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Effect of a Hockey Season on Pulmonary Function and Arterial Desaturation

by

Alex B. Game 

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Master of Science

Department of Physical Education and Recreation

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ABSTRACT

The purpose of this study was to examine changes in pulmonary function and arterial desaturation in hockey players before and after a competitive season. Sixteen male subjects (age 22 ±1.4) were tested pre- and post-season for anaerobic power, VO_2max and pulmonary function in the ice arena environment (Temp. 13-16°C and relative humidity 30-45%). A pulse oximeter was used to monitor arterial oxygenation (SaO_2) during the VO_2max test. Pulmonary function was performed following the anaerobic power test and the VO_2max test. There was no difference in anaerobic power or VO_2max after the season. SaO_2 at VO_2max was significantly lower after the season. Forced expiratory flow in 1 sec ratio (FEV_1rest/ FEV_1 exercise) and forced vital capacity (FVC) measured 15 and 25 minute post VO_2max were significantly lower after the season . In conclusion, there was a significant reduction in lung function after completion of a varsity hockey season that coincided with a significant decrease in SaO_2. This may impair performance in varsity ice hockey players.

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LIST OF SYMBOLS, NOMENCLATURE OR ABBREVIATIONS

CFU	Colony Forming Units
CO	Carbon monoxide
COHb	Carboxyhemoglobin
EIA	Exercise-induced asthma
EIB	Exercise-induced bronchospasm
EIH	Exercise-induced hypoxemia
FVC	Forced Vital Capacity
$FEF_{25-75\%}$	Mean Forced Expiratory Flow during the middle half of the FVC
FEV_1	Forced Expiratory Volume in one second
Hb	Hemoglobin
HbO_2	Oxygen saturation of hemoglobin
MetHb	Methemoglobin
NO	Nitric oxide
NO_2	Nitrogen dioxide
O_2	Oxygen
$PaCO_2$	Arterial carbon dioxide tension
PaO_2	Arterial oxygen tension
PAO_2	Alveolar oxygen tension
PEF	Peak Expiratory Flow
PvO_2	Venus oxygen tension
RBC	Red Blood Cell

SaO_2	Arterial oxygen saturation of hemoglobin
SO_2	Sulfur dioxide
VA/Qc	Ventilation-perfusion inequality
VO_2 max	Maximal oxygen consumption

CHAPTER 1

INTRODUCTION

1.1 Introduction

Exercise-Induced Bronchospasms (EIB) are characterized by a transient airflow obstruction that begins to occur after several minutes of strenuous exercise (Bar-Yishay, 1984). EIB can be triggered by the intensity of exercise, cold and dry environments, chronic asthma, or by a variety of air pollutants (Virant, 1992). EIB can be identified in 90% of patients with chronic asthma and in 35-40% of allergic nonasthmatics (Virant, 1992). Studies of athletic populations suggest that 3-10% of athletes experience EIB (Rice *et al.*, 1985). Prior to the 1984 Olympic games, the United States Olympic Committee found 67 of 597 athletes (11%) had a history of EIB and / or symptoms suggesting EIB (Voy, 1984). Recent research by Provost-Craig *et al.*(1996) and Mannix *et al.* (1996) showed that 30% and 35% of figure skaters had EIB. EIB is the physiological response and if diagnosed by a physician after an exercise treatment, the condition is called exercise-induced asthma (EIA). A recent study by Clark and Reid (1999) found previously undiagnosed incidents of EIA in 23% of an Alberta hockey team. As well, a study by Leuppi *et al.* (1998), suggested that hockey players could be at greater risk of asthma and EIB than athletes in other sports due to the cold, dry arena environments that they practise and compete in.

EIB results in increased airway resistance which reduces the ventilation-perfusion ratio in the lung and may lead to a drop in partial pressure of arterial oxygen content

(PaO_2). A decrease in PaO_2 during high intensity exercise produces a decrease in oxygen saturated with hemoglobin ($\%HbO_2$). If this level drops 4 % below the resting level, a condition termed exercise-induced hypoxemia (EIH) occurs (Powers *et al.*, 1989). This condition has been most often observed in elite aerobic endurance athletes with very high maximal oxygen consumption (VO_2 max) levels and may lead to a decrease in performance (Dempsey *et al.*, 1984; Powers *et al.*, 1989). Powers *et al.* (1989) found that for every exercise-induced reduction in $\%HbO_2$ there was an equal corresponding decrease in VO_2 max of elite endurance athletes. It has also been shown that athletes who experienced EIH at sea level suffer more gas exchange impairments at higher altitudes than healthy untrained individuals (Lawler, 1988).

The incidence of EIH in athletes with high maximal oxygen consumption levels such as elite endurance runners and cyclists does not seem applicable to hockey players for a number of reasons. Primarily, hockey players do not exhibit the same level of VO_2 max: Hockey players = 62 $ml \cdot kg^{-1} \cdot min^{-1}$ (Cox *et al.*, 1993) vs elite endurance athletes, >70 $ml \cdot kg^{-1} \cdot min^{-1}$ (Powers *et al.*, 1992). It has also been suggested that on-ice practise time and game play does not provide a sufficient physiological challenge to maintain or improve maximal oxygen consumption (Green *et al.*, 1979 and Cox *et al.*, 1993). However, Clark and Reid (1999) have suggested that the combination of environmental conditions such as lower temperature, lower relative humidity, poor air quality (including molds and chemicals such as carbon monoxide) and the amount of time spent practising and playing games under these conditions contribute to EIB in hockey players. Consequently, any observed EIB could then ultimately result in a decrease in

performance by reducing the ventilation perfusion ratio leading to EIH.

1.2 Significance of the Study

This study examined the existence of EIB and EIH in hockey players before and after a competitive season and investigated the relationship of these factors to arena air quality. If significant health issues related to EIB and EIH are detected in hockey players, this could result in the need for further screening for EIB in young athletes involved in hockey as well as other related winter sports such as figure skating, cross country and downhill skiing. Thus, diagnosed athletes would be able to seek medical treatment to offset any performance decrements. Results could also lead to new regulations in the monitoring and evaluation of the ice arena environment to reduce air borne pollutants and molds.

1.3 Purpose

The purpose of this study was to examine pulmonary function and arterial desaturation in the University of Alberta Golden Bears varsity hockey team before and after a complete CIAU competitive hockey season. Arterial desaturation or exercise-induced hypoxemia (EIH) was measured by an oximeter, which indirectly measured the amount of hemoglobin saturated with oxygen during a maximum oxygen consumption test (VO_2 max). Pulmonary function tests were conducted before and after the anaerobic power and VO_2 max tests to assess exercise-induced bronchospasm (EIB). All physiological testing was done at ice level in the Clare Drake arena at the University of Alberta to ensure that the air conditions the hockey players are normally exposed to were experienced during testing. Simultaneous measurements of air temperature,

humidity, and barometric pressure were made at ice level and a sample of arena air was tested for chemical and biological contaminants. These same testing procedures were repeated at the conclusion of the hockey season to determine the chronic effect of participation in hockey and exposure to arena conditions throughout the season.

1.4 Hypothesis

The hypothesis was that the combination of participation in competitive hockey and exposure to the type of environment in ice arenas has a negative impact on physiological function. Pollution, molds, cold and dry air may contribute to a condition of exercise-induced bronchial constriction which could limit performance. This bronchial constriction may cause a decrease in the supply of oxygen to the alveoli in the lung resulting in a decrease in PaO_2 that can result in exercise-induced hypoxemia.

1.5 Delimitation

- The study was delimited to members of the University of Alberta Golden Bears varsity hockey team. The sample consisted of 16 males.
- Subjects where delimited to non smokers. Non smoking was defined to mean that the subjects have not smoked in the last year.
- The study was delimited to measurements that the Horizon Metabolic Cart, the Biox IIa Oximeter and the Microloop 3535 spirometer are capable of measuring.
- The study was delimited to the subjects performing two testing sessions, one before the season and one following the completion of the hockey season. All subjects performed the tests with a minimum of 24 hours rest from any other intense exercise.

1.5 Limitations

- Motivation of the subjects was limited to investigator encouragement and individual motivational levels. Motivation to continue to perform when the work level became uncomfortable was entirely left to the subjects perception of whether the intensity was still tolerable.
- Attrition could be a problem due to the high intensity nature of the testing, the long hockey season and the possibility of injury in-between testing periods. Attrition may affect the statistical power and furthermore affect the validity of the study.
- Experimenter and equipment error could have limited the study.
- The intention to study a group of hockey players means that volunteers were invited to participate, thereby precluding random selection of subjects from the target population.
- Players practiced and played in other arenas throughout the season that were not monitored for mold and air pollution. The effect the air of the other arenas could limit the study.

1.6 Definitions

- VO_2 max: This is the maximum amount of oxygen which the body can take in, distribute to the working muscles, and use to perform maximal exercise. VO_2 max was indicated by a < 100 ml/min change in VO_2 with continued exercise prior to exhaustion. Additional supportive criteria included; a heart rate of 10% more or less of age predicted maximum; respiratory exchange ratio (RER) > 1.1 was

observed; and/or subject was too fatigued to continue exercise.

- Exercise-induced Hypoxemia: This was a decrease in $\%HbO_2$ of 4.0% or more below resting values in arterial blood during exercise. $\%SaO_2$ is the estimation of $\%HbO_2$ as measured by oximetry (Powers *et al.*, 1988).
- Exercise-induced Bronchospasm: This is a clinical syndrome characterized by transient airflow obstruction following several minutes of strenuous activity. Exercise-induced bronchospasm was diagnosed if the athlete had a decrease in forced expiratory volume at 1 s (FEV_1) or peak expiratory flow (PEF) of 10% or greater after exercise. Smaller airway bronchospasm was diagnosed by a 20% change in forced expiratory flow at 25-75% of vital capacity ($FEF_{25-75\%}$) (Mcfadden, 1995).
- Exercise-Induced Asthma: Is the same obstruction of airways as the EIB but it has been diagnosed by a doctor and treatment has been prescribed to alleviate the condition.

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CHAPTER 2

REVIEW OF LITERATURE

2.1 Introduction

Historically in the field of exercise science, pulmonary gas exchange was considered a minor limiting factor in maximal oxygen uptake (VO_2 max) in healthy individuals. In most cases efficient alveolar ventilation and alveolar-to-arterial exchange of O_2 and CO_2 are among the strongest links in the gas transport chain during exercise. However a weakness in this chain has been found in elite endurance athletes who exhibit a phenomena called exercise-induced hypoxemia (EIH). EIH refers to arterial oxygen-hemoglobin desaturation usually 21 to 35 mmHg below resting levels of PaO_2. This phenomena has been associated with exercise intensities in excess of 70% of VO_2 max in highly trained runners and cyclists who exhibit VO_2 max above 60 $ml \cdot kg^{-1} \cdot min^{-1}$ (Dempsey *et al.*,, 1984, Martin, 1992a, b, Pedersen, 1992, Warren, 1991, Lawler, 1988, Powers 1988, 1989b, and 1992, Williams, 1986, and McCusker, 1992). There seems to be a high incidence of EIH in endurance cyclists measured during VO_2 max testing (Brown, 1993). Conversely, untrained healthy individuals exhibit little or no EIH (Dempsey *et al.*, 1984; Powers, 1992). This suggests that EIH may be related to the amount of training done by althletes and/or physiological limitations due to the anatomical structure of the pulmonary system (Lawler, 1988). It is also possible that a genetic predisposition may influence EIH.

Hockey players present a unique athletic group to investigate the incidence of

EIH. Hockey players have significantly lower VO_2 max scores than elite endurance athletes (Cox, 1995) and have exhibited some level of detraining or overtraining during the long competitive hockey season (Green, 1994) . Some research has attempted to link this decrease in performance to inadequate supplementary endurance training throughout the course of the season (Cox, 1995). Others have linked a reduction in performance to acute and chronic fatigue over the season (Jones *et al.*, 1983). A study by Koistinen (1995) demonstrated that athletes levels of aerobic fitness have an impact on their recovery rate from intense exercise. This is perhaps where EIH has an effect on hockey players. Lowering the $\%HbO_2$ could lead to inadequate recovery between shifts and games. Recent publications in the field of exercise-induced asthma may point to a mechanism for EIH in hockey players. Two studies performed on figure skaters (Prevost-Craig *et al.*, 1996 and Mannerix *et al.*, 1996) and two on hockey players (Clark, 1998 and Leuppi, 1998) found that 30-35% of figure skaters and 19-23% of a hockey team studied, exhibited exercise-induced bronchospasms (EIB). This reduction in airflow following several minutes of intense exercise may reduce the ventilation-perfusion ratio in the lung which could lead to a drop in PaO_2 which could lead to EIH and ultimately limit performance.

2.2 Exercise-Induced Hypoxemia

EIH has been observed in healthy athletes since 1958 when Holmgren and Linderholm first reported a decline in PaO_2 during heavy exercise. This research found considerable variability of blood gas response across subjects as some athletes demonstrated EIH (eg. PaO_2 = 57 mmHg below resting values) whereas others

maintained PaO_2 within 5 to 8 mmHg of resting values (eg. PaO_2 = 93 mmHg). Rowell *et al.* (1964) demonstrated that arterial oxyhaemoglobin saturation (%HbO_2) declined from a resting mean of 98% to 85% during heavy exercise in highly trained endurance athletes. Dempsey *et al.* (1984) provided evidence that EIH exists in highly trained endurance athletes and proved that this phenomenon was not an isolated case or measurement artifact. Dempsey *et al.* (1984) tested sixteen male endurance athletes who had VO_2 max between 58 to 82 mlkg/min. and showed that PaO_2 was reduced to 21 to 35 mmHg below resting values in 50% of the athletes. Since this initial work by Dempsey *et al.*, (1984), additional research has measured PaO_2 during exercise and has provided further evidence of EIH (Martin, 1992b, Pedersen, 1992, Powers, 1992, and Warren, 1991).

2.3 Mechanism of Hypoxemia

Dempsey *et al.*, (1984) suggested four possible mechanisms of EIH: hypoventilation, venoarterial shunt, ventilation-perfusion mismatch, and diffusion limitation. At present, no one mechanism has been put forward as the cause of EIH. This section will discuss these four mechanisms and current research.

Hypoventilation is defined as alveolar ventilation below the rate metabolically required to maintain arterial blood gases at normal values (Powers, 1993). Clinically, hypoventilation is diagnosed when there is an increase in arterial carbon dioxide tension ($PaCO_2$) above normal values. Both Dempsey *et al.*, (1984) and Powers (1992) found a decrease of $PaCO_2$ during exercise in elite athletes exhibiting EIH, not an increase. From this perspective EIH was not caused by hypoventilation. However, Dempsey *et al.*, has

suggested that the magnitude of EIH is in part caused by an unequal hyperventilatory response contrary to the result of decrease in $PaCO_2$. In his later study there was little or no hyperventilatory response in subjects who exhibited severe hypoxemia. Dempsey *et al.*, (1984) suggests that this could result in a low alveolar oxygen tension (PAO_2) and a reduction in the driving force for oxygen transfer across the blood-gas barrier. Powers (1992) found that there was low correlation between PaO_2 at VO_2 max and ventilatory indices, and could not establish a relationship between hyperventilation and EIH.

There are some important methodological differences between the studies that may affect the interpretation of previous research. Dempsey *et al.*, (1984) studied runners during a treadmill exercise while Powers *et al.*(1992) investigated cyclists during cycle ergometry. The differences in exercise modality between runners and cyclists in ventilatory control or in efficiency of gas exchange, make comparisons tenuous. It has been reported that work on a treadmill elicits a higher VO_2 max than work on a cycle ergometer and this could account for nearly a five percent difference in VO_2 max scores. The second difference has been the magnitude of EIH observed. Dempsey *et al.*, (1984) reported that 8 out of 16 subjects had a PaO_2 at VO_2 max of 75 mmHg or less, whereas the lowest recorded in Powers (1992) was 79 mmHg. This is further evidence that differences in modality may affect EIH as treadmill exercise may elicit a greater level of hypoxemia compared to the cycle ergometer. This was later confirmed by Harris (1995) who found that subjects exercising on the treadmill had a slower hyperventilatory response which may be one of the factors contributing to magnitude of EIH.

The second proposed mechanism of EIH is that of venoarterial shunt. This refers

to areas in the lung where venous blood is not channeled through ventilated areas of the lung. (West, 1983). The result is poorly oxygenated blood being transported into arterial circulation without being reoxygenated in the alveoli which causes a decline in PaO_2. At rest, this shunt accounts for approximately 50% of the alveolar-arterial oxygen tension difference (A-a PO_2 diff.) (Whipp and Wasserman, 1969). If this effect was maintained during exercise or increased, it may account for EIH observed at intense exercise workloads. However, research by Dempsey *et al.*, (1984) and Powers (1992) demonstrated that when athletes exhibiting EIH switched from breathing a normoxic gas mixture (room air, 20% O_2) to a mild hyperoxic gas mixture (24-25% O_2) the PaO_2 rose back to normal levels. If there had been no effect on PaO_2 then venoarterial shunt may have been a cause of decreasing PaO_2 levels in EIH conditions. Since oxygen tension was restored, this eliminated the shunt as a possible cause of EIH.

Ventilation-perfusion inequality (VA/Qc) is a mismatch of ventilation and blood flow in the lung (West, 1983). At rest VA/Qc is well matched and does not have a negative impact on pulmonary gas exchange (West 1991). The key question is whether heavy exercise results in a greater VA/Qc mismatch and contributes to EIH. Hammond (1986) demonstrated an increase VA/Qc inequality during exercise, up to an oxygen consumption of 3 L/min. At higher work rates VA/Qc remained constant but A-a PO_2 Diff. continued to increase. It seems that VA/Qc does not explain the widening of the A-a PO_2 diff. at high intensities and cannot be considered solely as a factor contributing to EIH.

Exercise presents a unique challenge for pulmonary diffusion because of a

decrease in both the partial pressures of oxygen in mixed venous blood (PvO_2) and red blood cell (RBC) transit time in the pulmonary capillary. This leads to the possibility that diffusion limitations may play a role in EIH. Under normal conditions, two key adjustments in the cardiovascular system are made during exercise to improve the efficience of gas transfer: hyperventilation results in a increased PAO_2 which increases the driving force of oxygen into the blood; and the pulmonary capillary blood volume rises which increases the surface area available for diffusion and prolonged RBC transit time. However, the demand on pulmonary diffusion during heavy exercise in elite endurance athletes may be too great for the aforementioned adjustments. The major determinants of alveolar capillary diffusion are: surface area available for diffusion; distance required for diffusion from alveolar membrane to the RBC; RBC transit time; and rate of equilibrium in the pulmonary capillary. Dempsey (1987) has suggested that a combination of any two of the above mentioned determinants could result in EIH in elite endurance athletes. Warren (1991) estimated cardiac output, pulmonary capillary blood volume, and pulmonary diffusion during heavy exercise and concluded that the decrease in mean transit time doesn't explain the drop in PaO_2 in elite endurance athletes experiencing EIH. The more plausible explanation is that some barrier increases the diffusion time between alveolar membrane and capillary. As exercise intensity increases, stroke volume and cardiac output increase to meet the growing demands of the exercising muscle. This creates a great increase in the pressure of the blood as it enters the pulmonary capillaries. This increase in pressure has two effects, one is leakage and the other is actual mechanical disruption of the capillary walls. Wagner (1986) found an

increase in transcapillary fluid flux which could result in a low-grade pulmonary edema in the lung that may increase the diffusion distance. In a recent study, Hopkins *et al.*(1997) found that the capillary walls can fail under the increased blood pressure brought on by intense exercise. This latter study found that after exercising elite endurance cyclists, significant traces of RBCs and blood proteins were found in a bronchoalveolar lavage of the lungs. This would increase the diffusion distance as well as the available surface area available for diffusion. However they did not test oxygen consumption to determine if there were was a drop in PaO_2 along with the structural damage in the lungs.

2.4 Exercise-induced Hypoxia and VO_2 Max

A key question remains; does EIH result in any significant reductions in endurance performance? To date, three key investigations have found that the magnitude of EIH has an effect on maximal oxygen consumption (Powers *et al.*, 1989, O'Kroy, 1989 and Pedersen, (1992). The first study by Powers (1989) found that for every 1% decrement in $\%HbO_2$ there was an equal 1% decrement in VO_2 max. These findings have been corroborated by O'Kroy (1989) and Pedersen (1992) who found similar decrements in VO_2 max with EIH. This has also been recorded in a study by Lawler (1988) who demonstrated that endurance athletes who exhibit EIH at sea level suffer more gas exchange impairments at higher altitudes than healthy untrained individuals.

2.5 Exercise-induced Hypoxemia and Pulse Oximetry

Historically, measurement of $\%HbO_2$ required an arterial catheter. Furthermore, given the cost, time, and potential risk associated with this practice a noninvasive means

of estimating $\%HbO_2$ during exercise is beneficial. Pulse oximetry has been used to detect lung disease, assess the need for O_2 therapy (Ramanathan *et al.*, 1987) and evaluate the severity of disease and symptoms associated with exercise (Orenstein *et al.*, 1993). A large number of the studies investigating EIH have used the technique of pulse oximetry to estimate $\%HbO_2$ in athletes (Lawler *et al.* 1988, Martin and O'Kroy 1992, Powers *et al.* 1988, 1989a, and Williams *et al.* 1986).

Pulse oximetry is based on the different light-absorbing characteristics of oxyhemoglobin (HbO_2) and hemoglobin (Hb) at two different wavelengths; 660 nm (red) and 940 nm (infrared). A light emitting diode is located on one side of the probe and a photodetector is located on the other side. The probe is attached to the earlobe or finger of the subject. The transmitted light is divided into two components; component A is transmitted light of variable intensity that occurs during a systole and is a function of the pulsations of oxygenated arterial blood. Component B is transmitted light during diastole that has a constant intensity and is a function of the various tissues (skin, muscle, fat bone) (Alexander *et al.*, 1989). The oximeter divides component A by the background light absorption of component B, at the two different wavelengths, to obtain an absorption ratio (R):

$$R = \frac{A_{660} / B_{660}}{A_{940} / B_{940}}$$

Based on R, an algorithm within the pulse oximeter calculates $\%SpO_2$. Thus $\%SpO_2$ estimates the arterial oxygen saturation of available hemoglobin ($\%SaO_2$)

(Alexander *et al.*, 1989).

$$\%SaO_2 = \frac{HbO_2}{HbO_2 + Hb} \times 100\%$$

One source of error with this method is that the oximeter can distinguish only HbO_2 and Hb. However, two other hemoglobin compounds are present in small amounts: carboxyhemoglobin (COHb) and methemoglobin (MetHb). COHb and HBO_2 absorb a similar amount of light which could lead to falsely elevated $\%SaO_2$. Powers *et al.* (1989) found that subjects with COHb levels > 4% the pulse oximeter, overestimated HBO_2 saturation. COHb levels can be increased by smoking or being exposed to high concentrations of carbon monoxide levels. Methemoglobin has a large absorption at both wavelengths and tends to increase $\%SaO_2$ (Tremper, 1989).

Other sources of variability come from skin pigment (Cahan *et al.*, 1990), motion artifact (Tremper, 1989), and peripheral perfusion levels (Clayton, *et al.*, 1991). There is some debate as to the accuracy of using pulse oximetry during exercise. In a review of pulse oximetry studies, Mengelkoch *et al.* (1993) found that 70% of pulse oximeters have been shown to be accurate predictors of $\%HbO_2$ when $\%HbO_2$ is $\geq 85\%$ in nonsmoking subjects. The data also suggests that finger-probe models of oximeters may be more accurate than ear-probe models.

2.6 Exercise-induced Bronchospasm (EIB)

Exercise-induced Bronchospasm (EIB) occurs in up to 90% of all asthmatics and in approximately 3-10% of athletes (Mahler, 1993). EIB is characterized by a transient

airflow obstruction that begins to occur after several minutes of strenuous exercise (Bar-Yishay, 1984). Maximal airflow depression occurs 5-15 minutes post exercise, and it takes 20 to 60 minutes for air flows to return to baseline. Under normal, non-exercise conditions, air is effectively heated (37°C) and humidified (44 mg water/l) by the nasal passage (Hahn, 1984). During exercise, nasal breathing is rapidly bypassed in favor of mouth breathing to meet increasing oxygen demands. This subsequent loss in nasal function results in cooler, drier, inhaled air and combined with increased ventilation, promotes an even greater loss of water and heat from the airway during exercise. This airway environment provides the pathology for EIB (Virant, 1992).

In a survey conducted on the 1984 USA summer Olympic team, 67 of 597 athletes were diagnosed with EIB (Voy, 1984). Of the 67 athletes, 41 (61.2%) were previously undiagnosed with EIB. Prevost-Craig *et al.*(1996) studied figure skaters and found that 30% of their sample experienced EIB. As well, Mannix *et al.*(1996) found that 35% of their sample experience EIB. Clark and Reid (1999) along with Leuppi *et al.*(1998) showed a similar incidence rate among hockey players. Mcfadden *et al.*(1982, 1986) have shown that the severity of EIB depends on initial upper airway heat loss but also on the rapidity and magnitude of airway rewarming after exercise. Anderson *et al.*(1982) found that after subjects inhaled 100% humid air, the severity of EIB diminished, and that inhalation of dry air exacerbated EIB. The actual mechanism for EIB is most likely a combination of the two (water and heat loss). During inhalation, cold dry air results in airway drying. This fluid lose causes a change in osmolarity of airway lining fluid, which in turn triggers the release of a variety of chemical mediators

including mast cells, histamine, leukotriences, prostaglandins, thromboxanes, and platelet-activating factor (Virant, 1992). These mediators cause smooth muscle contraction, either by direct or indirect actions, leading to bronchospasm, an increase in airway secretions along with an increase in vascular permeability that may cause a low-grade pulmonary edema which could lead to increased diffusion distance of O_2 in the alveoli and a decrease in oxygen supply.

There are several factors which have been shown to exacerbate the severity of EIB, including the degree of chronic asthma, nasal obstruction, exercise conditions, and air pollution. The possible mechanisms that may relate to the increase in incidence in figure skaters and hockey players is that of exercise conditions and air pollution. Provost-Craig *et al.* (1996), recorded air temperatures and relative humidities during the summer months when the testing was performed (15.6 ± 2.5°C, 75-85%), however these conditions could be considerably worse during the winter which is the main competitive season for figure skaters. A study completed on Swedish cross-country skiers found that 55% of the subjects tested had asthma and 78% were diagnosed with bronchial hyperresponsiveness. The prevalence of asthma and bronchial hyperresponsiveness has been estimated to be 6-8% in Sweden. This suggests that athletes exposed to cold, dry air may suffer chronic changes in lung function which result in asthma or asthma related symptoms. It has been suggested that any form of exercise would be better tolerated in a warmer, more humid environment (Noviski, 1983). In fact a study by Drobnic *et al.*, (1992) found a relatively high rate of asthma in swimmers, suggesting that athletes with asthma may gravitate to the ambient swimming pool conditions.

In addition to ambient temperature and humidity, air pollutants may also be factors that could aggravate EIB. Pollutants such as molds, CO, NO_2, and other chemical compounds have been found in ice arenas with inadequate ventilation or malfunctioning resurfacing equipment (Lee *et al.*, 1993, Levy *et al.*, 1998).

Exposures to elevated NO_2 and SO_2 concentrations have been associated with several respiratory ailments including; chest tightness, cough, shortness of breath, and hemoptysis (Hedberg *et al.*, 1989, Peden, 1997). Healthy individuals have shown increased pulmonary airway resistance after 2 hours of exposure to 2.5-5ppm of NO_2. Several studies have shown an increase in airway resistance or a decrease in FEV1 with NO_2 concentrations as low as 0.1-0.3 ppm in asthmatics (Orehek *et al.*, 1976, Kleinman *et al.*, 1983, Bauer *et al.*, 1986). A recent study by Strand *et al.*, (1998) found that after repeated exposure to ambient levels of NO_2 (500 $\mu g \cdot m^{-3}$) subjects with mild asthma had an increased airway responsiveness to a small dose of an allergen.

Unlike NO_2, SO_2 exerts a rapid effect on lung function in asthmatics. Significant responses occur within 2 minutes and maximal response is noted within 5-10 minutes. Some asthmatics experience lung function decrements at levels of 0.25 ppm while others have no response at 2.0 ppm (Pedden, 1998). Exercise and SO_2 have additive effects on asthmatics (Kehrl *et al.*, 1987, Hackney, *et al.*, 1984). Work by Pierson *et al.*, (1992) observed that cold dry air exacerbates the bronchoconstriction effect of SO_2 exposure. However a study by Howard *et al.*, (1987) found that asthmatics participating in intermittent exercise had a more attenuated response to SO_2 than a group who performed continuous exercise. This may be due to the recovery of the surface liquid quantities in

the respiratory tract. SO_2 is highly water soluble and is readily absorbed. A break in exercise would have the effect of recovering the airway mucosa and result in a decreased delivery of SO_2 to the lower airways. Another study by Devalia *et al.*, (1994) found that when 0.2 ppm SO_2 and 0.4ppm NO_2 where combined there was a 60% decrease in the amount of allergen dose needed to induce a 20 % fall in FEV1. These findings suggest that combinations of air pollutants and environmental factors can pose significant health risk to asthmatics.

The other form of air pollution that may be found in the ice arena's environment is the presence of colonizations of molds and fungi. Molds have been considered possible triggers of allergic respiratory reactions since the 1930's (Munir and Bjorksten, 1997). Molds and fungi produce allergens, mytoxins, alcohols, aldehydes and ketones that have a varied effect on humans, from presence of moldy odors to active carcinogens (Munir and Bjorksten, 1997). Molds such as Penicillium, Alternaria, and Cladosporium have all been found to have an effect on the respiratory tract of humans (Licorish *et al.*, 1985). The prevalence of mold sensitivity in patients with asthma varies between 5 and 80% in different studies (Lopez and Salvaggio, 1985). Lack of agreement between studies may be due to the identification and culturing of molds and fungi. Counts are not expressed as a number of individual mould spores but as number of colony forming units (CFU). Some colonies inhibit the growth of others and some spores may not be living at time of sampling (Munir and Bjorksten, 1997). A low CFU count of mould species does not exclude a relationship between exposure and sensitivity. Recent work by Taskinen *et al.*, (1997) found an association between indoor mold exposure and respiratory symptoms

in school children by collecting data in the children's school and home environments.

2.6 Physiology of Hockey

Ice hockey is characterized by intense bouts of intermittent skating consisting of rapid changes in direction and velocity as well as upper body work consisting of stick handling, shooting, and frequent body contact. The high intensity bursts require the hockey player to develop muscle strength, power, and anaerobic endurance as well have a well developed aerobic system to enhance recovery and meet the demands of a full game. Thus, hockey represents a unique challenge to the athlete (Green, 1994).

The degree to which anaerobic processes predominate are ultimately dependent on the intensity, duration, frequency of the activity, and the recovery time. Because of the nature of the sport, direct measures are difficult to collect. However, physiologists have attempted to estimate activity through two procedures; time motion analysis and telemetric heart rate monitoring. Green et al. (1979) performed time motion analyses on a college hockey team and found that a typical shift duration averaged 85 seconds followed by 225 seconds recovery period. Within each shift there was an average of 2-3 stoppages in play which averaged 27 seconds each, so playing time was approximately 40 seconds. The number of shifts averaged 14-21 producing a playing time of 21-28 minutes, over the course of a 60 minutes game. Cox (1993) found that the typical National Hockey League Player (NHL) receives less than 16 minutes of playing time while some of the more skilled players received as much as 35 minutes playing time per game. In a later study, Cox et al (1995) telemetered three NHL players for heart rate during game situation and another four players during two on-ice practice sessions. The

telemetry results were compared with data from each player's laboratory assessments. They found that during a game, the percentage of time at or above threshold heart rate (lactate threshold) was 8.5 to 19.1%, which accounted for 6 minutes of playing time. During practices, the players were above threshold heart rate from 9 to 33% of the first practice session and during the subsequent practice, the players were above threshold for 0 to 9% of the practice time.

Since lactic acid is the terminal metabolite in anaerobic glycolysis, blood measures of lactate concentration have been used to evaluate the contribution of the anaerobic system. Studies have found between an eight- and ten-fold increase in blood lactate concentrations during a game (Green et al., 1976 and Wilson and Hedberg, 1975). These values were taken following each period so they can provide only an estimation of the contribution of the anaerobic system and cannot accurately describe what occurs within each shift. Blood lactate concentrations vary substantially between games and possibly between positions, reflecting the diversity that occurs in the nature of the play (Wilson an Hedberg, 1975).

With ice hockey, the involvement of the anaerobic system may be dependent on the efficiency of the aerobic system (Green, 1994). The time delay associated with the uptake and delivery of oxygen to the muscle, necessitates the involvement of anaerobic processes. During recovery, restoration of adenosine triphosphate (ATP) and creatine phosphate (CP) are aerobically dependent (Harris et al., 1976) requiring VO_2 to remain elevated during recovery. This is perhaps when hockey players may be effected by factors leading to EIB and EIH. After an intense hockey shift, bronchoconstriction,

increased airway secretions and vascular permeability resulting from environmental factors and may cause EIH. The lower O_2 concentrations in the arterial blood would cause inadequate recovery from the shift and result in reduced performance in subsequent shifts and possible accumulating game to game into observable chronic fatigue.

Cox (1993) gathered physiological data from 5 NHL teams and found that VO_2 max has progressed from 54 $ml \cdot kg^{-1} \cdot min^{-1}$. in 1980 to 62.4 $ml \cdot kg^{-1} \cdot min^{-1}$ in 1991. These improvements in aerobic power occurred independently of an increase in body mass which has progressed from 85.6 to 89.0 kg. This indicates that there has been a change in conditioning methods employed by NHL teams and players. Green (1987; 1994) has suggested that the amount of playing and training hockey players do currently, may result in chronic fatigue. Jones *et al.* (1983) found that after one hockey practice, maximal voluntary force decreased by 28% below baseline and remained depressed during the following two practices. During the following three days, which included hockey games, force gradually recovered but still remained depressed. Thus, hockey players seem to have the necessary physiological and environmental prerequisites for an increased incidence of pulmonary issues that may negatively influence performance.

2.7 Conclusion

Could the drop in force as a result of a hockey practice reported by Jones *et al.*, (1983) have been exacerbated by performing in the arena environment? Was the recovery of the working muscles inhibited by a low PaO_2 due to EIB? If training and performing in the arena environment produces EIB in hockey players, leading to a drop in the ventilation-perfusion ratio or diffusion limitations in the lungs, this could hamper the

transport of O_2 from the alveoli into the blood. This may result in EIH that can limit VO_2 max and have a negative impact on the recovery rate from intense exercise.

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CHAPTER 3

Effect of a Hockey Season on Pulmonary Function and Arterial Desaturation

3.1 Introduction

Exercise-induced bronchospasm (EIB) is defined as a decrease in lung function, usually characterized by a 10% fall in FEV_1 or a decrease of 20% in $FEF_{25-75\%}$ that occurs within 15 minutes after vigorous exercise (Virant, 1992). EIB can be triggered by intense exercise, cold dry environments, chronic asthma, or by a variety of air pollutants (Virant, 1992). EIB occurs in 90% of all asthmatics and in approximately 3-10% of athletes (Mahler, 1993). Research by Prevost-Craig *et al.* (1996) and Mannix *et al.* (1996) found a higher incidence rate of EIB among figure skaters than that of the typical athletic population. Also, Clark and Reid (1999) along with Leuppi *et al.* (1998) found a similar incidence rate among hockey players.

The bronchospasm caused by EIB results in an increased airway resistance which could reduce the ventilation-perfusion ratio in the lung and may lead to a drop in partial pressure of arterial oxygen content (PaO_2). As well, EIB can cause an increase in airway secretions along with increased vascular permeability which could produce a low grade pulmonary edema and may lead to an increased diffusion distance for O_2. These two factors are possible causes of exercise-induced hypoxemia (EIH). EIH has been most often observed in elite aerobic endurance athletes with very high maximal oxygen consumption levels and has been thought to limit performance (Dempsey *et al.*, 1984, Powers *et al.*, 1989).

Clark and Reid (1999) have suggested that the combination of environmental conditions such as temperature, humidity, air quality (including moulds and chemicals), along with the time spent practising and playing games in arenas, may contribute to EIB in hockey players. Consequently, EIB could result in a decrease in performance by reducing the ventilation perfusion ratio leading to EIH. Therefore, the purpose of this study was to determine if participation in competitive ice hockey and exposure to the type of environment that the game of hockey is played in, has a negative impact on physiological function. Spirometry was performed before and after a maximal oxygen consumption in which pulse oximetry was recorded. Testing was performed before and after a competitive season in the ice arena to evaluate the impact of this type of environmental exposure on physiological function. The hypothesis was that the combination of participation in competitive hockey and exposure to the type of environment in ice arenas has a negative impact on physiological function.

3.2 Subjects and Methods

Subjects consisted of volunteers from the University of Alberta varsity hockey team who were non-smokers and free of injury (n=16). All subjects completed an informed consent form prior to participation. The study was reviewed and approved by the Research Ethics Committee of the Faculty of Physical Education and Recreation.

In order to identify subjects with a prior history of asthma or atopic disease, all subjects were asked to complete a questionnaire developed by Clark and Reid (1999) (Appendix A). Body mass and height were recorded and percent body fat was obtained using the Yuhasz calculation (Yuhasz, 1966). All subjects completed a modified Wingate

anaerobic power test developed for hockey players on a Monark (Sweden) cycle ergometer. The subjects pedaled maximally against a resistance setting of 0.095 kg/kg body weight for 5 seconds followed by 10 seconds of submaximal cycling at 100 watts. This procedure was repeated for a total test time of 60 seconds (4* 5 s sprint: 10 seconds recovery). Maximal oxygen consumption (VO_2 Max) was determined by using a continuous incremental protocol to exhaustion on a Monark (Sweden) cycle ergometer. Subjects maintained a speed of approximately 75 rpm and resistance was increased by half a kilopond (kp) every two minutes until ventilatory threshold (VT) was reached as indicated by a decrease and plateau in VE/VCO_2 ratio prior to a systematic increase with increasing power outputs (Bhanbhani & Singh, 1985). Subsequently, resistance but not rpm was increased by half a kp, every minute until volitional exhaustion. During the test, expired gases where collected by a low-resistance, two way valve (Hans Rudolph 2700) and analyzed with a Horizon Metabolic Cart. Subject's heart rates were continuously monitored using a telemetry heart rate monitor system (Polar). Oximetry was measured with Biox IIa Oximeter (Ohmeda, Boulder, Colorado), to assess oxygen saturation at rest, during and after each test. As well, pulmonary function (FEV_1,FVC, FEF_{25-75}, and PEF) was measured using a Microloop 3535 Spirometer (Micro Medical Ldt., Novaitus Quedec, Canada) at rest, and at 1, 10, 15, 25 minutes following each test.

A monthly sample of arena air was analyzed for carbon monoxide, sulfur dioxide, nitrogen oxide, and nitrogen dioxide using a Miran IB analyser (Foxboro, Massachusetts, USA). The analyser was calibrated for each gas and then left to sample for 10 minutes inside the players box at ice level. A second sample of air was obtained three times

throughout the season using a Biotest Reuter Centrifugal Sampler (RCS) (RWR Scientific Inc., Ottawa, Canada) that was impacted onto Biotest rose bengal agar strips (RWR Scientific, Ottawa, Canada) for a complete airborne mold assessment. Samples were obtained from the dressing room, the ice surface and outside the arena as a control. The impacted agar strips were incubated for 5 days at 28 to 30°C (Siegler *et al.*, 1996). Micobial colonies were cultured and identified by the University of Alberta Microfungus Hervarium and Collection department.

Results were analyzed using a two-way analysis of variance for repeated measures (ANOVA). The Newnam Keuls test was used for post hoc comparisons. Results were deemed significant at the $p < .05$. A complete description of the experimental procedures is provided in Appendix B.

3.3 Results

Effect of hockey season on the physical fitness parameters:

The team had a mean age of 22.67 ± 1.5 years and height of 184.85 ± 8.6 cm. There was no significant difference between the body mass scores or the sum of six skinfolds before and after the season (Table 3-1). As well, there was no change in peak anaerobic power or in the percent drop off following the season (Table 3-1).

There was no significant change in absolute or relative VO_2 max scores (Table 3-2). There was a significant decrease in the resting arterial desaturation levels before and after the season (96.5 % ± 1.24 vs 95.2 % ± 1.74). There was a corresponding significant decrease between the arterial saturation levels at VO_2 max following the season (90.9 % ± 2.6 vs. 87.5 ± 1.2) (Table 3-2). There was no change in maximal heart rate or maximum

ventilation rate following the season.

Effect of hockey season on pulmonary function:

The presence of EIB (a decrease from baseline FEV_1 of at least 10% and/or 20% decrease in $FEF_{25-75\%}$) was observed in 5 of the 16 players (31%). Two of the sixteen players had positive responses to the questionnaire and were being treated for asthma. Both players used a beta-adrenergic aerosol metered dose inhaler to atenuate respiratory problems. One of these two players had no further pulmonary function problems while subject A (Table 3-3) still tested positive for EIB after the season. One other player tested positive for EIB at the beginning and end of the season (Subject B, Table 3-3) and had no prior history of asthma or allergies as determined by the questionnaire. Following the hockey season, 3 additional players tested positive for EIB (Subject C, D, E Table 3-3).

There was no change in the resting or the pulmonary function values (FVC, FEV_1,and $FEF_{25-75\%}$) obtained following the anaerobic power test (Table 3-6). The $FEF_{25-75\%}$ and PEF values did not change following the VO_2 max test either (Table 3-4). There was no change in FVC or FEV_1 1 and 10 minutes after the VO_2 max test. However after 15 and 25 minutes after the VO_2 max test, there was a significant decrease in FVC (5.69 L ± .71 vs. 5.38 L ± .80 and 5.73 L ± .64 vs. 5.37 L ± .90) but there was no significant change in FEV_1 (Table 3-4). There was a significant seasonal difference in the FEV_1 rest/ FEV_1 post VO_2 max ratio before and after the VO_2 max test. The 10, 15, and 25 minute ratios were all significantly lower then the 1 minute ratio. When the players who tested positive for EIB were excluded from the analysis, there was still a significant

decrease in FVC 15 and 25 minute post exercise (Table 3-5).

A Pearson correlation coefficient was performed to describe the relationship between EIH and EIB. Oxyhemoglobin saturaton (%SaO_2) at VO_2 max was significantly correlated before and after the season(r = .58). As well, the %SaO_2 was significantly correlated with the FVC obtained 10, 15 and 25 min post VO_2 max test (r = .57, .55, .57 respectively).

Air quality testing:

During the season, air temperature recorded at ice level ranged between 13-16°C and relative humidity ranged between 30-45%. Analysis of the air for SO_2, CO, NO, and NO_2 found no significant seasonal changes (Table 3-7). SO_2 was highest in September with a reading of 4.5 ppm and CO had a high reading in December with 5.6 ppm. There was only trace amounts of NO_2 found during the year and no NO was found in the arena air. Various species of molds found in the arena and outdoor air are listed in Table 3-8. In total, there were 20 different mold species found growing indoor and outdoor over the course of the season as measured pre, mid and post season. Of these, 8 different species were discovered in the dressing room over the course of the season. Three of the molds where also present in the outdoor sample at higher concentrations. Four of the species found where unique to the dressing room environment but where under 50 cfu/m^3. Eurotium amstelodami was the only species to be show an increase over the course of the entire season inside the arena. However, the levels were higher in the outdoor sample. Also, Eurotium amstelodami and Alternia alternata were the only species to be recorded a concentration of 50 cfu/m^3 or higher but again these high concentrations were in the

outdoor sample.

3.4 Discussion

It was hypothesized that the combination of participation in competitive hockey and exposure to the type of environment in ice arenas has a negative impact on physiological function. The findings of the study partially support this hypothesis. There was a significant reduction in arterial saturation levels at rest and during the maximal oxygen consumption test (VO_2 max) and evidence of an increase in EIB after the VO_2 max test following the season. However, there was no reduction in fitness parameters such as anaerobic power and the VO_2 max test as a result of these changes. In addition there was an increase in certain molds in and outside the arena and fluctuations in SO_2 throughout the season that could contribute to the increased incidence of EIB and EIH after the season. Thus it is plausible that the combined effects of participation in hockey over the season and the environmental conditions inside and outside the arena contributed to an impairment in pulmonary function in some players.

An important finding of this study was that the incidence of EIB among highly trained hockey players increased as the season progressed. At the begining of the season two out of the sixteen players (13%) tested positive for EIB. After the season, there were an additional three players who presented with EIB. This resulted in thirty one percent of the team experiencing EIB following a year's participation in hockey. These findings are consistent but higher than those of Clark and Reid (1999) and Leuppi *et al.* (1998) who found an EIA incidence rate of 23% and 19% in hockey players respectively. However their could be a difference in those who display EIB and those who would test positive

for EIA during a methacholine challenge test. As well, these finding are similar to those found in figures skaters and cross country skiers (Prevost-Craig *et al.*, 1996, Mannix *et al.*, 1996 and Larson *et al.*, 1993). The possible seasonal effect was explored by Larson *et al.* (1993) who found that there was no seasonal variation in bronchial responsiveness or lung function in cross country skiers. The latter researchers conducted their tests in the winter and the summer months, but no evaluations were made during the actual outdoor skiing environment. In the present study, spirometry was conducted in the arena both in early fall before the season started and in early spring when the hockey season was complete. It does seem that training and competition in ice arena environment may contribute to an increase in the incidence of EIB. As well, a significant drop in mean FVC for the entire team over the season at the 15 and 25 minute post VO_2 max test (Table 3-4), may provide evidence for an overall increase in bronchial hyperresponsiveness. The increased bronchoconstriciton results from a decrease in both the FEV_1 and the FEF_{25-75} (Table 3-4) 10, 15 and 25 min post VO_2 max test. There was a significant drop in the percent change in FEV_1 from the resting to the post VO_2 max FEV_1 after the season (Table 3-4). The combination of change in the large and small airway may account for the overall significant drop in FVC.

There have been a number of explanations for the high incidence of EIB in hockey players. One possible explanation is the environment that hockey players are exposed to during training and competition. Air temperature during the study ranged between 13- 16°C and the relative humidity was between 30-45%. There have been several studies that have addressed the correlation between the incidence of EIB and

respiratory heat and water loss (Mcfadden *et al.*, 1982, and Anderson *et al.*, 1982). The cold, dry air combined with several months of high-intensity exercise may provide a sufficient stimulus to trigger EIB. This type of air is also experienced outside the arena due to the winter conditions experienced by living in a northern Canadian city. However, other environmental factors may also play a role in EIB. Previous studies have shown an increase in airway resistance or a decrease in FEV_1 with exposure to NO_2, NO, and SO_2 (Bauer *et al.*, 1986 and Pierson *et al.*, 1992). In the present study, there were only trace amounts of NO_2 found throughout the year and no trace of NO (Table 3-7). However, concentrations of SO_2 were found to range from .3 to 4.5 ppm throughout the season. Peden (1998) noted that some asthmatics experience pulmonary function problems with SO_2 concentrations of 0.25 ppm while others have no problems at concentrations of 2.0 ppm. Pierson *et al.*, (1992) observed that cold dry air exacerbates the bronchoconstriction effect of SO_2 exposure. Work by Hackney *et al.*, (1984) found that exercise and SO_2 had an additive effect on asthmatics. The combination of a cold, dry environment, high intensity exercise and SO_2 exposure may also contribute to the increased occurrence of EIB in hockey players.

Another suggestion has been the possible presence of colonization of molds and fungi in the ice arena. Molds can produce mytoxins that can be triggers of bronchoconstriction (Platt *et al.*, 1989). Alternria alternata and Eurotium amstelodami were found periodically in the dressing rooms and in the arena but there was also a substantial amount found in the outdoor sample. According to recommendations by Health Canada, as long as airborne species found are qualitively similar and

quantitatively lower than outdoor air there is no cause for further assessment. Alternria alternate has been found to grow at low temperatures (1-10°C), so no continued colonization throughout the year suggests that the levels found inside were from the outdoors (Siegler *et al.*, 1996). Species of penicillium viridicatum are known to produce myotoxins which could irritate the respiratory tract, were found in the dressing room at the beginning of the season (Solley and Hyatt, 1989). However, no further colonization occurred throughout the season suggesting that it was not a contributor to the decreasing pulmonary function throughout the season. None of the species found indoors had counts over 50 cfu/m^3 which is the maximal allotment under Health Canada's Guide for indoor air quality. However, Health Canada's Guide lines for indoor air quality may be set with normal human activity and not humans exercising. So the lower levels of molds present may have an effect on individuals who are exercising. As well, even though no one mold was discovered in high enough quantities to contribute to increase pulmonary impairment, the additive effect of all the molds in the environment may be enough of a stimulus to cause increased impairment. The results suggest that there is the potential for indoor mold growth, and the predominant types found in the arena were also found in the outdoor environment suggesting that there was no unique exposure in the arena environment to these molds during the present study. However, it is possible that the combination of mold growth in the playing environment (arena) and living environment (outdoor) may be additive in its influence on pulmonary events.

One of the purposes of the study was to determine if there was a link between impaired respiratory function and incidence of EIH as the hockey season progressed.

There was a significant decrease in both the resting and the maximal $\%SaO_2$ during the season. EIH was determined by a decrease of 4% or more from resting oximetry values (Powers *et al.*, 1989) . At the start of the season there was a mean drop in $\%SaO_2$ of 5.6% (Table 3-2) which corresponded to ten players experiencing some form of EIH. At the conclusion of the season there was a mean drop in $\%SaO_2$ of 7.7% in fifteen players. Previous studies have found approximately 50% of endurance athletes tested experienced EIH (Powers *et al.*, 1989, Dempsey *et al.*, 1984). This higher incidence rate among hockey players provides evidence of some kind of pulmonary limitation that may be operating as a result of the environment that they practice and compete in and this may be different from the elite endurance athletes in the latter studies (Powers *et al.*, 1989, Dempsey *et al.*, 1984). The drop in resting $\%SaO_2$ may be explained by the effect of prior exercise and the environment on arterial desaturation.

There are two possible links between EIB and EIH. The bronchospasm caused by EIB results in increased airway resistance which could reduce the ventilation-perfusion ratio in the lung and may lead to a drop in partial pressure of PaO_2. During the preseason testing there was a significant correlation between the degree of arterial desaturation and the post exercise FVC's. However, there was no significant correlation following the season. The difference could be in the concentrations of SO_2 found in the arena at the time of testing. During the preseason testing the concentration of SO_2 was higher compared to levels found after the season (Table 3-7). The lack of correlation of arterial desaturation and FVC's is supported by Awe *et al.* (1999) who found that individuals with the greatest post exercise airflow restrictions had the highest SaO_2 during maximal

exercise. EIB causes an increase in airway secretions along with increased vascular permeability which could cause a low grade edema and may lead to an increased diffusion distance for O_2. A study by Hopkins *et al.* (1997) found that capillary walls can fail under the increased blood pressure brought on by intense exercise. During maximal exercise a combination of increased blood pressure and the increase in vascular permeability induced by EIB could result in the development of EIH.

3.5 Summary

The increase in incidence rate of EIB along with the overall increase in bronchial hyperreponsiveness over the course of the season supports the hypothesis that participation in a season of hockey has a negative impact on physiological function. As well, the same seasonal effect on arterial saturation levels also suggests that the environment the players compete and live in may have a negative impact on pulmonary function. However, the type of training that occurred throughout the season could also have an effect on arterial saturation levels. The cold, dry air and presence of some molds inside and outside the arena and the presence of SO_2 in the arena air combined with intense exercise during practices and competition throughout the season may trigger pulmonary impairment and a decrease in sport performance. However, it is important to note that there were no impairments in lab fitness test scores suggesting that the effect may not be too serious or may be of an acute transitory nature.

Table 3-1. Body composition and anaerobic power before and after a season of hockey.

	Pre Season	**Post Season**
Weight (kg)	89.0 ± 8.34	89.1 ± 8.00
Sum of Skinfolds (mm)	69.18 ± 28.8	67.7 ± 23.2
Percent Fat (%)	9.9 ± 2.3	10.1 ± 2.3
Peak 5 sec (W)	1124.7 ± 121.7	1107.5 ± 138.4
Peak 5 sec. (W/kg)	12.6489 ± 0.88	12.62 ± 0 .99
Percent drop off (%)	26.72 ± 6.55	27.13 ± 7.20

Values are means ± SD.

Table 3-2. Oxygen consumption before and after a hockey season.

Variable	Pre Season	Post Season
VO_2 Max (L/min.)	4.8 ± 0.45	4.9 ± 0.43
VO_2 Max ($ml \cdot kg^{-1} \cdot min^{-1}$.)	54.0 ± 5.2	55.1 ± 5.4
Max Heart Rate (b/min.)	188 ± 12	186 ± 12
Max Ventilation (L/min.)	192.5 ± 23.3	188.4 ± 23.2
Resting Arterial Saturation (%SaO_2)	96.5 ± 1.24	95.2 ± 1.74*
Arterial Saturation (%SaO_2) at VO_2 max	90.9 ± 2.6	87.5 ± 1.2*

Values are means ± SD. * Denotes significant difference from pre season, $p < 0.05$.

Table 3-3. Percent drop in FEV_1 and FEF_{25-75} pre and post season for 5 asthmatic players.

		Pre Season				Post Season			
Subjects	Measure	1 min.	10 min.	15 min.	25 min.	1 min.	10 min.	15 min.	25 min.
A	FEV_1(%)	8.92	13.15	8.92	-4.38	-2.98	9.4	5.28	8.02
	$FEF_{25-75\%}$ (%)	33.9	35.68	4.44	22.98	-8.8	20.46	3.82	5.35
B	FEV_1 (%)	-0.9	-1.32	1.1	11.57	-1.97	-17.98	32.3	22.04
	$FEF_{25-75\%}$ (%)	-57	-6.09	-27.4	29.27	16.89	-8.801	38.07	2.317
C	FEV_1 (%)	-4.6	-6.09	-5.89	-1.27	-12.9	1.18	5.1	12.85
	$FEF_{25-75\%}$ (%)	-27	-9.79	-4.97	-7.92	-31	6.56	8.38	11.11
D	FEV_1 (%)	-14	-2.45	-1.78	8.02	-11.2	3.23	4.96	14.34
	$FEF_{25-75\%}$ (%)	-1.8	-1.96	1.8	-4.47	-25.4	6.41	6.92	6.15
E	FEV_1 (%)	-9.8	-4.47	-3.83	4.84	-1.66	6.02	7.47	10.82
	$FEF_{25-75\%}$ (%)	-3.3	-3.5	-0.97	-2.72	-1.94	7.13	7.34	10.58

Table 3-4. Pulmonary function following the VO_2 max test pre and post season for all subjects.

	Pre Season					Post Season				
Measure	Rest	1 min.	10 min.	15 min.	25 min	Rest	1 min.	10 min.	15 min.	25 min.
FVC (L)	5.59 ± .68	5.63 ± .57	5.67 ± .68	5.69 ± .71	5.73 ± .64	5.57 ± .67	5.59 ± .62	5.44 ± .74	5.38* ± .80	5.37* ± .90
FEV_1 (L)	4.93 ± .59	5.21 ± .66	5.02 ± .64	4.90 ± .63	4.94 ± .64	4.91 ± .60	5.09 ± .66	4.87 ± .58	4.64 ± .76	4.77 ± .76
FEV_1/ FEV_1 (%)		-5.15 ± 5.69	-1.46‡ ± 5.23	-.67‡ ± 5.00	4.58‡ ± 7.38		-3.63*‡ ± 5.49	.93*‡ ± 6.45	6.02*‡ ± 8.54	7.03*‡ ± 7.07
$FEF_{25-75\%}$ (L)	5.73 ± .78	6.22 ± 1.40	5.48 ± 1.22	5.27 ± 1.02	5.17 ± 1.35	5.35 ± .98	5.98 ± 1.35	5.26 ± .92	4.91 ±1.16	4.09 ±1.06
$FEF_{25-75\%}$/ FEF (%)		-8.85 ± 22.4	3.28 ± 14.9	4.14 ± 15.6	9.02 ± 17.5		-10.95 ± 13.0	-3.0 ± 25.6	5.59 ± 11.1	3.18 ± 5.92
PEF (L)	606.6 ± 84.3	630.7 ± 89.8	555.0 ± 100.6	586.9 ± 92.4	562.6 ± 115.0	607.7 ± 89.3	638.4 ± 95.6	596.0 ± 127.1	558.8 ± 121.6	571.9 ± 116.2
PEF/PEF rest (%)		-4.46 ± 11.6	9.20 ± 9.8	3.13 ± 11.7	7.03 ± 14.3		-5.52 ± 10.9	2.26 ± 17.0	7.10 ± 13.6	5.88 ± 13.0
FEV_1/ FVC (%)	87.6 ± 4.6	91.5 ± 5.3	87.8 ± 5.4	86.7 ± 5.2	86.1 ± 8.1	87.7 ± 5.4	90.2 ± 6.5	88.7 ± 5.7	85.9 ± 6.9	88.1 ± 5.34

Values are means ± SD.

* Denotes significant difference from pre season, $p < 0.05$.

‡ Denotes significant difference from 1 minute value, $p < 0.05$.

Shading indicates no data required.

Table 3-5. Pulmonary function after the VO_2 max in all non EIB subjects.

	Pre Season					Post Season				
Measure	Rest	1 min.	10 min.	15 min.	25 min.	Rest	1 min.	10 min.	15 min.	25 min.
FVC (L)	5.65 ± .78	5.72 ± .65	5.75 ± .78	5.66 ± .80	5.77 ± .72	5.64 ± .69	5.63 ± .67	5.61 ± .79	5.48* ± .69	5.56* ± .76
FEV_1 (L)	4.99 ± .66	5.25 ± .71	5.06 ± .72	4.91 ± .69	4.98 ± .66	5.03 ± .55	5.17 ± .59	4.98 ± .62	4.86 ± .50	4.98 ± .60
FEV_1 % (%)		-5.19 ± 5.94	-.98*‡ ± 6.02	.14*‡ ± 5.03	4.73*‡ ± 7.18		-2.70 ± 4.55	2.00*‡ ± 3.14	2.07*‡ ± 4.25	3.71*‡ ± 3.88

Values are means ± SD.

Shading indicates no data required.

* Denotes significant difference from pre season, $p < 0.05$.

‡ Denotes significant difference from 1 minute value, $p < 0.05$.

Table 3-6. Pulmonary function following the anaerobic power test pre and post season in all subjects.

	Pre Season					Post Season				
Measure	Rest	1 min.	10 min.	15 min.	25 min.	Rest	1 min.	10 min.	15 min.	25 min.
FVC (L)	5.66 ± .71	5.78 ± .69	5.66 ± .97	5.74 ± .89	5.74 ± .85	5.82 ± . 86	5.90 ± .74	5.76 ± .93	5.89 ± .85	5.79 ± .92
FEV_1 (L)	4.86 ± .54	5.16 ± .50	4.90 ± . 72	4.84 ± .70	4.89 ± .72	4.93 ± .60	5.09 ± .70	4.85 ± .84	4.91 ± .80	4.87 ± .83
FEV_1 % (%)		-6.39 ± 4.16	-.96 ± 10.7	.937 ± 6.19	-.62 ± 10.1		-2.96 ± 3.16	2.59 ± 7.26	.96 ± 7.17	1.78 ± 8.55
PEF (L)	609.4 ± 102	646.2 ± 105	603.4 ± 116	585.5 ± 114	578.2 ± 118	608.8 ± 88	651.2 ± 104	580.7 ± 108	598.0 ± 85	595.1 ± 116
PEF/PEF rest (%)		-6.84 ± 13.6	1.57 ± 14.5	4.33 ±19.3	4.25 ±15.1		-5.12 ± 14.1	5.10 ± 16.0	.78 ± 18.0	3.70 ± 18.8
FEV/FVC (%)	86.2 ± 7.3	89.5 ± 4.7	84.9 ± 8.1	85.2 ± 4.9	86.3 ± 5.0	85.2 ± 6.5	86.3 ± 5.1	84.1 ± 6.0	84.1 ± 5.6	84.4 ± 5.9

Values are means ± SD. Shading indicates no data required.

Table 3-7. Concentration of selected gases found in the ice arena throughout the season.

Date	SO_2	CO	NO	NO_2
Sept.	4.5	1.2	0	0.1
Oct.	1.1	1.4	0	0
Nov.	1.2	1.6	0	0.3
Dec.	0.7	5.6	0	0
Jan.	0.3	0.8	0	0
Mar.	0.9	1.6	0	0

All gases are in ppm.

Table 3-8. Mold species present and there levels of fungal colony during the course of a hockey season.

	Pre-Season Levels (cfu/m^3)			Mid-Season Levels (cfu/m^3)			Post-Season Levels (cfu/m^3)		
Molds	**Dressing Room**	**Ice Surface**	**Outside**	**Dressing Room**	**Ice Surface**	**Outside**	**Dressing Room**	**Ice Surface**	**Outside**
Alternria alternata	25 (26%)	13 (22%)	99 (75%)						13 (5%)
Epicoccum nigrum	25 (26%)	13 (22%)							
Penicillium chrysogenum	25 (26%)								
Penicillium viridicatum	19 (20%)								
Claudosporium sphaerospermum		6 (11%)							
Ulcladium consorticale		6 (11%)	13 (10%)		6 (33%)				
Acremonium persicinum		6 (11%)							
Yeast		6 (11%)							
Bacteria		6 (11%)							

Eurotium amstelodami			13 (10%)	19 (75%)		90 (60%)	25 (80%)	19 (100%)	154 (60%)
Sterile basidiomycete			6 (5%)						
Scopulariopsis brumptii				6 (25%)					
Rhizomucor pusillus					6 (33%)		6 (20%)		
Myceliophthora lutea					6 (33%)				
Scopulariopsis brevicaulis						37 (25%)			6 (2%)
Aspergillus versicolor						8 (5%)			
Aspergillus Fumgatus						8 (5%)			6 (2%)
Emericella nidulans						8 (5%)			26 (10%)
Unidentified Coelomycete									44 (17%)
Penicillium sp.									6 (2%)

Blank spaces denote no presence of mold species.

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CHAPTER 4

GENERAL DISCUSSION AND CONCLUSION

4.1 Discussion

The findings from the present study seem to support the hypothesis that participation in ice hockey has some negative impact on physiological function as evidenced by a decrease in lung function over the course of the season. The results showed a significant decrease in mean FVC at the 15 and 25 min after the VO_2 max test (Table 3-4). There was a seasonal decrease in the FEV_1 rest/ FEV_1 post VO_2 max ratio resulting in a change from general state of bronchial dilation to bronchial constriction after the course of a hockey season. As well, the results also showed that this ratio changed significantly from a 3.63% bronchodilation immediately following the VO_2 max test, to a 7.03% bronchoconstriction 25 minutes later. This resulted in an overall 10.63% decrease in lung function following the VO_2 max test after the season.

Following the season of hockey, 31% (n = 5) of the team tested positive for EIB. This indicated that a third of the team had possible pulmonary function limitations to performance. However, this was limiting to some of the laboratory fitness tests. In the present study, two players were being treated for asthma. One of the players (Subject A) continued to have EIB based on spirometry suggesting that his medical treatment was unsatisfactory (Table 3-3). Spirometry testing revealed four other athletes with EIB but none of these had positive responses on the pre testing screening questionnaire (Table 3-3). This points to the need for other forms of screening of these athletes as recommended by other studies conducted on ice arena participants (Clark and Reid, 1999, Mannix *et al.*,

1996, Provost-Craig *et al.*, 1996 and Larson *et al.*, 1993). Studies by Clark and Reid (1999) and Mannix *et al.*, (1996) have found that questionnaires may not be the solution. Clark and Reid (1999) suggested that questionnaires are adequate to register athletes who are known asthmatics but found that three out of the five players diagnosed with asthma were not detected by the questionnaire. Mannix *et al.*, (1996) found that few of the figure skaters who experienced EIB reported any history of breathing difficulties. However, many of the individuals revealed significant symptoms with exercising upon close personal questioning. Both studies agree that the questionnaires for detection of EIB/EIA are limited and athletes such as hockey players and figure skaters should also undergo screening for EIB in an ice arena environment. Mannix *et al.* (1996) also suggested that the poor ability of the questionnaires to detect EIB may be due to elite athletes reluctance to complain of symptoms which could be conceived as "psychological" or as not having an obvious basis in physical disability. The questionnaire approach maybe more useful in screening for EIB in recreational settings or other sporting environments where these perceptions may not be as negatively viewed.

A close examination of the individual responses to the spirometry measures after the VO_2 max test revealed two different patterns of EIB (Table 3-3). Subject A and B, who had EIB at the start of the season as well as after the completion of the season, had significant decreases in both FEV_1 (>10%) and $FEF_{25-75\%}$ (>20%). Subject A was identified as being treated for EIA by the pre test screening questionnaire, but still experience EIA. This indicates that the medication used to control asthma was not protecting him while he was exercising in the arena. The three players who tested postive

for EIB at the end of the season showed a significant decrease in FEV_1 but not in $FEF_{25-75\%}$. This may indicate an increase in seasonal obstruction in the large and not in the small airways of the lung.

It is important to discuss the effect of team performance when 31% of the players on the hockey team experienced EIB near the end of the season. The players who comprise the 31% could be key players on the team. This may possibly limit the number of shifts a player has and/or the length of the shift. However, because there was no pulmonary impairment found after the anaerobic power test suggesting one hockey shift may not be enough of a stimulus to cause EIB. As well the main pulmonary impairment found was after the VO_2 max test which may be at a greater intensity then players experience during a hockey shift. Cox *et al.* (1995) found players were above threshold heart rate for only 6 minutes of playing time out of the entire game. So perhaps a series of repeated shifts may be enough of a stimulus to cause EIB. EIB may obstruct the adequate recovery ability of the players by perhaps a lower SaO_2 and EIH, resulting in more time required for the player to recover from the shifts. If this fatigue accumulates, the performance in subsequent shifts and perhaps games could be impaired resulting in chronic fatigue as described by Green (1994, 1987).

In normal subjects there is airway bronchodilation during exercise. However after high intensity exercise when the exertional levels are maximal and the minute ventilations are very high, there has been mild bronchoconstriciton detected (Farley *et al.*, 1988). In asthmatic subjects there are similar improvements in airway function during exercise. Beck *et al.* (1994) monitored the airway response during an incremental

protocol similar to that used in a VO_2 max test and found a progressive bronchodilation to maximal exercise immediately followed by bronchoconstriction. This tends to agree with the initial bronchodilation in most subjects 1 minute after the VO_2 max test which was followed by a bronchoconstriction after 10 minute post, described by a drop in FEV_1 and $FEF_{25-75\%}$ (Figure 3-2,3-3). Beck *et al.* (1994) also found that during exercise that varied in intensity, (speed or grade was changed) there was significant bronchodilation during the intense phase of the exercise. When the intensity of exercise was lowered, there was a significant drop in both FEV_1 and FEF_{50}. This reduced lung function during the lower intensity exercise could be the mechanism which would affect the performance of the hockey player. The intermittent bouts of maximal intensity followed by recovery on the bench could be enough of a stimulus during the game to cause a player's performance to decrease from EIB and perhaps contribute to the chronic muscular fatigue.

The possible drop in lung function during variable exercise does not explain the drop in arterial desaturation found during the VO_2 max test. As Beck *et al.*, (1994) reported, there is a steady bronchodilation until a maximum and then bronchoconstriction. This may be where the environment has an effect on the exercise response. The SO_2 and cold, dry air may produce an abnormal response during exercise which causes an increase in airway resistance or a diffusion limitation caused by the increase in vascular permeability accompanying the bronchoconstriction. This could result in an increase in the diffusion distance for O_2 which could result in a decrease in arterial desaturation.

It is also important to note that while the players as a group experienced significant arterial desaturation, there was no change in the maximal oxygen consumption scores of the team. The increasing desaturation over the course of the season could have a negative impact on their VO_2 max scores. Powers *et al.*, (1989) found for every 1% of arterial desaturation experienced there is a corresponding 1% decrease in VO_2 max. There was a 3.4% drop in maximal desaturation from pre to post season. Using the calculations of Powers *et al.*, (1989) the final season VO_2 max scores would have been 57.0 $ml \cdot kg^{-1} \cdot min^{-1}$ instead of 55.1 $ml \cdot kg^{-1} \cdot min^{-1}$. If we were to compare the preseason score of 54.0 $ml \cdot kg^{-1} \cdot min^{-1}$ with the corrected value of 57.0 $ml \cdot kg^{-1} \cdot min^{-1}$ based on the decrease in SaO_2, there would have been a gain over the season. This suggests that the chronic fatigue suggested by Green (1987, 1994) may in part be EIH.

The studies by Dempsey *et al.*, (1984) and Powers *et al.*, (1989) found that about half of the endurance athletes tested experienced EIH. In the present study there was 10 out of 16 (63%) players that experienced EIH. After the season there was 15 out of the 16 (94%) players that experienced EIH. The dramatic difference in the results between the studies suggests that hockey players may be more vulnerable to EIH than endurance athletes previously reported (Dempsey *et al.*, 1984 and Powers *et al.*, 1989). The environment and the type of intense exercise is different from the endurance sports reported by Dempsey *et al.*, (1984) and Powers *et al.*, (1989) and may not be the same mechanism for the incidence of EIH in elite endurance athletes. The effect EIB has on the lungs are similar to those proposed by Dempsey *et al.* (1984) to cause EIH. EIB causes an increase in airway secretions along with an increased vascular permeability and

could result in a low grade edema leading to an increased diffusion distance for O_2. Similarly, Dempsey *et al.* (1984) suggested an increase in diffusion distance in the lungs could be a cause of EIH. Hopkins *et al.* (1997) found that capillary walls can fail under the increased blood pressure brought on by intense exercise in endurance athletes. The role of inadequate ventilation has also been suggested as a mechanism of EIH. Powers *et al.* (1992) and Dempsey *et al.* (1984) found that ventilation did not play a role in the development of EIH. Awe *et al.* (1999) found that individuals with the greatest post exercise airflow restrictions had the highest SaO_2 during maximal exercise. The increase in airway resistance experienced by hockey players in the arena enviroment may cause a further obstacle for O_2 to overcome in order to reach the blood and may be the cause of the greater incidence of EIH.

4.2 Conclusion

The increase in incidence rate of EIB along with the overall increase in bronchial hyperresponsiveness over the course of the season supports the hypothesis that participation in a season of hockey has some negative impact on pulmonary function. As well, the same seasonal effect on arterial saturation levels also points to some negative impact on physiological function. It is still unclear whether there is a direct connection between the decrease in lung function and the decrease in $\%SaO_2$ over the season. However, the proposed mechanism of EIB agrees with the same mechanisms forwarded as a potential cause of EIH. The cold, dry air experienced in and out of the arena, the presence of SO_2 in the arena air, the presence of some molds in and out of the arena in combination with intense exercise during practices and competition throughout the

season may trigger pulmonary impairment. Despite these negative responses observed after the season, performance of laboratory fitness tests was not decreased. Nor was there significant increase in fitness which may also suggest some negative effect of the impaired pulmonary function on the lab tests. Whether hockey performance was hindered is not known.

It would appear that evidence from the present study and other studies involving athletes involved in winter sports atest to the need for pulmonary function assessments to be conducted in the competition and/or training environment (Clark and Reid, 1999, Mannix *et al.*, 1996, Provost-Craig *et al.*, 1996 and Larson *et al.*, 1993). The great number of undiagnosed asthmatics and exercise-induced asthmatics in winter sports points to the need for an effective screening program for athletes involved in such sports as hockey, figures skating, and cross country skiing. As well, a program should involve follow up testing to ensure individuals with EIA are being treated successfully. With effective treatment these athletes have an increased chance of performing up to their potential. Coaches, parents, and athletes themselves should be informed of the symptoms of EIB, or EIA. As well, the ice arenas used for recreational and minor league programs should be checked for airborne mold and chemical compounds found in the air to ensure that safe levels are maintained throughout the season.

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APPENDIX A

QUESTIONNAIRE

NAME_____

	YES	NO
1. Have you ever received treatment for asthma?		
2. Are you currently taking asthma medication?		
3. Are you currently taking allergy shots?		
4. Are you currently taking antihistamine tablets?		
5. Are you currently taking decongestants?		
6. Have you ever received treatment for eczema?		
7. Do you suffer from allergies?		
8. Have you every missed school or work because of chest tightness/cough/wheezing or porlonged shortness of breath?		
9. Do you smoke tobacco?		
10. Do you ever have chest tightness?		
11. When you exercise do you often have chest tightness?		
12. Do you cough or wheeze after strenuous exercise?		
13. Do you ever experience "Locker Room" coughing after strenuous exercise?		
14. Do you suffer from frequent chest infections during the competitive seasons?		
15. Do you become easily "winded" during pre-season conditioning drills, or out of shape by mid-season?		

APPENDIX B

Physiological Testing

Anthropometric Measurements

Standing height was measured by placing a metric measuring tape vertically against a wall. The subject where instructed to stand against the wall without footwear with heels together and in contact with the wall. A plane was placed on the head depressing the hair while the subject took a deep breath. A mark was made at the level of the lower border of the plane on the wall for measurement of height in cm (± 0.1cm).

Weight was measured by having the subject stand on a Heathometer beam (Continental Scale Corporation, USA) scale without foot wear and recorded in kilograms (± 0.1 Kg).

Skinfold thickness was measured using Harpenden skinfold calpers (Batz International, UK). The Yuhasz method was used consisting of the following six skinfold sites: triceps, subscapular, iliac crest, abdominal, chest and front thigh (Yuhasz, 1966). The skinfolds sites were first marked with a pen and then the fold was raised between the thumb and index finger at the mark. The calpers were placed at right angles to the fold. Readings were done at all six sites and then repeated a second time and recorded in millimeters (± 0.2). If there was a discrepancy greater then 0.4 between the two reading at a skinfold site a third was taken and a mean of the closest two reading was taken as the skinfold thinkness. Percent body fat was calculated using the following formula (Yuhasz, 1966):

$$\% \text{ Body Fat} = (\text{sum of six skinfolds} \times 0.097) + 3.64$$

Aerobic Fitness

Maximal oxygen consumption (VO_2 max) was determined by using a continuous incremental protocol to exhaustion on a Monark cycle ergometer (Varberg, Sweden). The subjects pedaled at 75 rpm at a resistance equal to 1.5 kp and progressed every two minutes by half a kp (37 w) until ventilatory threshold was reached as indicated by the plateau in VE/VCO_2 ratio prior to a systematic increase with increasing power outputs (Bhanbhani & Singh, 1985). Subsequently resistance was increased every minute until VO_2 max was achieved. VO_2 max was indicated by a < 100 ml/min change in VO_2 with continued exercise prior to volitional exhaustion. Additional supportive criteria included: a max heart rate of 10% more or less of age predicted maximum; respiratory exchange ratio (RER) > 1.1 was observed; and/or subject was to fatigued too continue exercise. During the test, expired gases where collected by a low-resistance two way valve (Hans Rudolph 2700) and analyzed by a Horizon Metabolic Cart (Anaheim, Ca). The metabolic cart was calibrated before and checked after each test using known gas concentration (Prax Aito, Edmonton, AB). Subjects heart rates where continuously monitored using a telemetry heart rate monitor system (Polar, USA, Inc., Stanford Connecticut) varified for accuracy against an ECG (Cambridge).

Pulmonary Function Tests

All subjects underwent a series of pulmonary function tests (PFT) to measure forced vital capacity (L), forced expired volume in 1 second (L), peak expiratory flow (L) and forced expiratory flow at 25-75% (L) of vital capacity that were measured using a Microloop 3535 Spirometer (Micro Medical Ldt., Novaitus, Quebec, Canada) Subjects wore a nose clip and where connected to the spirometer by way of a mouthpiece to perform three trials of maximum inspiration followed by maximum expiration followed by two trials of the maximum number of breaths that can be performed as rapidly and deeply as possible in 12 seconds. PFT was measured at rest prior to any exercise test and 1, 10,15, 25 minutes after the anaerobic power test and the VT/VO_2 max test. Exercise-induced bronchospasm was indicated by a decrease in forced expiratory volume at 1 s (FEV_1) or peak expiratory flow (PEF) of 10% or greater after exercise. Smaller airway bronchospasm was diagnosed by a 20% change in forced expiratory flow at 25-75% of vital capacity ($FEF_{25-75\%}$) (Mcfadden, 1995).

Arterial-Saturation

Oxyhaemoglobin desaturation ($\%HbO_2$) was measured by Biox IIa Oximeter (Ohmeda, Boulder, Colorado, USA). The finger probe and interface cables where secured to the subject and the bike so as to minimize movement artifact. The IIa Oximeter has been previously validated by Powers (1989) for measurement of arterial blood oxygen concentration under intense exercise conditions. The Oximeter has an internal calibration procedure which was performed before each test was conducted and checked after each test. The Oximeter was used during the VT/VO_2 max test.

Measurements where taken every minute for 5 minutes at rest, during VO_2 max and for 5 min after the tests. Exercise-induced hypoxemia was indicated by a decrease in $\%HbO_2$ of 4.0% or more below resting values during exercise (Powers *et al.*, 1988). Percent HbO_2 was calculated as followed $\%HbO_2$ = exercise $\%HbO_2$- rest $\%HbO_2$.

Anaerobic Power Test

Anaerobic power was assessed as peak 5 second power output (PO) and the mean of 4 repeats of 5 second intervals on a maximal Monark cycle ergometer. The test began with a 5 min warm-up on the cycle ergometer at a PO of 100 watts. At the end of the warmup the subject pedaled maximally against a resistance setting of 0.095 kg/kg body weight for 5 seconds followed by 10 seconds of submaximal cycling at 100 watts. This procedure was repeated for a total test time of 60 seconds (4* 5 s sprint: 10 seconds recovery). A fatigue index (%) was also calculated as follows:[(peak 5 s - lowest 5 s)/ peak 5s] * 100.

Air quality test

A sample of arena air was analyzed for carbon monoxide, sulfur dioxide, nitrogen oxide, and nitrogen dioxide using a Miran IB analyser (Foxboro, Massachusetts, USA). The analyser was calibrated for each gas and then left to sample for 10 minutes. A second sample of air was obtained using a Biotest Reuter Centrifugal Sampler (RCS) (RWR Scientific Inc., Ottawa, Canada) that was impacted onto Biotest rose bengal agar strips (RWR Scientific, Ottawa, Canada) for a complete airborne mold assessment. Samples were obtained from the dressing room, the ice surface and outside the arena. The impacted agar strips were incubated for 5 days at 28 to 30°C (Siegler *et al.*, 1996).

Enumeration of the colonies was performed using the following equation from the Biotest RCS Centrifugal air sampler instruction manual.

$$CFU = \frac{Colonies\ on\ Agar\ Strip\ x\ 25}{Sampling\ Time(Min.)}$$

Culturing and identification was perfomed by the University of Alberta Microfungus Hervarium and Collection department.

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