Epidemiology of Exercise-Induced Hypoxemia in Elite and Non Elite Athletes

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***Abstract*—**Exercise-induced arterial hypoxemia (EIAH) is a recognized phenomenon in healthy subjects, although its epidemiology and mechanisms remain incompletely understood. In this study, we assessed the prevalence rates of EIAH among various populations. Eighty-two subjects underwent incremental exercise protocols to maximum volitional fatigue while being monitored using forehead pulse oximetry. Pre- and post-exercise spirometric testing as well as serum lactate measurements were utilized. Results were stratified by severity of oxygen desaturation, gender, age, training, fitness level, and mode of exercise, and statistically compared to determine EIAH prevalence and characteristics of the desaturating populations. The prevalence of EIAH in our study population was 35.4% (37.1% for males and 30% for females), which was significantly lower than that reported previously. We additionally observed a higher prevalence of EIAH in moderately fit individuals than previously documented (32.7%) and a low prevalence in the least trained subjects (36.8% vs. 12.5%). Younger subjects displayed a trend towards greater development of EIAH, contrary to past observations. Spirometric analysis revealed no statistically significant differences in lung volumes and flow rates between individuals with normal saturation and those who displayed desaturation. Serum lactate measurements did not differ significantly between normal and desaturating populations. Our documented prevalence of EIAH is significantly lower, but more common in less fit and younger individuals, compared to previously published findings.

***Keywords*—**Exercise-induced arterial hypoxemia, EIAH, desaturation in athletes, exercise limitation.

I. Introduction

The phenomenon of exercise-induced arterial hypoxemia (EIAH) has been conclusively demonstrated in a variety of populations, in contrast to traditional physiology teachings [1]. EIAH is defined as a reduction in the blood oxygen level measured as hemoglobin oxygen saturation (SaO_2) or partial pressure of arterial oxygen (PaO_2), and can be classified as mild (93-95% SaO_2), moderate (88-93% SaO2) or severe (SaO2 <88%) (14). The concept of exercise-induced arterial hypoxemia (EIAH) has been developed and refined significantly in recent years. Contrary to longstanding beliefs, research has shown that the respiratory system is not always structurally and homeostatically capable of providing consistent hemoglobin oxygenation (SaO_2) during intense exercise in healthy individuals. Prior to these observations, conventional theories suggested that the cardiovascular system is the primary contributor to the body's inability to meet metabolic demands placed upon it during strenuous exercise. Failure to adequately supply mitochondria with O_2 would trigger anaerobic metabolism and muscle fatigue, leading to negative effects on exercise performance. However, the cardiovascular and metabolic systems are highly adaptable to the demands of high exercise performance whereas the respiratory system has shown little capacity to enhance in response to training or exercise.

Limitations of the respiratory system were initially demonstrated in some populations of very highly trained endurance athletes. During intense and even moderate levels of exercise, these elite athletes were unable to maintain normal arterial partial pressure of oxygen (PaO_2) [2], [3]. Further investigations are in progress to establish population prevalence across all levels of fitness, age, and gender. The prevalence of this phenomenon is still poorly understood in more sedentary and moderately active populations, although it is thought to be less common than in highly active populations. Interestingly, there is established evidence of EIAH occurrence in a variety of animals, particularly, thoroughbred race horses [1]. Recent demonstrations of the negative impact of even relatively small amounts of EIAH on maximal oxygen uptake ($VO_{2\ max}$) and exercise performance have compounded the significance of clarifying its effects on performance and the underlying causative factors [4], [5].

II. Causes of EIAH

Several potential mechanisms have been linked to EIAH, but no one model has been conclusively established as the major contributor. To date, the true implications of this phenomenon on exercise performance have never been unequivocally described, but there is evidence both for and against a negative impact [4]–[6]. Present theories generally attempt to explain the increased A-aDO_2 gradient commonly associated with EIAH. Some factors that prevent adequate airflow to the

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alveoli include lack of an appropriate ventilatory response, air flow limitation (potentially due to the presence of airway hypersensitivity), as well as respiratory muscle fatigue [7] –[18]. Other contributors to the decrease in A- aDO_2 include intrapulmonary or intracardiac shunting and transient pulmonary edema [7], [8]. However, there is little conclusive evidence to facilitate confirmation of the mechanism or combination of mechanisms causing EIAH at present. It is likely that several processes act in concert to induce EIAH. Further studies are required to identify these factors and establish the relative contribution of each mechanism.

III. Epidemiology of EIAH

Since limited studies have provided direct measurements of arterial blood gases in sufficiently large sample sizes to date, the true prevalence of EIAH has been difficult to determine [1]. Earlier research has shown evidence of EIAH in ~50% of young male athletes [2, 3]. These studies focused on elite athletes capable of extreme workloads with $VO_{2\ max}$ >60 ml/kg/min. Research on female athletes revealed a prevalence of 67% [12], [17]. Trained male athletes experienced EIAH at $VO_{2\ max} \geq 150\%$ of their age, size, and gender-predicted values; whereas female athletes experienced EIAH at significantly lower VO_{2max}, within 15-20% of their predicted values [17]. Hopkins et al. (2000) found that only 24% of fit female subjects desaturated when tested randomly with respect to their menstrual cycles [19]–[21]. A unique group of older, trained "master athletes" showed more significant EIAH than other populations [11]. In this study, all 10 subjects desaturated significantly at lower exercise intensities, with a more severe drop in PaO_2, which appeared with regimens involving lower levels of training.

Historically, EIAH has been investigated in relatively small populations of young, highly trained, male endurance athletes [2]. Its prevalence in these populations is documented as ~50% [2], [3]. However, these populations are not representative of society as a whole. While EIAH has additionally been examined in older individuals and females, there is still a dearth of evidence on its occurrence in these population groups [11], [12], [17]. This paucity is also observed with regard to investigations on EIAH in individuals with only moderate exercise history, i.e., average non-sedentary subjects. However, it is commonly accepted that EIAH occurs in some capacity in all the above populations. Difficulties in comparing these studies and extrapolating the data to society in general limit our knowledge of the true prevalence of EIAH. Sample populations in the majority of these studies rarely exceed 30 individuals, and most commonly involve less than 15. In addition, exercise protocols vary significantly between institutions, and standardizing the training levels and abilities of individuals between studies presents a considerable problem.

IV. Research objectives

The main aim of our study was to examine the prevalence and characteristics of EIAH in a large population of adult male and female individuals of varying levels of fitness and exercise training history. Our specific objectives were as follows:

1. To study EIAH in a population of high-level endurance athletes as well as more moderately active individuals with a view to establishing its true prevalence.
2. To determine whether a specific exercise protocol, such as either cycling or running, is more likely to produce EIAH.
3. To establish the significance of general population variables associated with EIAH, including gender, age, fitness level, and mode of exercise.
4. To evaluate the correlation between lactate levels and EIAH.

V. Materials and methods

A. Experimental Protocol

The study was approved by the Biomedical Ethics Board, University of Manitoba. Written informed consent was obtained from all participants. Eighty- two individuals were recruited prospectively from the local recreational and amateur athletic community. Participants were 19 to 74 years of age, and screened for activity levels as well as cardiac and pulmonary health using a standardized interview. Individuals were categorized as either highly (HT) or moderately trained (MT) based upon the results of this interview. Highly trained individuals included those who participated in a year-round exercise regimen, had been exercising for at least 3 years, and had participated in competitive events in the past 6 months. The moderately trained group included individuals who exercised between 3–7 times per week but were not competitive. Other factors that were considered included the types of exercise (commonly including either cycling or running, but also cross country skiing and swimming), the frequency of exercise per week (HT mean =8.0; MT mean = 5.6) and the number of hours per week (HT mean = 10.6; MT mean = 6.5), competitions per year (HT mean = 11.1; MT mean = 2.6) the intensity of the exercise, and years of consistent exercise (HT mean = 13.9; MT mean = 13.3).

The study utilized both cycling and running exercise protocols. Pre and post-exercise measurements were identical for both protocols. Prior to testing, blood pressure measurements were completed either manually or electronically (GE Dinamap Pro 100). Measurements of SaO_2 and heart rate were obtained using a Nellcor N-595 pulse oximeter and OxiMax Max-Fast adhesive forehead reflectance sensor, respectively. The capillary blood lactate concentration was measured using a simplified test meter (Arkray LT1710). $VO_{2\ max}$ was estimated using a heart rate monitor (Polar T31 N2965). Pre- and post-exercise arterial blood gas (ABG) samples were drawn from consenting subjects tested after June 2009. Subjects completed spirometry, prior to initiating exercise. A second measurement of the blood lactate concentration was obtained immediately following exercise via capillary sampling. Measurements of SaO_2, heart rate and blood pressure were taken at 5 and 15 minutes post- exercise.

Spirometry was performed at baseline, 5 and 15 minutes post-exercise. To assess the development of expiratory flow limitation and exercise-induced airway hyperresponsiveness,

spirometric parameters, including percent predicted forced expiratory flow in 1 second (FEV_1 PP), percent predicted forced vital capacity (FVC PP), ratio of FEV_1/FVC, and percent predicted forced expiratory flow rates at 25% to 75% of FVC (FEF_{25-75} PP), were recorded. A 10% decrease in FEV_1 post-exercise signified exercise- induced bronchoconstriction.

B. Cycling Protocol

A friction-braked cycle ergometer was utilized for the cycling protocol (SensorMedics Ergoline 800). Subjects were permitted to use their own saddle and/or pedals if they wished. Heart rate and SaO_2 were continuously monitored with a forehead oximeter. The warm-up consisted of three stages. The first stage comprised 10 minutes of low-power (60-120 watts) exercise, the second was 1 minute at high power (180-400 watts), and the third involved 4 minutes of low-power (60-120 watts) exercise. The subject's wattage during the second stage was determined based on exercise history and perceived and stated efforts utilized to complete the warm-up. The period of high-power exercise during warm-up was used to estimate the subjects' potential during the test protocol and potentiate their output. Completion of the warm-up was immediately followed by transition to the test protocol. In the cycling test protocol, the power output was amplified every minute from low baseline by increments of 20, 25, or 30 watts until exhaustion. Exhaustion was defined by the inability of the subject to maintain a minimum cadence of 90RPM. Individual baselines and increases were determined to optimize the test protocol between 10 and 15 minutes of exercise

C. Running Protocol

During the running protocol, SaO_2 and heart rate were continuously monitored using forehead oximetry. Running was performed on a slow buff treadmill. The warm-up period was conducted at a pace of between 4 and 6 mph with a 0% gradient. Following initiation of the test protocol, subjects ran at a constant pace of between 5 and 8 mph, based on their individual comfort level. Some individuals increased their pace during the later stages of exercise to induce fatigue more rapidly. The gradient of the treadmill was increased from a baseline of 0% or 2% by 2% every 3 minutes until volitional fatigue.

D. Statistical Analysis

Pre- and post-exercise measurements were compared using the two tailed student's T-test. Populations with normal saturation and varying degrees of exercise- induced desaturation were compared. Other populations, stratified by age, gender, training, fitness and mode of exercise, were additionally examined. The differences between the various populations were analyzed using ANOVA. A probability value of <0.05 was considered statistically significant.

VI. Results

Demographic characteristics of all study participants and stratified populations are presented in Table 1. Exercise performance data are recorded in Table 2. The prevalence of EIAH in our study population was 35.4%. Subjects who desaturated moderately (93%-94% SaO_2) and severely ($\leq$92% SaO_2) comprised 23.2% and 12.2 % of the total population, respectively. Individuals displaying EIAH achieved significantly greater maximum power output as well as greater output of power/kg, compared to those who did not desaturate ($p<0.05$). We observed no statistically significant variations in pre or post-exercise capillary lactate between subjects with and without EIAH. Individuals who desaturated severely achieved greater maximum wattage compared with those who desaturated mildly (391.7±126.7 vs. 348.3±68.7, $p<0.05$) and VO_{2max} (53.5±13.73 vs. 49.9±9.0, $p<0.05$). Post-exercise capillary lactate was not significantly different between the two groups.

Individuals classified as highly trained had a prevalence of EIAH equaling 40.7%, whereas the incidence of EIAH in the moderate training group was 34.6%. As expected, highly trained individuals exhibited significantly greater maximum power (power/kg) and estimated $VO_{2\,max}$. Again, we observed no significant variations in post-exercise capillary lactate (indicating severity of anaerobic metabolism) between the two groups.

We recorded an EIAH prevalence of 37.1% and 30% in males and females, respectively. Women appeared to desaturate more severely, although this finding was not statistically significant ($p = 0.19$). As expected, performance outcomes were significantly greater in the male population. In general, women displayed significantly lower average post-exercise lactate, compared to their male counterparts ($p= 0.001$).

Individuals were additionally stratified by age. Specifically, EIAH occurred in 40.0% of participants aged 19-29 years, 37.5% of those aged 30-39 years, 25% of those aged 40-49 years, and 33.3% of subjects aged 50+ years. The variations between age stratifications may be attributed to sample size, with the average number (n) = 20.5 per age group.

Fitness was categorized based on estimated $VO_{2\,max}$. The ranges utilized in this analysis included: Elite (>60 ml/kg), High (50-59 ml/kg), Moderate (40-49 ml/kg), and Low (30-39 ml/kg). We observed a marked increase in EIAH prevalence among subjects classified as elite (60.0%), compared with those in the high, moderate, and low groups (36.8%, 39.1%, and 12.5%, respectively); $p<0.05$ between elite and moderate $VO_{2\,max}$ versus low $VO_{2\,max}$. Our results clearly indicate a marked decrease in the prevalence of EIAH in low fitness individuals, in relation to the whole study population.

Exercise modality did not seem to affect whether, and to what degree, subjects exhibited EIAH. EIAH prevalence amongst cycling subjects was 34.4%, compared to 38.1% of runners ($p>0.05$). The average lowest O_2 saturation values were not significantly different between the exercise types. Significantly higher average lactate measurements were obtained with the cycling population ($p < 0.001$).

Table 3 contrasts the post exercise changes in heart rate and O_2 saturation recovery between normal and desaturating populations. Interestingly, the population that displayed EIAH showed a statistically significant decreased baseline O_2 when compared to normal ($p=0.015$). At every stage post exercise (0,

5, and 15 minutes) the desaturating population showed a significantly lower O2 saturation (All $p < .001$). A trend of increased post exercise HR was noted in the desaturating population, however this trend was only significant at the 5 minutes post exercise interval.

No statistically significant differences were noted between our EIAH and normal populations in respect to pre or post arterial blood gas values (including pH, pCO_2, pO_2, $cHCO_3$-).

VII. Discussion

The current investigation includes the largest population analyzed to date, with an estimated EIAH prevalence of ~35%, distinct from 50% documented earlier for high endurance male athletes and addresses some of the concerns about the generalizability of the data obtained from selected groups to the general population [1]–[3]. The subjects recruited for our study included both recreational and amateur athletes, among which only some were highly competitive, possibly similar to other studies. Therefore, the relevance of these findings highlights the importance of EIAH in compromising performance in individuals other than elite athletes. Our prevalence rates among males and females were distinct from those obtained with other studies where a higher proportion of women appeared to develop EIAH [3], [5], [15], [17]. It is important to note that these previous studies specifically targeted the follicular phase of the menstrual cycle for exercise testing. Indeed, Hopkins *et al.* (2000) noted that only 24% of fit female subjects desaturated when tested randomly with respect to their menstrual cycles, similar to our findings [21].

Upon stratification by age, younger age groups displayed greater prevalence of EIAH, which may either reflect a lower number of subjects in each group or lower level of maximal power output and $VO_{2\ max}$ achieved by older individuals. Data obtained with our 50+ age stratification supported previous findings that older athletes desaturate more severely than younger individuals, though our findings were not statistically significant [11].

Training levels influenced the incidence of EIAH in our population, with a significant increase in prevalence amongst the most highly trained individuals, consistent with earlier findings [1], [2], [22], [23]. Another study showed a greater prevalence of EIAH in females than males among moderately trained individuals, which was not evident in our study [15]. We additionally observed a greater prevalence of EIAH in moderately fit individuals than that previously reported (32.7%).

In our study, EIAH prevalence rates were high in elite athletes, whereas highly and moderately fit individuals (stratified based on estimated $VO_{2\ max}$) exhibited prevalence closer to the study population average. Subjects classified in the low fitness group displayed significantly lower prevalence rates. Our findings support previous literature showing the EIAH is highly prevalent in elite athletes [2], [3], [23]. There has been little evidence of EIAH in young male volunteers with $VO_{2\ max}$ values below 50 ml/kg/min, a finding that was not as evident, but still apparent in our study [23], [24].

Comparison of the cycling versus running protocols revealed similar rates of EIAH development. We conclude both protocols can be effectively utilized for the assessment of EIAH, in contrast to the conclusions of an earlier study suggesting that treadmill-based protocols reproduce EIAH more consistently [1].

There have been no efforts to correlate capillary lactate concentration and severity of anaerobic metabolism with EIAH development. Although we did not observe a correlation between the lactate levels of the non-desaturating and desaturating populations, significantly lower average post- exercise lactate concentrations were observed in females, compared to their male counterparts ($p=0.001$). The significance of this finding is not clear, and requires further investigation.

The lack of difference noted in ABG values may be attributed to small population size of tested individuals ($n = 15$) or to a lack of detectable difference in respiratory physiology in both populations. The similar pH and bicarbonate values may be attributed to the lack of an increased severity of anaerobic metabolism between desaturating and normal populations. This observation would also likely be associated with a lack of significant change in post exercise serum lactate levels between populations, a finding we also noted.

The post-exercise recovery data shows that those individuals who exhibit EIAH did not reach baseline O_2 saturations at 15 minutes post exercise, despite recovering to a saturation that would not be considered to be EIAH. This may suggest that the mechanism causing EIAH persists at least a short while post exercise. Additionally, the significant increase in heart rate at 5 minutes post exercise may be attributable to the greater degree of baseline fitness exhibited by the desaturating population or may be the result of other factors, such as increased post exercise cool down intensity among desaturating individuals.

VIII. Conclusion

Several issues regarding EIAH and the underlying mechanisms are poorly understood at present. In this study, we have attempted to gain a broader perspective of the prevalence of EIAH in a variety of population groups [25-28]. Our results disclosed a lower prevalence (~35%) than that previously reported, with a larger sample size comprising a more varied group of individuals in terms of training, fitness, age, and gender. Experiments on the population subgroups led to conclusions that both support and contradict the existing literature. Clearly, there is still a lack of consensus on the prevalence of EIAH in a variety of age groups, training levels, and even genders. Population prevalence data are frequently applied to support the proposed mechanisms of EIAH. Thus, considerable efforts should be made to determine the prevalence of EIAH in various groups, so that investigators can accurately use the specific physiologic properties of a given group to clarify the underlying mechanisms.

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Table 1: Average Total and Stratified Population Demographics

	n	%	Age Yr	Height cm	Weight kg	Exercise/week hr	Consistent Exercise yr
Total Population	82	100	36.4±13.0	175.3±8.9	74.9±8.9	7.9±3.8	13.5±10.9
Normal Saturators	53	64.6	37.2±13.1	175.0±9.0	74.3±11.9	7.3±3.5	14.5±11.5
Total Desaturators	29	35.4	35.0±12.7	175.8±8.8	75.9±10.9	8.9±4.2	11.6±8.7
Mild Desaturators	19	23.2	34.7±12.4	176.1±8.4	77.1±8.0	8.3±2.7	10.1±8.9
Severe Desaturators	10	12.2	35.5±14.0	175.2±10.0	73.6±15.3	9.9±6.1	14.5±11.0
Sex							
Male	62		35.4±13.5	178.8±6.7	78.8±9.4	7.9±3.5	13.1±11.1
Normal	39	62.9	36.6±13.9	179.2±5.9	78.3±9.8	7.2±3.0	14.9±11.9
Desaturators	23	37.1	33.4±12.7	178.1±7.9	79.6±8.7	9.0±3.9	10.1±9.2
Female	20		39.7±10.9	164.4±5.0	62.7±8.6	7.8±4.8	14.7±10.5
Normal	14	70	39.1±10.9	163.2±4.0	63.1±10.0	7.6±4.8	13.5±10.8
Desaturators	6	30	41.2±11.8	167.1±6.4	61.6±4.3	8.3±5.3	17.5±10.4
Age							
≤29	35		24.3±2.7	176.1±8.0	74.5±11.0	7.7±4.4	9.7±6.3
Normal	21	60	24.4±2.4	176.1±9.1	72.2±11.1	7.1±4.0	9.5±5.5
Desaturators	14	40	24.1±3.1	176.1±6.4	77.9±10.4	8.6±4.9	10.0±7.5
30-39	16		35.0±2.5	178.9±8.6	77.1±13.8	7.5±3.5	12.3±7.5
Normal	10	62.5	34.8±2.9	176.8±8.9	75.7±14.7	6.0±3.1	16.7±5.3
Desaturators	6	37.5	35.3±2.0	182.4±7.9	79.4±7.5	10.1±2.8	4.8±3.3
40-49	16		45.9±2.3	174.4±9.4	74.5±12.9	9.1±3.6	15.1±11.4
Normal	12	75	45.3±2.3	173.3±9.4	75.3±14.1	9.3±3.2	15.8±11.8
Desaturators	4	25	47.8±0.5	177.6±10.1	71.8±9.1	8.8±5.3	13.3±11.5
50+	15		56.3±6.8	170.3±8.8	73.8±9.0	7.2±2.8	21.9±16.8
Normal	10	66.7	57.0±7.5	172.7±8.6	76.0±8.0	6.9±2.8	21.4±19.6
Desaturators	5	33.3	54.8±5.4	165.4±7.8	69.5±10.4	8.0±2.9	22.8±11.4
Fitness							
Elite	10		28.9±7.1	179.6±6.9	70.9±7.9	11.6±4.4	10.9±7.4
Normal	4	40	28.3±7.7	180.3±7.7	72.9±7.5	9.5±1.9	11.0±6.2
Desaturators	6	60	29.3±7.5	179.1±7.1	69.6±8.6	13.0±5.1	10.8±8.7
High	19		38.6±10.5	176.4±7.2	76.4±8.4	9.1±3.0	14.1±10.5
Normal	12	63.2	39.5±10.6	176.4±7.4	76.1±8.9	9.0±3.5	14.9±10.7
Desaturators	7	36.8	37.1±11.1	176.5±7.3	77.0±8.2	9.2±2.4	12.9±10.8
Moderate	23		38.7±16.4	173.5±9.0	77.2±14.8	7.5±3.1	13.9±13.8

Normal	14	60.9	42.5±16.3	173.3±9.5	74.4±16.6	7.3±3.6	17.4±15.4
Desaturators	9	39.1	32.9±15.5	173.8±8.8	81.6±10.7	7.7±2.5	8.4±9.3
Low	16		36.1±11.9	172.0±9.4	73.3±10.4	5.4±2.3	14.4±10.6
Normal	14	87.5	35.9±11.3	173.0±8.9	74.4±9.9	5.5±2.4	14.0±10.1
Desaturators	2	12.5	38.0±21.2	165.5±14.8	65.7±14.4	4.3±2.5	17.0±18.4
Exercise Type							
Bike	61		35.2±13.3	175.5±9.1	76.3±10.8	7.9±3.6	13.2±11.5
Normal	40	65.6	36.8±13.8	175.4±9.2	75.9±11.6	7.5±3.4	14.2±12.4
Desaturators	21	34.4	32.1±12.1	175.6±9.0	76.9±9.3	8.7±3.9	11.1±9.6
Treadmill	21		40.0±11.4	174.6±8.3	70.8±12.8	7.7±4.5	14.4±9.2
Normal	13	61.9	38.5±11.4	173.6±8.3	69.3±11.9	6.8±4.0	15.4±8.5
Desaturators	8	38.1	42.5±11.8	176.2±8.8	73.3±14.7	9.3±5.0	12.9±10.6
Training							
High	27		38.7±12.4	176.8±9.2	74.2±9.9	10.6±4.1	13.9±13.0
Normal	16	59.3	40.2±13.2	176.2±9.8	74.4±11.6	9.6±3.8	13.4±14.8
Desaturators	11	40.7	36.5±11.6	177.7±8.6	73.8±7.0	12.1±4.2	14.5±10.6
Moderate	55		35.3±13.2	174.5±8.7	75.2±12.3	6.5±2.8	13.3±9.9
Normal	36	65.5	36.3±13.1	174.3±8.7	74.1±12.3	6.3±3.0	15.2±10.0
Desaturators	19	34.6	33.5±13.4	174.9±8.7	77.3±12.4	6.8±2.5	9.6±8.8

Mean±SD

Table 2: Average Total and Stratified Population Exercise Performance

	O2 Saturation, %		Max	Max Speed	Max Incline	Max	Estimated VO_{2max}	Lactate, mmol/l	
	Resting	Lowest	Watts	MPH	%	Watts/kg	ml/kg/min	Resting	Final
Total Population	99.3±1.1	95.0±3.2	329.0±88.9	6.1±0.8	8.0±2.3	4.26±1.06	47.8±9.9	1.5±0.5	11.6±3.0
Normal Saturators	99.6±0.9	96.7±1.3	312.4±85.9	6.0±0.6	8.2±2.2	4.04±0.94	46.2±9.4	1.4±0.4	11.7±2.8
Total Desaturators	98.8±1.3	92.0±3.3	360.7±87.9	6.3±1.1	7.8±2.5	4.67±1.18	50.8±10.2	1.5±0.6	11.5±3.5
Mild Desaturators	99.2±1.1	93.6±0.5	348.3±68.7	6.7±1.4	9.0±1.2	4.45±0.91	49.9±9.0	1.5±0.8	11.9±3.4
Severe Desaturators	98.2±1.4	88.9±4.0	391.7±126.7	5.9±0.6	6.5±3.0	5.19±1.64	53.5±13.7	1.5±0.3	10.8±3.5
Sex									
Male	99.4±1.0	95.2±2.4	351.5±77.9	6.1±0.9	8.5±2.3	4.4±1.0	48.9±9.9	1.4±0.5	12.1±3.0
Normal	99.6±0.9	96.6±1.2	338.2±76.0	5.9±0.4	8.3±2.7	4.2±0.9	48.0±9.8	1.3±0.4	12.1±2.7
Desaturators	99.0±1.2	92.7±1.8	373.2±78.2	6.4±1.7	9.0±1.2	4.8±1.2	50.3±10.1	1.5±0.7	12.1±3.6
Female	99.1±1.4	94.6±4.9	226.8±60.7	6.1±0.6	7.3±2.2	3.5±0.9	44.3±9.2	1.6±0.5	9.8±2.2
Normal	99.5±1.1	96.9±1.6	223.3±53.2	6.2±0.9	8.0±1.4	3.4±0.9	41.3±6.1	1.7±0.5	10.0±2.5
Desaturators	98.0±1.6	89.2±5.8	242.5±116.7	6.1±0.3	6.5±3.0	3.8±1.4	53.5±11.6	1.5±0.4	9.4±1.8
Age									
≤29	99.0±1.3	95.0±2.6	315.8±91.9	6.1±0.2	9.2±2.3	4.1±1.1	47.6±10.6	1.6±0.6	12.7±2.6
Normal	99.5±1.0	96.7±1.3	286.8±87.7	6.0±0	9.0±2.6	3.9±1.0	46.4±10.5	1.5±0.5	12.2±2.9
Desaturators	98.4±1.4	92.6±1.9	353.8±85.9	6.5	10	4.4±1.3	49.2±10.9	1.7±0.8	13.5±1.9
30-39	99.6±1.2	95.8±2.5	365.0±83.7	6.5±1.2	7.7±2.0	4.8±1.1	51.5±12.5	1.2±0.3	10.8±3.7
Normal	99.4±1.5	97.2±1.5	351.3±88.3	6.0±0	6.0±2.8	4.5±1.2	47.4±13.3	1.3±0.3	12.3±2.7
Desaturators	99.9±0	93.3±0	420.0±28.3	6.7±1.1	8.5±1.2	5.5±0.6	58.2±2.8	1.0±0.1	8.9±3.6
40-49	99.5±0.7	95.4±2.5	375.0±86.3	6.1±0.8	7.7±2.3	4.7±1.0	48.7±7.5	1.5±0.4	10.8±2.5
Normal	99.6±0.7	96.5±1.4	354.3±93.8	6.2±0.9	8.4±1.7	4.3±0.9	47.4±7.3	1.4±0.4	10.5±2.8
Desaturators	99.3±1.0	92.0±1.8	423.3±46.2	6	4	5.7±0.1	56.5±3.5	1.6±0.2	11.7±1.0
50+	99.3±0.9	94.0±5.2	290.5±66.2	5.5±0.6	7.5±3.0	3.9±0.7	43.3±5.4	1.4±0.5	10.8±3.2
Normal	99.6±0.5	96.6±1.3	291.3±50.8	5.5±0.7	8.0±2.8	3.8±0.6	42.8±5.3	1.3±0.4	11.6±2.6
Desaturators	98.8±1.3	88.8±6.3	288.3±113.4	5.5±0.7	7.0±4.2	4.1±1.0	44.3±6.3	1.5±0.6	8.7±3.9
Fitness									
Elite	99.3±1.4	93.5±2.9	455.0±35.5	6.8±1.0	9.3±1.2	6.2±0.8	65.3±3.0	1.2±0.4	12.1±3.8
Normal	100.0±0.0	95.8±1.0	438.8±31.7			5.8±0.6	67.0±2.3	1.2±0.5	15.2±1.2
Desaturators	98.8±1.6	92.0±2.8	476.7±32.1	6.8±1.0	9.3±1.2	6.5±0.9	64.2±3.0	1.2±0.3	10.0±3.4
High	99.5±1.0	95.5±1.8	362.3±59.0	6.0±0.8	10.0±1.6	4.7±0.7	53.3±3.1	1.3±0.4	11.9±2.9
Normal	99.8±0.6	96.6±1.4	349.4±64.8	6.0±0.8	10.0±1.6	4.5±0.6	52.7±2.6	1.3±0.4	11.2±3.4
Desaturators	99.1±1.5	93.7±0.5	377.1±52.3			4.9±0.8	54.3±3.7	1.4±0.5	12.9±1.6
Moderate	99.0±1.4	95.3±2.3	310.0±69.6	5.7±1.2	8.7±1.2	4.0±0.6	43.4±3.2	1.6±0.7	12.1±3.2
Normal	99.1±1.4	96.8±1.2	308.8±74.8	7	8	4.0±0.6	44.0±3.4	1.4±0.4	12.5±1.7
Desaturators	98.7±1.3	92.9±1.3	312.1±64.4	5.0±0	9.0±1.4	3.9±0.6	42.4±2.7	2.0±1.0	11.4±5.1
Low	99.7±0.6	96.0±3.2	259.2±69.0	5.7±0.5	8.0±1.6	3.4±0.7	36.8±1.4	1.4±0.3	11.2±2.5
Normal	99.7±0.6	96.9±1.6	264.0±66.4	5.7±0.5	8.0±1.6	3.3±0.7	36.9±1.5	1.4±0.3	11.2±2.5
Desaturators	99.5±0.7	89.5±4.9	235.0±106.1			3.5±0.9	36.5±0.7	1.4±0.3	11.7±3.7
Exercise Type									
Bike	99.3±1.1	95.3±2.7	329.0±88.9			4.3±1.1	47.7±9.9	1.5±0.5	12.8±2.1
Normal	99.5±1.0	96.8±1.3	312.4±85.9			4.0±0.9	46.4±9.7	1.4±0.4	12.6±2.2
Desaturators	98.9±1.2	92.5±2.3	360.7±87.9			4.7±1.2	49.9±10.1	1.6±0.7	13.3±1.7
Treadmill	99.4±1.2	94.4±4.3		6.1±0.8	8.0±2.3		48.4±10.1	1.5±0.5	7.9±2.2

	O2 Saturation, %		Max	Max Speed Estimated VO_{2max}	Max Incline	Max Lactate, mmol/l		
	Resting	Lowest	Watts	MPH	%	Watts/kg		
			ml/kg/min	Resting	Final			
Normal	99.9±0.3	96.6±1.5		6.0±0.6	8.2±2.2		45.2±8.7	1.5±0.5
Desaturators	98.6±1.7	90.8±5.0		6.3±1.1	7.8±2.5		54.2±10.8	1.4±0.5
Training								
High	99.4±0.9	94.7±2.6	393.0±75.1	6.9±0.9	9.5±1.0	5.2±0.9	56.6±7.9	1.3±0.4
Normal	99.7±0.6	96.3±1.3	377.1±79.4	6.5±0.7	9.0±1.4	4.9±0.7	54.9±8.2	1.3±0.3
Desaturators	99.0±1.2	92.4±2.2	417.8±64.2	7.3±1.1	10.0±0	5.6±0.9	59.1±7.0	1.3±0.4
Moderate	99.2±1.2	95.2±3.4	290.3±73.4	5.9±0.7	7.6±2.4	3.7±0.8	42.7±6.9	1.5±0.6
Normal	99.5±1.0	96.9±1.3	276.6±69.2	5.9±0.5	8.0±2.4	3.6±0.8	41.8±6.5	1.5±0.5
Desaturators	98.7±1.4	92.1±3.9	316.5±76.6	6.0±1.0	7.0±2.4	3.9±0.8	44.5±7.6	1.6±0.7

									12.2±
									12.7±
									11.6±
									11.3±
									11.2±
									11.5±

Table 3: Average Post Exercise Recovery Data

	n	HR Resting	HR 0 min	HR 5 min	HR 15 min	O2 Sat Resting	O2 Sat 0 min	O2 Sat 5 Min	O2 Sat 15 Min
Total Population	82	60.3±10.9	147.5±30.6	104.7±16.4	95.6±17.5	99.3±1.1	97.7±4.2	98.1±1.7	97.7±2.2
Normal Saturators	53	58.4±9.1	146.7±32.8	100.6±14.9	93.1±17.2	99.6±0.9	98.8±4.5	98.8±1.2	98.3±2.2
Total Desaturators	29	63.5±12.9	149.1±26.6	111.5±16.7	99.7±17.7	98.8±1.3	95.8±2.6	96.9±1.8	96.6±1.8
Mild Desaturators	19	62.8±12.7	148.4±26.5	113.9±15.0	103.2±15.0	99.2±1.1	95.9±2.8	97.1±1.8	96.9±1.7
Severe Desaturators	10	64.7±13.7	152.5±30.8	106.4±19.9	91.8±21.4	98.2±1.4	95.4±2.1	96.6±1.7	95.7±1.8

Mean±SD. All times are minutes post-exercise.