

Does Oxygen Uptake Before Physical Exercise Affect Tear Osmolarity?

by

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Recently, it has been reported that tear osmolarity (Tosm) is correlated with plasma osmolarity and will increase during exertion. We aimed to assess whether inhaling oxygen-enriched air between exercises could significantly change the Tosm value. Thirty men aged 24.9 years were included in the study. A cycloergometer was used to perform the exercise protocol. We recorded the participants' Tosm (mOsm/L), heart rate (HR, beats/minute), oxygen saturation, and blood pressure values. After the first exhaustive exercise (T1), participants inhaled oxygen in the experimental group and a placebo in the control group. After the second exercise (T2), another set of measurements was obtained. The Tosm value before exercise was 297.4 ± 1.21 and 296.53 ± 1.11 mOsm/L ($p = 0.61718$) and the HR was 72.6 ± 2.59 and 73 ± 2.59 beats/minute ($p = 0.39949$) in the study and the control group, respectively. At T1, Tosm was 303.67 ± 1.25 and 302.2 ± 1.25 mOsm/L ($p = 0.41286$) and the HR reached 178.04 ± 2.60 and 176.4 ± 2.60 beats/minute ($p = 0.65832$), respectively. At T2, Tosm in the study group reached 305.73 ± 0.86 mOsm/L (correlation with the use of oxygen: $r = -0.3818$), and in the control group, it was 308.4 ± 0.86 mOsm/L ($p = 0.0373$), while the HR reached 172.20 ± 2.53 beats/minute in the study group and 178.2 ± 2.53 beats/minute in the control group ($p = 0.057$). It was concluded that inhaling oxygen before and after exercise could increase the rate of recovery after exhaustive exercise.

Key words: tearing, body water, sport, lacrimation, TearLab, Tosm.

Introduction

Over the years, oxygen therapy has become a well-established method in the treatment of cardiovascular and respiratory disorders. Nowadays, the use of oxygen is not limited to the treatment of various disorders, but is also considered to increase the mental and physical abilities of the human body (Wylęgała, 2016).

Oxygen deficit in the body during exercise leads to fatigue, which is caused by inadequate energy supply. This leads to an increase in both the heart rate (HR) and the breathing rate (Chung et al., 2008). Exercise beyond 40% of the maximal oxygen uptake

significantly increases plasma osmolarity (Convertino et al., 1981).

Recently, the use of disposable tanks with 5 L of pure oxygen to be used *before* exercise has been marketed; producers claim that oxygen can significantly increase performance during short-term training. Inhalation of 30% oxygen increases minute ventilation by 21% (Chung et al., 2008). Inhaling oxygen for 2 min causes transient hyperoxia that lasts approximately for six minutes (Suchý et al., 2010). Inhaling oxygen-enriched air increases exercise performance. However, literature shows mixed evidence regarding whether inhaling oxygen during rest and before

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exercise can increase such performance. In sports sciences and medical therapy, there is always a need to find a reliable, fast, and noninvasive method of assessing body hydration. Previously, the measurement of tear osmolarity (Tosm) was time-consuming and required large quantity of tears; however, with the introduction of new clinical devices, these drawbacks have been overcome. Currently, Tosm is more frequently used in both research and clinical settings (Wylęgała et al., 2019). Tosm can replace plasma and urine osmolarity as a standard form of measuring hydration. Many authors have linked plasma osmolality with Tosm (Fortes et al., 2011; Sollanek et al., 2012; Ungaro et al., 2015; Vera et al., 2017). However, previous studies have provided mixed results regarding the correlation between Tosm and plasma osmolality. It is possible that changes in plasma volume, due to an increased amount of the blood flow through the muscles, skin, and pulmonary tissue, are responsible for the increase in the Tosm value during exercise.

Oxygen supplementation increases exercise performance (Moore et al., 1992); thus, we hypothesized that the use of oxygen during rest intervals would decrease changes in Tosm inherent to physical exercise. Therefore, in this study, we aimed to investigate whether commercially available oxygen could have an effect on Tosm value during exercise.

Methods

Participants

This study was approved by the Ethics Committee of the Silesian Medical University KNW/0022/KB1/47/I/17. All the participants provided their written informed consent and received a leaflet describing the procedures before conducting the study.

A total of 30 healthy males participated in the study. Their mean (\pm standard deviation) age was 24.9 ± 1.69 years. The day before the experiment, all participants underwent a slit-lamp examination of the anterior segment and completed a Polish version of the Ocular Surface Disease Index (OSDI) questionnaire. Contact lens wearers, individuals with dry eye disorder, and any other ocular conditions, as well as individuals with systemic disease e.g. (diabetes, allergies, hormonal disorders) were excluded from the

study.

Furthermore, only participants with a score ≤ 12 of a self-assessed OSDI were selected.

In this study, we used 5 L of orange-flavored oxygen Oxywatt (Schothelt, Bielsko-Biała, Poland) containing 95–99.5% oxygen. The control group inhaled compressed atmospheric air with the same scent as the study group. Participants were randomly assigned to one of the groups.

Measures

As it has been already shown, evaporation during physical exercise is related to altered blinking that might lead to an unnatural increase in Tosm, thus exercises were performed with the participants' eyes shut (Wylęgała et al., 2019). The workload was calculated individually using the following mathematical formula: %Heart rate capacity (HRC) = $100 \times (\text{HR after exercise} - \text{HR sitting}) / ((220 - \text{age}) - \text{HR sitting})$ (Kinoshita et al., 2016).

Tosm was measured using the TearLab system (TearLab Osmolarity System, TearLab Corp., San Diego, CA, USA). It utilizes temperature-corrected electrical impedance to measure the Tosm within 60 s (Bunya et al., 2015). According to the manufacturer's instructions, this device needs to be calibrated every day with the control 334-mmol/L card before the measurements. Furthermore, two random cards were verified with osmolarity control solutions after opening a new box of 42 cards. Using the patient monitor FX 3000 (Emtel, Zabrze, Poland), we measured the HR, blood pressure, and blood oxygen saturation. Next, Cycloergometer BH Spada (Beistegui Hermanos S.A, Vitoria-Gasteiz, Spain) was used to perform the interval test. The study was conducted at a constant temperature of 22°C and 40% humidity, between 9 am and 12 noon. The temperature and humidity were measured with a thermo-hygrometer (Beurer HM 16, Beurer GmbH, Ulm, Germany).

Design and Procedures

Participants were seated and listened to a lecture 30 min before the start of the experiment. Before the beginning of the exercise, i.e., at T₀, Tosm, HR, blood pressure, and blood oxygen saturation were measured. Participants performed two exercises at an interval of 10 min. After a 2 min warm-up, participants were asked to cycle up to 70% of their maximal HR capacity for 2 min.

Then, Tosm, HR, and blood pressure were measured. During 10 min of recovery, they took 5 deep breaths of either the placebo or oxygen at the first and the fifth and in the last minute of recovery; then, they performed the 2-min 70% maximal HR capacity exercise protocol again on the basis of the Watt value measured in the first exercise. During testing, participants were not allowed to drink. Tosm was measured before the warm-up and directly after completing each exercise. The data were analyzed using Statistica v10.2.1 (Statsoft, Cracow, Poland). The descriptive data included the mean (\pm standard deviation) and standard error values. Data normality was evaluated using the Levene's test for the homogeneity of variances and the Shapiro-Wilk test. The values at T1 and T2 were compared with the control values using a one-way ANOVA followed by the Fisher's LSD posthoc test.

Results

Before the first bout of exercise, Tosm reached 297.4 ± 1.21 and 296.53 ± 1.11 mOsm/L in

the study and control group, respectively (Figure 1) ($p = 0.61718$).

After the first exercise bout, Tosm in the study group reached 303.67 ± 1.25 mOsm/L, and in the control group, it was 302.2 ± 1.25 mOsm/L ($p = 0.41286$). After the second exercise bout, Tosm was elevated in both groups; 305.73 ± 0.86 and 308.4 ± 0.86 mOsm/L in the study and control group, respectively, which was statistically significant ($p = 0.0373$) when compared with the control. Tosm measured at T2 correlated with oxygen uptake ($r = -0.3818$). Before exercise, the HR in the control and in the study group was 75.73 ± 2.59 and 72.6 ± 2.59 beats/min, respectively. After the first exercise bout, the HR increased to 176.4 ± 2.60 and 178.04 ± 2.60 beats/min in the control and in the study group, respectively. After the second exercise bout, the HR reached 172.20 ± 2.53 beats/min in the study group and 178.2 ± 2.53 beats/min in the control group. The results were not statistically significant ($p = 0.057$) (Figure 2).

Table 1. Values of the systolic and diastolic pressure measured during the exercise protocol.

		Control	Study	<i>p</i> -value
Systolic T0 mmHg	Mean	132.27	126.93	$p = 0.36058$
	SE	4.06	4.06	
Systolic T1 mmHg	Mean	147.47	144.07	$p = 0.571$
	SE	4.20	4.20	
Systolic T2 mmHg	Mean	146.60	152.07	$p = 0.255$
	SE	3.32	3.32	
Diastolic T0 mmHg	Mean	75.27	73.67	$p = 0.719$
	SE	3.11	3.11	
Diastolic T1 mmHg	Mean	74.60	73.27	$p = 0.699$
	SE	2.41	2.41	
Diastolic T2 mmHg	Mean	78.13	76.20	$p = 0.477$
	SE	1.89	1.89	

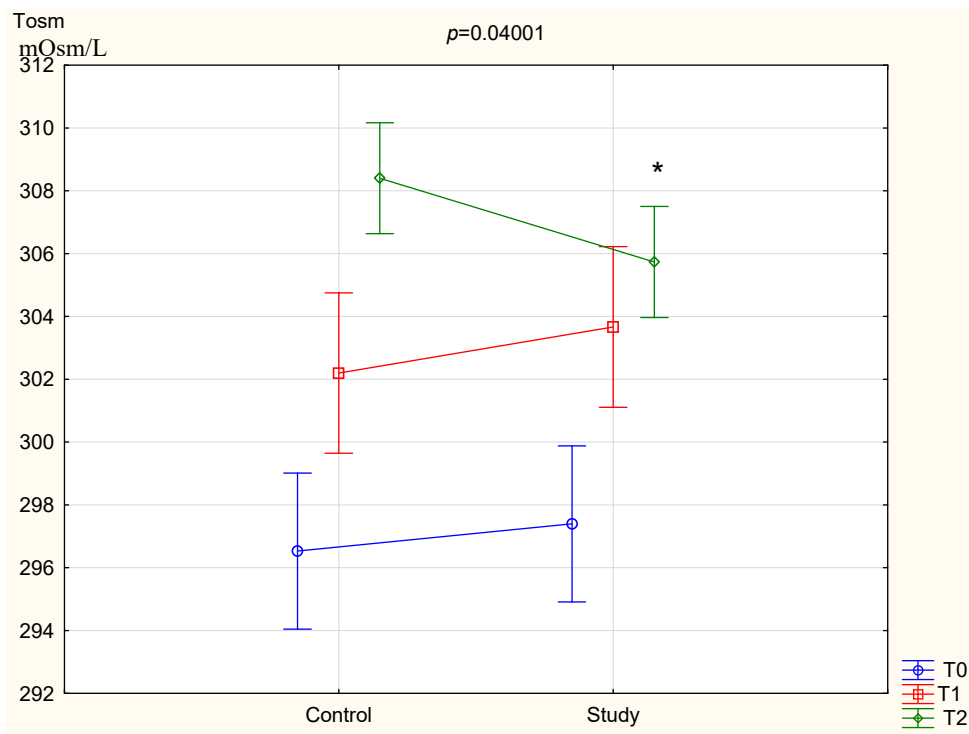


Figure 1. Changes in Tosm measured before the exercise T0, in the rest interval T1 and after the second exercise bout T2 in the group that inhaled oxygen and in the control group.

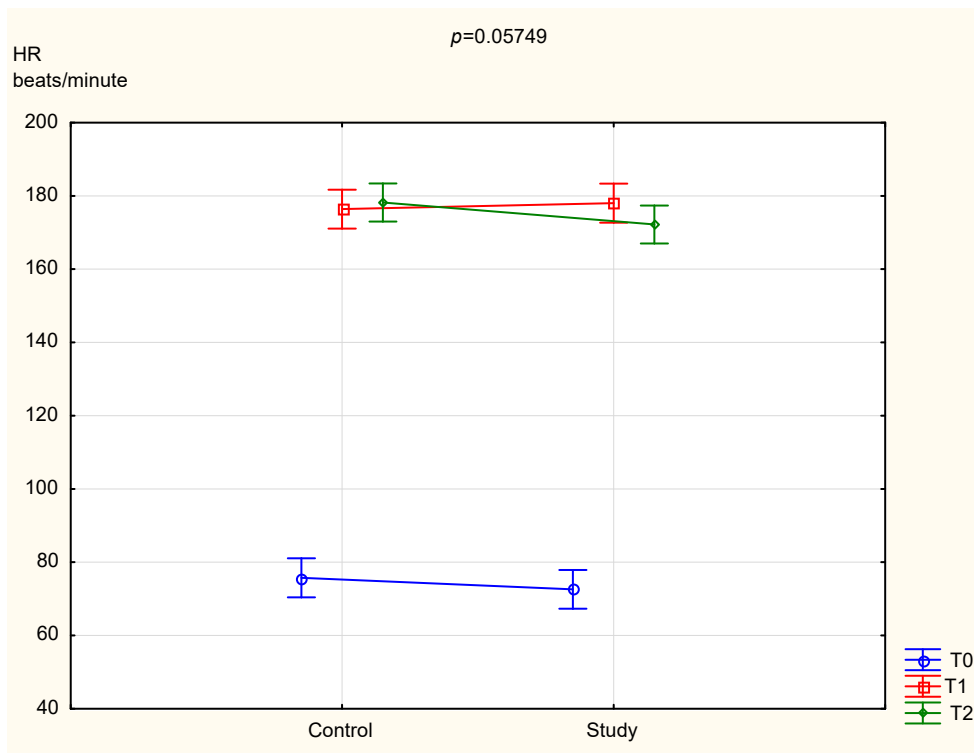


Figure 2. Changes in HR measured before the exercise T0, in the rest interval T1, and after the second exercise bout T2 in the experimental and in the control group.

Table 1 presents the results of the diastolic and the systolic blood pressure before and after the first and the second exercise bouts; these differences were found to be non-significant.

Discussion

In this study, we investigated the effect of inhaled oxygen on physical exercise by administering it at various concentrations to the study group during cycloergometer exercise. Inhaling oxygen during exercise can be beneficial to the participant. Our study showed that short term exercise could lead to an increase in *Tosm*, which was smaller in the study group.

When participants inhaled 40% oxygen, their running time on a treadmill increased to 300 s from 250 s before inhalation (Wilson and Welch, 1975). The inhalation of oxygen increased the partial pressure of oxygen in rowers (Nielsen et al., 1998) and cyclists by 10% (Knight et al., 1993), and in runners (Nummela et al., 2002); in the latter group, breathing 40% oxygen decreased the drop in pH (physiological acidity) during exercise (Nummela et al., 2002). The inhalation of 30% oxygen increased the CO₂ emission, which was interpreted as an increase in the production of ATP in the oxidative phosphorylation process (Prieur et al., 1998). Inhaling oxygen can decrease the number of breaths and the subjective scores of fatigue and breathlessness, as well as increase blood saturation (Moore et al., 1992). Furthermore, inhalation of oxygen before exercise decreases the level of lactate accumulation in muscle tissue (Pupiš et al., 2010). Most of the previous studies have been performed in laboratory settings where participants inhaled oxygen during exercise (Chung et al., 2006; Han et al., 2011; Peltonen et al., 1995; Prieur et al., 1998; Pupiš et al., 2010). Only few studies described the utilization of oxygen before exercise (Chung et al., 2006; Suchý et al., 2010). In this study, we attempted to create settings that would resemble the possible use of oxygen in sports, using metal canisters containing 5 L of oxygen in concentrations ranging from 95 to 99.5%, which are now commercially available. By decreasing both the respiratory rate and the HR, we could increase the frequency and the length of effort. In another study (Suchý et al., 2010), athletes inhaled 30% oxygen during rest intervals between maximum effort exercise on a cycloergometer.

After the warm-up, a 30-s Wingate test was conducted, followed by a recovery period of 8 min. During this time, athletes took eight breaths lasting approximately 2 s. In that study, participants in the study group inhaled oxygen from a disposable canister, whereas the control group inhaled atmospheric air. After the rest interval, another bout of physical exercise was performed. The results showed a decrease in the difference in maximum power output between the first and the second tests in the study group. Moreover, the average power observed during the experiment was found to be higher in the study group than that of the control group. Participants in the experimental group also experienced lower fatigue, along with a lower concentration of lactate and a lower HR (Suchý et al., 2010). These results are contradictory to those reported by Robins et al. (1992) as they showed that breathing oxygen after an exercise bout had no effect on peak VO₂ and the HR (Robbins et al., 1992). However, their study was conducted on 13 highly trained individuals, while in our study, we included only participants with moderate weakly sports activity. Winter et al. (1989) conducted a study on 12 soccer players and reported no changes neither in the level of lactate accumulation nor in their performance (Winter et al., 1989). However, the exercise protocol lasted much longer, what could explain no changes in performance. After intensive exercise, excess post-exercise oxygen consumption (EPOC) caused by an increase in circulation and ventilation heat, but mostly by a change of metabolism from carbohydrates to fatty acids, is observed (Børsheim and Bahr, 2003). High-intensity exercise will significantly increase oxygen consumption during EPOC (Thornton and Potteiger, 2002).

Many studies have reported that during exercise *Tosm* increased significantly (Fortes et al., 2011; Vera et al., 2017). Physical exertion leads to body water shortage produced by increased breathing, diuresis, and sweating (Zwierko et al., 2015). To the best of our knowledge, this is the first study examining the effects of oxygen on *Tosm*. *Tosm* did not increase as much in the study group as it did in the control group. *Tosm* can be linked to the changes in the hydration status of the body. Ungaro et al. (2015) showed a relationship between plasma osmolality and

Tosm (Ungaro et al., 2015). In participants exercising with fluid restriction, during exertion, Tosm was found to be 299 ± 9 when compared with 293 ± 9 mOsm/L in the fluid-intake group. After a rest interval, it returned to 288 ± 7 when compared with 289 ± 8 mOsm /L in the fluid-restriction group and the control group, respectively (Ungaro et al., 2015). Another study showed a high correlation between plasma osmolality and Tosm ($r = 0.93$). The basal Tosm was 293 ± 9 mOsm/L and after exercise, it increased to 305 ± 13 mOsm/L (Fortes et al., 2011). Similarly, during a kinetic exercise, there is a significant drop in the intraocular pressure, which was linked with an ion shift due to sweating, evaporation, and plasma expansion (Opara et al., 2016; Wylęgała, 2016). We advocate an increase in Tosm to be linked with a similar alteration. In another study on 19 helicopter pilots who performed a maximal incremental test on a treadmill, Tosm was found to be significantly increased from 303.72 ± 6.76 to 310.56 ± 8.80 mOsm/L. The researchers observed that changes were present only in the untrained group and correlated with the level of body fat (correlation coefficient = 0.77–0.89) (Vera et al., 2017). In our study, we observed an increase in Tosm in both groups. TearLab provides reliable and stable outcomes of monitoring patients with dry eye disease (Bunya et al., 2015). It provides a correlation of 0.96 with standard solutions and provides more accurate results than its current market competitor (Rocha et al., 2017).

As exercising leads to an increase in body temperature (Lim et al., 2008), we hypothesized

that changes in body temperature would contribute to an increase in evaporation. Therefore, we decided to perform our experiment with the participants' eyes shut. We also hypothesized that during physical exercise with increased body temperature, evaporation would also increase. In addition, participants performing a new exercise in a laboratory setting may alter their blinking rate (Bentivoglio et al., 1997).

One of the limitations of our study was that we measured only one eye (the same eye throughout the exercise). Previous studies have also conducted tests on a single eye (Kobayashi et al., 2018; Vera et al., 2017; Walsh et al., 2012), which is due to high costs of probes and time needed to obtain the results. The other limitation was that regular training affected the body's response to intensive exercise, and we chose to include only people with normal sports activity, not competitive athletes. In our study, we did not show the results of oximetry. During the exercise, the results of oxygen saturation decreased to 70% SpO₂ and were extremely variable. We believe this may be a result of an increased hand movement during the test.

Conclusion

We may conclude that short-term intensive exercise leads to an increase in Tosm. Inhaling oxygen before exertion leads to a relatively smaller increase in Tosm as well as HR. Our study indicated a relationship between the level of oxygen and the Tosm value.

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