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# Measurements of lactate in exhaled breath condensate at rest and after maximal exercise in young and healthy subjects

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**Abstract**

Arterial lactate concentrations, taken as indicators of physical fitness, in athletes as well as in patients with cardio-respiratory or metabolic diseases, are measured invasively from arterialized ear lobe blood. Currently developed micro enzyme detectors permit a non-invasive measurement of hypoxia-related metabolites such as lactate in exhaled breath condensate (EBC). The aim of our study is to prove whether this technology will replace the traditional measurement of lactate in arterialized blood. Therefore, we determined the functional relation between lactate release in EBC and lactate concentration in blood in young and healthy subjects at rest and after exhausting bicycle exercise. During resting conditions as well as after exhausting bicycle exercise, 100 L of exhaled air along with blood samples from the ear lobe was collected after stationary load conditions in 16 healthy subjects. EBC was obtained by cooling the expired air volume with an ECoScreen I® (FILT GmbH, Berlin) condenser. The analysis was performed within 90 min using an ECoCheck® ampere meter (FILT GmbH, Berlin). Lactate measurements were performed using a bi-enzyme sensor after lactate oxidase-induced oxidation of lactate to pyruvate and $H_2O_2$. The rates of lactate release via the exhaled air were calculated from the lactate concentration, the volume and the collection time of the EBC. The functional relation of lactate release in exhaled air and lactate concentration of arterial blood was computed. At rest, the mean lactate concentration in arterialized blood was $0.93 \pm 0.30$ mmol $L^{-1}$. At a resting ventilation of $11.5 \pm 3.4$ L $min^{-1}$, the collection time for 100 L of exhaled air, Ts, was $8.4 \pm 2.9$ min, and $1.68 \pm 0.40$ mL EBC was obtained. In EBC, the lactate concentration was $21.4 \pm 7.7$ $\mu$mol $L^{-1}$, and the rate of lactate release rate in collected EBC was $4.5 \pm 1.7$ nmol $min^{-1}$. After maximal exercise load ($220 \pm 20$ W), the blood lactate concentration increased to $10.9 \pm 1.8$ mmol $L^{-1}$ and the ventilation increased to $111.6 \pm 21.4$ L $min^{-1}$. The EBC collection time decreased to $3.9 \pm 1.9$ min, and $1.20 \pm 0.44$ mL EBC were obtained in the recovery period after termination of exercise. The lactate concentration in EBC increased to $40.3 \pm 23.0$ $\mu$mol $L^{-1}$, and the lactate release in EBC increased to $13.6 \pm 8.6$ nmol $min^{-1}$ ($p < 0.01$). Assuming a volume of 4.3 mL water in 100 L of exhaled air (saturated with water at 37 °C), we calculated a lactate release at rest of $11.5 \pm 4.3$ nmol $min^{-1}$ and $48.6 \pm 30.7$ nmol $min^{-1}$ ($p < 0.01$) after exhausting exercise. Detectable releases of lactate in exhaled breath condensate were found already under resting conditions. During exhausting external load on a bicycle spiroergometer, an increase in the lactate

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concentration was found in arterialized blood along with an increased lactate release in EBC. The correlation between expiratory lactate release via EBC and lactate concentration in arterialized blood is studied in pursuing investigations.

## Introduction

Determinations of metabolic conditions in patients with cardio-respiratory disease by measurements of lactate in blood are traditionally performed on samples from arterialized earlobe blood. Exhaled breath condensate (EBC), obtained non-invasively by leading exhaled air through a cold trap, contains water, metabolites and various mediators of airway inflammation and oxidative stress. Compositions and concentrations of these substances are altered in lung disease, and they return to normal during therapy or they increase during metabolic adaptations to exercise [1].

### *Background*

Currently developed micro-enzyme detectors permit non-invasive measurement of metabolites of anaerobic glucose degradation (lactate) and markers of inflammation or oxidative stress ($H_2O_2$) in EBC. In young and healthy subjects, it was proved whether lactate release in EBC correlates with arterialized blood lactate concentrations at rest and after exhausting exercise.

Under hypoxic conditions, glucose and glycogen are degraded to pyruvate, and metabolites such as lactic acid accumulate in circulating blood and pass through the capillary bed of the lungs. Lactic acid is produced in small amounts already under resting conditions. During exhausting exercise, higher amounts of lactate are produced and cause acidification of tissues and blood, resulting in weakening of working muscle function. Increased levels of lactate in blood are found during severe exercise as well as in patients with tissue hypoxia due to acute lung injury. During first investigations in patients with pulmonary diseases, one could find lactate release in EBC of patients with pulmonary diseases [2].

The half time of lactate catabolism is 10–15 min. One part of lactate is degraded in an oxidative process to $CO_2$ and $H_2O$, another part is metabolized by the heart muscle and the major part is resynthesized to glycogen in the liver [3].

### *Lactate concentration in arterialized blood*

Resting values of 0.25–1.5 mmol $L^{-1}$ lactate are found in arterial and arterialized ear lobe blood samples of healthy subjects [4]. When external load exceeds 60% of the maximal oxygen capacity, the slope of lactate concentration continuously increases with increasing load. The aerobic–anaerobic transition range is defined at 2–4 mmol $L^{-1}$. A further increase in values depends on an increasing anaerobic metabolism with lactic acid production. Glycolysis produces two molecules of lactic acid for every glucose molecule. Most of the lactate is used for gluconeogenesis: one part is metabolized in anaerobic working muscles, and a smaller amount is degraded by brain, kidneys, heart and red skeleton muscles to $H_2O$ and $CO_2$. These mechanisms of lactate metabolism depend on a rapid transport from unaerobic working muscles into lactate-consuming cells. Since lactic acid at physiological pH is almost completely dissociated into lactate and protons, the charged lactate ions can only cross the plasma membranes by specific transport mechanisms and not by free diffusion. A rapid lactate exchange across the mitochondrial and plasma membrane is effectively catalyzed by a family of proton-linked monocarboxylate transporters (MCTs) found in tissue and membranes of metabolic active cells [5]. These transporters catalyze the facilitated diffusion of lactate with protons and do not need further energy input, solely depending on the concentration gradients of lactate and protons. So far, determination of the quantitative kinetics of lactate in- and efflux in intact skeleton muscles is not possible. Studies on sarcolemmal membrane vesicles confirm the exchange of monocarboxylates across the skeleton muscle membrane by means of a saturable and stereospecific transport system which shows a 1:1 coupling between lactate and $H^+$ [6]. Using current technology, a quantitative measurement of lactate production at rest and during external load cannot be performed.

### *Aims of investigation*

The correlation between lactate concentration in arterial blood and lactate release in exhaled air was studied in young and healthy subjects performing exhausting bicycle exercise. The aim of the present investigation was to prove whether the rate of lactate release in EBC correlates with lactate concentrations in arterial blood and whether, therefore, the lactate analysis in blood samples in future can be replaced by non-invasive measurement in exhaled breath condensate.

## Methods

### *Anthropometrical data*

The investigation was carried out on 16 healthy sporting subjects (9 males, 7 females), age 23 ± 1.4 years (range 22–26 years), 175 ± 8.4 cm height and normal BMI 22.2 ± 1.7 kg $m^{-2}$ (range 19.7–24.8 kg $m^{-2}$). All subjects were free from acute airway infections and had, according to the European Community for Coal and Steel (ECCS) reference formulae for spirometry [7], highly normal values for the forced vital capacity (FVC). For males 113 ± 11.4% of the ECCS predicted values (%pred.) and for females 108 ± 10.3%pred. were measured. The mean values for forced expiratory volume in 1 s ($FEV_1$) were also highly normal, 105.7 ± 8.8%pred. in females and 119.1 ± 10.5%pred. in males. Tiffeneau index, another marker for airway obstruction,

was also highly normal in all subjects (males 107 ± 4.5%pred. and 102 ± 5.9%pred.).

*Study protocol*

The subjects performed bicycle exercise (Ergoline E900, Bitz Germany), preferentially in the morning hours in an air-conditioned room, following the BAL protocol widely used in performance evaluation of athletes, starting with 5 min cycling at 50 W and increasing external load by 50 W every 3 min. Respiratory parameters, using a medium-sized face mask and heart rate meter (Polar system), were continuously recorded by a ZAN 600 USB CPA spiroergometer (ZAN Oberthulba, Germany). Before each exercise test, appropriate calibrations of the air-flow sensor were performed with a 3.0 L syringe and calibrations of the $O_2$ and $CO_2$ sensors with calibration gases from cylinders (5% $CO_2$, 16% $O_2$, 79% $N_2$) and ambient air.

*Collection of exhaled breath condensate (EBC)*

At rest and after reaching the break-off exercise level, 100 L of exhaled air and samples of arterialized blood from the ear lobe were collected [8]. The subjects were instructed to breath normally and to avoid contamination with saliva. During quiet breathing, 99% of the condensate is derived from water vapour; only up to four small particles per mL exhaled air with a mean diameter of 0.3 $\mu$m add to the condensed water vapour [9, 10]. EBC was obtained by cooling 100 L of expired air in a −20 °C cold trap (ECoScreen I, FILT GmbH, Berlin). The trap inlay was Teflon coated and the condenser cooled the expired air in a counter-flow principle. At rest, 100 L of exhaled air was collected in 8.4 ± 2.0 min and 1.68 ± 0.39 mL EBC was obtained. After exhausting exercise at 220 ± 23.5 W, 100 L of exhaled air was collected in 3.9 ± 1.8 min and 1.20 ± 0.44 mL EBC was obtained. 100 L of exhaled air at 37 °C saturated with water vapour at standard atmospheric pressure (101 kPa) should contain 4.4 mL water [11]. The collected EBC volumes represent 39.1% and 27.9% of the theoretical water vapour content of 100 L of exhaled air. From the EBC sample, three aliquots were taken for lactate, $H_2O_2$ and pH and acid–base measurements.

The rates of expiratory lactate release (nmol $min^{-1}$) were calculated and obtained from the concentrations of lactate in EBC, EBC volume and the time of collection. Since the condenser extracts only up to 40% of the water vapour during quiet breathing, the release of lactate in the theoretical amount of 4.4 mL water from 100 L water vapour saturated exhaled (alveolar) air at 37 °C was calculated in addition to lactate release in collected EBC [11]. A functional relation of lactate releases in EBC and lactate concentrations of arterial blood was computed.

*Lactate measurements in EBC*

Lactate analysis was performed after conversion of lactate to pyruvate and $H_2O_2$ using miniaturized bi-enzyme sensors (ECoCheck, FILT GmbH, Berlin). In the second step, the resulting $H_2O_2$ was analysed amperemetrically after conversion of $H_2O_2$ by peroxidase ($H_2O_2 \rightarrow 2H^+ + O_2 + 2e^-$). The released $H_2O_2$ is oxidized at the platinum electrode and induces an electrical current proportional to the lactate concentration. The sensor is highly specific for $H_2O_2$ and two- to threefold more sensitive than chemoluminescence or fluorescence photometry. Measurements of $H_2O_2$ can be performed in an effective range between 30 and 3000 nmol $L^{-1}$. Calibrations were performed for every subject. The method was described in detail in previous papers [12, 13]. Acid–base parameters were measured in crude EBC samples, equilibrated with 5% $CO_2$. Lactate was measured in 0.3 mL EBC diluted in a 0.3 mL buffer solution. The rate of release from the lung (mol $min^{-1}$) is calculated from the concentration of lactate, the collection time and EBC volume. Lactate concentrations can be measured in the range between 5 and 150 $\mu$mol $L^{-1}$. There is no relevant cross sensitivity between lactate and $H_2O_2$ since lactate concentrations are 100- to 1000fold the natural $H_2O_2$ concentrations. For lactate measurements, sensitivity is adequately reduced.

*Lactate measurements in earlobe blood*

The right earlobe was arterialized by hyperaemization with Finalgon® (Böhringer, Mannheim, Germany) before the EBC collection at rest. Blood samples were collected in 20 $\mu$L heparin-coated glass tubes before the exercise challenge, immediately after break-off and 3, 6 and 9 min later. The samples were haemolyzed in a 1.0 mL reaction solution in 2 mL Eppendorf caps. Lactate measurements were performed photometrical on the same day with an automated Eppendorf Ebio plus photometer (Eppendorf, Wesseling-Berzdorf, Deutschland).

*Statistical analysis*

Data were compiled in tables (Microsoft, Excel 2003). The final statistical analysis was performed using the SPSS 11.5 statistic program. After testing the data for homogeneity and normal distribution, means and standard deviations were calculated and presented in tables and box plots. Using Fisher's *t*-test for paired data, respiratory and cardiovascular data were tested for significant alterations before and after exercise testing ($p < 0.05$).

## Results

*Respiratory and cardiac responses*

At exhausting exercise levels ventilation in males and females increased by nearly tenfold to 123 ± 21.3 L $min^{-1}$ and 97.7 ± 10.7 L $min^{-1}$ respectively compared to resting ventilation. At maximum load, females reached a mean heart rate of 186 ± 9.5 beats $min^{-1}$ at 200 ± 23.6 W and males 180 ± 6.3 beats $min^{-1}$ at 241 ± 24.9 W. The oxygen consumption increased to 44.9 ± 3.3 mL $min^{-1}$ $kg^{-1}$ in females and 49.6 ± 6.6 mL $min^{-1}$ $kg^{-1}$ in males. Oxygen pulse reached its peak value at 14.6 ± 1.5 and 21.0 ± 1.8 mL/heart beat in females and males. In all subjects, the respiratory exchange rate (RER) exceeded 1.00, and reached a mean value of 1.15 ± 0.05 at the break-off point in females and 1.14 ± 0.07 in males.

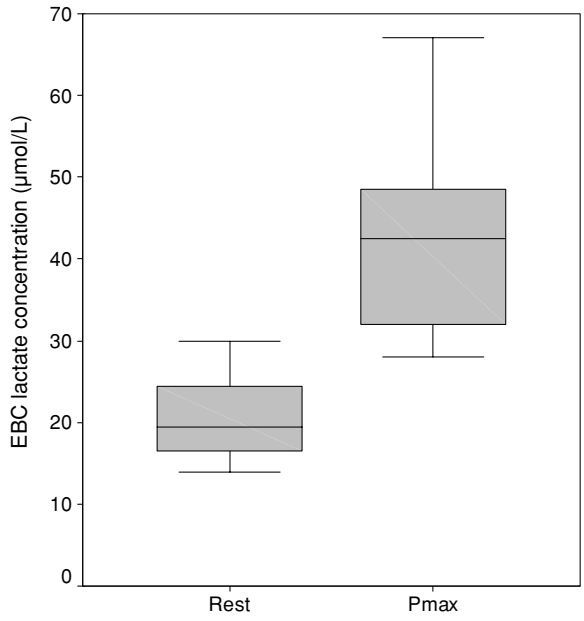


**Figure 1.** Lactate concentration in collected EBC volume at rest and after exhausting bicycle exercise ($P_{max}$).

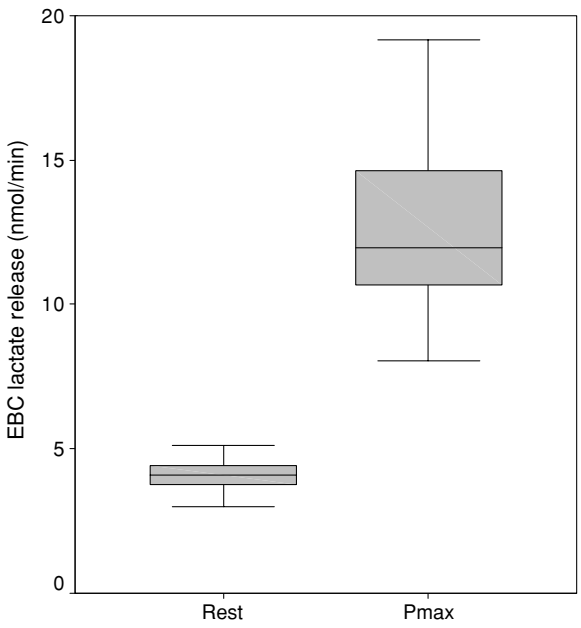


**Figure 2.** Lactate release per minute derived from collected EBC volume at rest and after exhausting exercise ($P_{max}$).

*Exhaled breath condensate*

Under resting conditions, 100 L of exhaled air was collected within 8.4 ± 2.0 min according to a ventilation of 11.5 ± 3.4 L min$^{-1}$, which delivered 1.68 ± 0.39 mL EBC. The collected EBC volume represents 39.1% of the theoretical water content of 4.4 mL in 100 L of air, saturated with water vapour at 37 °C and normal barometric pressure (101 kPa). In the recovery period, after exhausting exercise, the collection time for 100 L of exhaled air was significantly shortened to 3.9 ± 1.8 min ($p < 0.05$) according to a mean ventilation of 25.6 ± 11.7 L min$^{-1}$. The EBC volume was decreased to 1.20 ± 0.44 mL, or 27.9% of the theoretical water content.

*Lactate release in EBC compared to arterialized blood*

At rest, the lactate concentration in arterialized blood was 0.93 ± 0.30 mmol L$^{-1}$ at a resting ventilation of 11.5 ± 3.4 L min$^{-1}$. The lactate concentration in EBC at rest was 21.4 ± 7.7 $\mu$mol L$^{-1}$, and the lactate release rate in the collected EBC volume was 4.5 ± 1.7 nmol min$^{-1}$ (figures 1 and 2).

After reaching the exercise break-off point (external load 220 ± 20 W), the blood lactate concentration increased to 10.9 ± 1.8 mmol L$^{-1}$ (figure 4) and the ventilation increased to 111.6 ± 21.4 L min$^{-1}$. The lactate concentration in EBC increased to 40.3 ± 23.0 $\mu$mol L$^{-1}$, and the lactate release in EBC increased to 13.6 ± 8.6 nmol min$^{-1}$ ($p < 0.01$) in the collected EBC volume (figures 5 and 6).

Taking the theoretical water volume at 37 °C of 4.4 mL EBC into account, the following rates of lactate release were calculated: 11.5 ± 4.3 nmol min$^{-1}$ at rest and 48.6 ± 30.7 nmol min$^{-1}$ ($p < 0.01$) after termination of maximal exercise, corresponding to a 4.2-fold increase (figure 3). Lactate release in EBC significantly correlated to lactate concentration in arterialized blood ($p > 0.01$) (figures 7 and 8).

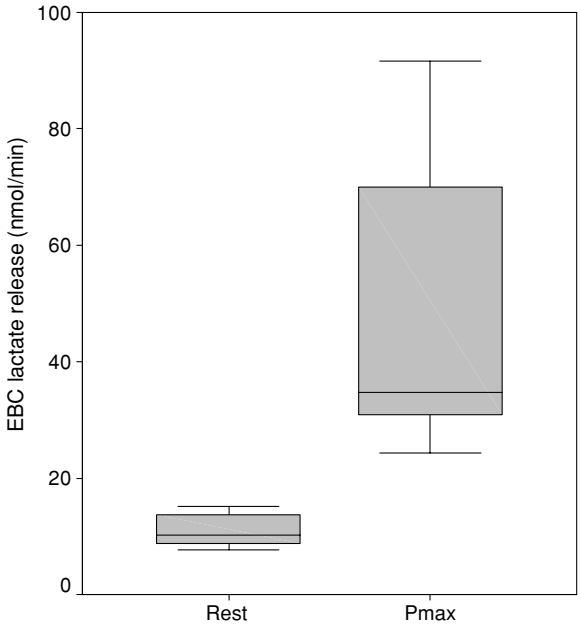


**Figure 3.** Lactate release per minute in total amount of water of 4.4 mL derived from 100 L of exhaled air at 37 °C and saturated with water vapour at rest and after exhausting exercise ($P_{max}$).

## Discussion

In young and healthy subjects, a detectable release of lactate in exhaled breath condensate (EBC) is already found under resting conditions. After exhausting external load, increasing lactic acid production results in an increase in lactate concentration in arterialized blood along with an increased lactate release in EBC. Lactate release in EBC increased more than fourfold after exhausting exercise while lactate concentrations in arterialized blood increased nearly

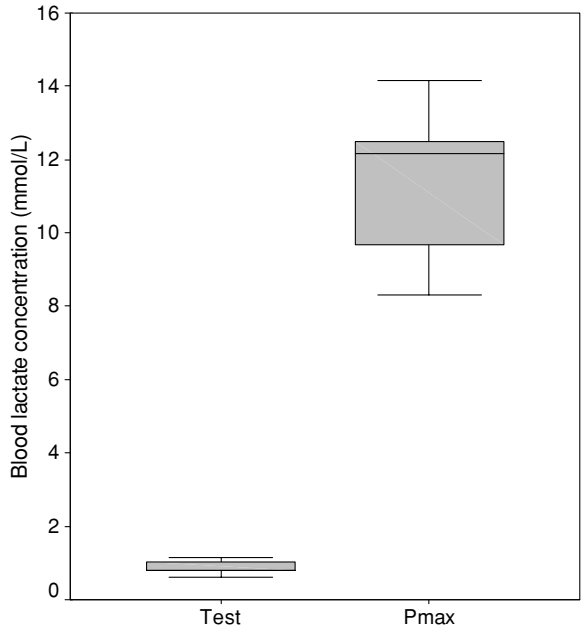


**Figure 4.** Lactate concentration in arterialized earlobe blood at rest and after exhausting bicycle exercise ($P_{max}$).

tenfold. The relation between the rate of lactate release in EBC and lactate concentration in arterialized blood at various defined levels of mechanical loads of bicycle exercise is studied in pursuing investigations. From our results we have got evidence that lactate concentrations in blood can be predicted on the basis of the lactate release in EBC. The present results support the aim of the investigation that lactate measurements in EBC, after further development of enzyme sensors, may in future replace invasive measurements in earlobe blood.

*Collection of EBC*

At rest, 100 L of exhaled air was collected in 8.4 ± 2.0 min (11.9 L $min^{-1}$) and 1.7 mL EBC was extracted by the −20 °C cold water trap. A ventilation of 11.9 L $min^{-1}$ is within the flow range obtained from healthy adults under resting conditions [10]. 1.7 mL EBC collected at rest represents only 39% of the theoretical water content of 4.4 mL in 100 L of exhaled air, saturated with water vapour at 37 °C and standard atmospheric pressure [11]. As reported by Proctor in *Handbook of Physiology* [14], in the alveolar space inspired air is warmed up to body temperature and water vapour saturated.

Gessner and co-workers reported that 40% of the water vapour was extracted by the ECoScreen I cold trap with no differences in healthy subjects and patients with COPD [15], which is in line with our results (39%) of EBC collection at rest [8]. Anderson estimates a water loss of only 3.0–3.5 mL per 100 L of exhaled air [16], taking into account that inhaled room air is water vapour saturated by about 60%.

After termination of exercise, ventilation gradually decreased from a maximal value of 120 L $min^{-1}$ to a normal value at rest. The time needed for the collection of 100 L exhaled air in this period of recovery decreased to 3.9 ± 1.8 min, which corresponds to a mean ventilation of 25 L $min^{-1}$. From this volume only 1.2 mL EBC was condensed which is only 28% of the total water.

It is unknown whether exhaled air is still saturated with water vapour at exhausting exercise levels. In the recovery period when the mean ventilation decreased to 25 L $min^{-1}$, it does not seem to be probable that exhaled air was not saturated with water vapour at the recorded flow rates. The cooling trap becomes less effective at higher flow rates and smaller water volumes are recovered. This observation is in accordance with the results of Reinhold and co-workers who found in calves that the EBC volume derived from 100 L of exhaled air decreased with increasing ventilation of the elder calves [17]. An insufficiency of the condenser was also reported by McCafferty and co-workers altering ventilation and breathing pattern in humans [18]. As known from the end tidal $CO_2$ partial pressure or the concentration of $H_2O_2$ in exhaled air [19], the concentration of lactate in exhaled air and in EBC should depend on alveolar ventilation. In order to standardize the measurements, lactate release per minute was calculated from the amount of EBC collected and for the theoretical amount of 4.3 mL water that can be extracted from 100 L of exhaled air between the body temperature of 37 °C and −20 °C in the cold trap. Even at −20 °C, 0.1 mL water vapour will remain in 100 L of expired air. There are no experimentally based arguments against the assumption that lactate release is proportional to the amount of EBC volume collected. The capacity of the cold trap is limited, but the concentration of volatile substances should not depend on the recovery ratio. So, in addition to the lactate released in EBC collected, lactate release was calculated for the theoretical amount of water that can be extracted from 100 L of exhaled air, and not only due to the limited capacity of the cold trap.

Our assumption is supported by the results of McFadden and co-workers on intra-airway thermodynamics during exercise and hyperventilation in healthy subjects and asthmatics. In healthy and asthmatic subjects, the intra-airway temperature fell progressively as ventilation increased, and there were no significant differences between the thermal profiles of the two populations investigated at rest or during exercise. Calculation of water losses and the osmolality of the airway surface fluid failed to demonstrate significant airway drying in either group [20].

During each provocation, intra-airway temperatures fell equivalently, thereby producing similar intrathoracic water fluxes and heat transfers. Neither stimuli was associated with airway drying, and both resulted in similar distributed losses of thermal energy from the tracheo bronchial tree despite small regional heat and water exchanges [21].

*Lactate release in EBC*

The subjects investigated reached a $P_{max}$ at 220 W (3.24 W $kg^{-1}$) at the break-off point with lactate concentrations of 8–14 mmol $L^{-1}$ at an oxygen uptake rate of 47 mL $kg^{-1}$ $min^{-1}$, and a respiratory exchange rate of 1.15. The half life period of lactate in peripheral blood is 10–15 min, long enough to be measured in blood samples and in exhaled breath condensate collected within the first 6 min after

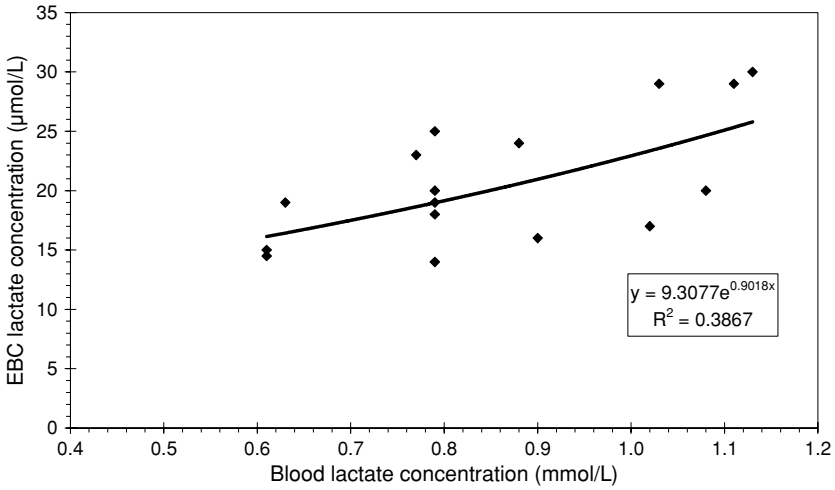


**Figure 5.** Correlation between blood lactate concentration and EBC lactate concentration under resting conditions.

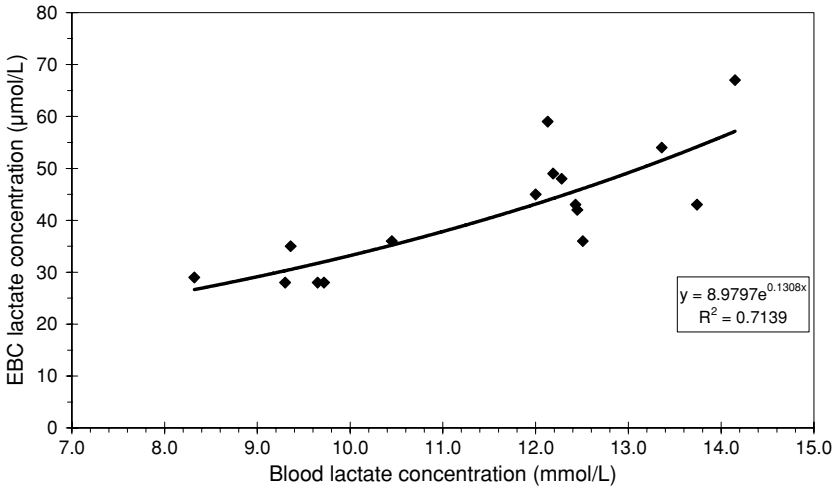


**Figure 6.** Correlation between blood lactate concentration and EBC lactate concentration after exhausting exercise ($P_{max}$).

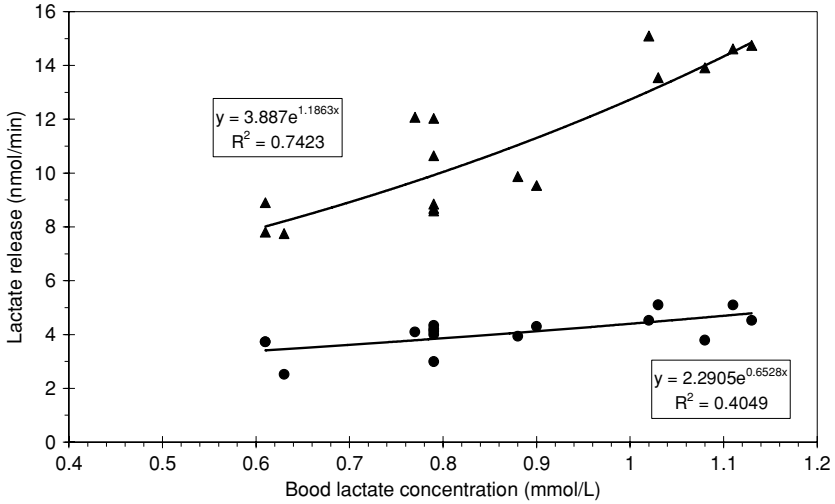


**Figure 7.** Correlation between blood lactate concentration and lactate release per minute in collected EBC (●) and in theoretical amount of 4.4 mL water of 100 L of exhaled air (▲) under resting conditions.

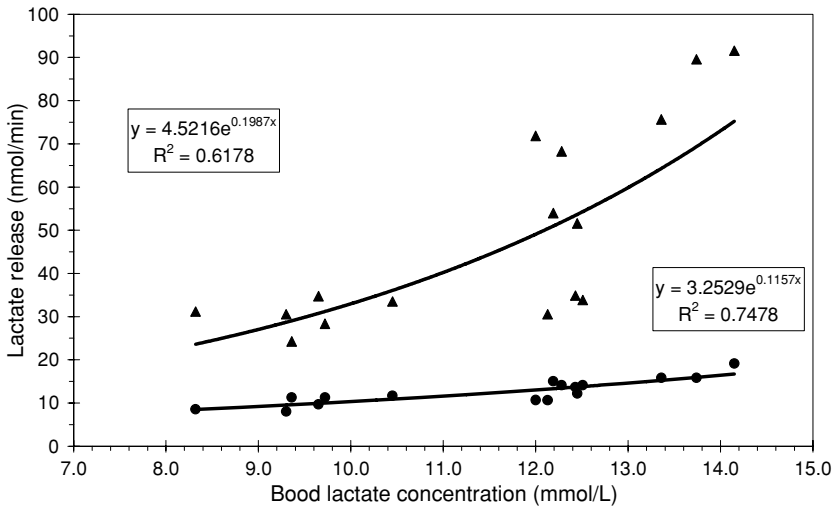


**Figure 8.** Correlation between blood lactate concentration and lactate release per minute in collected EBC (•) and in theoretical amount of 4.4 mL water of 100 L of exhaled air (▲) after exhausting exercise ($P_{max}$).

termination of exercise. As observed in humans, lactate release in EBC is small in other species too. In experiments on calves, lactate concentrations were beyond the detection limit (0.5 $\mu$mol $L^{-1}$) [22]. In healthy subjects, lactate concentrations in EBC of 2.5 ± 0.4 $\mu$mol $L^{-1}$ were found [2]. In patients with acute lung injury, lactate and $NH_4^+$ closely correlated to EBC pH [23]. Greenwald and co-workers studied ionic concentrations of numerous substances including lactate in EBC of adolescent endurance runners before and after track practice. They did not find significant effects of exercise on lactate concentration in EBC, but an acidification of EBC pH and an increase in ammonia concentration. Compared to exhausting bicycle exercise performed in the present investigation, track practice is not as intensive and lactate concentrations in blood should not increase as much as found in our study. However, the rate of lactate release was not measured in their investigation [24].

It is still unknown how and how much lactate enters the alveolar space along with $CO_2$ and other volatile substances and is released to ambient air. The release of lactate could be due to the equilibrium between the non-dissociated lactic acid of the gas mixtures in the respiratory tract and blood and tissue.

Lactate, which is found in exhaled air, should reflect the amount of non-dissociated lactic acid in blood and tissue. After diffusion into the alveolar space, lactic acid should dissociate almost completely into its conjugated base lactate and $H^+$ ions (dissociation constant $1.4 \times 10^{-4}$). According to the law of mass action, the concentration of lactic acid is equal to the ratio: (lactic acid concentration × $H^+$ ion concentration)/dissociation constant.

At a $H^+$ ion concentration of 40 nmol $L^{-1}$ (pH = 7.40) and a lactate concentration of 1 mmol $L^{-1}$ blood, the concentration of non-dissociated lactic acid is 316 nmol $L^{-1}$ blood. From our measurements, we calculate a lactate release of 11.5 nmol $min^{-1}$ at a resting ventilation of 11.5 L $min^{-1}$, yielding a lactate concentration of the expired air of 1 nmol $L^{-1}$. This concentration is even two orders of magnitude lower than in the blood compartment.

The relationship of release and uptake of the lactic acid–lactate system in the lung can be assessed as follows: lactate release in EBC increased from 11.5 nmol $min^{-1}$ at rest to 48.6 nmol $min^{-1}$ after exhausting exercise. Regarding a body weight of nearly 70 kg and a blood volume of 5.0 L (7% of the body mass), during a mean circulation time at rest of 1 min, 5 mmol $min^{-1}$ lactate will perfuse the lungs, but only two lactate molecules per million will be released into exhaled air (outflux = 11.5 nmol $min^{-1}$, influx = 5 mmol $min^{-1}$, outflux/influx = 2.3/1000000) under the assumption of an anionic lactate transport system.

Assuming an electro neutral lactic acid transport in the lung, we yield the following result: about 8 out of 1000 lactic acid molecules are released (outflux = 11.5 nmol $min^{-1}$, influx = 5 × 300 nmol $min^{-1}$, outflux/influx = 7.7/1000).

## Conclusion

A lactate concentration of 1.0 nmol $L^{-1}$ was found in exhaled air of young and healthy non-smokers already under resting conditions. The rate of lactate release (11.5 nmol $min^{-1}$) at rest represents only a minor fraction of about 5 mmol $min^{-1}$ lactate, perfusing the capillary pulmonary vessels. Lactate release in EBC increased 4.2-fold after exhausting exercise, while lactate concentration increased nearly tenfold in arterialized blood. However, lactate release in EBC correlated to oxygen consumption. The results support the idea that non-invasive lactate measurements in EBC may replace measurements in earlobe blood. The correlation of lactate released in EBC and lactate concentration in arterialized blood under different levels of bicycle exercise is studied in pursuing investigations.

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