OXYGEN DEFICIT IS RELATED TO THE EXERCISE TIME TO EXHAUSTION AT MAXIMAL AEROBIC SPEED IN MIDDLE DISTANCE RUNNERS

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ABSTRACT

The purpose of this study was to show the relationship between oxygen deficit and the time to exhaustion (tlim) at maximal aerobic speed (MAS). The minimum speed that elicits \( V_{\text{O}_2\text{max}} \) was assumed to be the maximal aerobic speed (MAS). Fourteen subelite male runners (mean (SD: age = 27 ± 5 yrs: \( V_{\text{O}_2\text{max}} = 68.9 ± 4.6 \text{ ml kg}^{-1}.\text{min}^{-1} \); MAS = 21.5 ± 1 km h\(^{-1}\)) participated in the study. Each subject performed an incremental test to determine and MAS. The subjects ran to exhaustion at velocities corresponding to 100 and 120 % MAS. Oxygen deficit was measured during the period exercise to exhaustion at 120% of MAS and was calculated from the difference between \( O_2 \) demand and the accumulated \( O_2 \) uptake. The tlim values at 100% MAS were correlated with the values of tlim at 120% MAS (r = 0.52). The results reveal that the oxygen deficit was related to the time to exhaustion at MAS and indicate that the greater the oxygen deficit, the greater the time to exhaustion at MAS. It was also noted that the adjustment of oxygen consumption is related to the oxygen deficit. In other words, the subjects who have an important anaerobic capacity are the most efficient during an exercise time to exhaustion at MAS. The time limit values can be expressed by a linear regression making intervene MAS and anaerobic capacity. This conclusion could be of great interest in the training of middle distance runners.

KEYWORDS: Anaerobic capacity, maximal aerobic speed, oxygen deficit, time to exhaustion.

INTRODUCTION

The concept of \( O_2 \) deficit was introduced by Krogh and Linhard in 1920 as the difference between the curve of actual \( O_2 \) uptake at the beginning of exercise and the subsequent steady-state level of \( O_2 \) uptake. In 1969, Hermansen used this concept to determine the accumulated oxygen deficit by calculating the area between the curves representing the oxygen consumed and the actual oxygen consumption. Over the last fifteen years, accumulated oxygen deficit has also been represented as the quantity of ATP formed by anaerobic processes during an exhaustive exercise (Medbo et al., 1988). Accumulated oxygen deficit is proportional to the intensity of the exercise (Astrand & Rodhal, 1986) and is generally described as being the time necessary for circulatory and respiratory adjustments to imposed exercise (Astrand & Rodahl, 1986). It is generally considered that \( O_2 \) transport is not a limit for its consumption within the cells at the beginning of the exercise.

\( O_2 \) deficit represents a measurement of maximal power provided by an anaerobic process (di Prampero, 1981; Medbo et al., 1988). Number of authors (Medbo et al., 1988; Camus & Thys, 1991) have attempted to quantify \( O_2 \) deficit during running time at submaximal and supramaximal intensities by using the \( O_2 \) deficit measurement developed by Medbo et al. (1988). This involves assessing the portion of \( O_2 \) deficit which may influence the variability in the time limit observed at maximal aerobic speed (MAS). The interruption of supramaximal exercise intensity can be attributed to two physiological factors. The first is directly linked to the accumulated oxygen deficit. Exercise is stopped
when the oxygen deficit reaches a critical value which represents a global and approached measure of maximal energy liberated by anaerobic processes (Karlson & Saltin, 1970; Medbo et al., 1988). According to Camus and Thys (1991), the values corresponding to maximal anaerobic energy capacity (EANmax) are included between 1086 and 1317 J per kg of body weight.

The second factor determining the interruption of supramaximal exercise is linked to acidosis. Muscular contraction is stopped when intramuscular pH reaches 6.3–6.4 (Hermansen & Osnes, 1972). The production of lactate during the first minute of exercise is the result of a transient imbalance between the produced and eliminated lactate. The inertia of the Krebs cycle is lower during moderate activity than at rest. Indeed, the increase in time to obtain a stable \( \text{VO}_{2\text{max}} \) from a resting state to a submaximal state may be explained by the utilization of oxygen reserves and by the production of lactate. We were also interested in measuring the adjustment time of oxygen consumption. Indeed, when a subject performs a physical exercise, an increase in \( \text{VO}_{2\text{max}} \) at the onset of the exercise is observed. This increase is an exponential function of the time, the reaction half-time of this process being approximately 30 s. The same is observed at the recovery period (Margaria et al., 1933; Margaria et al., 1965). For submaximal exercises (approximately 80% of \( \text{VO}_{2\text{max}} \)) involving low weak lactate accumulation, \( \text{O}_2 \) debt increases (Saiki et al., 1967; Wassermann et al., 1967). According to di Prampero’s study (1970), of \( \text{O}_2 \) kinetics during intensive exercises, it can be observed that \( \text{O}_2 \) kinetics is an approximately exponential function of time regardless of the type and/or intensity of the exercise. The half-time required to reach a steady state is approximately 27 s, and is independent of the intensity of the exercise.

Furthermore, in the transition from a light exercise to a more intensive exercise one, this value increases rapidly, the half-time being 17 s (di Prampero & Ferreti, 1970). After having measured the adjustment time of oxygen consumption, an attempt was made in the present study to determine if this parameter was correlated to the oxygen deficit. Moreover, it appeared of interest to determine if \( \text{O}_2 \) deficit was correlated with the time limit, and if the \( \text{O}_2 \) deficit values were in agreement with other studies concerning \( \text{O}_2 \) deficit during maximal exercises. A final goal was to determine if there was a correlation between \( \text{O}_2 \) deficit and some the physiological variables (pH, lactate).

**MATERIALS AND METHODS**

**Subjects**

Fourteen subelite male runners participated in the study. Their average (± SD) age, height and weight was 27 ± 5 yrs, 178.2 ± 4.9 cm and 70.2 ± 6 kg, respectively. All the subjects were informed about the possible risks and discomforts involved in participating in the study and signed a statement of informed consent.

**Determination of MAS and \( \text{VO}_{2\text{max}} \)**

Each subject performed 3 exercise tests on a treadmill (Gymrol 1800). All of the tests were performed within a 3-week period. During this period, subjects’ training programmes were maintained. The tests were separated by a minimum of 7 days, and subjects abstained from high intensity exercise for a least 24 h before each test. In the first test, a continuous incremental protocol was used for the determination of MAS (maximal aerobic speed) and \( \text{VO}_{2\text{max}} \). The initial test speed was about 12 km h\(^{-1}\) (slope 0%) and was increased by 2 km h\(^{-1}\) every 3 min up to 80% of the running speed in a 1.5 km race, and subsequently by 1 km h\(^{-1}\) to exhaustion. In the last 30 s of each workload, a fingertip blood sample was obtained and analyzed for lactate concentration [L]. Heart rate (HR) was also monitored (Siemens electrocardioscope) and recorded (Sport tester PE 3000) throughout each test.

The criteria used to determine \( \text{VO}_{2\text{max}} \) were: (a) a plateau in \( \text{VO}_2 \) despite an increase in running speed, (b) a respiratory exchange ratio above 1.1 and (c) a heart rate (HR) over 90% of the predicted maximal HR. MAS was defined as the lowest running velocity which elicited a \( \text{VO}_2 \) value equal to \( \text{VO}_{2\text{max}} \).

**Time to exhaustion at 100% and 120% of MAS**

The time to exhaustion at 100% (tim 100) and 120% (tim 120) of velocity at MAS were measured during two weeks, respectively. The tests were spaced out by a minimum interval of 7 days. Following a 15 min warm-up period at 60% of MAS, the speed was quickly increased (maximum 15 s) up to MAS. The subject was then verbally encouraged to run to exhaustion. During the 100% MAS test, the slope was 0%, but for the second test at 120% MAS the slope was increased by 1.5% to 1 km h\(^{-1}\) as recommended by Margaria et al. (1965). The time of the test was measured when the speed of
\( \text{VO}_{2\text{max}} \) was obtained with an electronic stopwatch. Lactate concentration was determined as described above and an analysis of blood gases was also realized to determine blood pH. These data were acquired using an AVL 993 analyzer. Arterialized blood samples were taken at the ear lobe before and after the test.

**Determination of O\(_2\) deficit**

The \( O_2 \) deficit (DO\(_2\)) was determined and measured during the exercise time to exhaustion at 120% MAS according to Medbo et al. (1988) and Scott et al. (1991). The \( O_2 \) deficit was estimated based on the criteria of Medbo et al. (1988). For this method \( O_2 \) demand is assessed by performing an extrapolation of the linear relationship between the running speed and \( O_2 \) uptake at submaximal intensity. The \( O_2 \) deficit was calculated from the difference between \( O_2 \) demand and the accumulated \( O_2 \) uptake. The \( O_2 \) deficit was expressed in ml \( O_2 \) kg\(^{-1}\) or as a percentage of total volume oxygen consumed.

**The half time of adjustment (\( t_{1/2} \))**

A graphic method was used to determine the adjustment half-time of oxygen consumption. The objective was to identify the instant where the curve of the relationship \( V_{\text{O2}}/\text{time} \) reached the beginning of the plateau corresponding to the intersection of the two tangents of the curve. The half time of adjustment of kinetic oxygen is calculated by dividing this point by 2.

**Data analyses**

Descriptive data are presented as the average \( \pm \) standard deviation (\( \pm \) SD). The coefficient of variation (CV = [SD / mean] \( \times \) 100) for each response was calculated. Correlations between bioenergetic characteristics were determined. The significance level was set at 0.05. The linear regression was calculated according to the least squares method.

**RESULTS**

Table 1 shows the maximal oxygen uptake of subjects (and coefficient of variation expressed as a %, CV), with an average of 68.9 \( \pm \) 4.6 ml kg\(^{-1}\) min\(^{-1}\) (7%), and a range of 61.7 to 75. The average velocity of MAS was 21.5 \( \pm \) 1 km h\(^{-1}\) (5%). The time to exhaustion at MAS, and at 120% MAS, was found to be equal to 269 \( \pm \) 77 sec (29%) and 86 \( \pm \) 25 sec (29), respectively. The \( O_2 \) deficit was equal to 32.31 \( \pm \) 7.1 ml kg\(^{-1}\) and 10.62 \( \pm \) 1.92%, respectively and the half time of adjustment (\( t_{1/2} \)) was equal to 36 \( \pm \) 8 s (Table 1).

**Correlation between \( t_{\text{lim}} \) 100 and \( t_{\text{lim}} \) 120**

Values for \( t_{\text{lim}} \) at 100% and 120% MAS were positively correlated (\( r \) 0.52).

**Correlation between \( DO_2 \) expressed in ml \( O_2 \) kg\(^{-1}\) and \( t_{\text{lim}} \) 100**

Time to exhaustion at MAS was significantly correlated with \( O_2 \) deficit expressed in ml kg (\( r \) 0.63) (Fig. 1), and negatively correlated with \( O_2 \) deficit expressed as a percentage (\( r \) –0.68). In a previous study (Billat et al., 1994), it was demonstrated that the MAS was related to \( t_{\text{lim}} \) at MAS. As a result, the values of \( t_{\text{lim}} \) were defined by a linear regression in the present study.

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**Table 1. Individual data.**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>( t_{\text{lim}} ) 100 s</th>
<th>( t_{\text{lim}} ) 120 s</th>
<th>MAS km h(^{-1})</th>
<th>( \text{VO}_{2\text{max}} ) ml min(^{-1}) kg(^{-1})</th>
<th>DO(_2) ml kg(^{-1})</th>
<th>DO(_2) %</th>
<th>pH</th>
<th>[L] mM</th>
<th>( t_{1/2} ) s</th>
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using MAS (km h$^{-1}$) and O$_2$ deficit (ml O$_2$ kg$^{-1}$). The value of $r^2$ was equal to 0.831. The linear relation (1) was defined by:

$$t_{lim\ 100} = -43,841 \ MAS + 7,276 \ DO_2 + 977.12$$ (1)

**Correlation between DO$_2$ expressed as a percentage and both VO$_{2\max}$ and MAS**

There was a significant correlation between the maximal oxygen consumption and O$_2$ deficit expressed as a percentage ($r = 0.61$). There was also a correlation between DO$_2$ expressed as a percentage and the maximal aerobic speed ($r = 0.65$).

**DO$_2$ and metabolic variables (pH, [L])**

The levels of blood lactate and blood pH at the end of the time limit at MAS were equal to 6.8 ± 1.9 and 7.3 ± 0.1 mM, respectively. There was a significant negative correlation between the O$_2$ deficit expressed in ml kg$^{-1}$ and blood pH ($r = -0.66$). Moreover, there was a significant correlation between the O$_2$ deficit expressed in ml kg$^{-1}$ and [L] responses ($r = 0.62$).

**DO$_2$ and the half time of adjustment ($t_{1/2}$)**

The values of $t_{1/2}$ were equal to $36 \pm 8$ s. There was a significant correlation between $t_{1/2}$ and O$_2$ deficit ($r = 0.55$) (Fig. 2).

**DISCUSSION**

The measured values of $t_{lim}$ at 100% MAS are in accordance with the different studies of $t_{lim}$ (McLellan & Skinner, 1985; Lavoie & Mercier, 1987; McLellan & Cheung, 1992; Billat et al., 1994). The results show that

![Fig. 1. Correlation between time limit at MAS (s) and DO$_2$ (ml O$_2$ kg$^{-1}$).](image1)

![Fig. 2. Correlation between DO$_2$ (ml O$_2$ kg$^{-1}$) and the half time of adjustment (s).](image2)
there was a significant positive correlation between tim  
100 and tim 120 which would appear to indicate that  
an aerobic capacity plays a role in the ability to sustain  
running at MAS, as previously suggested by Billat et  
al. (1994). The study of oxygen deficit and the half-  
time to adjustment should provide some insight as to  
the exact role of anaerobic capacity in sustaining run- 
ning at MAS. The values of O2 deficit were lower than  
those proposed in other studies dealing with oxygen  
deficit during supramaximal exercises (Medbo et al.,  
1988; Camus & Thys, 1991; Scott et al., 1991).

In these studies, O2 deficit ranged from 18 to 40 ml  
kg\(^{-1}\), even when these authors suggested that the O2  
deficit ranged from 52 to 90 ml kg\(^{-1}\). The results of  
the present study are best explained by the fact that the  
exercise time to exhaustion was realized after a warm-up,  
which affected the steady state, whereas the other stud- 
ies began from a resting state. It would appear, however,  
that the results given here are in accordance with the  
classical data on muscular exercise. Indeed, in accor- 
dance with this data the supramaximal exercise was  
interrupted when the O2 deficit reached a critical value  
equal to the maximal anaerobic energy (Karlsson &  

In fact, we found a decrease in blood pH, which cor- 
responds to a muscular acidosis, as explained by Hern- 
mans and Osnes (1972). The average values of  
\(V_{\text{O2 max}}\) and DO2 were not equal as suggested by Marg- 
garia (1976) and by Camus and Thys (1991), but there  
was a significant correlation between these two charac- 
teristics when DO2 was expressed as a percentage \((r =  
0.61)\). The relationship between average values of  
\(V_{\text{O2 max}}\) and DO2 would tend to confirm Margaria’s  
hypothesis (1976) according to which the existing rela- 
tionship is defined by \(DO_2 = f(V_{\text{O2 max}})\) as Camus and  
Thys (1991) suggested. Moreover, there is a significant  
correlation between oxygen deficit and blood responses  
([L]), thus confirming the results of di Prampero (1981).  
The average value in this last study were 9.78 \pm 1.5  
mM, whereas in the present study the average value was  
lower \((6.8 \pm 1.9 \text{ mM})\). This difference can be explained  
by the fact that the maximal exercise was preceded by a  
warm-up which made glycolysis occur as soon as the  
exercise began. It is possible that the lower DO2 values  
and blood responses could stem from the fact that the  
subjects were specialists in long distance running, and  
that the anaerobic power was thus inferior to that of a  
sprinter (Komi et al., 1977; Schnabel & Kinderman,  
1983). In accordance with Komi et al. (1977), this  
phenomenon would be due to the different typology of  
muscular fibers among different type of training.

The time limit was significantly positively corre- 
lated with O2 deficit expressed in ml O2 kg\(^{-1}\) \((r =  
0.63)\), and negatively correlated when expressed as a  
percentage of total volume oxygen consumed \((r =  
-0.68)\). Two conclusions can be reached based on this  
information. Firstly, that the O2 deficit increased with  
the increasing time limit. Secondly, the time limit  
decreased with increasing levels of anaerobic metabo- 
lism. Moreover, there is a significant correlation  
between MAS and O2 deficit \((r = 0.65)\) expressed as a  
percentage, showing that when MAS was high, the role  
of anaerobic metabolism was greater.

The results show that a relationship exists between  
the O2 deficit and \(t_{1/2}\), also showing that the greater O2  
deficit, the greater the \(t_{1/2}\). These results suggest that the  
adjustment of oxygen consumption intervenes at the  
maximal values of the time limit. In other words, the  
subjects who had the maximal value of anaerobic  
capacity were those who had a high \(t_{1/2}\), and greater  
time limit values. It can be observed that the average \(t_{1/2}\)  
values were in accordance with those of di Prampero  
and Ferreti (1970), who observed a \(t_{1/2}\) equal to 27 s  
which was independent of the work rate.

In a previous study (Billat et al., 1994; Renoux et al.,  
1999), it was demonstrated that there is a relationship  
between the time limit at MAS and at 120% MAS thus  
supporting the model of Monod and Sherrer (1954).  
The value of ‘a’ in this model can be defined as the  
aerobic running capacity expressed by the oxygen  
deficit as described by Medbo et al. (1988). Indeed,  
these authors explain that the O2 deficit during an  
 exhaustive exercise inferior to 5 min is not statistically  
different to the accumulated O2 deficit between exhaus- 
tive bouts of exercise lasting 2 or 4 min. The time limit  
values can be expressed by a linear regression (1) in  
which two independent values interfere, namely the  
maximal aerobic speed and anaerobic capacity.

In conclusion, we could say that these results  
increase our comprehension of the physiological mech- 
anisms active in middle distance runners.

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