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Lung function and oxygen saturation after participation in Norseman Xtreme Triathlon

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ABSTRACT

Objectives: To examine development of exercise-induced bronchoconstriction (EIB) defined as ≥ 10% reduction in forced expiratory volume in one second (FEV_1) and exercise-induced arterial hypoxemia (EIAH) defined as ≥ 4% reduction in oxygen saturation (SpO_2) from before to after participation in the Norseman Xtreme Triathlon. Secondarily, to assess if changes in FEV_1 and SpO_2, are related to respiratory symptoms, training volume and race time.

Methods: In this quasi-experimental non-controlled study, we included 63 triathletes (50♂/13♀) aged 40.3 (±9.0) years (mean ±SD). Fifty-seven (46♂/11♀) measured lung function and 54 (44♂/10♀) measured SpO_2 the day before the race, 8-10 minutes after finishing the race (post-test 1) and the day after the race (post-test 2). Respiratory symptoms and weekly training volume were recorded with a modified AQUA questionnaire. ANOVA for repeated measures was used to detect differences in lung function and SpO_2. Statistical significance was accepted at 0.05 level.

Results: Twenty-six participants (46%) developed EIB at post-test 1 and 16 (28%) at post-test 2. All lung function variables were significantly reduced from baseline to post-test 1 and 2. Thirty-five participants (65%) developed mild to moderate EIAH. No significant correlations were observed except a weak correlation between maximal reduction in FEV_1 and respiratory symptoms ($r=0.35$, $p=0.016$).

Conclusion: Our results demonstrated that 46% of the participants developed EIB and 65% developed EIAH after the Norseman Xtreme Triathlon. Changes in FEV_1 and SpO_2 were not correlated to weekly training hours or race time. We observed a weak correlation between maximal reduction in FEV_1 and respiratory symptoms.

INTRODUCTION

Increasing interest of participation in extreme long-distance races such as ultra-marathon, open water swim and extreme triathlon, is reported over the last decades (1). One of these is the Norseman Xtreme Triathlon, held in Norway in August. Participation has become more and more popular and in 2019 about 4300 athletes, representing 84 nations, registered for 250 slots. The race is known as one of the toughest triathlons in the world (2) and consists of a 3.8 km open water swim in Eidfjord followed by 180 km cycling, crossing two mountain plateaus (Hardangervidda and Mt. Imingfjell), and a full distance marathon (42 km) finishing at the top of Mt. Gaustatoppen, 1883 meters above sea level (m.a.s.l.). However, the current knowledge of the acute effect of extreme and long-lasting endurance exercise on physiological variables, including pulmonary responses, in recreational triathletes is scarce.

A significant reduction in lung function, measured as the forced expiratory volume in one second (FEV_1), has previously been reported after long distance endurance competitions such as triathlons, marathons, and ultra-marathons, including ultra-mountain marathons (3-5). Respiratory muscle fatigue has been suggested as one possible mechanism of reduced lung function (3, 6, 7). Blaber et al. (4) investigated changes in pulmonary function and autonomic cardiovascular control in eight ultra-marathon athletes. They observed that 30s maximal voluntary ventilation (MVV_{30s}) and MVV_{30s} tidal volume (TV) were reduced after the race as compared to baseline measurements. In addition, the ratio of FEV_1 and forced vital capacity (FVC), FEV_1/FVC, was reduced after the race. However, only baseline MVV was correlated to race performance and not the changes in MVV from pre to post-race (4). Moreover, Hue et al. (8) demonstrated that a cycle-run succession (30 minutes cycling followed by 20 minutes running) induced changes in pulmonary function in 8 national level and 6 elite level triathletes. They reported an increase in residual volume (RV) and functional residual capacity (FRC) in the elite level triathletes after the cycling session and a decrease in diffusion capacity (DLCO) in both groups. The decrease in DLCO persisted at the end of the cycle-run session in the national level group only (8).

In elite endurance athletes, bronchial hyper-responsiveness (BHR) and exercise-induced bronchoconstriction (EIB), defined as ≥ 10% reduction in FEV_1 from before to after exercise, are frequently observed, especially in cold air athletes and swimmers (9) but also in triathletes (9, 10). Airway inflammation due to the mechanical, osmotic and thermal stress from repeated high ventilation rates during training and competitions in unfavourable environments is suggested as the mechanism for development of BHR and EIB in endurance-trained athletes (11). Couto et al. (12) suggested "sports asthma" as a new distinct asthma phenotype, based on the presence of respiratory symptoms, BHR/EIB and associated with "autonomic dysautonomy" induced by systematic high-intensity endurance exercise (12). Previous studies have shown increased parasympathetic activity in endurance-trained athletes (13) as well as associations to BHR (14)

Exercise-induced arterial hypoxemia (EIAH) has been observed in high level endurance athletes (15) and this may be due to EIB and hypoventilation or exercise-induced pulmonary edema (EIPE) (16) Exercise-induced pulmonary edema is most common in water sport athletes, including triathletes, and is often reported as swimming-induced pulmonary edema (SIPE) (17, 18).

As triathlon consists of swimming, cycling and running, we hypothesized that triathletes are at significant risk to develop EIB and EIAH due to the increased ventilatory demands of the three different sports and environmental conditions during the competition. Therefore, the primary aim of the present study was to examine lung function and oxygen saturation by pulse oximetry (SpO_2) the day before the race, immediately after the race and 12-18 hours after finishing the Norseman Xtreme Triathlon. Secondarily, we aimed to assess possible relationships between changes in lung function and oxygen saturation as well as respiratory symptoms, training volume and race time.

METHODS

The Regional Ethics Committee for Medical and Health Research (REK-number 2016/932) approved the present study and it was carried out according to the principles stated in the Declaration of Helsinki. Each subject provided a signed consent before inclusion.

The present study is part of a larger study aiming to assess core temperature, changes in biomarkers from blood samples and changes in lung function from before to after the Norseman Xtreme Triathlon race. Lung function and oxygen saturation results from the races in 2016 (n=36) and 2017 (n=27) are combined. Data was collected with identical protocols before and after both races. According to the Norwegian Meteorological Institute the range in temperature in this location and time period was 3.2-11.3°C and 4.4-14.2°C in 2016 and 2017, respectively. Relative humidity ranged from 69-88% in 2016 and 72-93% in 2017, respectively. There were no observed differences between the baseline characteristics, nor the lung function and SpO_2 results between the participants in 2016 and 2017.

We collected baseline data in Eidfjord, about 7 m.a.s.l. The first 160 of the 250 participants in the Norseman cross the finish line at Mt. Gaustatoppen (1883 m.a.s.l.) and the remaining 90 participants cross the finish line at Gaustablikk Hotel (1100 m.a.s.l.) due to safety reasons. However, all participants complete an equal distance of 3.8 km open water swim, 180 km cycling, and a full distance marathon (42.2km). The air temperature and humidity during the races ranged between 3.8 - 10.8°C and 71-90%, respectively. The mean water temperature during the swim was 14.3°C. We performed all measurements indoor before and after the race. The mean room temperatures were 21°C (±2°C).

Design and subjects

Sixty-three Norseman participants (50♂/13♀) with a mean age of 40.3 years, ranging from 22-62 years, were included in the present quasi-experimental non-controlled study with pre-post testing **(Table 1)**. Data were collected at baseline (the day before the competition, in Eidfjord), 8-10 minutes after crossing the finish line at Mt. Gaustatoppen or Gaustablikk hotel (post-test 1) and the day after the race

(post-test 2). Twenty-seven participants came from Norway, 22 from 9 other European countries and 14 participants from 7 different countries outside of Europe. Participants from all continents except Africa and Antarctica are included in the present study.

Two participants aborted the race due to respiratory disorders, one of whom was hospitalized. Two participants aborted the race due to injuries after cycle accidents, and two of the included participants missed the time deadline to finish at Gaustatoppen and thus we were not able to measure them after the race. Hence, the total sample includes 57 participants (46♂/11♀) with lung function and 54 participants (44♂/10 ♀) with oxygen saturation test results.

Measurements of lung function and oxygen saturation by pulse oximetry (SpO_2) were performed at all three time points **(Table 2 and Figure 1)** and blood pressure (BP) and heart rate (HR) were measured at baseline **(Table 1)**. Prior diagnoses of asthma and allergy, use of anti-asthmatic medication the last year and the presence of respiratory symptoms (cough, phlegm, wheeze and heavy breathing) during or after exercise, as well as use of diet supplements and training volume were recorded with the modified AQUA-questionnaire (19) at baseline. Ten participants reported to use anti-asthmatic medication ($beta_2$-agonists) regularly and used their asthma treatment as prescribed at all time points.

Lung function

Lung function was measured by maximum expiratory flow-volume loops using a MasterScreen Pneumo spirometer (Jaeger GmbH, Würzburg, Germany). The best of three measurements, regarding FEV_1 and FVC, with acceptable technique was used in the analysis. The following variables were recorded: FVC, FEV_1, forced expiratory flow at 50% of vital capacity (FEF_{50}) and FEV_1/FVC ratio. All manoeuvres complied with the general acceptability criteria of the European Respiratory Society (ERS) (20). Lung function values are expressed as percentage of predicted, according to Quanjer et al. (21). A reduction in FEV_1 from baseline to after the race was defined as exercise-induced bronchoconstriction (EIB) according to the ERS (21). The spirometer was corrected for temperature, humidity and altitude before

volume calibration, according to the suppliers' manual, prior to all lung function measurements in Eidfjord, at Mt. Gaustatoppen and at Gaustablikk Hotel.

Oxygen saturation, blood pressure and heart rate

Oxygen saturation (SpO_2), blood pressure (BP), systolic blood pressure (SBP) and diastolic blood pressure (DBP) and resting heart rate (HR) were measured with a Welch Allyn Spot Vital Signs (LXi, New York, USA) recorder after sitting upright on a chair for 5 minutes. The mean of two BP measures, the mean of the two highest stable SpO_2 measures and the lowest measure of HR were used in our analyses. Exercise-induced arterial hypoxemia (EIAH) was defined as a reduction in SpO_2 of 4% or more from baseline according to Prefaut et al. (22)

STATISTICAL ANALYSIS

Demographic data are presented with mean and standard deviation (SD). Results are expressed as mean with 95% confidence intervals (CI), unless otherwise stated. Categorical data are presented as counts and proportions (%).

Power calculations are based on the changes in FEV_1. Twenty participants were estimated to be a sufficient number to detect a difference of 10% with 80% power.

The changes in FEV_1 and SpO_2 from baseline to post-race are analysed using per protocol analysis. Differences between males and females are analysed by independent T-test and differences from baseline to post-test 1 and 2 are analysed using repeated measures ANOVA. Categorical variables are compared by Chi Square or Fisher’s Exact tests and Spearman's correlation coefficient is used for analysing correlations between continuous variables. Statistical analyses are performed with Statistical Package for Social Sciences (SPSS, version 21.0; Chicago, IL, USA). A p-value less than or equal to 0.05 is considered statistically significant.

RESULTS

Participants

The characteristics of the participants are presented in **Table 1**. Sixty-three participants were recruited and performed lung function measurements at baseline, 54 participants measured BP, HR and SpO_2 at baseline and 55 participants answered the questionnaire. Six (4♂/2♀) dropped out as previously described. The 57 included participating triathletes had a mean race time of 14 hours (h) and 55 minutes (min), ranging from 10 h and 5 min to 18 h and 15 min.

[Insert Table 1]

Twenty-six participants (46%) developed EIB immediately after the race and 16 participants (28%) still had bronchial constriction (≥ 10% reduction in FEV_1 from baseline) the day after the race (**Table 2**).

[Insert Table 2]

In total, 35 participants (65%) developed mild to moderate EIAH defined as ≥ 4% reduction in SpO_2, of which 32 participants from baseline to post-test 1 and 3 participants from baseline to post-test 2, respectively. Mean oxygen saturation was significantly reduced from baseline, 99.0% (98.7, 99.3) [95%CI] to post-test 1, 94.4% (93.7, 95.1 by 4.6%) and to post-test 2, 96.6 (96.2, 97.0) by 2.4%, respectively (**Figure 1**).

[Insert Figure 1]

[Insert Table 3]

Self-reported doctor diagnosed asthma and allergy, respiratory symptoms, nutritional supplements and training volume are presented in **Table 3**. Ten participants (18%) reported doctor diagnosed allergy and all used antihistamines regularly or as needed. In addition, 10 participants (18%) reported to use $beta_2$-agonists regularly or as needed. However, only 4 of them reported a previous doctor diagnosed asthma and used both inhaled steroids and $beta_2$-

agonists. Thirty-six participants (66%) reported respiratory symptoms during or after training/competition and the most frequently reported symptoms were cough (29%) followed by phlegm (13%) and wheeze (13%), respectively. All participants used one or more dietary supplements. Multivitamins were most frequently reported (31%) followed by Omega 3 (26%) and magnesium (26%). Male and female participants reported similar training volume per week, 13.2 and 13.6 hours per week, respectively (**Table 3**).

A weak, but significant correlation was found between maximal reduction in FEV_1 and respiratory symptoms ($r = 0.35$, $p=0.016$). We did not observe any significant correlation between maximal reduction in FEV_1 and maximal reduction in SpO_2 nor between maximal reduction in FEV_1 and training hours per week, race time or use of dietary supplements.

DISCUSSION

The main results from the present study were that nearly half of the Norseman participants developed EIB immediately after the race and almost two thirds of the athletes developed EIAH. Moreover, 28% had still reduced lung function (EIB) the day after the race. All lung function variables and SpO_2 were significantly reduced from baseline to post-test 1 and 2 **(Table 2 and Figure 1)**. A weak, but significant, correlation was observed between maximal reduction in FEV_1 post-race and self-reported respiratory symptoms at baseline.

Exercise-induced bronchoconstriction and lung function

The pulmonary response after a long-distance triathlon such as the Norseman is, to our knowledge, scarcely described in the literature. However, the high prevalence of EIB in our group of recreational triathletes is in line with previous studies (10, 23). Knöpfli et al. (10) investigated seven elite triathletes in the Swiss national team and found that three out of seven (43%) developed EIB after a sport-specific 8-minute running test on a 400 meters athletics track. Langdeau et al. (23) reported a prevalence of BHR (measured as a positive methacholine provocation test <16mg/ml) of 32% among well-trained triathletes. In addition, a high prevalence in the use of $beta_2$-agonists among triathletes (24.9%) are reported from the summer Olympics in 2004 and 2008 (9). The low sample size in the study of Knöpfli et al. (10), different methodology of diagnosing EIB and that the participants were elite or well-trained triathletes in the above mentioned studies makes it challenging to compare with our group of recreational triathletes completing an extreme triathlon competition.

In line with studies from Hill et al. (3) and Blaber et al. (4), we found significant reductions in all lung function variables after the race **(Table 2)**. Blaber et al. (4) observed that FEV_1/FVC,

MVV_{30s} and MVV_{30s} TV were reduced from baseline to after an ultra-marathon in eight runners and Hill et al. (3) reported declines in FVC (7,1%), FEV_1 (8,4%) and FEF_{50} (18,6%), but not in MVV in 12 participants after a long distance triathlon. Contrary to our results (Table 2), Hill et al (3) found that only FEV_1 was significantly reduced the morning after the race.

Mechanisms

The mechanisms leading to reduced lung function after long distance endurance races are not known. Only four participants (7.3%) in the present study reported doctor diagnosed asthma and 10 participants (18.2%) used anti-asthmatic medication regularly **(Table 3)**. However, none of the asthmatic triathletes developed EIB after the race, which may be due to the use of medication. Participants reporting respiratory symptoms during or after exercise **(Table 3)** had significantly greater mean reduction in FEV_1 after the race (15.7%) as compared to participants who did not (8.4%). Although previous studies have shown that respiratory symptoms are not associated with BHR or EIB (12, 24) we cannot exclude asthma as a contributing factor to EIB in the present study. We could have measured fractional exhaled nitric oxide (FE_{NO}), an eosinophilic inflammatory marker from the airways, and thus have a measure of eosinophilic inflammation and /or atopic asthma. However, according to Couto et al. (12), FE_{NO} is not associated with "sports asthma" and we could then probably lost potential asthmatic athletes. As previously mentioned, Hue et al. (8) observed an increase in RV and FRC and a decrease in DLCO after a cycle-run succession in 14 high-level triathletes. As we did not measure MVV, RV, FRC or DLCO in the present study, we cannot confirm or exclude similar findings among our participants. Further, increased parasympathetic activity (13, 25), respiratory muscle fatigue (3, 5) and EIPE/SIPE (17, 18, 26) have previously been proposed as possible mechanisms to reduced lung function in connection with exercise.

Parasympathetic activity measured as heart rate variability (HRV) and pupillometry, is shown

to be increased (13, 27) and correlate with VO_{2max} in endurance trained athletes (28). In addition, Pichon et al. (29) reported increased parasympathetic activity in subjects with BHR to methacholine. This is in line with Stang et al. (14) who observed a strong association between BHR and parasympathetic activity in high-level swimmers, but not in cross country skiers. However, in the present study, we did not measure parasympathetic activity, but speculate that increased parasympathetic activity is involved in the mechanisms of EIB in our participants.

It has been suggested in the literature that respiratory muscle fatigue may cause reduced lung function after long duration endurance competitions, such as triathlon (3, 5, 7). However, results regarding the change in respiratory muscle strength, measured as maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP), after such competitions are conflicting. Warren et al. (30) did not find significant reduction in spirometry variables after a 24-hour run, but Mahler & Loke (31) did after an ultra-marathon. However, neither group observed changes in MIP and MEP (30, 31). Others have reported significantly reduced MIP (3, 6, 32) and MEP (32, 33) after triathlons and marathons, respectively. Hence, it seems likely that increased ventilation over time causes respiratory muscle fatigue and reduced lung function (7). However, of practical reason we did not measure MIP or MEP in the present study, but we agree with Wüthrich et al. (32) who urges more investigation of the association between reduced lung function and respiratory muscle strength during and after long duration endurance races.

Oxygen saturation

A significant reduction ($p<0.05$) in SpO_2 from baseline to post-test 1 (4.6%) and 2 (2.4%), was seen in the present study. Thirty-five of 54 participants (29♂/6♀) (64.8%) developed mild EIAH immediately after the race, defined as > 4% reduction in SpO_2 from baseline

(Figure 1). Reduction in SpO_2 has previously been reported among triathletes after competitions and shorter intense exercise sessions (34-36). Guenette et al. (37) reported that 61.9% of well-trained cyclists and triathletes had > 4% reduction in SpO_2 after a maximal endurance bike test using a finger sensor. Laursen et al. (38) observed a surprisingly high prevalence of EIAH among triathletes (84.6%) after a treadmill endurance test as well as on a cycle ergometer, using an ear sensor. They defined EIAH as SaO_2 < 93% and resting values were not accounted for. A meta-analysis by Jensen et al. (39) showed that finger-sensors were more accurate than ear-sensors for measuring SpO_2 and this may explain the difference in prevalence of SpO_2 between our results and Laursen et al.(38) . Different methodology and definitions have been used to identify EIAH among athletes (22, 40), which makes it challenging to compare our results with others. It is debated whether reduced PaO_2 of 8-10 mmHg during intensive exercise can be defined as EIAH (22). Nielsen (40) proposed that EIAH should be defined as SaO_2<95%, but he does not take individual resting values into account. However, other authors recommend defining EIAH as >4% reduction from baseline (22, 41).

In the present study, 49 out of 57 triathletes passed the finish line at 1883 m.a.s.l. and reduced oxygen pressure (PiO_2) probably influenced the desaturation (22, 40, 42). In 1982, Squires & Buskirk (43) showed significantly reduction in SaO_2 after maximal treadmill exercise in runners at increasing altitudes. This is in line with Siegler et al. (44) who reported similar results among cyclists. Gaston et al. (42) reported that athletes with EIAH at sea level had a greater aerobic impairment than non-EIAH athletes after maximal exercise at 2150 m.a.s.l., probably due to hypoventilation. In contrast, Vernillo et al. (45) did not find any significant reduction in SpO_2 after a mountain marathon with a peak altitude of 3329 m.a.s.l. However, they measured SpO_2 at 1224 m.a.s.l. both at baseline and post marathon. Hence, it seems that there is individual adaptation to exercise at

altitude. Most studies include EIAH after maximal short-term exercise (41) and different test protocols makes it challenging to compare. Unfortunately, there is limited research of EIAH after prolonged endurance competitions at different altitudes.

PERSPECTIVES

In the present sport specific study, we demonstrate that 46% of the recreational triathletes developed EIB and 65% developed EIAH after the Norseman Xtreme Triathlon. The day after the race, 28% still had obstructive features with a reduction in FEV_1 ≥10%. This is in line with previous studies in triathletes (3, 8, 10, 16, 37) as well as in marathon and ultra-marathon runners (4, 7). However, the sample size is small in most studies and the included athletes are often at elite level (3, 4, 7, 8, 10). Research performed in recreational triathletes participating in a real competition is scarce and thus a strength of the present study. Compared to marathon and ultra-marathon, long distance triathlon consists of three different exercises and further investigations are needed to confirm our results in recreational triathletes. We did not observe correlations between changes in FEV_1 and SpO_2, nor between changes in FEV_1 and training hours per week or race time, only a weak correlation between maximal reduction in FEV_1 and self-reported respiratory symptoms. The mechanisms of reduced lung function and oxygen saturation in long distance triathletes should thus be emphasized in further research. The practical impact of the present study is that both athletes, crew and medical staff, should be aware of pulmonary challenges, both the obstructive pattern observed in the present study as well as the common symptoms of severe lung disease such as exercise/swimming-induced pulmonary edema (EIPE/SIPE). Prior to the event, the race organizers should recommend triathletes with respiratory symptoms to be thoroughly examined for EIB by a doctor to ensure effective treatment. Asthmatic athletes should be encouraged to use their anti-asthmatic medication as usual before the race.

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DISCLOSURES

The authors have no conflicts of interest or financial ties to disclose.

DECLARATION

The authors declare that the results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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Table 1. Subject demographics, heart rate (HR) and blood pressure (BP) at baseline. Results are given as mean with standard deviation (SD) (n=63 and 54[a]).

	Males n=50 (44)[a]	**Females n=13 (10)[a]**	**All n=63 (54)[a]**	**p**
Age *(years)*	41.2 (9,2)	36.9 (7.5)	40.3 (9.0)	0.125
Height *(cm)*	179.1(6.2)	166.7 (4.3) *	176.5 (7.7)	<0.001
Weight *(kg)*	76.7 (7.8)	61.4 (6.2) *	73.6 (9.7)	<0.001
BMI *(w/h^2)*	23.9 (2.0)	22.1 (1.9) *	23.6 (2.1)	<0.005
[a]HR *($beats \cdot min^{-1}$)*	57.9 (8.0)	60.2 (6.7)	58.3 (7.8)	0.415
[a]BP_{sys} *(mm Hg)*	131.7 (14.3)	119.1 (9.5)*	129.4 (14.3)	<0.05
[a]BP_{dia} *(mm Hg)*	78.4 (9.0)	75.5 (4.8)	77.9 (8.5)	0.330

*= significant different ($p \leq 0.05$) from males; [a] n= 54 (♂=44, ♀=10); BMI= body mass index; HR= heart rate; BP_{sys}= systolic blood pressure; BP_{dia} = diastolic blood pressure

Table 2. Lung function (forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), forced expiratory flow at 50% of vital capacity (FEF_{50}) and FEV_1/FVC measured at baseline, 5-10 minutes after the race (post-test 1) and the day after the race (post-test 2). All

variables are expressed in absolute values and percent predicted (% pred.) except for FEV_1/FVC. Values are given as mean with 95% confidence intervals (CI) (n=57).

	Baseline ***n*=57(45♂/12♀)**	**Post-test 1** ***n*=57(45♂/12♀)**	**Post-test 2** ***n*=57(45♂/12♀)**
FEV_1*(L)*	4.24 (3.99, 4.48)	3.76 (3.48, 4.04)**	3.90 (3.64, 4.17)$^{\#\#}$
FEV_1 *(%pred)*	110,9 (106.7, 115.1)	98.7 (92.3, 105.1)**	102.3 (96.7, 107.9)$^{\#\#}$
FVC *(L)*	5.56 (5.26, 5.86)	5.07 (4.77, 5.36)**	5.24 (4.92, 5.56)$^{\#\#}$
FVC *(%pred)*	120.4 (116.7, 124.2)	110.0 (105.1, 115.1)**	113.4 (108.8, 118.1)$^{\#\#}$
FEF_{50} *(L·sec)*	4.32 (3.93, 4.72)	3.75 (3.26, 4.24)**	3.82 (3.43, 4.23)$^{\#\#}$
FEF_{50} *(%pred)*	86.7 (79.4, 94.1)	75.4 (65.7, 85.0)**	76.8 (68.9, 84.8)$^{\#\#}$
FEV_1/FVC *(%)*	76.16 (74.51, 77.82)	74.03 (71.03,77.02)*	74.49 (72.26, 76.72)$^{\#}$

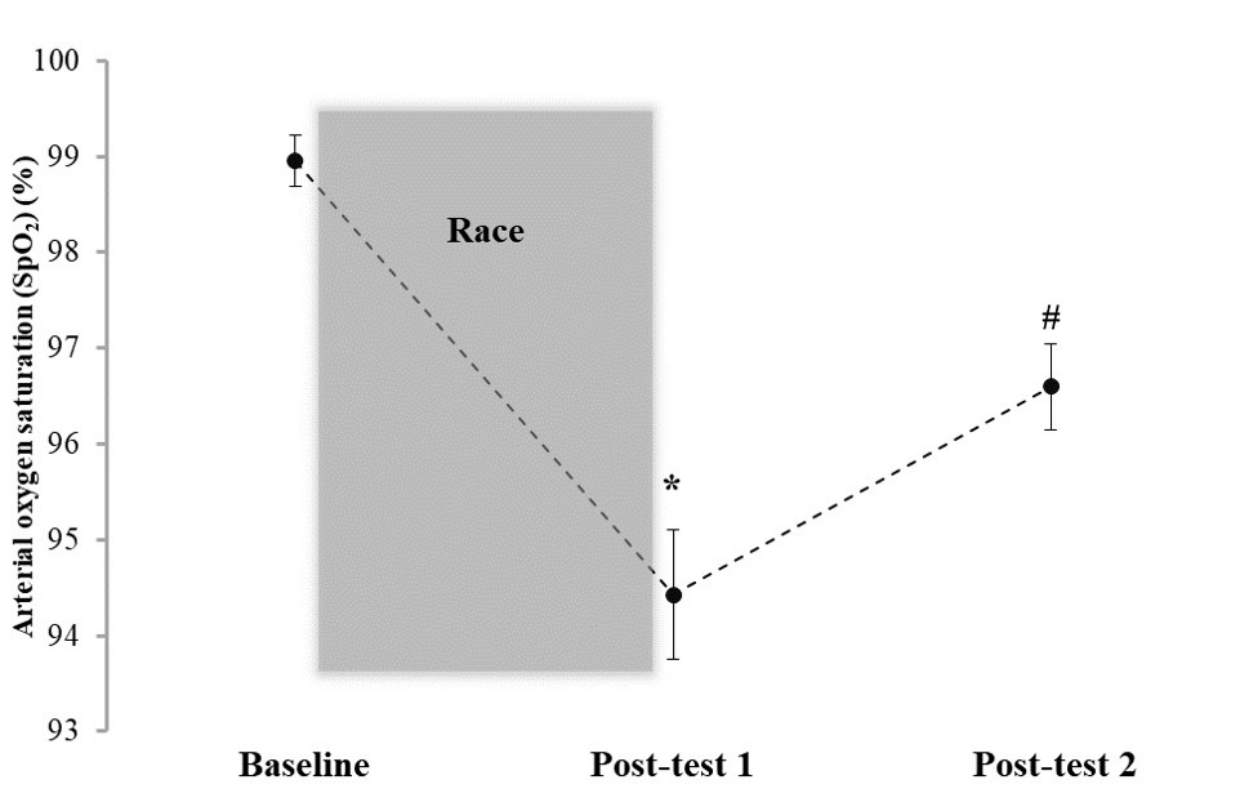
* = significantly different from baseline to post-test 1 ($p \leq 0.05$), **= $p \leq 0.001$, # = significantly different from baseline to post-test 2 ($p \leq 0.05$), ## = $p \leq 0.001$, ♀= female, ♂= male

Table 3. Self-reported doctor diagnosed allergy and asthma, most frequent respiratory symptom during or after exercise, use of diet supplements and training hours/week, (*n*=55). Results are given as numbers with percentage (%) for categorical data and mean ± standard deviation (SD) for continuous data.

	Males *n*=43	**Females *n*=12**	**All *n*=55**
Doctor diagnosed allergy	7 (16.3%)	3 (25.0%)	10 (18.2%)
Doctor diagnosed asthma	3 (7.0%)	1 (8.3%)	4 (7.3%)
Anti-asthmatic medication	9 (20.9%)	1 (8.3%)	10 (18.2%)
Most frequent respiratory symptom during exercise:			
-Cough	13 (30.2%)	3 (25.0%)	16 (29.1%)
-Phlegm	7 (16.3%)	0 (0%)	7 (12.7%)
-Dyspnea	4 (9.3%)	2 (16.7%)	6 (10.9%)
-Wheeze	6 (14.0%)	1 (8.3%)	7 (12.7%)
Use of diet supplements:			
-Magnesium	11 (25.6%)	3 (25.0%)	14 (25.5%)
-Iron	4 (9.3%)	0 (0%)	4 (7.3%)
-Omega 3	12 (27.9%)	2 (16.7%)	14 (25.5%)
-Omega 6	0 (0%)	1 (8.3%)	1 (1.8%)
-Multivitamins	11 (25.6%)	6 (50.0%)	17 (30.9%)
-Protein	7 (16.3%)	1 (8.3%)	8 (14.5%)
-Others	5 (11.6%)	1 (8.3%)	6 (10.9%)
Training hours per week	13.2 (±4.2)	13.6 (±7.6)	13.3 (±5)

Caption figure 1

Figure 1. Arterial oxygen saturation measured at baseline, 5-10 minutes after the race (post-test 1) and the day after the race (post-test 2). Results are given as mean with 95% confidence intervals (CI), *= significant changes from baseline to post-test 1, #= significant changes from baseline to post-test 2 ($p \leq 0.05$), (n=54).


100
99
98
97
96
95
94
93
Arterial oxygen saturation (SpO_2) (%)
Race
*
#
Baseline
Post-test 1
Post-test 2