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The influence of cardiorespiratory fitness on the decrement in maximal aerobic power at high altitude

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Summary. There are conflicting reports in the literature which imply that the decrement in maximal aerobic power experienced by a sea-level (SL) resident sojourning at high altitude (HA) is either smaller or larger for the more aerobically "fit" person. In the present study, data collected during several investigations conducted at an altitude of 4300 m were analyzed to determine if the level of aerobic fitness influenced the decrement in maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) at HA. The $\dot{V}_{O_{2\max}}$ of 51 male SL residents was measured at an altitude of 50 m and again at 4300 m. The subjects' ages, heights, and weights (mean ± SE) were 22 ± 1 yr, 177 ± 7 cm and 78 ± 2 kg, respectively. The subjects' $\dot{V}_{O_{2\max}}$ ranged from 36 to 60 $\mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$ (mean ± SE = 48 ± 1) and the individual values were normally distributed within this range. Likewise, the decrement in $\dot{V}_{O_{2\max}}$ at HA was normally distributed from 3 $\mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$ (9% $\dot{V}_{O_{2\max}}$ at SL) to 29 $\mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$ (54% $\dot{V}_{O_{2\max}}$ at SL), and averaged 13 ± 1 $\mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$ (27 ± 1% $\dot{V}_{O_{2\max}}$ at SL). The linear correlation coefficient between aerobic fitness and the magnitude of the decrement in $\dot{V}_{O_{2\max}}$ at HA expressed in absolute terms was $r = 0.56$, or expressed as % $\dot{V}_{O_{2\max}}$ at SL was $r = 0.30$; both were statistically significant ($p < 0.05$). Therefore, only 31 and 9%, respectively, of the variability in the decrement at HA could be attributed to the $\dot{V}_{O_{2\max}}$ at SL. Thus, while the more aerobically fit individuals do tend to suffer a larger decrement in maximal aerobic power at altitude, the level of aerobic fitness *per se* accounts for a relatively small amount of the variability between individuals in this decrement.

Key words: Hypoxia – $\dot{V}_{O_{2\max}}$ – Physical fitness

Introduction

The maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) of sea level residents is reduced by exposure to hypoxia. The amount of reduction is proportional to the reduction in the partial pressure of oxygen in the inspired air. The relationship between the reduction in $\dot{V}_{O_{2\max}}$ and altitude has been estimated to be on the order of 10% (of sea level $\dot{V}_{O_{2\max}}$) for every 1000 m ascended beyond an altitude of 1500 m above sea level, with little or no apparent decrement in $\dot{V}_{O_{2\max}}$ between sea level and 1000 m (Buskirk 1969; Hartley 1971). There is, however, considerable individual variability in the magnitude of this decrement at high altitude. This variability is particularly significant at moderate altitudes (1000-2000 m) where the decrement is small (Squires and Buskirk 1982) and close to the limit of precision of measurement. The degree of aerobic fitness has been said to modify the relationship between altitude and the decrement in $\dot{V}_{O_{2\max}}$ (Buskirk 1969; Grover et al. 1967; Saltin 1967). In two studies (Grover et al. 1967; Saltin et al. 1967) the decrement in $\dot{V}_{O_{2\max}}$ of highly trained athletes was reported to be greater than would have been expected based on observations made on less fit individuals. On the other hand, Buskirk (1969) studied the results of a number of investigations, and concluded that the decrement was smaller for fit persons than for less fit. The purpose of this investigation was to determine the extent to which aerobic fitness influenced the size of the decrement in $\dot{V}_{O_{2\max}}$ at high altitude.

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Methods

Data collected from fifty-one male soldiers participating in several investigations were used in this study. Each subject had been fully informed as to the nature, requirements and potential risks of participation. All were permanent residents of sea level (SL) who had not sojourned at high altitude (HA) for at least three months prior to their participation. The subjects' ages, heights, and weights were (mean ± SE) 22 ± 1 yr, 177 ± 7 cm and 78 ± 2 kg, respectively.

$\dot{V}_{O_{2max}}$ was determined at SL (50 m) and at HA (4,300). The amount of time spent at HA prior to determination of $\dot{V}_{O_{2max}}$ varied between subjects from one to eight days. The $\dot{V}_{O_{2max}}$ was measured using either a discontinuous cycling protocol (Kamon 1972) for 29 subjects, or a continuous cycling protocol (McArdle et al. 1973) for 22 subjects. For the discontinuous tests, subjects performed exercise bouts (approximately 4 min) of increasing intensity until an increase of 30 W produced an increase in oxygen uptake ($\dot{V}_{O_2}$) of less than 150 ml · min^{-1}. For the continuous tests, exercise intensity was progressively increased at regular intervals until the subject could no longer continue. The highest $\dot{V}_{O_2}$ measured during exercise was taken as maximum, and a plateau of $\dot{V}_{O_2}$ with increased exercise intensity was obtained in the majority of these tests.

Open-circuit spirometry was used to measure $\dot{V}_{O_2}$ and carbon dioxide production ($\dot{V}_{CO_2}$). For the discontinuous tests, timed collections of expired air were made. Expired gas volumes were measured using a Tissot spirometer, and O_2 and CO_2 concentrations were measured using an oxygen fuel cell (Applied Electrochemistry S-3A) and infrared CO_2 (Beckman LB-2) gas analyzers, respectively. The gas analyzers were calibrated before each experiment using gases of known composition (mass spectrometer). For the continuous tests, respiratory exchange was determined every 30 s using a semi-automated system. Expired gas volumes were measured with a flow transducer (Hewlett-Packard) and a 3 pneumotach; O_2 and CO_2 concentrations were determined as before. Analog signals from the flow transducers and gas analyzers were digitized, and $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ (STPD) and $\dot{V}_E$ (BTPS) were calculated using modified computational procedures of Sue et al. (1980).

Standard statistical procedures were used to analyze the data. The decrement in $\dot{V}_{O_{2max}}$ at 4300 was calculated both as the absolute difference (ml · kg^{-1} · min^{-1}) between SL and HA, as well as the % of $\dot{V}_{O_{2max}}$ at SL which was lost at HA. Body weight, $\dot{V}_{O_{2max}}$ at SL and decrement in $\dot{V}_{O_{2max}}$ at HA (both methods of computation) were tested for normality of distribution using the Chi-square goodness of fit test (Snedecor and Cochran 1967). Linear regression was used to determine the correlation between aerobic fitness and the decrement (both computations) in $\dot{V}_{O_{2max}}$ at HA (Snedecor and Cochran 1967). The level of significance was $P < 0.05$.

Results

The subjects' weights ranged from 55 to 109 kg with a mean ± SE of 78 ± 2 kg, and the values were normally distributed ($\chi^2 = 2.10$, $df = 2$, $0.25 < P < 0.50$) within this range. The values of $\dot{V}_{O_{2max}}$ at SL were also normally distributed ($\chi^2 = 5.11$, $df = 4$, $0.25 < P < 0.50$) ranging from 36 to 60 ml · kg^{-1} · min^{-1} with a mean ± SE of 48 ± 1 ml · kg^{-1} · min^{-1}. The distribution of decrement in $\dot{V}_{O_{2max}}$ at HA is shown in Fig. 1. The absolute decrement (Fig. 1A) in $\dot{V}_{O_{2max}}$ at HA was normally distributed ($\chi^2 = 3.38$, $df = 3$, $0.25 < P < 0.50$) between 3 and 29 ml · kg^{-1} · min^{-1} with a mean ± SE of 13 ± 1 ml · kg^{-1} · min^{-1}. Similarly, the % of $\dot{V}_{O_{2max}}$ at SL which was lost at HA (Fig. 1B) was normally distributed ($\chi^2 = 1.80$, $df = 5$, $0.75 < P < 0.90$) from 9 to 54% and averaged 27 ± 1%.

In Fig. 2, the absolute decrement in $\dot{V}_{O_{2max}}$ at HA is plotted as a function of the $\dot{V}_{O_{2max}}$ at SL. The linear regression equation was $Y = 0.52\ X - 11.39$, where Y is the absolute decrement and X is the $\dot{V}_{O_{2max}}$ at SL. The linear correlation coefficient (r) between these two variables was 0.56,

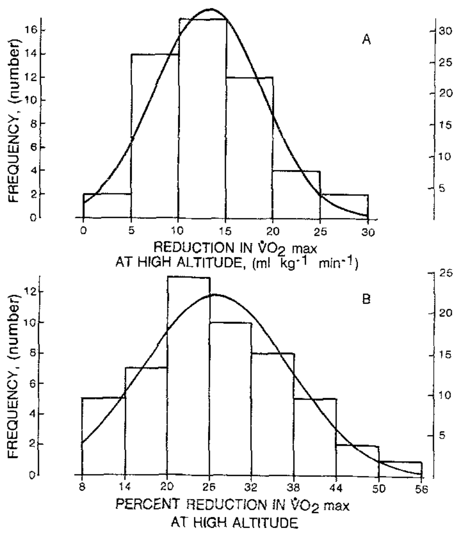

Fig. 1. Distribution of the decrements in maximal oxygen uptake ($\dot{V}_{O_{2max}}$) experienced by the subjects expressed as the absolute difference between sea level and high altitude **A** or expresses as the % difference **B**

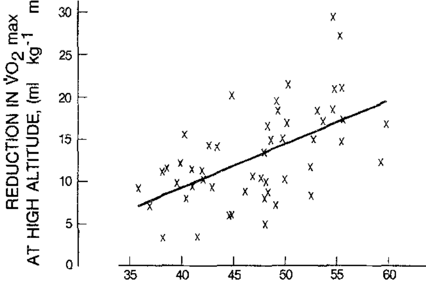

Fig. 2. The absolute decrement in maximal oxygen uptake ($\dot{V}_{O_{2max}}$) at high altitude plotted as a function of $\dot{V}_{O_{2max}}$ at sea level. Solid line denotes regression line ($Y = 0.52\ X - 11.39$, $r = 0.56$) and individual values are denoted on the graph by X

indicating that the slope of the regression line was significantly ($p < 0.05$) different from zero. However, only 31% of the variance in the absolute decrement was accounted for by the $\dot{V}_{O_{2\max}}$ at SL. Figure 3 shows the decrement expressed as % $\dot{V}_{O_{2\max}}$ at SL plotted as a function of the $\dot{V}_{O_{2\max}}$ at SL. The corresponding regression equation was $Y = 0.50\ X + 3.49$. The r for these two variables was 0.30. While this again indicated that the slope of the regression line was significantly ($p < 0.05$) different from zero, only 9% of the variance in the relative decrement was accounted for by the $\dot{V}_{O_{2\max}}$ at SL. Thus, the more aerobically fit men did tend lose a greater portion of their maximal aerobic power at HA.

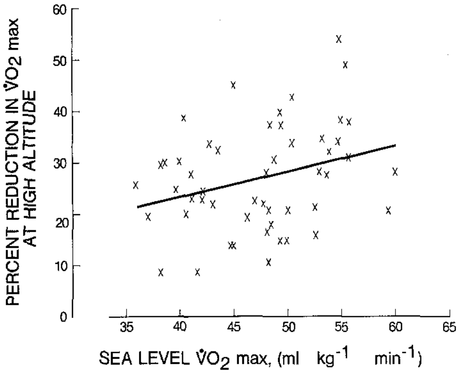

Fig. 3. The % decrement in maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) at high altitude plotted as a function of $\dot{V}_{O_{2\max}}$ at sea level. Solid line donotes regression line ($Y = 0.50\ X + 3.49$; $r = 0.30$) and individual values are denoted on the graph by X

Discussion

Maximal oxygen uptake is directly proportional to the maximum rate of systemic oxygen transport, which is the product of arterial oxygen content (C_aO_2) and maximum cardiac output ($\dot{Q}_{\max}$). With acute (<24 h) hypoxic exposure, $\dot{Q}_{\max}$ is unchanged from SL, but arterial oxygen saturation, thus C_aO_2, is reduced resulting in a decrement in $\dot{V}_{O_{2\max}}$ (Horstman et al. 1980). After some days at HA, C_aO_2 returns towards normal (due to an increased hematocrit), but $\dot{Q}_{\max}$ becomes reduced (due to an decreased maximum stroke volume and heart rate) with the net effect that $\dot{V}_{O_{2\max}}$ remains depressed (Saltin et al. 1968).

Both Buskirk (1969) and Hartley (1971) have collated data from a number of studies conducted at different altitudes, and have constructed graphs in which $\dot{V}_{O_{2\max}}$ is expressed as a function of altitude. From these graphs it may be estimated that, beginning at 1500 m above SL, $\dot{V}_{O_{2\max}}$ declines about 10% for each additional 1000 m ascended. There is, however, a good deal of variability in the decrement experienced, especially when moderate altitudes are considered. For example, Saltin (1967) reported that the decrement in $\dot{V}_{O_{2\max}}$ experienced by eight male athletes at 2250 m ranged from 9 to 22% of their $\dot{V}_{O_{2\max}}$ at SL, and in another study (Saltin et al. 1968) of four subjects at 4300 m, decrements were reported which ranged from 19 to 32%. In the present study, the decrement at 4300 m averaged 27% of the $\dot{V}_{O_{2\max}}$ at SL, almost exactly the 28% which would be predicted based on the graphs constructed by Buskirk (1969) and Hartley (1971).

Although some investigators have reported that the level of aerobic fitness influences the magnitude of the decrement in $\dot{V}_{O_{2\max}}$ at HA, consensus is lacking. However, the previous investigations reporting a relationship between fitness and $\dot{V}_{O_{2\max}}$ at altitude were all based on studies of small numbers of subjects having a limited range of aerobic fitness. The present study indicates that the degree of aerobic fitness, as indicated by $\dot{V}_{O_{2\max}}$, accounts for relatively little of the variability in the decrement in $\dot{V}_{O_{2\max}}$ at HA. This finding is based on results from a very large sample size having a wide range of individual aerobic fitness, body weight, and decrement in $\dot{V}_{O_{2\max}}$ at HA. The subject group was normally distributed with respect to these parameters. Irrespective of how the decrement is expressed ($\mathrm{ml \cdot kg^{-1} \cdot min^{-1}}$, or % $\dot{V}_{O_{2\max}}$ at SL), most of the variability in the decrement at HA must be accounted for by factors other than physical fitness, *per se*.

A number of other such factors can be postulated. The physiological responses to extreme environmental heat (Drinkwater and Horvath 1979) and cold (Leblanc et al. 1978) are influenced to some degree by the age of the subject, so perhaps responses to hypoxia are also age-related. The present study cannot adequately address this issue since the age range of subjects was extremely narrow (18 to 31 years), but for this group there was no significant correlation between age and the decrement in $\dot{V}_{O_{2\max}}$ ($r = 0.03$). It could be suggested that the decrement in $\dot{V}_{O_{2\max}}$ is related to the length of time spent at HA, but the $\dot{V}_{O_{2\max}}$ of SL residents does not change during the first 15 days of HA residence (Saltin et al. 1968; Young et al. 1982). The degree of arterial oxygen saturation during maximal exercise at altitude may vary between individuals. Dempsey et al. (1978) have reported that certain individuals at SL, notably elite track athletes, show significant hypoventilation and significant hypoxemia at or near their $\dot{V}_{O_{2\,max}}$. These individuals possibly suffer a larger decrement in $\dot{V}_{O_{2\,max}}$ at altitude as compared to normal individuals who do not experience arterial desaturation during maximal exercise. Individuals such as these were not included among the subjects of the present study, where only five subjects had $\dot{V}_{O_{2\,max}}$ between 55 and 60 $ml \cdot kg^{-1} \cdot min^{-1}$ and none exceeded 60 $ml \cdot kg^{-1} \cdot min^{-1}$.

In conclusion, the results of this study indicate that the average decrement in maximal aerobic power of a group of SL residents acutely exposed to HA is reasonably well predicted by the relationship previously described by Buskirk (1969) and by Hartley (1971). There is a tendency for the more aerobically fit individual to incur a larger decrement in maximal aerobic power at high altitude. However, there is considerable interindividual variability in the observed decrement, whether expressed in absolute terms or relative to $\dot{V}_{O_{2\,max}}$ at SL, and a relatively small amount of this variability can be accounted for by the individual's level of aerobic fitness.

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