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Effects of Hypoxic Training on Normoxic Maximal Aerobic Power Output

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Abstract. The responses to 1-leg submaximal and maximal exercise have been studied in four male subjects before and after a 5 week training programme. One leg was trained under normoxic conditions and the other under hypoxic ($F_{IO_2} = 0.12$) conditions for 30 min/day, 3 times/week at a fixed absolute work load which approximated to 75 % of the limb's normoxic $\dot{V}_{O_2max}$. Before and after training both limbs were measured in normoxia, one limb was additionally measured in hypoxia. The aim of the experiments being to use each subject as his own control and to try and elucidate the effects of hypoxia per se as a training stimulus to the improvement of maximal aerobic power output ($\dot{V}_{O_2max}$) measured in normoxia.

The results showed that before training the responses to exercise at submaximal and maximal levels were identical in each limb; the effects of hypoxia being to raise $\dot{V}_{E\,1.5}$ and $f_{H\,1.5}$, to reduce $\dot{V}_{O_2max}$ and to leave $\dot{V}_{O_2\,450}$ unchanged. The effects of the two types of training were to reduce $\dot{V}_{O_2\,450}$, decrease $f_{H\,1.5}$ and increase $\dot{V}_{O_2max}$, the effects being independent of the F_{IO_2}. The changes in $\dot{V}_{O_2max}$ of the hypoxic and normoxic trained legs were related to the initial $\dot{V}_{O_2max}$ of each subjects' limb. It was concluded that our investigation lends no support to the view that hypoxia has either an additive or potentiating effect with exercise during a training programme on the improvement of aerobic power output measured under normoxic conditions.

Key words: Maximal Aerobic Power — Training — Hypoxia — 1-Leg Work.

Exposure to a reduced fractional concentration of oxygen in the inspired air over a prolonged period of time is known to give rise to an increased cardiac output at given oxygen intake, a rise in pulmonary ventilation, an enhanced plasma level of haemoglobin (and possibly myoglobin) concentration and a change in the degree of capillarisation in working muscle (see [1] for general review). All these factors are associated (under normoxic conditions) with a rise in maximal aerobic power output ($\dot{V}_{O_2max}$), but whether hypoxia itself, acts as a training stimulus is unknown. Some studies [15] have claimed to have shown an increase in sea level aerobic performance following prolonged exposure to and work at high altitude, but others [3] have not—the question remains open.

In the present study in order to overcome certain genetic and environmental (*e.g.* the initial starting level of fitness of different individuals [22]) factors which are known to confound the results of even the best planned training studies, we have studied four subjects during 1-leg exercise using each subject as his own control. One limb of each subject was trained under normoxic conditions and the other under hypoxic conditions ($F_{IO_2} = 0.12$) for 30 min/day; 3 times/week over a 5 week period at 75% of the limbs $\dot{V}_{O_2max}$ under normoxic conditions. Each limb was subjected to exactly the same absolute work intensity and since we have shown that a pair of limbs from a given subject are physiologically similar, the only variable factor in the training regimen was the degree of hypoxia. Thus any differences in the aerobic power output of a pair of limbs following training should be attributable to this factor alone and not masked by other biological variables.

Material and Methods

The material for this investigation were four healthy male subjects aged 24 to 36 years. Their physical characteristics were as follows: weight, 79.4 ± 11.6 kg; height, 180.2 ± 8.0 cm; lean body mass (estimated from skinfold thickness and body weight), 68.0 ± 7.6 kg; and limb muscle (plus bone) volume, 7.74 ± 0.67 l (right leg) and 7.34 ± 0.56 l (left leg). The anthropometric measurements were taken by methods previously described in detail [8]. The subjects were all interested in physical activity but none was in training before the commencement of the investigation. In preliminary experiments they were allowed to become accustomed to pedalling the bicycle with 1-leg and to breathing hypoxic gas mixtures. The details of the bicycle used for these experiments have been given elsewhere [8]. It was a fixed wheel ergometer and the subject's foot was placed in a plimsoll which was attached firmly to the pedal with bolts. No attempts were made to return the crank to the upright position with springs or by any other means (*c.f.* Freyschuss and Strandell [12]). The subjects were measured either in triplicate (SM and WP) or duplicate (AT and PI) during submaximal and maximal exercise before and after training. The criteria used as evidence for maximal aerobic power ($\dot{V}_{O_2max}$) having been reached has been outlined previously [6, 8, 9] and the same method was used in this study. The subject's legs were measured in normoxia and in addition the leg to be trained under hypoxic conditions was measured in hypoxia at the onset and end of the training programme. Thus a total of 60 measurements of 1-leg exercise performance was made on the four subjects during the period of the investigation. The subjects trained 3 times/week for 30 min at a set absolute work load (approximately 75 % of their normoxic 1-leg $\dot{V}_{O_2max}$ before training) for 5 weeks. One leg was trained under normal environmental conditions ($F_{IO_2} = 0.21$) and the other in hypoxia ($F_{IO_2} = 0.12$). Each training session was rigidly supervised and the work output and cardiac frequency recorded for each subject throughout the training sessions.

The physiological responses to exercise were measured in a conventional way by the open circuit technique using a mixing chamber to sample mixed expired gases [10]. Cardiac frequency was monitored using a differential amplifier coupled to a linear instantaneous rate meter [19]. The results for submaximal exercise were analysed and expressed in terms of minute ventilation ($\dot{V}_E$) at a $\dot{V}_{CO_2}$ of 1.5 l · min^{-1} ($\dot{V}_{E\,1.5}$), tidal volume (V_T) at $\dot{V}_E$ of 30 l · min^{-1} ($V_{T\,30}$), oxygen intake

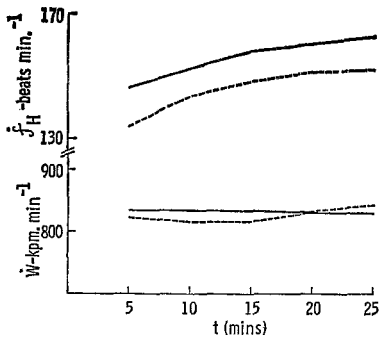

Fig. 1. The work output ($\dot{W}$ — kpm · min^{-1}) and cardiac frequency (f_H) of the leg trained in hypoxia (———) and normoxia (- - - -). The lines represent the mean of 15 training sessions for each limb of the four subjects

($\dot{V}_{O_2}$) at a work output ($\dot{W}$) of 450 kpm · min^{-1} ($\dot{V}_{O_2\,450}$), cardiac frequency (f_H) at a $\dot{V}_{O_2}$ of 1.5 l · min^{-1} ($f_{H\,1.5}$) and a $\dot{V}_{O_2}$ at an f_H of 175 beats · min^{-1} [4, 8, 9]. During maximal exercise the mixing chamber was replaced by a Douglas bag and the subjects breathed directly into this via a low resistance (Otis-McKerrow) mouthpiece and a short piece (internal diameter $1^1/_2$") of smooth-bore tubing. The hypoxic mixture during the measurements before, during and after the training period was delivered to the subjects by rotameters (arranged in a way similar to that described by Cunningham *et al.* [5]) via a 500 l Douglas bag. The gas concentrations of the mixture was monitored continuously by automatic (paramagnetic O_2 and infra red CO_2) analysers.

Results

Throughout the period of the investigation the physical characteristics of the subjects showed no significant changes.

Submaximal Responses to Exercise

Before training the submaximal responses to training of each limb were identical under normoxic conditions (Table 1). The effects of hypoxia were to raise $\dot{V}_{E\,1.5}$ from 45.48 ± 4.85 to 57.59 ± 6.61 l · min^{-1} ($P < 0.001$) and $f_{H\,1.5}$ from 127 ± 11 to 146 ± 9 beats · min^{-1} ($P < 0.001$) but $\dot{V}_{O_2}$ for given $\dot{W}$ of 450 kpm · min^{-1} remained unchanged. In three subjects the increased $\dot{V}_{E\,1.5}$ was mainly due to an increase in V_T; f_H remaining unchanged, but in the remaining subject the opposite was true. Hypoxia reduced the $\dot{V}_{O_2}$ at an $f_{H\,175}$ from 2.51 ± 0.23 to 2.02 ± 0.18 l · min^{-1} ($P < 0.001$).

The effects of the two types of training were to reduce $\dot{V}_{O_2\,450}$ and $f_{H\,1.5}$, increase $\dot{V}_{O_2\,175}$ and leave $\dot{V}_{E\,1.5}$ and $V_{T\,30}$ unchanged. These changes appeared to be independent of whether the leg was trained in

Table 1. Submaximal training responses to 1-leg exercise. One leg of each subject was trained under normoxic conditions ("normoxic" leg), the other under hypoxic conditions ("hypoxic" leg). Both were measured in normoxia before and after training. In addition the "hypoxic" leg was measured in a similar way in hypoxia. The mean (± SD) data have been expressed in terms of a ventilation rate at a $\dot{V}_{CO_2}$ of 1.5 l · min^{-1} ($\dot{V}_{E\,1.5}$), tidal volume at a $\dot{V}_E$ of 30 l · min^{-1} ($V_{T\,30}$), a $\dot{V}_{O_2}$ at a work rate of 450 kpm · min^{-1} ($\dot{V}_{O_2\,450}$), cardiac frequency at a $\dot{V}_{O_2}$ of 1.5 l · min^{-1} ($f_{H\,1.5}$) and a $\dot{V}_{O_2}$ at an f_H of 175 beats/min^{-1} ($\dot{V}_{O_2\,175}$)

Variable	Units	Normoxia ($F_{IO_2} = 0.21$)				Hypoxia ($F_{IO_2} = 0.12$)	
		"normoxic" leg		"hypoxic" leg		"hypoxic" leg	
		before	after	before	after	before	after
$\dot{V}_{E\,1.5}$	l · min^{-1} BTPS	49.08 ±5.67	47.73 ±3.27	45.48 ±4.85	47.81 ±4.21	57.59**** ±6.61	61.40**** ±8.59
$V_{T\,30}$	l · BTPS	1.75 ±1.10	1.61 ±0.52	1.50 ±0.76	1.50 ±0.37	1.75 ±0.47	1.51 ±0.15
$\dot{V}_{O_2\,450}$	l · min^{-1} STPD	1.31 ±0.07	1.18*** ±0.07	1.31 ±0.06	1.14*** ±0.07	1.31 ±0.04	1.20 ±0.14
$f_{H\,1.5}$	beats · min^{-1}	131 ±10	118** ±10	127 ±11	118* ±13	146**** ±9	132**** ±8
$\dot{V}_{O_2\,175}$	l · min^{-1} STPD	2.36 ±0.21	2.86*** ±0.18	2.51 ±0.23	2.71* ±0.28	2.02**** ±0.18	2.44**** ±0.17

Significance: Before—after *** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$. Normoxia—hypoxia **** $P < 0.001$.

Table 2. Maximal responses to 1-leg exercise before and after training. Absolute ($\dot{V}_{O_2max}$) and net ($\dot{V}_{O_2max\ net}$) maximal aerobic power maximal ventilation ($\dot{V}_{E\,max}$) and cardiac frequency ($f_{H\,max}$). Mean (± SD) data as Table 1

Variable	Units	Normoxia ($F_{IO_2} = 0.21$)				Hypoxia ($F_{IO_2} = 0.12$)	
		normoxic leg		hypoxic leg		hypoxic leg	
		before	Δ after	before	Δ after	before	Δ after
$\dot{V}_{Emax}$	l · min^{-1} BTPS	129.0 ±2.4	+ 9.8 ±10.9	129.4 ±4.8	+11.3 ±11.4	129.3 ±8.8	— 2.45 ±20.90
$\dot{V}_{O_2max}$	l · min^{-1} STPD	2.83 ±0.12	+ 0.24** ± 0.20	2.79 ±0.13	+ 0.34** ± 0.19	2.25**** ±0.11	+ 0.21** ± 0.13
$\dot{V}_{O_2max\ net}$	l · min^{-1} STPD	2.34 ±0.12	+ 0.28** ± 0.16	2.34 ±0.09	+ 0.36** ± 0.19	1.87**** ±0.13	+ 0.18** ± 0.08
f_{Hmax}	beats · min^{-1}	177 ±7	+ 4 ± 5	177 ±8	+ 4 ± 7	179 ±7	— 1 ± 5

Significance: Before normoxic—hypoxic leg in normoxia, NS. Before normoxia—hypoxia ****$P < 0.001$.
Before and after training ** $P < 0.001$.

Analysis of variance before and after training in normoxia and hypoxia

	Df	SS	MS	F-ratio	Sign
Between subjects	3	0.0508	0.0169	2.32	NS
Between types of training	1	0.0149	0.0149	2.04	NS
Error	2	0.0146	0.0073		

hypoxia or normoxia (Table 1). Thus the relationship of the various submaximal physiological variables to each other for each limb remained unchanged before and after training.

Maximal Responses to Exercise

The changes in responses to maximal exercise before and after training are summarised in Table 2. Before training there was no significant differences between the right and left legs of the subjects. Following training the $\dot{V}_{E\max}$, $\dot{V}_{O_2\max\ net}$ and $f_{H\max}$ of the leg trained in normoxia changed by $+ 9.8 \pm 10.9\ l \cdot min^{-1}$; $+ 0.28 \pm 0.16$ ($P < 0.01$); and 4 ± 5 beats $\cdot$ min^{-1} respectively. The corresponding figures for the opposite limb trained in hypoxia were $+ 11.3 \pm 11.4\ l \cdot min^{-1}$; $0.36 \pm 0.19\ l \cdot min^{-1}$ ($P < 0.01$); and $+ 4 \pm 7$ beats $\cdot$ min^{-1}. An analysis of variance revealed no significant differences between the two limbs following the two different types of training.

The effects of hypoxic training on the aerobic power output of the leg which was measured before and after training in hypoxia (in addition to normoxia—see "Material and Methods") are also shown in Table 2. Before training, maximal exercise in hypoxia ($F_{IO_2} = 0.12$) resulted in 0.54 l $\cdot$ min (19%) decrease in 1-leg $\dot{V}_{O_2\max}$.

Following training the changes in "hypoxic" $\dot{V}_{O_2\max}$ both in absolute (l $\cdot$ min) and relative (%) terms was less than the corresponding figures for the same "hypoxic trained" leg measured in normoxia. As a consequence of this the difference between the normoxic and hypoxic $\dot{V}_{O_2\ max}$ widens after training.

Discussion

Interest in hypoxia as a possible training stimulus was revived by the decision to hold the Olympic games at Mexico City in 1968 but since the initial spate of publications on the subject (see Margaria [18] for general review) discussion of the topic in the literature appears to have declined. Nevertheless, the major question remains: does exercise and hypoxia have an additive effect and thereby produce an improvement in performance over and above that produced by the same training in normoxia? If proven, the advantages to athletes are clear but an affirmative answer to the question may also have application to other fields, such as rehabilitation and occupational medicine, where one is often concerned with facilitating the patients' return to an active normal life, within a short a time-span as possible.

Klausen *et al.* [17] were the first to show that altitude induced hypoxia may have a substantial affect on $\dot{V}_{O_2\max}$ on return to sea level. They observed in 12 ♂ subjects a 14% increase in $\dot{V}_{O_2\max}$ after a 5 week stay

at altitude of 3800 m. Their results found agreement in the later work of Dill and Adams [11] and the study of Bannister *et al.* [2] who used an environmental chamber to simulate altitude conditions. In contrast, Hansen *et al.* [16] and Buskirk *et al.* [3] found no change in $\dot{V}_{O_2max}$ on return to sea level following training and exposure to altitude. However, all these studies are difficult to interpret for various reasons. The investigations of Klausen, Buskirk, Dill and Bannister used no controls, whereas the study of Hansen took this precaution but failed to standardise the training regimen adequately at altitude and sea level so that the effects of training were not directly comparable. The only study, to our knowledge, which used control subjects and made attempts to gain equivalence between the hypoxic training at altitude and normoxic training at sea level is that of Roskamm *et al.* [20]. They related the training programme to the initial fitness of their subjects and measured three groups of ♂s under simulated conditions at altitudes of 3450 m, and 2250 m and at 260 m. Their data indicate mean changes of + 10.0% and + 17.5% in $\dot{V}_{O_2max}$ measured at 260 m for those who trained at the simulated altitudes compared with 6.4% for those who were trained and measured at 260 m.

We have approached the problem in an entirely different manner. Using our previous experience in 1-leg work [8] we designed our experiments to see if we could assess the influence of hypoxia per se on maximal aerobic power output. The use of 1-leg exercise enabled us to use each subject as his own control whilst training under both normoxic and hypoxic conditions. The training stimulus in terms of absolute work output performed was constant for both legs, throughout the training programme and the $\dot{V}_{O_2max}$ of each subjects' right and left leg before training commenced was closely similar (Table 2). Thus the two most important criteria for assessing the effects of a training programme were met: a constant training stimulus under controlled environmental (F_{IO_2}) conditions and the standardisation of the confounding influence of the initial level of fitness of a subject on his subsequent improvement in $\dot{V}_{O_2max}$ performance [22]. Under these circumstances it is clear that the effect of hypoxia per se on aerobic power improvement is minimal. We find no clear evidence that training in reduced F_{IO_2} enhances or potentiates the improvement of $\dot{V}_{O_2max}$ as observed under normoxic conditions (Table 2).

Hypoxia per se, as has been shown many times [1], increases $f_{H\,1.5}$ and $\dot{V}_{E\,1.5}$ for given $\dot{V}_{O_2}$ and reduces $\dot{V}_{O_2max}$ but has little or no effect on the O_2 cost of work. Training in hypoxia though it has the effect of changing the former three parameters in the opposite direction in the "hypoxic" leg (see Table 1) when the magnitude of the changes are compared to normoxia, the differences between f_H and $\dot{V}_E$ in the two conditions

remain unchanged whilst the *difference* in $\dot{V}_{O_2max}$ actually shows a small (4%) but significant increase after training (Table 2; *c.f.* Saltin [21]). The reduction of $\dot{V}_{O_2max}$ in hypoxia as found by Gleser [14] is associated with the fall in F_{IO_2} (Table 2). This is in direct contrast to the findings for the effect of hyperoxia on 1 limb performance [8].

The effects of hypoxia per se on $\dot{V}_{O_2max}$ must be borne in mind when interpreting our observations on the respective merits of training under hypoxic and normoxic condition. We chose to require each subject to exercise at a constant absolute work level which represented approximately 75% of his normoxic $\dot{V}_{O_2max}$. Thus under hypoxic conditions the relative work performed rose to nearer 90% during the greater part of the training period. Since the relative (as well as the absolute) work intensity is known to have an effect on the improvement of $\dot{V}_{O_2max}$ observed in a training programme [7] a critic of our results may argue that our data are biased in favour of the limb trained in hypoxia. However, though we would accept such criticism as fair, we would point out that the additional benefits of training at intensities of exercise beyond 80% $\dot{V}_{O_2max}$ has not been documented and is open to question [1] and in any case such arguments would only strengthen our case. Since despite the possibility of a training bias towards hypoxia analysis of co-variance of our data clearly reveals no statistical evidence for a difference in the two forms of training stimuli.

This is an important finding but one must be careful not to extend beyond the experimental conditions under which it has been established. Firstly, it must be emphasised that our data were obtained from exercise with 1 limb. We have argued [8] previously that the essential limitation to such exercise is probably peripheral and not central and more dependent upon flow to, and utilization of oxygen within, the leg's muscle rather than the output of the heart. It therefore seems unlikely that our results can be applied directly to other forms of work and in particular, normal 2-legged cycling or running. Secondly, the fact that we have shown no differences to exist between "hypoxic" and "normoxic" training cannot be interpreted to mean that exercise in hypoxia is necessarily without effect. Our experiments were of relatively short duration (5 weeks) and Fusancho *et al.* [13] have clearly shown the importance of duration of exposure to hypoxia and age at which it is initiated on the changes observed at altitude. One cannot say what the effects might have been if our experiments had been prolonged, or younger (or older) subjects had been used. Certainly it would be difficult to explain the high values of $\dot{V}_{O_2max}$ found in high altitude natives [23] without invoking some of these factors. Thus, we cannot refute unequivocally the theory that training in hypoxia or at altitude has a beneficial effect on sea level physical performance but our present results seriously question

its physiological basis. Our experiments indicate that provided the absolute work stimulus is constant, the training effect of cycling is independent of the F_{IO_2} in which the limb is trained.

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