preliminary (unpublished) tests<br>ists also demonstrated that the  $\dot{V}O_{2\text{peak}}$  during 5-<br>ork tests  $\text{WO}_{\text{2peak}}$  during 5-<br>
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1.12) from the  $\dot{V}O_{2\text{max}}$  obtained during<br>
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thlon IV 20 gauge, 1.5 mm OD × 32 mm<br>
on, USA) was inserted with the tip up-<br>
netrery, which was anaesthetize VO<sub>2max</sub> obtained during<br>ge, 1.5 mm OD × 32 mm<br>inserted with the tip up-<br>as anaesthetized locally<br>rmaceuticals, Australia)<br>od samples (2.5 ml) were<br>est and after 1, 3 and 5<br>mmediately and stored<br>g, were analyzed in tri-<br>c  $\bar{x} = 5.161 \cdot \text{min}^{-1}$ ).<br>
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gases An indwelling catheter (Cathlon IV 20 gauge,  $1.5 \text{ mm OD} \times 32 \text{ mm}$ ) length, Johnson and Johnson, USA) was inserted with the tip upstream into the brachial artery, which was anaesthetized locally (lignocaine hydrochloride, 2%, Astra Pharmaceuticals, Australia) immediately prior to each test. Arterial blood samples (2.5 ml) were drawn into heparinized glass syringes at rest and after 1, 3 and 5 min of exercise. Samples were capped immediately and stored vertically on ice and, after thorough mixing, were analyzed in triplicate for blood gases [P<sub>a</sub>O<sub>2</sub>; arterial carbon dioxide tension  $(P_aCO_2)$ ] and pH using a Ciba Corning Blood Gas Analyser (model 860, Medfield, Mass., USA). Quadruplicate analyses of functional oxyhaemoglobin saturation (HbO<sub>2</sub> = HbO<sub>2</sub>/HbO<sub>2</sub> + reduced Hb), fractional oxyhaemoglobin saturation  $[S_aO_2 = HbO_2/(HbO_2 +$ reduced Hb + metHb + COHb)] and [Hb] were conducted using a Ciba Corning CO-oximeter (model 270 Medfield). The blood gas analyser was calibrated every 2h using standards purchased from the manufacturer, with two quality controls (BioRad, Anaheim, Calif., USA) assayed every hour. The CO-oximeter was calibrated prior to the first test, and quality controls were assayed on each morning of testing. Calibration and control solutions were purchased from Ciba Corning. Blood gas and  $S_aO_2$  values were measured at 37°C with no temperature correction factor applied, since in a repeated measures design each subject acts as their own control and results are directly comparable. The ideal alveolar gas equation was used to calculate alveolar oxygen tension  $(P_A O_2)$  and the alveolar – arterial oxygen tension difference  $(AaDO<sub>2</sub>)$  (Otis 1964). The arterial oxygen content  $(c_aO_2)$  values were calculated using functional  $HbO_2$ , according to the method of Siggaard-Andersen et al. (1988).

#### Expression of results and statistical analysis

A three-way repeated measures analysis of variance (ANOVA) (Statistica/W – StatSoft, Tulsa, Okla., USA) was used to determine the interactions and main effects of time, chamber pressure and gender on blood (*P*aO2, *P*aCO2, *P*AO2, AaDO2, *S*aO2, pH, [Hb],  $\tilde{c}_aO_2$ ), metabolic [ $\tilde{V}O_2$ , expired carbon dioxide production ( $\tilde{V}CO_2$ )],  $VO_2$ , expired carbon dioxide production ( $VCO_2$ )],<br>tata [expired minute volume of gas at standard<br>pressure dry ( $V_E$  srpp), expired minute volume of<br>erature and pressure saturated ( $V_E$  srps), venti-<br>for  $O_2$  ( $V_E$  srps and ventilation data [expired minute volume of gas at standard temperature and pressure dry ( $V_{\text{E STPD}}$ ), expired minute volume of  $V_{\text{E STPD}}$ , expired minute volume of<br>ressure saturated ( $V_{\text{E BTPS}}$ ), venti-<br> $_{\text{PS}}$ / $\dot{V}O_2$ ), ventilatory equivalent for<br>way ANOVA was used when time gas at body temperature and pressure saturated ( $V_{\rm E|BTPS}$ ), venti- $V_{\rm E}$  BTPS), venti-<br>y equivalent for<br>used when time latory equivalent for  $O_2$  ( $V_E$   $_{BTPS}/VO_2$ ), ventilatory equivalent for  $W_{\text{E E TPS}}/V\text{O}_2$ ), ventilatory equivalent for<br>two-way ANOVA was used when time  $\rm CO_2$  ( $V_{\rm E\ BTPS}/V$  $\ddot{ }$  $VCO<sub>2</sub>$ ]. A two-way ANOVA was used when time

was not an independent variable ( $\dot{V}O_{2\text{peak}}$ , total work). The Tukey HSD post-hoc test was performed to locate specific mean differences. Student's *t*-tests for independent samples were used to compare the physical characteristics of the men and women. The significance level was set at  $P < 0.05$ , with results expressed as mean (SEM), unless otherwise stated.

## Results

## Oxygen consumption

VO<sub>2peak</sub>, total work). The Tukey<br>d to locate specific mean differ-<br>pendent samples were used to<br>cs of the men and women. The<br>0.05, with results expressed as<br>ted.<br><br>Wight response to hypobaria<br> $\%$ : N, 5.21 (0.13)  $1 \cdot \text{min$  $\dot{V}\text{O}_{2\text{peak}}$  decreased significantly in response to hypobaria VO<sub>2peak</sub> decreased significantly in response to hypobaria<br>n both the men  $[-5.9 (0.9)\%$ : N, 5.21 (0.13) 1·min<sup>-1</sup>;<br>MH, 4.90 (0.12) 1·min<sup>-1</sup>; MH, 3.43 (0.13) 1·min<sup>-1</sup>; and the<br>decrease was significantly greater in men tha in both the men  $[-5.9 (0.9)\%; N, 5.21 (0.13) 1 \cdot min^{-1};$ MH, 4.90 (0.12)  $1 \cdot \text{min}^{-1}$  and women  $[-3.7 \ (1.0)\%; N, ]$ 3.56 (0.13)  $1 \cdot \text{min}^{-1}$ ; MH, 3.43 (0.13)  $1 \cdot \text{min}^{-1}$ ], and the decrease was significantly greater in men than women,  $F_{(1,18)} = 8.4, P = 0.01$ . During exercise,  $VO_2$  of the men at MH was reduced after 1, 2, 3, 4 and 5 min of exercise compared with the matched times at N (Fig. 1), while that of women was reduced after 3 and 4 min. The correlation between normobaric  $\dot{V}O_{2\text{peak}}$ % $\Delta V$ O<sub>2peak</sub> from N to MH failed to reach statistical significance for the pooled data of the men and women  $(r^2 = 0.15, P = 0.09).$ 

### Performance

 $VO_2$  of the men<br>min of exercise<br>(Fig. 1), while<br>nd 4 min. The<br> $\dot{VO}_{2\text{peak}}$  and<br>each statistical<br>each statistical<br>en and women<br>and women<br>and work and<br>that of the wo-<br>g. 1). However,<br>rcise decreased<br>n [- 3.9 (0.8)%:<br>d wom  $VO_{2\text{peak}}$  and<br>each statistical<br>en and women<br>and women<br>and women<br>and assessed for<br>that of the wo-<br>g. 1). However,<br>rcise decreased<br>al women  $[-3.8]$ ,<br>respectively,<br>a of men and<br>ant correlation<br>and either the VO<sub>2peak</sub> from N to MH failed to reach statistical<br>ificance for the pooled data of the men and women<br>= 0.15,  $P = 0.09$ ).<br>
Sormance<br>
men produced significantly less total work and<br>
ver than men at both N and MH. When asses Women produced significantly less total work and power than men at both N and MH. When assessed for each minute of exercise,  $\dot{W}$  of the men was reduced V of the men was reduced<br>3 min, while that of the wo-<br>hypobaria (Fig. 1). However,<br> $\ge$  5 min of exercise decreased<br>H in both men [- 3.9 (0.8)%:<br>3 (2.5) kJ] and women [- 3.8<br>H, 77.7 (2.1) kJ], respectively,<br>When the data significantly after 1, 2 and 3 min, while that of the women was not changed with hypobaria (Fig. 1). However, the total work for the entire 5 min of exercise decreased significantly from N to MH in both men  $[-3.9 \ (0.8)\%]$ : N, 114.8 (2.8) kJ; MH, 110.3 (2.5) kJ] and women [– 3.8 (1.2)%: N, 80.8 (1.8) kJ; MH, 77.7 (2.1) kJ], respectively,  $F_{(1,18)} = 31.4$ ,  $P < 0.0001$ . When the data of men and women were pooled there was no significant correlation between %∆ total work from N to MH and either the corresponding  $\Delta \dot{V}O_{2\text{peak}}$  ( $r^2 = 0.05$ ) or  $\Delta S_aO_{2\text{peak}}$  $(r^2 = 0.04)$ .

### Oxygen saturation of haemoglobin

 $VO_{2\text{peak}}$  (*r*<br>of haemog<br>ficant difference both showing<br>to mean value and 89.9 (0)<br>and woment at MH<br>ise,  $F_{(1,18)}$  is and woment is a manufact diffusional HbO<sub>2</sub><br>mificant diffusional HbO<sub>2</sub> was There was no significant difference in  $S_aO_2$  between men and women, with both showing a progressive decrease from rest ( $\approx 98\%$ ) to mean values after 5 min of exercise of 92.1  $(0.6)$ % (N) and 89.9  $(0.6)$ % (MH) (Fig. 2). When the data of men and women were pooled,  $S_aO_2$  was significantly lower at MH than at N, at rest and throughout exercise,  $F_{(1,18)} = 79.1$ ,  $P < 0.0001$ . For pooled data of men and women, the correlation between  $\Delta\%S_3O_2$  during the final minute of exercise at N and MH and the corresponding  $\Delta VO_{2\text{peak}}$  was not significant  $VO_{\text{2peak}}$  was not significant<br>
i was similar to fractional<br>
ference between men and<br>
as significantly lower at N  $(r^2 = 0.10)$ . Functional HbO<sub>2</sub> was similar to fractional  $S_aO_2$ , with no significant difference between men and women at N or MH.  $HbO<sub>2</sub>$  was significantly lower at N



**Fig. 1** Oxygen consumption  $(VO_2)$  power output (in watts, W) and  $VO_2$ ) power output (in watts, W) and<br>ance-trained male ( $n = 10$ ) and female<br>all-out performance tests on a cycle<br>33 kPa) and at mild hypobaria (92.66<br>lent to 580 m altitude referenced to sea<br>SEM). \*Significantly differe cardiac frequency  $(f_c)$  of endurance-trained male  $(n = 10)$  and female  $(n = 10)$  athletes during 5-min all-out performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). This hypobaria is equivalent to 580 m altitude referenced to sea level. Values are means (SEM). \*Significantly different from normobaria, within-group comparison. Note that, at all times, the values for women are significantly different from those for the men at the matched chamber pressure

than at MH, at rest and throughout exercise and, furthermore, decreased significantly between successive measurement times.

## Blood gases and associated variables

There was no significant difference in  $P_aO_2$ ,  $P_aCO_2$ , *P*AO2 and AaDO2 between men and women and, therefore, group data were pooled for each of these variables. At both N and MH, there was a significant decrease in  $P_aO_2$  from resting values after 1 min of exercise, after which  $P_aO_2$  was generally maintained



**Fig. 2** Changes in arterial oxyhaemoglobin saturation  $(S_a O_2)$ , haemoglobin concentration ([Hb]), arterial oxygen content  $(c_a O_2)$ and pH during 5-min performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Values are means (SEM) for ten male and ten female endurance-trained athletes. \*Significantly different from normobaria, within group comparison; † significantly different from previous time, within group comparison; § significantly different from men at matched chamber pressure

throughout the remainder of exercise (Fig. 3).  $P_AO_2$  and  $AaDO<sub>2</sub>$  increased significantly and progressively from rest values during exercise at both N and MH (Fig. 3). When data from all four measurement times were pooled,  $P_AO_2$  was significantly lower at N than MH,



**Fig. 3** Time course of arterial  $O_2$  tension ( $P_aO_2$ ), alveolar  $O_2$  tension  $(P_A O_2)$ , alveolar-arterial  $O_2$  tension difference  $(AaDO_2)$  and arterial  $CO<sub>2</sub>$  tension ( $P<sub>a</sub>CO<sub>2</sub>$ ) during 5-min performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Values are means (SEM) for ten male and ten female endurancetrained athletes. The data for the men and women are pooled since they are not significantly different. \*Significantly different from normobaria; †significantly different from previous time, withincondition comparison

 $F_{(1,18)} = 5.6$ ,  $P < 0.03$ , while the AaDO<sub>2</sub> was significantly widened with hypobaria  $F_{(1,18)} = 168.9$ ,  $P \le 0.0001$ .  $P_aCO_2$  exhibited a significant, progressive decrease from rest until after 5 min of exercise, with no significant difference between N and MH (Fig. 3). The pH response during exercise was different for men and

women, depending upon chamber pressure,  $F_{(3,54)} =$ 4.31, *P* < 0.01. Overall, pH decreased with exercise time but was significantly higher at MH than N for men only after 3 and 5 min of exercise. While in the early stages of exercise at both N and MH pH was not different between men and women; the pH of the women was higher after 3 min of exercise at N, and lower after 5 min of exercise at MH (Fig. 2).

## Oxygen content and haemoglobin

While there was no difference in resting values,  $c_aO_2$ during exercise was significantly lower at MH than at N,  $F_{(1,18)} = 11.93$ ,  $P < 0.01$ . At N for the pooled data of men and women,  $c_aO_2$  increased significantly [ $\approx 3.3$ (0.3)%] above resting values after 1 min and remained elevated throughout exercise (Fig. 2). At MH, however, men failed to increase  $c_aO_2$  above resting levels at any time during exercise, while women exhibited an increase after 1 min but not after 3 and 5 min. Overall, men had a significantly higher [Hb] than women at rest and during exercise at both N and MH. There was a significant haemoconcentration from rest to 5 min of exercise in both men [N, 10.6 (0.6)%; MH, 8.9 (0.9)%] and women [N, 9.4 (0.5)%; MH 10.4 (0.7)%], although, the haemoconcentration of men was significantly greater during exercise at N than at MH (Fig. 2).

# $f_c$  and  $O_2$  delivery

Although significantly lower in women than men,  $f_c$  was not significantly different between N and MH at each minute of exercise for both men and women (Fig. 1). Cardiac output is apparently unaffected by acute hypobaria (Stenberg et al. 1966) and, assuming cardiac outputs during 5 min of exercise of 30  $1 \cdot \text{min}^{-1}$  for men and 25  $1 \cdot min^{-1}$  for women, it can be calculated that there was a corresponding reduction in mean  $O_2$  delivery of 198 ml · min<sup>-1</sup> for men and 100 ml · min<sup>-1</sup> for women from N to MH. The reduction in mean  $\dot{V}O_2$  with hypobaria at min 5 was 297 ml·min<sup>-1</sup> in men and 131 ml·min<sup>-1</sup> in women, such that 76% of this reduction in men and 67% in women could be accounted for by a decrease in  $O_2$  delivery.

### Ventilation

 $VO_2$  with hypobaria at min<br>1 131 ml·min<sup>-1</sup> in women,<br>in men and 67% in women<br>crease in O<sub>2</sub> delivery.<br>creased significantly and<br>arcise in both the men and<br>and, throughout exercise,<br>ventilation than women.<br>of exercise wa Both  $V_{\text{ESTPD}}$  and  $V_{\text{EBTPS}}$  increased significantly and  $V_{\text{ESTPD}}$  and  $V_{\text{E BTPS}}$  increased significantly and<br>ssively after 1 min of exercise in both the men and<br>a at N and MH (Fig. 4) and, throughout exercise,<br>ad significantly higher ventilation than women.<br> $\dot{V}_{\text{E BTPS}}$  at progressively after 1 min of exercise in both the men and women at N and MH (Fig. 4) and, throughout exercise, men had significantly higher ventilation than women. While  $V_{\text{E BTPS}}$  at any minute of exercise was not sig- $V_{\text{E BTPS}}$  at any minute of exercise was not sig-<br>ly different between N and MH,  $V_{\text{E STPD}}$  was<br>antly decreased with hypobaria in both men and<br>. During exercise both  $V_{\text{E BTPS}}/VO_2$  and<br> $\sqrt{V}CO_2$  of the women were s nificantly different between N and MH,  $\dot{V}_{\text{ESTPD}}$  was  $V_{\text{E STPD}}$  was<br>th men and<br> $\dot{VQ}_2$  and<br>antly higher<br>he response significantly decreased with hypobaria in both men and women. During exercise both  $V_{\text{E BTPS}}/VO_2$  and  $V_{\text{E BTPS}}/V_{\text{O}_2}$  and<br>ignificantly higher<br>roup, the response  $V_{\text{E BTPS}}/V_{\text{CO}_2}$  of the women were significantly higher  $V_{\text{E BIPS}}/V_{\text{CO}_2}$  of the women were significantly higher<br>than those of the men. Within each group, the response than those of the men. Within each group, the response



**Fig. 4** Ventilatory response of endurance-trained male  $(n = 10)$  and female  $(n = 10)$  athletes during 5-min all-out performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Ventilation is expressed as expired gas at standard temperature and pressure dry ( $V_{\text{E STPD}}$ ), expired gas at body tempera- $V_{\rm E STPD}$ ), expired gas at body tempera-<br>E BTPS), ventilatory equivalent for  $O_2$ <br>equivalent for  $CO_2$  ( $V_{\rm E BTS}/\dot{V}CO_2$ ).<br>mificantly different from normobaria,<br>inficantly different from previous time,<br>ote that, at al ture and pressue saturated ( $V_{\text{E BTPS}}$ ), ventilatory equivalent for O<sub>2</sub>  $V_{\rm E\, BTPS}$ ), ventilatory equivalent for  $O_2$ <br>ry equivalent for  $CO_2$  ( $V_{\rm E\, BTPS}/VCO_2$ ).<br>ignificantly different from normobaria,<br>mificantly different from previous time,<br> $\sqrt{3}$ <br> $\sqrt{3}$  and the values for the men at th  $(\dot{V}_{\rm E\ BTPS}/\dot{V}{\rm O}_2)$ , and ventilatory equivalent for  ${\rm CO}_2$  ( $\dot{V}_{\rm E\ BTPS}/\dot{V}{\rm CO}_2$ ).  $V_{\rm E\,BTPS}/VO_2$ ), and ventilatory equivalent for  $CO_2$  ( $V_{\rm E\,BTPS}/VCO_2$ ).<br>alues are means (SEM) \*Significantly different from normobaria,<br>ithin group comparison; †significantly different from previous time,<br>inthin-gro Values are means (SEM) \*Significantly different from normobaria, within group comparison; †significantly different from previous time, winthin-group comparison. Note that, at all times, the values for women are significantly different from those for the men at the matched chamber pressure

of men and women was generally not different between N and MH, although the  $V_{E\ BTPS}/\dot{V}CO_2$  of women was significantly elevated after 4 and 5 min of exercise at MH compared with N (Fig. 4).

## **Discussion**

 $V_{\rm E\,BTPS}/VCO_2$  of women was<br>
4 and 5 min of exercise at MH<br>
emonstrate that a significant<br>
(total work) was concurrent<br>
and  $\dot{V}O_{\rm 2peak}$ . However, the<br>
these relationships failed to<br>
of the small and relatively<br>
tot This is the first study to demonstrate that a significant decrease in performance (total work) was concurrent with both reduced  $S_aO_2$  and  $\dot{V}O_{2\text{peak}}$ . However, the  $VO_{2\text{peak}}$ . However, the<br>relationships failed to<br>ne small and relatively<br>l work  $[-3.9 (0.9)\%]$ .<br>formance at hypobaria<br>rent for men or women,<br>a significantly greater<br>tudies (Johnson et al.<br>tin and O'Kroy 1993;<br>that hypoxic strength of association of these relationships failed to reach significance because of the small and relatively homogeneous decrease in total work  $[-3.9 \ (0.9)\%]$ . Additionally, the decrease in performance at hypobaria equivalent to 580 m was not different for men or women, even though men experienced a significantly greater decrement in  $\dot{V}\text{O}_{2\text{peak}}$ . Other studies (Johnson et al.  $VO_{2peak}$ . Other studies (Johnson et al.<br>
et al. 1988; Martin and O'Kroy 1993;<br>
992) have shown that hypoxic conditions<br>
lititudes of approximately 2000–3500 m<br>
land reduce  $\dot{VO}_{2max}$ . However,  $\dot{VO}_{2max}$ <br>
as an adequate as 1992; Lawler et al. 1988; Martin and O′Kroy 1993; Powers et al. 1992) have shown that hypoxic conditions that simulate altitudes of approximately 2000–3500 m accentuate EIH and reduce  $\hat{V}\text{O}_{2\text{max}}$ . However,  $\hat{V}\text{O}_{2\text{max}}$ has limitations as an adequate assessment of performance (Snell and Mitchell 1984) and the only other study which has directly assessed the performance consequences of EIH (Koskolou and McKenzie 1994) reported that moderate hypoxaemia, induced by 16.8% inspired oxygen, was associated with reduced performance ( $\approx 4.5\%$ ) but did not alter  $V_{\rm O2max}$ .

 $\dot{V}O_{2\text{max}}$ . However,  $\dot{V}O_{2\text{max}}$ <br>ate assessment of perfor-<br>1984) and the only other<br>ssed the performance con-<br>aemia, induced by 16.8%<br>ated with reduced perfor-<br>aemia, induced by 16.8%<br>ated with reduced perfor-<br>at  $WO_{2max}$ .<br>
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(b) the simulation to the haemon Koskolou and McKenzie (1994) suggested that their result may be a consequence of enhanced muscle blood flow as well as enhanced  $O_2$  extraction at the tissue. On the other hand, the present data support the proposal of Warren et al. (1991) that  $c_aO_2$  should decrease if exercise-induced hypoxemia is to have a negative impact on exercise performance. The normal response to exercise is haemoconcentration (Harrison 1985) with a consequent increase in  $c_aO_2$  above resting levels (Warren et al. 1991). This response was seen in both men and women at normobaria; however, both sexes had significantly lower  $c_aO_2$  after 1, 3 and 5 min of exercise at MH compared with N. At the corresponding times, the  $\dot{V}\text{O}_2$  of both men and women was reduced and W was VO<sub>2</sub> of both men and women was reduced and W was<br>ower, although only significantly so after 1 and 3 min<br>or men. After 5 min of exercise at MH, the women were<br>unable to increase  $c_aO_2$  above resting levels, despite a<br>sim lower, although only significantly so after 1 and 3 min for men. After 5 min of exercise at MH, the women were unable to increase  $c_aO_2$  above resting levels, despite a similar degree of haemoconcentration to that at N, while the men experienced both lower haemoconcentration than at N and  $c_aO_2$  that was not different from that at rest. It is likely that the decrease in  $S_aO_2$ ,  $c_aO_2$  and  $VO_2$  $VO_2$ <br>ntly<br>er 1<br>oser<br>hae-<br>om-<br>and<br>ooth<br>aric<br> $O_2$ . with hypobaria may be attributed to a significantly lower  $P_aO_2$  during exercise at MH, since the  $P_aO_2$  after 1 min of exercise in both men and women fell much closer to the steeper portion of the  $O_2$  loading curve for haemoglobin [N, 10.7 (0.2) kPa; MH, 9.4 (0.2) kPa]. Compared with N conditions, the reduction in  $c_aO_2$  and calculated oxygen delivery after 5 min of exercise in both the men and women when exercising under hypobaric conditions accounted for  $67-76\%$  of the decrease in  $VO_2$ .  $\overline{V}O_2.$ 

This range is similar to the 71.2% reported by Lawler et al. (1988) who used hypoxia (14% O<sub>2</sub>) to reduce  $c_aO_2$ and  $VO_{2\text{peak}}$ , and similar to the 70.5% reported by Gore et al. (1996) when using identical hypobaria for a progressive  $VO_{2\text{max}}$  test.

VO<sub>2peak</sub>, and similar to the 70.5% reported by Gore (1996) when using identical hypobaria for a pro-<br>ive  $\dot{V}O_{2max}$  test.<br>his is also the first study to demonstrate that EIH at<br> $_{\text{peak}}$  is not significantly different b  $\dot{V}O_{2\text{max}}$  test.<br>
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tery  $S_aO_$ This is also the first study to demonstrate that EIH at  $VO_{2\text{peak}}$  is not significantly different between endurance-VO<sub>2peak</sub> is not significantly different between endurance-<br>rained male and female athletes, either at N (92.0 versus<br>22.1%, respectively) or at MH (90.1 versus 89.9%, re-<br>pectively). Although McCusker and Brilla (1992) r trained male and female athletes, either at N (92.0 versus 92.1%, respectively) or at MH (90.1 versus 89.9%, respectively). Although McCusker and Brilla (1992) reported an  $S_aO_2$  of 85% at  $\dot{V}O_{2\text{max}}$  in trained female VO<sub>2max</sub> in trained female<br>tained indirectly using ear<br>mate the incidence of EIH<br>et al. 1992). In contrast,<br>d that five well-trained fe-<br>ce EIH at  $\dot{V}O_{2\text{max}}$ , with a<br>The present findings chal-<br>and Williams (1987) of athletes, their results were obtained indirectly using ear oximetry, which may overestimate the incidence of EIH (Brown et al. 1993; Norton et al. 1992). In contrast, Pedersen et al. (1992) reported that five well-trained female athletes did not experience EIH at  $VO_{2\text{max}}$ , with a radial artery  $S_aO_2$  of  $\approx$  96%. The present findings challenge the proposal of Powers and Williams (1987) of an absolute  $\overline{VO}_{2\text{max}}$  of 4–5 1 min<sup>-1</sup> for development of EIH, since the normobaric  $\dot{V}O_{2\text{peak}}$  of women was 3.6  $1 \cdot \text{min}^{-1}$ . Relative, rather than absolute,  $\dot{V}O_{2\text{peak}}$  may be a more appropriate predictor since the female athletes had a mean  $\hat{VO}_{2\text{peak}}$  of 61 ml·kg<sup>-1</sup>·min<sup>-1</sup>.

 $VO_{2\text{max}}$ , with a<br>t findings chal-<br>ns (1987) of an<br>evelopment of<br>vomen was 3.6<br> $'O_{2\text{peak}}$  may be<br> $[O_{2\text{peak}}]$ .<br>remains equi-<br> $[O_{2\text{peak}}]$ .<br> $[O_{2\text{beam}}]$ <br> $[O_{2\text{beam}}]$ .<br>alues at N and<br> $[O_{2\text{B}}]$ . These results<br>is et al. (  $\dot{V}O_{2\text{max}}$  of 4–5 1 · min<sup>-1</sup> for development of<br>
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iism of EIH remains equi-<br>
one theory suggests an in-<br>
response (Dempsey et al.<br>
marker is a rise in  $P_aCO_2$ <br>
resent  $VO_{2peak}$  may be<br>female athletes<br> $^{-1}$ .<br>I remains equi-<br> $^{-1}$ .<br>I remains equi-<br> $^{-1}$ .<br>A rise in  $P_aCO_2$ <br>a rise in  $P_aCO_2$ <br>aluring values at N and<br>E BTPS was not<br>H. These results<br>rs et al. (1992).<br>(Johnson et al.<br>I.l. 1986)  $\ddot{V}O_{2peak}$  of 61 ml·kg<sup>-1</sup><br>precise mechanism of<br>s et al. 1993), one the<br>perventilatory responsich the cardinal marke<br>values. In the present<br>values. In the present<br>rogressively below res<br>men and women, and<br>ifferent betw While the precise mechanism of EIH remains equivocal (Powers et al. 1993), one theory suggests an inadequate hyperventilatory response (Dempsey et al. 1984) for which the cardinal marker is a rise in  $P_aCO_2$ above resting values. In the present study,  $P_aCO_2$  during exercise fell progressively below resting values at N and MH in both men and women, and  $V_{\text{E BTPS}}$  was not  $V_{\text{E BTPS}}$  was not<br>H. These results<br>ers et al. (1992).<br>5 (Johnson et al.<br>al. 1986) is the<br>H. Although the<br>H. Although the<br>MH, AaDO<sub>2</sub> was<br>wughout exercise<br>to the 1.3-kPa<br>cerbated by ex-<br>effectiveness of<br> $D_2$  relative t significantly different between N and MH. These results are consistent with the findings of Powers et al. (1992). Also consistent with the work of others (Johnson et al. 1992; Powers et al. 1992; Wagner et al. 1986) is the widened  $AaDO<sub>2</sub>$  that accompanied EIH. Although the  $P_AO_2$  was not different between N and MH, AaDO<sub>2</sub> was significantly increased at rest and throughout exercise with hypobaria, but this was consequent to the 1.3-kPa fall in  $P_aO_2$  at rest, and was not exacerbated by exercise. The pH is an indicator of the effectiveness of alveolar ventilation  $(V_A)$  to reduce  $P_aCO_2$  relative to the  $V_A$ ) to reduce  $P_aCO_2$  relative to the ercise (Whipp and Pardy 1986). In H of the women was not different while the men had a significantly in N, after 3 and 5 min of exercise. at MH may be partially a function rk intens high  $VCO_2$  during exercise (Whipp and Pardy 1986). In  $VCO_2$  during exercise (Whipp and Pardy 1986). In<br>urrent study, pH of the women was not different<br>en N and MH, while the men had a significantly<br>r pH at MH than N, after 3 and 5 min of exercise.<br>the higher pH at MH may be the current study, pH of the women was not different between N and MH, while the men had a significantly higher pH at MH than N, after 3 and 5 min of exercise. While the higher pH at MH may be partially a function of lower absolute work intensity, enhanced EIH at MH has the ''benefit'' of improved buffering capacity of reduced haemoglobin. Although the pH of men and women was somewhat different (possibly related to different total Hb mass and [Hb]), metabolic acidosis was not increased with greater hypoxaemia, as speculated by Koskolou and McKenzie (1994). Rather than incurring the metabolic cost to the respiratory muscles of greater  $\dot{V}_A$  to reduce further  $P_aCO_2$  and limit the fall  $V_A$  to reduce further  $P_aCO_2$  and limit the fall<br>considerable buffering capacity of reduced<br>onsequence lower  $S_aO_2$  may be energetically<br>ent. Hopkins and McKenzie 1993) and Otis<br>calso speculated that increasing ventilat in pH, the considerable buffering capacity of reduced Hb as a consequence lower  $S_aO_2$  may be energetically more efficient. Hopkins and McKenzie 1993) and Otis (1964) have also speculated that increasing ventilation in

an attempt to lessen metabolic acidosis may actually worsen it by increasing the  $O_2$  consumption of the respiratory muscles.

In conclusion, this is the first study to show that a significant decrease in performance ( $\approx 3.7\%$  decrease in total work for both men and women) was concurrent with both reduced  $S_aO_2$  and  $\dot{V}O_{2\text{peak}}$  at hypobaria  $VO_{2peak}$  at hypobaria<br>is study also demon-<br> $k$  was not significantly<br>ned male and female<br>ather than absolute,<br>priate predictor of ex-<br>nen and women, it was<br>rease in  $VO_{2peak}$  could<br>in  $O_2$  delivery, which<br>wen mild altitude equivalent to 580 m altitude. This study also demonstrate that hypoxaemia at  $\hat{VO}_{2\text{peak}}$  was not significantly  $\overline{VO}_{2peak}$  was not significantly<br>ce-trained male and female<br>ive rather than absolute,<br>appropriate predictor of ex-<br>For men and women, it was<br>he decrease in  $\overline{VO}_{2peak}$  could<br>crease in  $O_2$  delivery, which<br> $\overline{P}_2$  at different between endurance-trained male and female athletes. Therefore, relative rather than absolute,  $VO_{2\text{peak}}$  may be a more appropriate predictor of ex- $VO_{2peak}$  may be a more appropriate predictor of ex-<br>recise-induced hypoxaemia. For men and women, it was<br>calculated that 67–76% of the decrease in  $\dot{V}\Omega_{2peak}$  could<br>be accounted for by a decrease in  $O_2$  delivery, which<br> ercise-induced hypoxaemia. For men and women, it was calculated that 67–76% of the decrease in  $\dot{V}O_{2\text{peak}}$  could VO<sub>2peak</sub> could<br>elivery, which<br>altitude may<br>ents with high<br>mts with high<br>gas exchange at<br>s. Acta Physiol<br>d hypoxaemia in<br>7–170<br>uestionnaire for<br>f asthma. Chest<br>SM, Stanef T,<br>V(1993) Aerobic<br>durance cycling be accounted for by a decrease in  $O_2$  delivery, which indicates that reduced  $P_1O_2$  at even mild altitude may have ramifications for performance in events with high aerobic energy demands.

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