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Revision 1

How important is $\dot{V}O_2$max when climbing Mt. Everest (8,849 m)?

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Abstract

The maximal rate of oxygen uptake ($\dot{V}O_2max$) of humans declines with increasing altitude, but represents the upper limit of aerobic endurance performance at low and high altitude as well. Before Reinhold Messner and Peter Habeler climbed Mt. Everest first (1978) without supplemental oxygen, physiologists have doubted whether this would be possible due to insufficient $\dot{V}O_2max$ remaining when approaching the summit (8,849 m). Subsequently, several studies evaluated the decline in the $\dot{V}O_2max$ levels at real and simulated extreme altitudes. However, the potential influence of the preexisting individual sea level $\dot{V}O_2max$ remained largely unconsidered. Based on available studies and case observations, here we discuss the observed and expected decline of $\dot{V}O_2max$ up to 8,849 m dependent on the individual sea level $\dot{V}O_2max$. It is concluded that a high sea level $\dot{V}O_2max$ and an only moderate decline of arterial oxygen saturation and associated $\dot{V}O_2max$ with increasing altitude, due to appropriate acclimatization and ascent strategies, enable certain mountaineers to climb 8,000er summits and even the Everest without supplemental oxygen.

1. Forty-three years ago, Mt. Everest was climbed first without bottled oxygen

The maximal rate of oxygen uptake ($\dot{V}O_2max$) of a human being dictates the upper limit of aerobic endurance performance and thus, how fast a given track distance can be accomplished. Barometric pressure (BP) and related partial pressure of oxygen (pO_2) decrease with increasing altitude, progressively reducing individual $\dot{V}O_2max$ and endurance performance. Therefore, it seems logical that a certain level of $\dot{V}O_2max$ is a prerequisite for climbing high mountains. Before Reinhold Messner and Peter Habeler climbed Mt. Everest first (1978) without supplemental oxygen, physiologists have doubted whether this would be possible. Doubts were primarily based on research findings obtained during the unique Himalayan Scientific and Mountaineering Expedition in 1960 to 1961 (Pugh et al., 1964). In that study, $\dot{V}O_2max$ data were collected up to an altitude of 7,440 m, which indicated an extremely low $\dot{V}O_2max$ of about 5 mL/min/kg when extrapolated to the summit of Mt. Everest (8,849 m) (Pugh et al., 1964; West and Wagner, 1980). Such $\dot{V}O_2max$ levels are barely larger than $\dot{V}O_2$ needed during resting conditions at sea level (about 3.5 mL/min/kg), absolutely not compatible with climbing activities close to the summit of Everest. Those calculations were based on an average sea level $\dot{V}O_2max$ of about 50 mL/min/kg (Pugh et al., 1964; West and Wagner, 1980). West and Wagner highlighted the critical importance of only small changes in barometric pressure and associated diffusion limitation of oxygen transfer by the blood-gas barrier in those extreme hypoxic conditions, likely explaining the unexpected success of Messner and Habeler (West and Wagner, 1980).

2. The importance of the individual sea level $\dot{V}O_2max$ to climb at extreme altitudes

The potential influence of the individual $\dot{V}O_2max$ remained largely unconsidered. This aspect has been addressed during the American Medical Research Expedition to Everest (AMREE) in 1981, where 2 male subjects (marathon runners) with an average sea level $\dot{V}O_2max$ of about 61mL/min/kg demonstrated a slightly left-shifted decline of $\dot{V}O_2max$ with increasing altitude (West et al., 1983), when compared to data reported by Pugh et al. (Pugh et al., 1964) (figure 1). Despite the initially steeper decline of $\dot{V}O_2max$, the higher $\dot{V}O_2max$ at sea level resulted in a clearly higher $\dot{V}O_2max$ of about 15 mL/min/kg at the top of Mt. Everest (simulated: breathing 14% O_2 at 6,300 m) (West et al., 1983) (Fig. 1). Some indications on the importance of sea level $\dot{V}O_2max$ are provided by the findings of the Operation Everest II study (OEII), where eight men participated in a simulated ascent to Mt. Everest (hypobaric altitude chamber) over the course of 40 days (Cymerman et al., 1989). Those participants, with an average sea level $\dot{V}O_2max$ of about 50 mL/min/kg, demonstrated a similar $\dot{V}O_2max$ decline up to about 7,500 m as reported by Pugh and colleagues (Fig. 1). However, further $\dot{V}O_2max$ reduction to 8,849 m (BP: 240 mbar) was less pronounced as derived from the extrapolation by Pugh et al. (15.3 vs. 5 mL/min/kg) (Cymerman et al., 1989; Pugh et al., 1964) (Fig. 1). Noteworthy, individual data from the OEII study suggest a correlation between baseline $\dot{V}O_2max$ and that on the

simulated Everest summit. In one subject with a baseline $\dot{V}O_2$max of 41.8 mL/min/kg, this value fell to 11.2 mL/min/kg at 240 mbar (Everest summit) compared to 17.4 mL/min/kg in another subject with a baseline $\dot{V}O_2$max of 58.1 mL/min/kg (Cymerman et al., 1989). The question arises whether even higher baseline $\dot{V}O_2$max would parallel higher $\dot{V}O_2$max values and superior performance when climbing Everest without the support of bottled oxygen? This may be actually assumed based on the fact that since 1978 more than 200 climbers summited the Everest without use of supplemental oxygen (https://www.nationalgeographic.com/adventure). Of course, sophisticated pre-acclimatization, improved light equipment, prepared and secured routes, optimal logistics, mental toughness, etc. may all play important roles, but most of those climbers are also extremely fit as demonstrated by the fast ascent times (M. Burtscher et al., 2009; Martin Burtscher et al., 2021; Millet and Jornet, 2019).

We studied a 40-years old male mountaineer who climbed Mt. Everest on the north route from 6,300 m to the top of Everest in 16 h and 42 min without supplemental oxygen (M. Burtscher et al., 2009). This well-acclimatized climber was characterized by above-average values of maximal voluntary ventilation (MVV), resting lung diffusion capacity for carbon monoxide (DL_{CO}) and DL_{CO} divided by the alveolar volume, and a baseline $\dot{V}O_2$max of 65 mL/min/kg (M. Burtscher et al., 2009). Compared to data from well-trained athletes (Treml et al., 2020), his peripheral oxygen saturation (SpO_2) during exercise at altitude was maintained at above-average values. Likely due to previous high-altitude acclimatization, maximal exercise testing at a simulated altitude of about 5,000 m revealed a $\dot{V}O_2$max reduction by only 25% from baseline, which is comparable to that determined in the OEII study, but is much less compared to data from the AMREE (West et al., 1983) (Fig. 1).

Figure 1. about here

To extrapolate the further decline to the top of Everest, two different models can be applied, (1) the $\dot{V}O_2$max decline follows the shape demonstrated in the OEII study, or (2) the decline would become considerably steeper above 5,000 m, as observed by Pugh et al. (Pugh et al., 1964) or West et al. (West et al., 1983). In the first case, a $\dot{V}O_2$max slightly above 30 mL/min/kg, and in the second case, a $\dot{V}O_2$max of about 20 mL/min/kg would result (Fig. 1). Both scenarios are compatible with at least slow uphill walking the last few hundred meters to the top of the world, as confirmed in a recent case report (Millet and Jornet, 2019). While a certain $\dot{V}O_2$max value, i.e., above 60 mL/min/kg may be considered as a prerequisite to climb 8,000er summits and in particular the Everest without supplemental oxygen (M. Burtscher et al., 2015), the upper limit of $\dot{V}O_2$max associated with performance benefits at extreme altitudes remains to be determined.

3. In addition to a high sea level $\dot{V}O_2$max, an only slight decrease of $\dot{V}O_2$max is important

A high sea level $\dot{V}O_2$max and a rather moderate decrease of $\dot{V}O_2$max with increasing altitude are main determinants for aerobic performance at extreme altitudes. Sea level $\dot{V}O_2$max values of climbers ascending successfully to the top of Mt. Everest without supplemental oxygen may vary between 55 and 90 mL/min/kg. In a previous study, $\dot{V}O_2$max of 60 (± 6) mL/min/kg were reported for six climbers who reached 8,500 m or higher without bottled oxygen, including Peter Habeler and Reinhold Messner (Oelz et al., 1986). However, these measurements have been performed between 2 and 12 months after the last ascent to high-altitude, and thus, body mass and related relative $\dot{V}O_2$max may have changed considerably compared to pre-ascent values. A climber, who reportedly performed the "fastest known time" to Mount Everest had a sea level $\dot{V}O_2$max of 92 mL/min/kg (Millet and Jornet, 2019). Thus, a minimum seal level $\dot{V}O_2$max of about 55 mL/min/kg will be necessary to reach the summit of Mt. Everest without supplemental oxygen, but higher levels are advantageous as they enable faster climbing and provide some reserve in severe environmental conditions or emergency cases.

Sufficient increase in minute ventilation is the most important factor to maintain SpO_2 when exercising in hypoxia. As the decrease in SpO_2 is strongly associated with the decrease in $\dot{V}O_2$max, explaining more than 80% of the $\dot{V}O_2$max loss at altitude (Ferretti et al., 1997; Treml et al., 2020; Wehrlin and Hallén, 2006), factors that help to maintain SpO_2 are of utmost importance. Beside inappropriate ventilatory acclimatization, low SpO_2 values may also result from particular anthropometric characteristics (small lung size), relative hypoventilation and diffusion limitation (Dempsey and Wagner, 1999). Exceeding a SpO_2 decline of 4% in men and 3% in women during maximal exercise, even at sea level, each additional 1% reduction is associated with a 1% loss in $\dot{V}O_2$max (Powers et al., 1993). Thus, athletes suffering from exercise-induced hypoxemia (EIH) are particularly prone to a pronounced SpO_2 drop and associated $\dot{V}O_2$max decline at altitude (Gaston et al., 2016). EIH occurs in a large proportion of elite endurance athletes (> 50%) (Powers et al., 1988) and is associated with abnormal gas exchange, i.e., widening of the alveolo-arterial oxygen pressure difference $(A\text{-}a)O_2$diff (Dempsey and Wagner, 1999; Prefaut et al., 2000). Although the provided exercising SpO_2 values of the 2 subjects (marathon runners) of the AMREE study (West et al., 1983) do not indicate EIH, it cannot be entirely excluded to be responsible for the steep $\dot{V}O_2$max decline with gain in altitude. Either way, the prevention of a too marked arterial desaturation by appropriate acclimatization to high (extreme) altitude (M. Burtscher et al., 2006; Dünnwald et al., 2021; Stoneham and Pethybridge, 1993), coupled with an initial high $\dot{V}O_2$max, will help to maintain $\dot{V}O_2$max at a level sufficient to climb the summit of Everest without supplemental oxygen (Millet and Jornet, 2019).

At extreme altitudes, the drop of maximal cardiac output contributes significantly to the $\dot{V}O_2$max decrease. It has been demonstrated that in contrast to moderate altitude, where the reduction of $\dot{V}O_2$max was entirely explained by the reduced arterial oxygen content, in severe hypoxia the reduced inspiratory pO_2, the impaired gas exchange and the lower cardiac output, associated with a lower leg blood flow, contribute equally to the drop in $\dot{V}O_2$max (Calbet et al., 2003a, 2003b). The decrement of cardiac output in severe hypoxia has been attributed to the low arterial pO_2, e.g., through modulating the output drive from cardiovascular nuclei in the central nervous system (Calbet et al., 2003a, 2003b). The lower maximal heart rates in chronic hypoxia seem to be mediated by hypoxia-induced stimulation of medullary cardiovagal neurons, which was restored by vagal blockade with glycopyrrolate (Boushel et al., 2001). In this context, future research is needed to better elucidate effects of cerebral hypoxia of various severities on determinants of $\dot{V}O_2$max. Nevertheless, the level of SpO_2 remains important, as the reduction of maximal cardiac output is also associated with the severity of hypoxemia.

At any condition, cerebral blood flow is exquisitely regulated to secure O_2 delivery to brain while simultaneously avoiding hyperperfusion. Because brain O_2 requirements are almost constant at any physiological condition, a higher basal $\dot{V}O_2$max could be beneficial in order to provide adequate O_2 delivery to central nervous system while exercising in extreme environmental hypoxic conditions. Curtelin et al. demonstrated during sprint exercise at severe acute hypoxia in humans that when a conflict exists between preserving brain O_2 delivery or restraining cerebral blood flow to avoid potential damage by an elevated perfusion pressure, the priority is given to brain O_2 delivery (Curtelin et al., 2018).

Only a few women have summitted the Everest without supplemental oxygen, probably also attributed to existing sex differences in pulmonary ventilation during intensive exercise (Dominelli et al., 2013; Horiuchi et al., 2019). As hyperventilation is likely more limited in women compared to men, due to mechanical ventilatory constraints, a more pronounced drop in SpO_2 and associated $\dot{V}O_2$max can rather be expected in women (Dominelli et al., 2013; Horiuchi et al., 2019), which was more pronounced in endurance trained women according to data by Woorons and colleagues (Woorons et al., 2005). Furthermore, Horiuchi et al. demonstrated that minute ventilation and energy expenditure during exercise in hypoxia had a stronger effect on the SpO_2 reduction in women compared to men (Horiuchi et al., 2019). Thus, a steeper decline of $\dot{V}O_2$max and aerobic performance with increasing altitude may occur in women, representing a particular challenge when climbing in extreme altitudes.

The main limitations of the suggested interpretation arise from the low numbers of subjects evaluated (N = 1 – 6), and from the fact that except in the Operation Everest II study (performed in a hypobaric chamber), the decline in $\dot{V}O_2$max to the top of Everest was extrapolated by calculation

from values obtained at a lower altitude (M. Burtscher et al., 2009; Pugh et al., 1964), or by reducing FiO_2 at a lower real altitude (6,300 m) (West et al., 1983). Moreover, the single case study (upper line in figure 1) was performed in normobaric hypoxia, but that should not have led to underestimation of the $\dot{V}O_2$max decline compared to hypobaric conditions (Treml et al., 2020) .

4. The impact of appropriate acclimatization and ascent strategies

Beside genetic realities (e.g., $\dot{V}O_2$max trainability, high tolerance to hypoxia, cold, pain, etc.) and long-term training regimens, optimal physiological adjustments during acclimatization and non-physiological factors (such as logistics, light-weight equipment, or simply fortune) contribute all to the extraordinary performance in climbers reaching the Everest summit without supplemental oxygen. Appropriate acclimatization is of utmost importance. Considerable improvements of the initially reduced SpO_2 values during maximal exercise have been reported after an 8-week acclimatization period to 4,100 m (Lundby et al., 2004). Pronounced hyperventilation, as occurring with acclimatization, and associated respiratory alkalosis cause a leftward shift of the oxygen dissociation curve, which can effectively prevent a large drop in SpO_2 (Lundby et al., 2006).

The complexity of hemoglobin concentration on $\dot{V}O_2$max at low and high altitude (before and after acclimatization; but not at extreme altitude) has been extensively discussed elsewhere (Calbet et al., 2006). Briefly, during acclimatization to high altitudes, blood hemoglobin concentration increases while plasma volume and maximal cardiac output decrease (Calbet et al., 2004). These authors demonstrated that neither the increase in hemoglobin concentration during the stay at high altitude (9 weeks at 5,260 m) nor subsequent hemodilution did increase pulmonary or leg $\dot{V}O_2$max, due to reciprocal changes in leg blood flow via local metabolic control of the muscle vasculature (Calbet et al., 2002). However, when breathing hyperoxic air (55% O_2) leg VO_2max was increased, indicating the greater importance of oxygen delivery to than the maximal oxidative capacity of skeletal muscles (Calbet et al., 2002). Interestingly, in chronic hypoxia (5,260 m) a reduced amount of the total oxygen transport capacity was made available to the exercising muscles, contributing (beside reduced cardiac output) to the lower $\dot{V}O_2$max despite normalization of arterial oxygen content (Calbet et al., 2003a, 2003b). A study by Lundby and colleagues demonstrated reduced skeletal muscle capillary conductance with acclimatization to altitude, primarily resulting from the reduced peak leg blood flow (Lundby et al., 2006). The capacity of peripheral oxygen extraction (skeletal muscle capillarization, density and function of mitochondria), which is usually pronounced in subjects with high $\dot{V}O_2$max, increases the alveolar-arterial pO_2 gradient as the oxygen-depleted blood arrives to lung. Whether and to which extent this is beneficial or not remains to be elucidated as it was shown to provoke hypoxemia during submaximal hypoxic exercise (Van Thienen and Hespel, 2016), but seems to be prevented by appropriate acclimatization strategies (Millet and Jornet, 2019). Moreover,

conditions, such as maintaining adequate hydration (affecting plasma volume and, in consequence, the degree of hemoconcentration), can alter the delicate balance between blood rheological properties and optimal peripheral and pulmonary perfusion. Plasma volume expansion in chronic extreme (simulated) altitude actually resulted in improved $\dot{V}O_2$max (Richalet et al., 1999).
Thus, type and time of (pre)acclimatization (Millet and Jornet, 2019), individual responses to and behavior at extreme altitude will modulate $\dot{V}O_2$max and climbing performance above 8,000 m.
Despite the excellent studies performed at real (Grocott et al., 2007; Pugh et al., 1964) and simulated (Cymerman et al., 1989; Richalet et al., 1999) extreme altitudes, future studies are needed to elucidate favorable individual characteristics, training and acclimatization strategies enabling people to climb the highest mountains in the world without supplemental oxygen.
Needless to say, the better the climbing economy and the lower the extra weight to carry (shoes, climbing equipment, etc.) the better is $\dot{V}O_2$max transformed into ascent performance. For instance, the extra work of opening a track in the snow supposes a notable increase in the energy requirement (Carceller et al., 2019).

5. Conclusion

From a physiological point of view and as repeatedly confirmed by high-altitude climbers, both high sea level $\dot{V}O_2$max and an only moderate $\dot{V}O_2$max decline with increasing altitude enable mountaineers to climb 8,000er summits and even the Everest without supplemental oxygen. Appropriate long-term training and acclimatization strategies are prerequisites to accomplish this extraordinary goal.

Declaration of Competing Interest

The authors report no conflict of interest.

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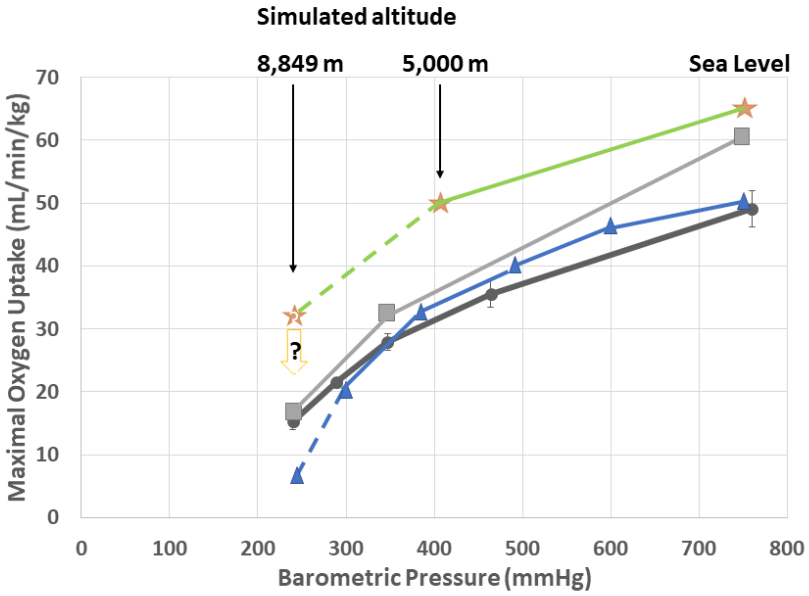
Figure legend

Figure 1. Decline of maximal oxygen uptake ($\dot{V}O_2max$) from baseline to the simulated Everest summit (barometric pressure: 240 mmHg), recorded in the Operation Everest II study (filled circles) (Cymerman et al., 1989), the Himalayan Scientific and Mountaineering Expedition (measured up to 7,440 m and further extrapolation; triangles) (Pugh et al., 1964), the American Medical research Expedition to Everest (measured up to 6,300 m, air, 16%O_2 and 14%O_2 – simulating the Everest summit; squares) (West et al., 1983), and a case study (measured up to 5,000 m of simulated altitude and further extrapolation - 2 models; stars) (M. Burtscher et al., 2009).

Figure 1.


Simulated altitude
8,849 m
5,000 m
Sea Level
Maximal Oxygen Uptake (mL/min/kg)
Barometric Pressure (mmHg)
?
0
10
20
30
40
50
60
70
0
100
200
300
400
500
600
700
800