Chapter 2
Accumulated Oxygen Deficit Issues

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1. Introduction

As detailed in the chapter 1 of section 1, muscles work by shortening the muscle fibers, a process driven by cross-bridge cycling. During each cycle one molecule of ATP is split to ADP and inorganic phosphate. Since the stores of ATP are very limited, ATP must be resynthesized as fast as it is broken down. The process of splitting and resynthezing ATP is called ATP-turnover. ATP can be resynthesized by aerobic and by anaerobic processes (see chapter 1 of section 1 and chapter 5 of section 5 for more details). The first ones require O_2, and aerobic ATP-turnover can thus be quantified by the O_2 consumption. Amounts of O_2 are thus an alternative measure of the ATP-turnover and of amounts of energy release. Anaerobic processes are per definition independent of O_2 consumption. However, since the stoichiometry between O_2 consumption and aerobic ATP-turnover is known from textbooks of biochemistry, also anaerobic ATP-turnover can be expressed in units of O_2.

At rest and during continuous exercise of moderate intensity, ATP is resynthesized by aerobic processes only. At onset of exercise or during high-intensity exercise also anaerobic processes contribute. There is thus a gap between the total ATP-turnover rate (called O_2 demand when expressed in units of O_2) and the measured O_2 uptake. This difference is called the O_2 deficit and reflects the anaerobic rate of the energy release. A central part of this presentation is to show how the O_2 deficit can be used to quantify rates of anaerobic ATP-resynthesis and further to address limitations of the principle.

This chapter focuses on how to quantify anaerobic energy release by the accumulated O_2 deficit principle during supramaximal exercise intensities (above that corresponding to maximal O_2 uptake). Thus, quantification of anaerobic energy release at the onset of exercise at moderate intensity proposed by Krogh and Lindhard [1] is not considered further. The basic ideas of the maximal accumulated O_2 deficit principle were worked out independently by three groups [2-5] in the 1980-is.

2. Quantitative concepts

When the anaerobic energy release is to be determined, terms both for the rate of energy release and for the amount of energy release are needed. We therefore suggested a set of terms of the aerobic, anaerobic and total energy release when the energy release is expressed in terms of units of O_2 to distinguish between rates and amounts of energy release [4]. These terms seem now largely accepted (Table 1).

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Table 1. Central terms of the accumulated O_2 deficit concepts

Rate of energy release	*Amount of energy release*
O_2 demand	Accumulated O_2 demand
– O_2 uptake	– Accumulated O_2 uptake
= O_2 deficit	= Accumulated O_2 deficit

Amounts of energy are the rates of energy integrated (by time) over the exercise period in question, indicated by adding "accumulated" to the corresponding term of rate. If the rate is constant, the integration reduces to a simple multiplication of rate and duration.

3. Principle of determining accumulated O_2 deficit

Accumulated O_2 deficit is in most studies apparently determined according to principles detailed elsewhere [4]. However, results obtained have been discussed, questioned, and criticized (e.g. [6,7]). The main reason for conflicting results appears to be that many studies have not paid proper attention to the main principles and possible problems and pitfalls addressed in detail elsewhere [4,8-10]. Thus, a presentation of the main principles and possible problems and limitations are needed.

The idea of using the accumulated O_2 deficit as a measure of the anaerobic energy release during exercise is based on the following four principles:

1. Energy release (ATP-resynthesis) is aerobic or anaerobic. The anaerobic part is thus the total energy release less the aerobic part. The aerobic part is taken from the measured O_2 uptake.
2. During exercise at moderate intensities where anaerobic processes are negligible, O_2 uptake increases linearly by exercise intensity taken as speed of treadmill running or as power of ergometer cycling at constant frequency (Figure 1, left panel). Since there is no anaerobic contribution, the measured O_2 uptake reflects the total rate of ATP-turnover or O_2 demand. Consequently, during these conditions the total ATP-turnover rate or O_2 demand increases linearly by exercise intensity. This linear relationship between exercise intensity and O_2 demand is extrapolated to supramaximal intensities where anaerobic contribution is considerable.
3. During exercise at constant intensity the rate of ATP-turnover is constant throughout the exercise even if continued to exhaustion (Figure 1, right panel).
4. The accumulated O_2 deficit is taken by integrating O_2 deficit over the exercise period in question (see grey area in Figure 1).

Points 1 and 4 above largely follow by definition. The basis for points 2 and 3 is summarized below and discussed in more detail elsewhere [4,8,9].

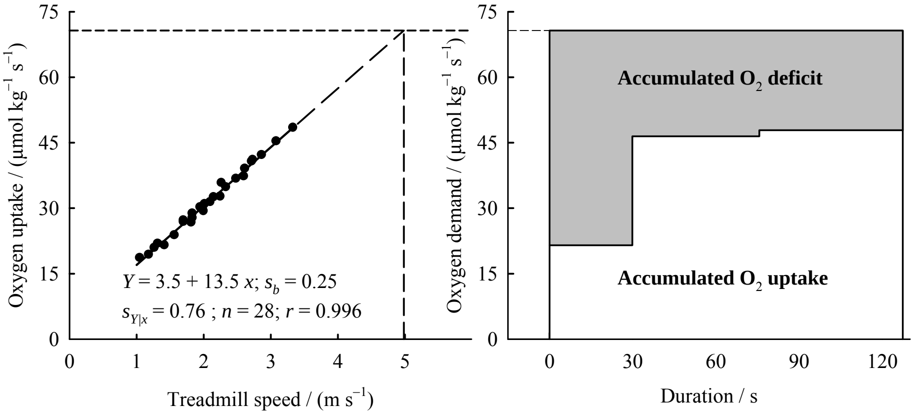

Figure 1. Principle of finding the accumulated O_2 deficit during supramaximal exercise. *Left*, the subject exercised repeatedly for 10 min at constant intensity (here speed) during several days, and the O_2 uptake was measured during the last 2 min of each exercise. A linear regression of steady state O_2 uptake on exercise intensity was calculated from the data. On a separate day the subject exercised at 4.98 $m.s^{-1}$ for 127.8 s to exhaustion. By linear extrapolation (long dashed lines) the O_2 demand of that exercise was estimated to be 70.7 $\mu mol.kg^{-1}.s^{-1}$ (95.0 $ml.kg^{-1}.min^{-1}$; short dashed lines). *Right*, during the run to exhaustion expired air was collected in three Douglas bags, and the O_2 uptake was measured. The difference between the estimated O_2 demand and the measured O_2 uptake was taken as O_2 deficit. That quantity was integrated over the whole run to exhaustion, giving the accumulated O_2 deficit of 3.76 $mmol.kg^{-1}$ for that exercise (grey area). The data are for a well-trained young man with a maximal O_2 uptake of 48.6 $\mu mol.kg^{-1}.s^{-1}$ (63.5 $ml.kg^{-1}.min^{-1}$) running on a treadmill at 10.5 % (6°) inclination.

4. Requirements for obtaining reliable values of the accumulated O_2 deficit

Common principles of statistics together with simulations have shown that a minimum of 8–10 measurements of the steady state O_2 uptake, taken as the O_2 uptake in the period 8–10 min of exercise at constant intensity, is needed to find reliable relationships of O_2 demand versus exercise intensity [4]. Intensities between 35 and 90% of maximal O_2 uptake were used. At intensities below ≈35% of maximal O_2 uptake there is often a disproportionately high O_2 uptake (Figure 2), and this effect may have a simple biomechanical explanation [4,8,9]. While O_2 uptake reaches a steady state within ≈3 min at moderate intensities, the presence of a slow component with a time constant of ≈3 min at higher intensities led us to use bouts of exercise of 10 min duration (see [10] for further details).

4.1. Proposed short-cut approaches

The relationships between exercise intensity and O_2 uptake vary between subjects, reflecting individual variations in exercise economy. Therefore a separate relationship should be established for each subject. This makes the procedure outlined above and detailed elsewhere [4] time consuming, and simpler procedures than that of at least 8–10 bouts of exercise each lasting 10 min for each subjects have been sought. Two possibilities appear acceptable, while others do not.

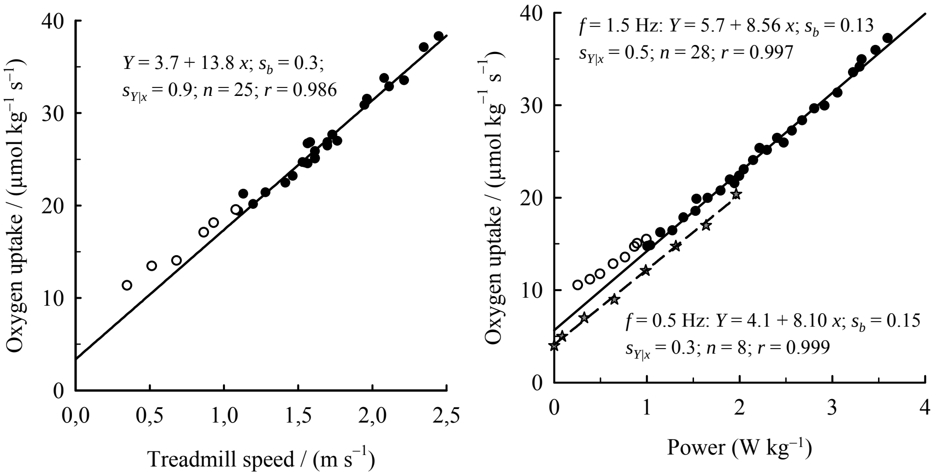

Figure 2. Nonlinearity in O_2 uptake versus exercise intensity at low intensities. *Left*, treadmill running at 6° (10.5%). At speeds less than ≈1.0 m s^{-1} the O_2 uptake is disproportionately high (open symbols, not included in calculating the regression line). *Right*, cycle exercise at 1.5 Hz (upper curve and circles) and at 0.5 Hz (lower curve and asterisks) for two different subjects. At powers less than ≈1.0 W.kg^{-1} the O_2 uptake is disproportionately high at 1.5 Hz (open symbols, not included in calculating the regression line). At 0.5 Hz the relationship was linear down to zero load, which equaled resting O_2 uptake. The data sets are from three different healthy young men: [4] with permission of *J Appl Physiol*, [9] with permission of *Can J Appl Physiol*/*Appl Physiol Nutr Metab* and [10] with permission of *Acta Kinesiol Univ Tartuensis*.

4.1.1. Less than 10 min durations

One might suggest reduce the time for each exercise for establishing relationships between exercise intensity and O_2 uptake. Reducing the duration to 6 min or less affected the relationships obtained significantly [11], probably because the slow component does not reach a steady state [10]. This approach is therefore not recommended.

4.1.2. Fixed Y-intercept

The *Y*-intercept does not vary much between subjects when properly established [4,10]. Thus, a fixed *Y*-intercept may be established for the specific exercise condition. Thereafter, for each subject 2–4 measurements of the O_2 uptake after 8–10 min of exercise at 70–90% of the maximal O_2 uptake is carried out. The O_2 demand is then taken to increase linearly by intensity from the fixed *Y*-intercept through the measured values. This approach has been used in many studies [12-21], apparently with reliable outcome. Since the *Y*-intercept varies considerably between exercise conditions and models, it must be established for the specific exercise conditions in question.

4.1.3. Step-wise increases in intensity with no rest between

Another possibility is to use stepwise increases in exercise intensity, for example every 4th min, with no rest or break between each step, thus obtaining ≈10 measurements in the recommended range of intensities within 1 h [10]. The basic idea is that while the O_2 uptake may not reach a complete steady state within 4 min, the mismatch for a small step is minimal and disappears almost completely during the next 4 min period, as shown mathematically [10]. Corresponding approaches have been used for humans using some steps of 4 min [22] or 2 min [23,24] and on horses running for 1–2 min at each step [25-29], but with no further validation and justification.

4.1.4. Reliable and unreliable short-cut approaches

The experiments for either optional procedure (points 4.1.2 and 4.1.3 above) can be carried out in less than one hour. One approach requires that the common *Y*-intercept has been established in advance, while the other one does not. A third and even better approach may be to combine the two summarized above.

Some studies claim that the relationships may be nonlinear. Consequently, second-order fits have been tried using only 6–12 measurements over a quite narrow range of exercise intensities [30-32]. However, second-order fits based on at least 20 measurements appear numerically unstable and thus unreliable even when covering a wide range of intensities [33]. Nonlinear fits with no further validation are therefore not recommended.

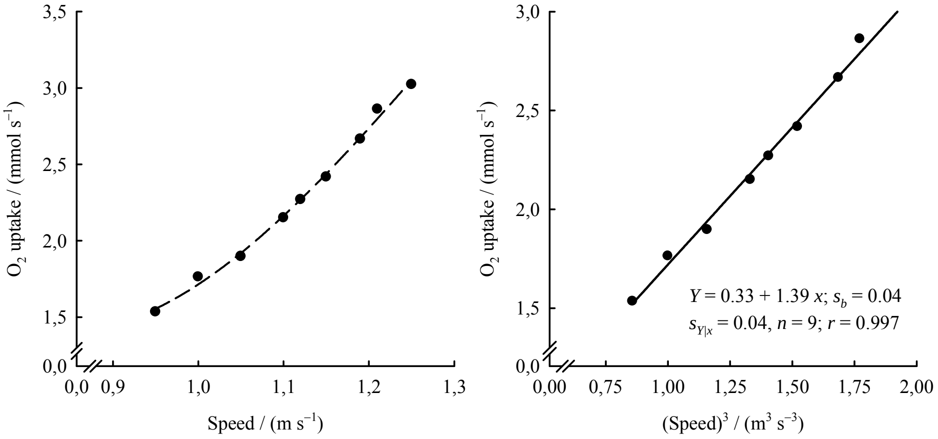

Figure 3. Relationship between swimming speed and O_2 uptake (left panel). The power needed to overcome water resistance increases by the cube of speed, and consequently the relationship is curved. When plotting O_2 uptake versus the cube of speed, the relationship is linear (right panel). Data on a top Japanese swimmer, kindly provided by Dr.Ogita.

4.2. Requirements to experimental design

The O_2 demand is calculated from exercise intensity, which therefore must be precisely known. That is usually the case in laboratory experiments using a treadmill with known speed and fixed inclination or ergometers exerting known power outputs. Corresponding experiments have also been carried out for swimming in a flume where the speed of water is accurately known [34-36]. The O_2 uptake was closely related to water speed, and by a proper mathematical transform linear relationships were obtained (Figure 3). Proper experiments have also been carried out on ergometers for kayaking [37-40]. The knee extensor model that has been used in some studies (e.g. [6,41]), does on the other hand have problems that make it unsuited for estimating the O_2 demand and thus to calculate the accumulated O_2 demand (see [8,9] for further details). One study has tried to determine the accumulated O_2 deficit during track running [42], but the results are seriously influenced by limitations with portable analyzers addressed below.

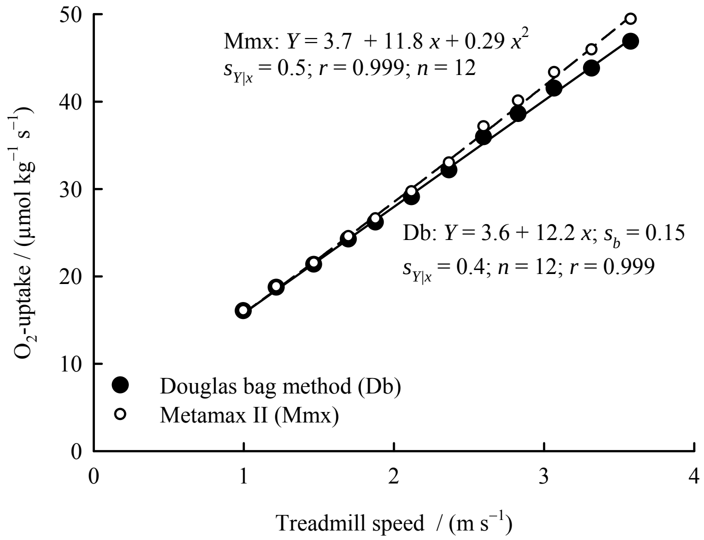

Figure 4. Relationship between treadmill speed and O_2 uptake measured by the Douglas bag method (Db) and the Metamax II instrument (Mmx). A healthy young man ran on the treadmill (10% inclination) while the speed was raised stepwise every 4th minute. O_2 uptake was measured simultaneously by both methods (Db and Mmx) as detailed elsewhere [43]. At low speeds and thus moderate O_2 uptakes the two sets of values were almost identical. At higher O_2 uptakes built-in algorithms in the Metamax introduced a nonlinearity in the output as indicated by the significant second-order term. Reproduced from [10] with permission of *Acta Kinesiol Univ Tartuensis*.

4.3. Requirements to instruments

We have in all our experiments measure O_2 uptake by the old-fashioned, manual Douglas bag method [3,4,8,10,33,44-47,47-52]. Air collected in Douglas bags was later analyzed for gas fractions by the Scholander technique [53] or by instruments from Applied Electrochemistry Instruments (AEI). Volumes of expired air were measured in a Tissot-type spirometer [54] or by a turbine-type gas flow meter from AEI. These methods and instruments are readily tested for accuracy and precision. Most laboratories today use fully automatic, computerized systems where the data are processed by unknown algorithms. Although not well explained in the instrument's manual, there may be built-in averagings and delays of 30–60 s in the recording. That is unacceptable when the accumulated O_2 deficit is to be determined during exercise [55]. This is clearly a problem in some studies [42,56,57], but most studies do not report adequate data. Moreover, some instruments may have built-in corrections that distort the values [55], and therefore need corrections [58,59]. Consequently, relationships between exercise intensity that are linear when properly measured by the Douglas bag technique, may turn out as curved when processed by an automatic system (Figure 4). Most studies give no information on possible quality control of their methods and instruments. Thus, it is not known whether the problems addressed here are limited to a few studies and instruments or perhaps are being quite widespread but unrecognized by most users.

5. Precision

The error in accumulated O_2 deficit increases by exercise duration. The largest terms in the error are those related to estimating O_2 demand by extrapolation. When adequately carried out, the accumulated O_2 deficit can be determined with an error of $\leq$ 4% for exercise lasting no more than 3 min [4,8,9]. Similar values have been reported by one study [60], while others have reported random errors twice as large [13,18,61].

5.1. Test-retest

During test-retest experiments accumulated O_2 deficit is calculated using the same relationship of O_2 demand versus intensity. This might reduce the test-retest-variability to less than that of single determinations since imprecision in the estimated O_2 demand is the main term. However, data from our studies [4,46] suggest a random error (SD) of 0.12 mmol.kg^{-1} (4%) in test-retest-experiments, similar to that of another group [60,62]. Others have found test-retests-variabilities $\approx$0.2 mmol.kg^{-1} [13,18,61]. Thus, the test-retest-variability is at least as large as the statistical error in single determinations.

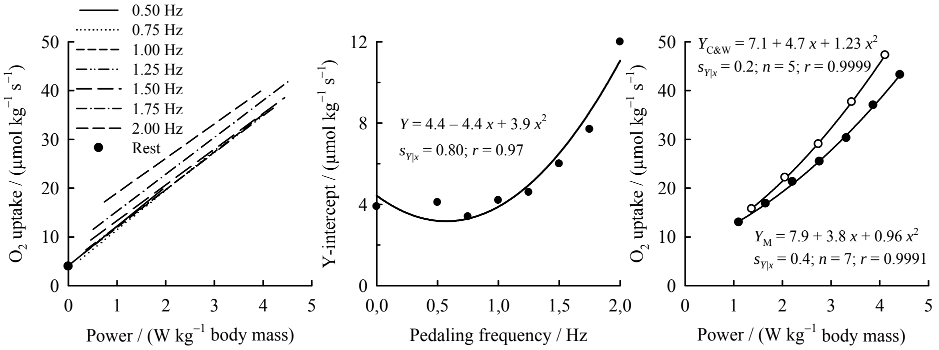

Figure 5. *Left*, O_2 uptake versus the power at seven different pedaling frequencies and at rest for one subject. Data points have been excluded for sake of clarity except for the rest value. The length of each line shows the range of exercise intensities used for each frequency. *Middle*, Y-intercept of each relationship in the left panel versus pedaling frequency. *Right*, O_2 uptake versus power if the braking force is kept constant and power is varied by varying the pedaling frequency only. Upper curve and open symbols are from ([63], C&W), while the lower curve and filled symbols are from ([10], M). Reproduced from [10] with permission of *Acta Kinesiol Univ Tartuensis*.

5.2. Variations in exercise conditions

For treadmill running O_2 demand increases both with inclination and speed, and consequently either inclination or speed is kept constant. For cycling O_2 demand likewise increases with power that can be varied by varying pedaling frequency or braking force. O_2 demand increases with pedaling frequency even if the power is kept constant (Figure 5). The Y-intercept increases with frequency in approximately a second-order manner that may have a straight-forward biomechanical explanation [10]. Consequently, pedaling frequency should be kept constant. Many studies have nevertheless allowed the frequency to vary [64-71]. The effect on the outcome is difficult to estimate. Two recent studies tried to correct for varying frequencies during exercise [30,32], but the values reported for accumulated O_2 deficit appear unreliably high, probably because the relationships obtained were based on too few measurements.

6. Accuracy

At onset of exercise O_2 stores, mainly in venous blood, are reduced. Consequently, use of stored O_2, that per definition is an aerobic contribution, is included in the accumulated O_2 deficit. This amounts to $\approx$0.25 mmol O_2.kg^{-1} body mass [4,8,9,45] and is thus a constant bias of $\approx$10% in most cases.

6.1 Comparisons with muscle metabolites

Anaerobic energy release in muscle can be determined directly from muscle biopsies taken before and immediately after exercise, and the values have been compared with accumulated O_2 deficit in five different studies on cycling exercise (Figure 6). The results agree quite well when the assumed working muscle mass was taken to be 25% of the body mass and 1 mol O_2 was taken equal to 6.5 mol of ATP (a value taken from textbooks of biochemistry). Bangsbo and coworkers [6,41] also reported an apparent excellent agreement between the accumulated O_2 deficit for knee extensor cycling and measured muscle metabolites. However, that is a forced agreement obtained by taking 1 mol of O_2 equal to 4.6 mol of ATP, a conversion factor at variance with that of textbooks of biochemistry.

The comparison of accumulated O_2 deficit with muscle metabolites in figure 6 does not include release of lactate from muscle to blood during exercise. However, released lactate amounts to only a few percents even for cycling lasting 2–3 min [51,52].

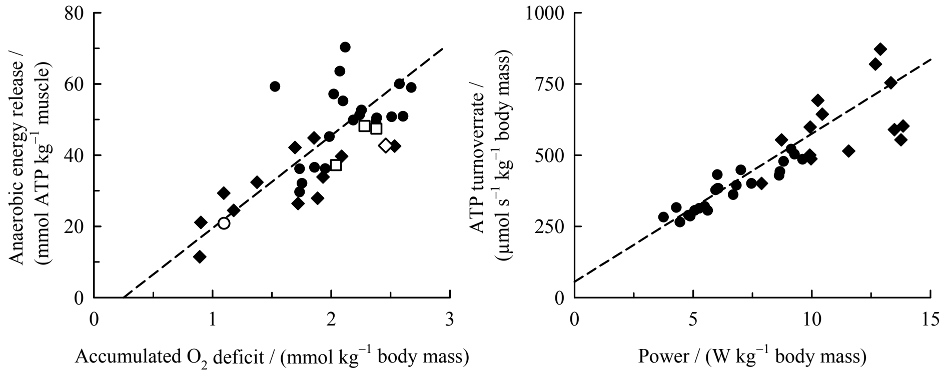

Figure 6. *Left*, anaerobic energy release in muscle versus accumulated O_2 deficit. The dashed line is the expected relationship provided the biopsy data are representative of a working muscle mass equal to 25% of the body mass and that use of stored O_2 amounted to 0.25 mmol.kg^{-1} body mass. □, individual data from [47]; □, individual data from [49]; □, mean of six subjects from [72]; □, mean of six subjects from [64]; □, mean of ten trained cyclists from [73]; all subjects were healthy young men. The data from Sahlin and coworkeres [72] are from measurements of the accumulated O_2 deficit at the onset of exercise where no extrapolation was involved (Krogh and Lindhard [1] principle). *Right*, total ATP-turnover rate versus power. The dashed line is a linear extrapolation of the subjects' relationship between power and O_2 demand expressed as an ATP-turnover rate: The slope of 52 µmol ATP.J^{-1} is the mean of the individual slopes and corresponds to a delta efficiency of 0.27 assuming ATP is produced by oxidation of glycogen. The line's Y-intercept of 55 µmol ATP.s^{-1}.kg^{-1} body mass is the mean of all subjects' intercept. The aerobic energy release was taken

from the measured O_2 uptake plus an assumed use of stored O_2 of 0.25 mmol.kg^{-1} body mass, while the anaerobic energy release was taken from measured muscle metabolites assuming these values were representative of a working muscle mass of 25% of the body mass. The data are taken from [47, 49] for subjects cycling at preset powers leading to exhaustion between 10 s and 3 min. The figure is modified from [49] and reproduced with permission of *Acta Kinesiol Univ Tartuensis*.

6.2. Studies in hypoxia and normoxia

Anaerobic energy release is per definition independent of aerobic metabolism and O_2. If the accumulated O_2 deficit reflects the anaerobic energy release accurately, it should be independent of the O_2 uptake. Determinations of accumulated O_2 deficit in hypoxia have given similar values to those on the same subjects in normoxia despite lower exercise intensity in hypoxia [4,74-76] and is illustrated further elsewhere [8,9]. Moreover, for exercise lasting 3–4 min or more the O_2 demand in hypoxia was taken directly from the measured steady-state O_2 during 10 min runs of the pre-tests in normoxia, and therefore no extrapolation was needed [4].

6.3. Comparisons with working muscle mass

The working muscle mass when cycling with one leg is half of that using two legs. The accumulated O_2 deficit when cycling to exhaustion using one leg only was 52% of that when using both legs [66].

Maximal accumulated O_2 deficit has been determined for resistance-trained, endurance-trained and untrained subjects [77]. The values for resistance-trained and endurance-trained were very close whether expressed relative to body mass (2.35 mmol kg^{-1}) or to leg muscle mass (8.3 mmol.kg^{-1}). The values for untrained subjects were on the other hand ≈25% less than those of the trained subjects. Thus, for trained subjects maximal accumulated O_2 deficit may be proportional to active muscle mass.

6.4. Justification of linear extrapolation

Assumption number 2 of linear extrapolation to supramaximal exercise intensities has been examined further [49]. The rate of energy release was taken from the measured O_2 uptake and changes in muscle metabolites during supramaximal exercise, and this rate was related to the power (Figure 6, right panel). The data obtained were distributed around linear extrapolations of relationships established at submaximal intensities, suggesting a constant mechanical efficiency and thus justifying the linear extrapolation.

To sum up, data summarized above and detailed elsewhere [4,8-10,47,49,64,73] suggest that when properly determined, accumulated O_2 deficit is an accurate measure of the anaerobic energy release during exercise when disregarding that use of stored O_2 is a component of the accumulated O_2 deficit. The random error in single determinations as well as the test-retest-variability is ≈4% when properly determined.

7. Physiological importance of accumulated O_2 deficit and implications

Accumulated O_2 deficit depends on the working muscle mass and thus expectedly increases by body mass or by working muscle mass. All data reported below have therefore been expressed relative to body mass or to estimated active muscle mass.

7.1. A maximal accumulated O_2 deficit

If exercise is carried out at different intensities and thus for different durations to exhaustion, accumulated O_2 deficit increases by duration before leveling off (Figure 7). For exercise lasting longer than 2 min there is no further increase [2,4,5,45,78-80], and that maximum is taken as the anaerobic capacity. For durations less than 1 min the accumulated O_2 deficit is less than the maximum both for treadmill running [4,81] and for cycling [2,5,45,64-67,70]. When running on a treadmill, a minimum of 2 min exercise appears to be needed to reach the maximum value and thus to exhaust the anaerobic capacity [4,81]. During all-out cycling anaerobic capacity may be exhausted in ≈1 min [64-67,69,70]. For cycling at constant power to exhaustion the time needed to exhaust the anaerobic capacity is in the range 1–2 min [2,5,45,66]. Other exercise models have not been examined for time needed to exhaust anaerobic capacity.

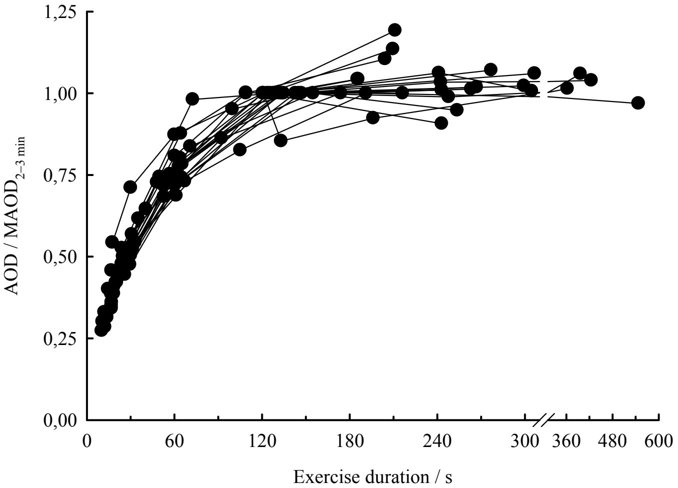

Figure 7. Accumulated O_2 deficit (AOD) for exhausting runs of different durations to exhaustion, relative to the maximum ($MAOD_{2\text{–}3\ \text{min}}$), taken as the accumulated O_2 deficit for a run leading to exhaustion in 2–3 min. The data are from 19 healthy young men with different background (sprint-trained, endurance-trained, untrained men [4,46]).

7.2. Relative importance of aerobic and anaerobic energy release

The relative aerobic and anaerobic contribution to exhausting exercise of different intensities and durations has been determined in many studies, and the results agree well (Figure 8). The hyperbolic curve fit is compatible with a constant anaerobic energy release plus an O_2 uptake at a constant rate throughout the exercise. However, for exercise of short durations the accumulated O_2 deficit is less than the maximum (see Figure 6). The reduced accumulated O_2 deficit seems to be largely compensated for by an accelerated increase in the O_2 uptake at the onset of exercise [45], probably because of the very high exercise intensity for short-lasting bouts.

7.3. O_2 uptake versus exercise duration – delay from onset of exercise

Determination of accumulated O_2 deficit requires that O_2 uptake is measured throughout the exercise. Krogh and Lindhard [82] showed in 1913 a major increase in O_2 uptake within 30 s of exercise (Figure 9, left). At moderate to high exercise intensities accumulated O_2 uptake increases linearly by time after a delay of ≈15 s (e.g. [45,70,83,84], see also Figure 8). In a further study on very intense exercise leading to exhaustion in 10–30 s the delay was only 8 s ([49], Figure 9, right). Thus, the traditional view that aerobic processes need considerable time before contributing, needs revision.

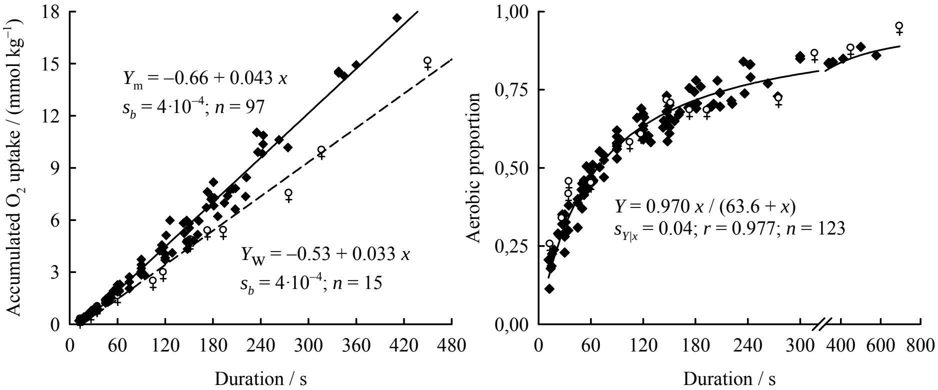

Figure 8. *Left*, accumulated O_2 uptake versus exercise duration. The regression lines (separate lines for men [Y_m] and women [Y_w]) suggest that after a delay of 15 s accumulated O_2 uptake rises linearly by time. *Right*, relative contribution of accumulated O_2 uptake to total energy release versus exercise duration. The curve is a hyperbola fit to the data, and the standard error in the two parameters is 0.014 and 2.8 s, respectively. For 60 s duration accumulated O_2 uptake is 47% and for 68 s duration 50% of the total as estimated from the fitted curve. The data have also been re-examined after subtracting 0.25 mmol.kg^{-1} of presumed use of stored O_2 from accumulated O_2 deficit and adding this amount to accumulated O_2 uptake. After that correction the calculated aerobic proportion is 0.57 for exercise of 60 s duration. The data are pooled from 32 studies [4,12-16,19,20,39,40,44-46,49,58,59,64,65,67-69,73,81,85-93] (right panel) and 30 studies (left panel; two studies reported only the relative aerobic contribution and not the accumulated O_2 uptake). Each value is the mean of 2–18 subjects. The subjects were endurance-trained, middle-distance trained, sprint-trained, or untrained healthy young adults. ☐, men; ♀women.

7.4. Influence of maximal accumulated O_2 deficit on performance

Testing of athletes may be of interest to predict their performance. Maximal accumulated O_2 deficit may predict the performance of 100–1500 m track running within ≈10% of the final time [94]. Two other studies suggest that the performance on a 100 m sprint may be predicted from maximal accumulated O_2 deficit with an error of ≈0.5 s [13] or <1 s [21]. Thus, although factors like running economy ("technique") and O_2 uptake (or lactate threshold) are important, maximal accumulated O_2 deficit alone appears to be an important predictor. There was on the other hand no relationship between maximal accumulated O_2 deficit and performance on 800 m running among a homogenous group of runners, but the variability (SD) in performance in that study was only 4 s (3% [90]). This is close to the random error in single determinations of accumulated O_2 deficit, and this example thus illustrates a limitation of the principle.

Accumulated O_2 deficit together with either maximal O_2 uptake or lactate threshold could predict the performance on 500 m kayaking with an error of only 1.6 s (1.3%) [37]. These parameters did on the other hand not predict the performance on repeated sprints among a group of Australian football players [17]. Thus, maximal accumulated O_2 deficit may predict performance for simple and physically demanding exercises, but for more compound or technically demanding tasks a possible effect of a large maximal accumulated O_2 deficit is not clear.

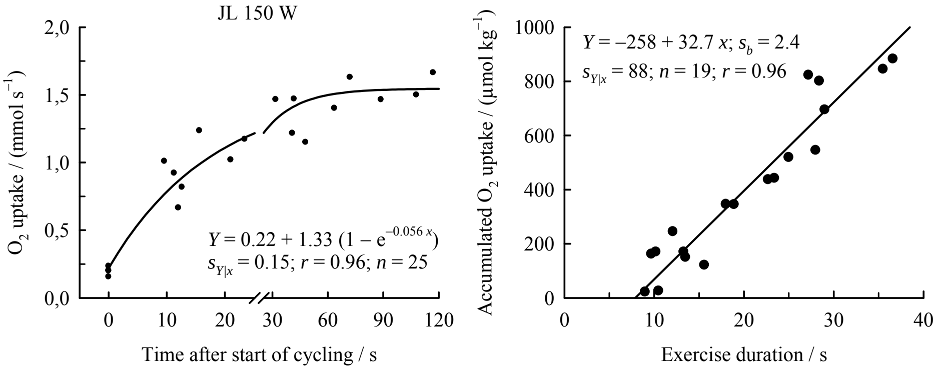

Figure 9. *Left*, O_2 uptake versus exercise duration [82]. Subject JL cycled at four different occasions at ≈150 W while the O_2 uptake was measured before exercise (time zero) and at intervals. x-axis is broken at 25 s for change of scale, and five measurements beyond 2 min of exercise are not shown for sake of clarity. At onset of exercise the major increase in O_2 uptake was seen before 30 s. *Right*, accumulated O_2 uptake versus exercise duration from [49] (individual data from nine subjects) for cycling at constant power to exhaustion in either ≈10 or ≈30 s. After a delay of 8 ± 1 s accumulated O_2 uptake rose linearly by duration at a rate ≥80% of maximal O_2 uptake. The Y-intercept of –0.26 mmol O_2.kg^{-1} equals (with opposite sign) the assumed use of stored O_2 at the onset of exercise. The data are compatible with no delay in the O_2 consumption in working muscles from the onset of exercise, and if present, the delay is no more than 2 s.

7.5. Between-subjects variations on maximal accumulated O_2 deficit

7.5.1. Gender differences

When groups of men and women with similar background have been examined, maximal accumulated O_2 deficit of women has been 12–17% less [46,95,96], 24% less [19] or 31–34% less than that of men [66,97]. Maximal accumulated O_2 deficit for 11 yr old girls was 14% less than that of boys of similar age [98]. When expressed relative to lean body mass or estimated active muscle mass, maximal accumulated O_2 deficit for women was 11–18% less than that of men [66,95].

7.5.2. Effect of age

Maximal accumulated O_2 deficit of 12 yr old boys [99] was half of that of young men in similar studies [74,78,79,100]. More recent studies have found a similar ratio of maximal accumulated O_2 deficit between 11 yr and 15 yr old boys, while the value of 11 yr old girls was 64% of that of 15 yr old girls [97,98]. Thus, during puberty maximal accumulated O_2 deficit seems to be doubled in males and increase by at least 50% in females even when expressed relative to body mass. This suggests an increase in maximal accumulated O_2 deficit as muscle mass increases during puberty.

The (maximal) accumulated O_2 deficit of 1.1 mmol.kg^{-1} of 60–70 yr old persons may be around half of that of healthy young people [101], suggesting that the value decreases during aging, perhaps related to decreased muscle mass.

7.5.3. *Effect of training background and physical condition on accumulated O_2 deficit*

The (maximal) accumulated O_2 deficit may be more than 30% higher for sprint-trained subjects than for endurance-trained or untrained subjects [12,44,46], but a 20% excess has also been reported [68]. The difference is at least partly due to a larger muscle mass, since endurance-trained and resistance-trained subjects had similar values when expressed relative to the leg muscle mass [77].

The (maximal) accumulated O_2 deficit of 0.5 mmol.kg^{-1} of elderly patients with chronic heart failure was only half of that of healthy, age-matched controls [101]. The low value for these patients may be no more than that accounted for by reduced O_2 stores and breakdown of phosphocreatine during exercise; 0.5 mmol.kg^{-1} is also the threshold for lactate appearance observed on healthy untrained young men [102].

7.6. *Effect of training on maximal accumulated O_2 deficit*

Regular aerobic type of training at moderate intensity for four months improved maximal accumulated O_2 deficit by 11% for 11–12 yr old boys [99], while similar training for healthy young men did not improve maximal accumulated O_2 deficit [103]. Intense interval-type of training three times per week for 6 wk, leading to considerable lactate production during each training session, may improve maximal accumulated O_2 deficit by ≈10% for relatively untrained young men and women [46]. The anaerobic energy release during each training session was ≈75% of the maximum. More severe training may increase the maximal accumulated O_2 deficit by 20–30% [96,104] or even more for relatively untrained subjects [19,105] and horses [106].

High-altitude training and training in hypoxia may improve aerobic performance. Maximal accumulated O_2 deficit does not seem to improve much during periods of high-altitude training [36,107,108].

7.6.1. *Proposed training for improving short-lasting, high-intensity performance – Irisawa-Tabata-protocol*

Aerobic processes are important even for exercise lasting less than 1 min (Figures 8 and 9). Consequently training principles improving both aerobic and anaerobic capabilities is wanted. A training program of 20 s intermittent exercise at 170% of maximal O_2 uptake, 10 s rest, repeated for 4 min (8 sets) was therefore designed [104]. During each training session the accumulated O_2 deficit over the eight sets equaled the maximal accumulated O_2 deficit, and the O_2 uptake rose to its maximum value. After 6 wk of training 4–5 d/wk maximal accumulated O_2 deficit rose by 28%, and in addition maximal O_2 uptake rose by 15% for a group of relatively untrained young men [103]. This protocol was first worked out by Irisawa, being coach of Japanese elite speed skaters, and further examined scientifically by Tabata and coworkers [103,104]. The principle is therefore called the Irisawa-Tabata protocol.

7.7. *Examination of effects of dietary supplements*

Maximal accumulated O_2 deficit rose by 9% after five days of creatine supplement, and the performance improved accordingly [109]. Caffein may improve performance, and maximal accumulated O_2 deficit rose by 11% [16] and by 17% [110] after ingestion of caffein. Thus, the accumulated O_2 deficit principle can provide information on why specific treatments do improve performance.

8. Final comments

In the 1990-is Saltin and Gastin stated that the accumulated O_2 deficit principle appears as the only likely candidate for a noninvasive method for determining anaerobic energy release during exercise [111,112]. The current principle of accumulated O_2 deficit has been used in more than 150 published studies according to searches in Pubmed and Sports discus. The principle and method has nevertheless been criticized and by some even been claimed invalid (e.g. [6,7]). The main reason for conflicting results and opinions may be that while nearly all studies refer to recommended principles [4], most studies have used short-cuts that have not been validated. Moreover, problems of nonlinearity at low exercise intensities shown above (see Figure 2) have been disregarded, resulting in apparent negative values of the accumulated O_2 deficit [7,22]. Despite the request for further methodological investigations [112], not much has been done.

Few studies give any information on quality and possible problems in their studies. Hardly any study reports pertinent statistics as the error of regression and error of the slope. Instead statistics that leading statisticians disprove for such contexts like the correlation coefficient [113] and intraclass correlation [114-116], are often used.

To sum up, referring to Gastin [112]: The accumulated O_2 deficit principle still remains a promising measure of the anaerobic energy release during exercise if properly carried out. Further investigation on methodological issues is warranted, and standardization of procedures is still necessary. Data from numerous studies have given unambiguous results on the importance of both aerobic and anaerobic energy release during short-lasting, strenuous exercise (Figure 8), and further that aerobic processes are important even for exercise lasting ≤ 30 s (Figure 9). Thus, as concluded by others [21,88,117,118], current textbook dogma need revision.

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