

**NEW INSIGHTS INTO RESPIRATORY MUSCLE FUNCTION
IN AN ATHLETIC POPULATION**

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Dissertation presented for the degree of
Doctor of Philosophy (Sport Science) at Stellenbosch University



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December 2008

DECLARATION

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SUMMARY

The aims of this study were (1) to determine the effect of concurrent respiratory muscle training (RMT) on respiratory muscle (RM) function and aerobic exercise performance in women competitive field hockey players, (2) to determine the effect and time duration of RM detraining on RM function in those who underwent RMT, and (3) to determine the predictors of RM strength and endurance in an athletic population.

Twenty two women hockey players underwent a series of kinanthropometric and respiratory muscle function measurements, and were then randomly assigned to an experimental group (EXP, n = 15) who underwent concurrent RMT, and a control group (CON, n = 7) who underwent sham training. Twenty subjects took part in the RM detraining study.

Significant improvements in pulmonary function and RM endurance (5 – 9%) were found in both groups after the HT-RMT and HT-ST interventions, while EXP also showed a significant improvement in RM strength variables (13% in MIP, 9% in MEP). $MEF_{50\%}$ was the only variable that showed a significant difference in the changes over time after 20 weeks of DT between EXP and CON. RM strength in both groups remained relatively unchanged over the DT period. RM endurance in both groups remained unchanged after 9 weeks of DT, but decreased significantly after 20 weeks of DT in EXP.

It was concluded that the intensity and duration of both the HT-RMT and HT-ST programmes were adequate to elicit training adaptations in the RM. In both groups there was a complete reversal in lung volumes after 9 weeks and a tendency of a reversal in RM endurance after 20 weeks of DT. It is suggested that a RMT programme should be incorporated every 9 weeks in the training schedule of field hockey players, to maintain improved RM function.

182 Subjects (160 athletes and 22 non-athletes) participated in the third part of the study. Men athletes had superior lung volumes, RM endurance and inspiratory muscle strength compared to men non-athletes, while women athletes showed superior RM strength

compared to women non-athletes. In the athletic population, the kinanthropometric and pulmonary function variables showed the strongest relationships with MVV ($r = 0.25 - 0.80$) followed by inspiratory muscle strength ($r = -0.18 - 0.56$) and weaker but statistically significant relationships with expiratory muscle strength ($r = -0.21 - 0.43$). Gender, mesomorphy and exercise sessions per week predicted 35% (SEE = 26.6 cmH₂O) of the variance in inspiratory muscle strength (MIP). Gender and mesomorphy predicted 24% (SEE = 28.3 cmH₂O) of the variance in expiratory muscle strength (MEP), while gender, relative sitting height, FEV₁ and PEF_R predicted 78% (SEE = 18.2 L.min⁻¹) of the variance in RM endurance (MVV).

Gender was the strongest predictor of RM function and kinanthropometry only added a small percentage to the prediction equations. Although the reference equations are still not adequate to predict RM strength, it does provide more suitable reference values than previously reported. The predicted values derived from the equation for MVV can be applied as adequate reference values for athletic populations of the same age range (17 – 34 years).

OPSOMMING

Die eerste doel van die studie was om die effek van respiratoriese spierinoefening (RSI) op respiratoriese spierfunksie (RS) en aërobiese kapasiteit in kompeterende vroue veldhokkiespelers te bepaal. Die tweede doel was om die effek en duur van respiratoriese dekontisionering (RDK) op RS te bepaal. Laastens is die voorspellers van respiratoriese spiersterkte en -uithouvermoë in 'n atletiese populasie bepaal.

Twee en twintig vroue hokkiespelers het 'n reeks kinantropometriese en respiratoriese toetse ondergaan en is daarna lukraak verdeel in 'n eksperimentele groep (EKS, $n = 15$) wat RSI ondergaan het, en 'n kontrole groep (KON, $n = 7$) wat plasebo inoefening ondergaan het. Twintig van hierdie hokkiespelers het aan die RDK-studie deelgeneem.

Albei groepe het statisties betekenisvolle verbeterings getoon in pulmonêre funksie en respiratoriese spieruithouvermoë (5 – 9%), terwyl die EKS ook 'n betekenisvolle verbetering in respiratoriese spiersterkte (13% in MIP, 9% in MEP) getoon het. $MEF_{50\%}$ was die enigste veranderlike wat 'n statisties betekenisvolle verskil tussen die twee groepe getoon het t.o.v. die verandering oor tyd na 20 weke van RDK. Respiratoriese spiersterkte het relatief onveranderd gebly in albei groepe gedurende die RDK tydperk, terwyl respiratoriese spieruithouvermoë betekenisvol afgeneem het in die EKS na 20 weke van RDK.

Die gevolgtrekking van die studie is dat die intensiteit en duur van die hokkie oefenprogram plus die RSI program en die hokkie oefenprogram plus plasebo inoefening doeltreffend was om inoefenings-aanpassings in die respiratoriese spiere te bewerkstellig. Na 9 weke van RDK was daar 'n totale omkering na die oorspronklike waardes in longvolumes en 'n tendens tot omkering in respiratoriese spieruithouvermoë. Dit word voorgestel dat die RSI-program elke 9 weke in die inoefenings-program van hokkiespelers ingesluit word om die verbeterde RS te behou.

182 Deelnemers (160 atlete en 22 nie-atlete) het aan die derde deel van die studie deelgeneem. Mans atlete het groter longvolumes, beter respiratoriese spieruithouvermoë en

beter inspiratoriese spiersterkte as mans nie-atlete getoon, terwyl vroue atlete beter respiratoriese spiersterkte as vroue nie-atlete getoon het. Die kinantropometriese en pulmonêre funksie veranderlikes het die sterkste korrelasies met respiratoriese spieruithou vermoë (MVV) getoon ($r = 0.25 - 0.80$), gevolg deur inspiratoriese spiersterkte (MIP, $r = -0.18 - 0.56$) en die swakste maar steeds betekenisvolle korrelasies met ekspiratoriese spiersterkte (MEP, $r = -0.21 - 0.43$). Geslag, mesomorfie en die hoeveelheid inoefenings-sessies per week het 35% (SEE = 26.6 cmH₂O) van die variansie in MIP voorspel, terwyl geslag en mesomorfie 24% (SEE = 28.3 cmH₂O) van die variansie in MEP kon voorspel. Geslag, relatiewe sithoogte, FEV₁ en PEF_R het 78% (SEE = 18.2 L.min⁻¹) van die variansie in respiratoriese spieruithou vermoë voorspel.

Geslag was die sterkste voorspeller van RS, terwyl kinantropometrie slegs 'n minimale bydrae tot RS gemaak het. Alhoewel die voorspelling-vergelykings nie voldoende is om respiratoriese spiersterkte te voorspel nie, bied dit wel 'n vergelyking wat meer relevant is vir 'n atletiese populasie as wat voorheen gerapporteer is. Die voorspelde waardes vir respiratoriese uithou vermoë, afgelei uit die voorspelling-vergelyking vir MVV, kan gebruik word as verwysingswaardes vir 'n atletiese populasie wat binne dieselfde ouderdoms-reikwydte val (17 – 34 jaar).

ACKNOWLEDGEMENTS

Thank you to all the subjects who willingly sacrificed their time and effort to participate in the study.

Thank you to my promoter, Prof. Elmarie Terblanche, for your excellent guidance and advice during the implementation of the research and your insightful suggestions, assistance and contributions to the preparation of this dissertation.

Thank you to the National Research Foundation of South Africa for your financial support through the NRF Prestigious Scholarship (2006 – 2008). Thank you to Stellenbosch University for the Postgraduate Merit Bursary (2006 – 2008) and the Ernst & Ethel Eriksen Trust (2006) for your financial assistance during the study. Thank you to *PowerLung*© Inc. (Houston, Texas, USA) for sponsoring the respiratory muscle training devices (*PowerLung*© – Sport), which made the implementation of the respiratory muscle training intervention possible. Opinions expressed and conclusions arrived at, are those of the author and do not necessarily reflect those of the above institutions.

Thank you to Prof. Martin Kidd, for your excellent knowledge and assistance with the statistical analyses. Thank you to Cecile Carsten, for your assistance with the data collection during the second part of the study.

Thank you to my parents, Albie and Helene Kroff, for your support throughout my years of education. I am forever grateful for your unconditional love, motivation, and acceptance of me, and all of my dreams and endeavors. Thank you to my partner, Carla, for your incredible love, invaluable motivation and support, and a very special thank you for your technical assistance with the preparation of this dissertation and the many sacrifices you selflessly made during this time. To God, thank you for blessing me with a healthy mind and the opportunities in life to learn. Thank you to my family and friends for your words of motivation.

DEDICATION

I dedicate this dissertation to Carla;
you are my inspiration and the treasure of my heart.

“The great use of life is to spend it on something that will outlast it”

- James Truslow Adams

LIST OF ABBREVIATIONS

ANOVA	:	analysis of variance
ATS	:	American Thoracic Society
BET	:	breathing endurance test
BMI	:	body mass index
BL	:	baseline
BSA	:	body surface area
BTPS	:	body temperature, ambient pressure, saturated with water vapour
C_{max}	:	highest possible pressure that could be maintained during an incremental resistive breathing test
cmH ₂ O	:	centimetres water
CMRT	:	concurrent respiratory muscle training
CMS	:	cervical magnetic stimulation
CNS	:	central nervous system
CON	:	control group
COPD	:	chronic obstructive pulmonary disease(s)
CR	:	coefficient of repeatability
CV	:	coefficient of variation
DT	:	respiratory muscle detraining of the present study
EELV	:	end expiratory lung volume
e.g.	:	for example
EMG	:	electromyography (electromyographic)
EMT	:	expiratory muscle training
EPTL	:	expiratory pressure threshold loading
ERS	:	European Respiratory Society
EX	:	exercise sessions per week
EXP	:	experimental group
FEV ₁	:	forced expiratory volume in 1 second (L)
FFM	:	fat free mass

FMEF _{25-75%}	:	mean forced expiratory flow during the middle half of the FVC
FRC	:	functional residual capacity
FT	:	fast-twitch
FVC	:	forced vital capacity (L)
HR	:	heart rate (bpm)
HT-RMT	:	hockey training plus respiratory muscle training
HT-ST	:	hockey training plus sham training
Hz	:	Hertz
ICC	:	intraclass correlation coefficient
i.e.	:	that is
IFRL	:	inspiratory flow resistive loading
IMT	:	inspiratory muscle training
IPTL	:	inspiratory pressure threshold loading
kcal.day ⁻¹	:	kilocalories per day
kPa	:	kilopascal
L	:	litre(s)
L.min ⁻¹	:	litres per minute
L.s ⁻¹	:	litres per second
LSD	:	least significant difference
m	:	metre(s)
MAP	:	mean arterial pressure (mmHg)
MEF _{50%}	:	maximum instantaneous forced expiratory flow where 50% of FVC remains to be expired.
MEP	:	maximum expiratory mouth pressure (cmH ₂ O)
MEP _{peak}	:	peak maximum expiratory mouth pressure (cmH ₂ O)
MESO	:	mesomorphic component
min	:	minute
MIP	:	maximum inspiratory mouth pressure (cmH ₂ O)
MIP _{peak}	:	peak maximum inspiratory mouth pressure (cmH ₂ O)
ml.kg ⁻¹ .min ⁻¹	:	millilitres per kilogram per minute

MMEF	:	maximum mid-expiratory flow
mmHg	:	millimeters mercury
MSVC	:	maximal sustainable ventilatory capacity
MVV	:	maximum voluntary ventilation (L.min ⁻¹)
n	:	number of subjects
N.m	:	Newton metres
P(A-a)O ₂	:	alveolar-arterial oxygen pressure difference
Pab	:	abdominal pressure
PAS	:	physical activity score
PCO ₂	:	partial pressure of carbon dioxide
Pdi	:	transdiaphragmatic pressure
Pdi,tw	:	transdiaphragmatic twitch pressure
PEFR	:	peak expiratory flow rate (L.s ⁻¹)
Pes	:	esophageal pressure
Pga	:	gastric pressure
pH	:	potential hydrogen
PI	:	post-intervention
PIFR	:	peak inspiratory flow rate (L.s ⁻¹)
PNS	:	phrenic nerve stimulation
Ppl	:	pleural pressure
PTL	:	pressure threshold loading
r	:	correlation coefficient
R ²	:	coefficient of determination
RBT	:	resistive breathing task
RM	:	respiratory muscle(s)
RMT	:	respiratory muscle training
RMTTP	:	respiratory muscle training programme of the present study
RPE	:	rate of perceived exertion
RV	:	residual volume
s	:	second(s)
SD	:	standard deviation

SEM	:	standard error of mean
ST	:	slow-twitch
TIRE	:	test of incremental respiratory endurance
TLC	:	total lung capacity
T_{lim}	:	exercise time to limitation/exhaustion
TRIMP	:	training impulse
VC	:	vital capacity
V_E	:	minute ventilation ($L \cdot min^{-1}$)
VIH	:	voluntary isocapnic hypernoea
VO_{2max}	:	maximum oxygen consumption ($ml \cdot min^{-1}$ or $ml \cdot kg^{-1} \cdot min^{-1}$)
vs.	:	against
W	:	Watts
wk	:	week

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CHAPTER ONE

INTRODUCTION

Historically, the lung and its diseases has been the focal point of interest in respiratory physiology research. Investigations into the role of the respiratory system during exercise in healthy individuals received little attention, since scientists believed that the respiratory system was actually “over-built”, compared to the rest of the oxygen transport system, to perform exercise. However, the theory by Dempsey (1986) illustrated that in highly trained individuals the respiratory system might become the limiting factor during exercise, since the respiratory system does not undergo training-induced adaptations to the same extent as the cardiovascular system and the locomotor muscles in order to increase their maximal work capacity. Due to this hypothesis more research on the respiratory system’s responses to exercise and the causes and consequences of these responses have been done in the past three decades.

In recent years, evidence of inspiratory (Volianitis et al., 2001b; McConnell et al., 1997; Babcock et al., 1996; Coast et al., 1990; Bye et al., 1984) and expiratory muscle fatigue (Taylor et al., 2006; Verges et al., 2006) have been reported after whole-body, high-intensity endurance exercise in healthy individuals of different fitness levels. Evidence of RM fatigue during exercise opened investigations into the effect of RM fatigue on athletic performance. This phenomenon was investigated by either inducing RM fatigue prior to exercise or partially unloading (with mechanical ventilator) the RM during exercise. During the past three decades these studies reported significant increases in exercise performance during partial unloading of the RM (Harms et al., 2000) and significant decreases in exercise performance after inducing RM fatigue prior to exercise (Taylor & Romer, 2008; Verges et al., 2007b; Harms et al., 2000; Mador & Acevedo, 1991; Martin et al., 1982).

The occurrence of RM fatigue during exercise and the limiting effect it had on exercise performance made coaches and scientists realize that interventions are needed to try and eliminate RM fatigue during high-intensity exercise. Previously, RMT interventions could

only be implemented in a laboratory setting, however, since the availability of hand-held, commercial respiratory muscle training (RMT) devices more thorough investigations into the ergogenic effect of RMT in sportsmen and –women became apparent. Studies that found no ergogenic effect of RMT can be attributed to methodological inadequacies (Fairbairn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987). The development of these specific RMT trainers made it possible for studies to incorporate more rigorous placebo-controlled study designs; and researchers took caution to conduct more reliable exercise performance tests that closely resemble real-life events. These RMT intervention studies found significant improvements in time trial performance in well-trained athletes (Johnson et al., 2007; Romer et al., 2002a; Volianitis et al., 2001b; Sonetti et al., 2001).

The majority of studies have investigated the ergogenic effect of RMT in endurance athletes (\pm 11 studies reported well-trained athletes) and only one study found a significant improvement in total recovery time during repetitive sprints in sprint athletes (Romer et al., 2002b). Thus, very little is known about the ergogenic effect of RMT in sports characterized as high-intensity, repetitive sprint sports. Field hockey is a high-intensity, repetitive sprint, team sport. Only two studies investigated the ergogenic effect of RMT in women athletes (Volianitis et al., 2001b; Wells et al., 2005). Thus, RMT and its ergogenic effect in women athletes competing in high-intensity, repetitive sprint sport were warranted.

Sonetti et al. (2001) was the first to investigate the effect of concurrent voluntary isocapnic hyperpnoea training and inspiratory threshold loading training on performance. Wells et al. (2005) investigated the effect of concurrent inspiratory and expiratory muscle training on exercise performance. The majority of pressure threshold loading training studies investigated the effect of inspiratory pressure threshold training on exercise performance and only one study investigated the effect of expiratory pressure threshold training on exercise performance (Suzuki et al., 1995).

New insights into concurrent inspiratory and expiratory muscle training in team sports and particularly in women will enable sport scientists and coaches to determine whether specific RMT will be beneficial to the fitness of team sport players and whether or not it needs to be

incorporated into their daily training programme. It will also reveal whether concurrent training would be more beneficial to improve performance as opposed to inspiratory or expiratory muscle training separately. It will also support the effect of RMT in women athletes specifically, which is a neglected area of research in the literature.

Romer & McConnell (2003b) are the only recently available study that investigated the effect of RM detraining. Although very little information on RM detraining is available, this phenomenon is extremely important to coaches and athletes. If RMT improves athletic performance, coaches should know how frequently their athletes need to undergo RMT to maintain optimal RM strength and endurance to positively influence athletic performance.

Since the 1960's studies have investigated the factors that determine RM strength and endurance in healthy individuals. However, only a few studies (Fuso et al., 1996; Cordain et al., 1987) determined those factors that influence the RM strength measurements in an athletic population. The determination of reference values for RM strength and endurance measurements in athletic populations will enable scientists to rate an athlete's RM strength and performance to a value that was derived from a similar population. From the comparison of predicted and observed values, scientists and coaches can determine whether an athlete has optimal RM strength and endurance or whether they need to undergo RMT to improve their RM strength and endurance and subsequently athletic performance.

CHAPTER TWO

RESPIRATORY MUSCLE FUNCTION AND EXERCISE

A. INTRODUCTION

Historically, the assessment and interpretation of the function of the respiratory system was investigated to diagnose respiratory weaknesses in patients with lung pathology. However, with the constant challenges placed upon sport scientists and exercise physiologists to determine those factors that limit superior athletic performance, the investigation into the role of the respiratory system during exercise in highly trained athletes have become more apparent.

The hypothesis by Dempsey (1986) states, that in an untrained, healthy individual, the pulmonary system is ideally designed and can easily regulate the homeostatic demands of exercise. Thus, the limiting factor for exercise performance lies within the other systems in the chain of oxygen transport and utilization, i.e. cardiac output, stroke volume, skeletal muscle vascularity and oxidative capacity of the locomotor muscles. He believed that the respiratory system did not improve its efficiency as an adaptation to regular training to the same extent as the cardiovascular and locomotor systems. Therefore, in highly fit individuals, the gas exchange capabilities of the lungs, the maximum chest wall response and the respiratory control system may become the limiting factor in exercise performance.

The unique structural and functional characteristics of the respiratory muscles in conjunction with the specific neural regulation of breathing by the respiratory control system, ensures that the capacity of these muscles for pressure generation usually exceeds the exercise demands placed upon them in moderately fit, healthy individuals. A brief explanation of this statement is given in the following two paragraphs as described by Romer & Polkey (2008):

The respiratory control system: The neural regulation of breathing is optimal during exercise. During moderate exercise the respiratory control system needs to regulate

ventilation to meet the metabolic requirements in order to maintain arterial blood-gas tensions and arterial pH near resting values. If exercise intensity is increased, a point will be reached for the onset of metabolic acidosis. When this happens, additional hyperventilation is required to minimize the drop in arterial pH and to prevent arterial hypoxemia. Simultaneously, the respiratory control system regulates ventilation and breathing frequency so that the work performed and subsequently, oxygen cost of the respiratory muscles, is minimized (Romer & Polkey, 2008).

The respiratory muscles: The respiratory muscles are specifically designed to deal with the increased ventilatory demands during exercise. The inspiratory muscles (mostly the diaphragm) have high oxidative capacities, a short capillary-to-mitochondrial diffusion path for oxygen and a velocity of shortening between that of fast-twitch and slow-twitch skeletal muscle fibers (Mizuno, 1991). The expiratory muscles are activated with increased exercise intensity, even though expiratory flow has not become limited. The activation of the expiratory muscles causes a decrease in end-expiratory lung volume (EELV) below resting values (Henke et al., 1988). A reduced EELV helps the inspiratory muscles during inspiration as follows: It increases tidal volume such that the respiratory system compliance remains high (Stubbing et al., 1980). It causes the diaphragm to be lengthened at the end of expiration which means the diaphragm is at optimal length to develop a greater force during inspiration. Lastly, the reduced EELV cause storage of elastic energy in the chest and abdominal walls during expiration. The stored elastic energy helps to carry a portion of the work required during inspiration that follows (Grassino et al., 1981). The activation of the accessory respiratory muscles during exercise of increasing intensities also helps to carry the load of work of breathing during increased ventilation.

The maximum ability of the respiratory muscles to generate adequate pressure gradients for air flow during exercise in the manner described above, can be referred to as the dynamic capacity of the respiratory system or the maximal dynamic pressure of the respiratory muscles.

This chapter elaborates on the characteristics and function of the respiratory muscles (RM) and the oxygen cost of RM work. The different methods for the assessment of RM function are briefly explained. Thereafter, the evidence of RM fatigue during exercise, the factors that could possibly be responsible for RM fatigue, as well as the implications of the assessment of RM fatigue are described. The last section of the chapter elaborates on the effect of RM fatigue on subsequent exercise performance, the methods to investigate this effect, and lastly, the possible mechanisms by which RM fatigue can limit performance.

B. THE RESPIRATORY MUSCLES

To fully understand the function of the respiratory system during exercise the function of the respiratory muscles (RM) and the demands placed upon them during exercise must be explained. The RM can be divided into inspiratory and expiratory muscles. Their characteristics (fiber types) are suited for their specific functions.

1. Characteristics and functions of the respiratory muscles

During rest, contractions of the diaphragm, the parasternal intercostals and external intercostals expand the chest cavity to induce air flow into the lungs (inspiration), while expiration is passive. However, whilst exercising, the accessory inspiratory muscles (sternocleidomastoids, scalenes, back and shoulder girdle) become active during inspiration and the expiratory muscles (*Rectus Abdominis*, internal and external obliques, *Transversus Abdominis*, internal intercostals) become actively involved during expiration in a proportional manner to the exercise intensity (Sheel, 2002).

EMG activity recordings showed that the diaphragm is recruited in proportion to increasing exercise hyperpnoea as exercise intensity increases. As exercise proceeds the diaphragm contributes less and less to the total pressure generated by the inspiratory muscles and the accessory muscles contribute more and more to the total pressure generated by the inspiratory muscles (Sheel, 2002).

The diaphragm contains approximately 60% slow twitch fibers (Polla et al., 2004) and has greater oxidative capacity and blood flow than most limb muscles. This is exactly the characteristics that the diaphragm needs, since it requires continuous activity throughout life and should therefore be resistant to fatigue. Human intercostal muscles also contain approximately 60% slow twitch fibers (Koulouris & Dimitroulis, 2001).

In contrast with the fibers of the diaphragm, the expiratory muscle has a higher proportion of fast twitch fibers than slow twitch fibers (Keens et al., 1978). Therefore, abdominal muscles (expiratory muscles) have a lower oxidative capacity and have less endurance (less resistance to fatigue) than inspiratory muscles (Uribe et al., 1992; Gandevia et al., 1983).

The fiber characteristics of the respiratory muscles are not fixed. Specific training, hypoxic conditions, age, respiratory diseases and pharmacological agents (B₂ agonists and corticosteroids) can modify respiratory muscle fiber properties (Secher et al., 1984). Studies in animals have shown that specific endurance training elicits a small but significant (20 – 30%) increase in mitochondrial enzyme activity and resting glycogen levels (Powers et al., 1994; Grinton et al., 1992; Powers et al., 1990). Vrabas et al. (1999) also found a significant decrease in type IIb and an increase in type I myosin heavy chains in rat diaphragms after 10 weeks of treadmill exercise training. These enzyme and fiber type changes increased the diaphragm's resistance to fatigue.

2. The energy demands of the respiratory muscles

To maintain a higher work rate the energy demands for locomotor muscle contractions increase as exercise intensity increases. The same happens with the energy demands for respiratory muscle contraction since the respiratory muscles have to work harder to maintain the higher levels of ventilation during exercise. The factors determining the energy demands of the respiratory muscles during exercise is the work of breathing, the strength of the inspiratory and expiratory muscles and their efficiency. Aaron et al. (1992) reported that the oxygen cost of breathing is approximately 2%, 3 – 5% and 10 – 16% of total body oxygen consumption during rest, moderate and high intensity exercise, respectively. Stronger and

more efficient respiratory muscles need less energy and therefore less oxygen at a given level of exercise intensity compared to weaker and inefficient respiratory muscles (Macklem, 1980).

The blood flow to the respiratory muscles must also be sufficient to meet the high energy demands of the respiratory muscles during exercise. Cardiac output is divided between the respiratory muscles and the working muscles to meet their energy demands for ventilation and exercise, respectively. The oxygen cost and blood flow demands of the respiratory muscles differ between untrained and highly trained individuals during maximal exercise, due to differences in ventilatory requirements.

The untrained individual: During maximal exercise in the untrained individual the pressure produced by the expiratory muscles are well within the limit for effective pressure generation; and the pressure produced by the inspiratory muscles are well within (40 - 60%) the dynamic capacity of the respiratory system (Johnson et al., 1992). Therefore, the metabolic requirements of the respiratory muscles during maximal exercise in the untrained individual are relatively low (Romer & Polkey, 2008). The oxygen cost of hyperventilation during heavy exercise amounts to ~ 10% of the VO_{2max} in untrained individuals (Aaron et al., 1992).

The trained endurance athlete: During maximum exercise, the highly fit individual can increase expiratory pressures that will exceed the maximum dynamic pressure where air flow becomes limited. The dynamic inspiratory pressures can reach 90% (or even more) of the dynamic capacity of the respiratory system (Johnson et al., 1992). Romer & Polkey (2008) gave the following explanations for the latter: When exercise intensity and subsequently ventilation increase, to a much higher extent than during maximal exercise in the untrained individual, it causes problems for adequate pressure generation by the respiratory muscles. Firstly, the increased dynamic compression in the airways causes a limitation in air flow. EELV is forced upward for air flow to increase further (Klas & Dempsey, 1989). Secondly, ventilation operates at high lung volumes, which makes it difficult for the inspiratory

muscles to overcome the increased elastic resistance of the lungs and chest wall (Milic-Emili & Petit, 1960). Thirdly, at higher lung volumes, the inspiratory muscles are shorter which means that their force generation is decreased during inspiration. This subsequently means a lesser pressure gradient for air flow (Johnson et al., 1992). The oxygen cost of hyperventilation during heavy exercise amounts up to 16% of the total $\text{VO}_{2\text{max}}$ and total cardiac output in fit individuals (Harms et al., 1998; Aaron et al., 1992).

The strength and maximal work rate of the respiratory muscles to generate adequate pressure gradients for ventilation during heavy maximal exercise in the trained individual, could become a limiting factor during strenuous exercise. Similarly to limb locomotor muscles, respiratory muscles undergo morphological and metabolic adaptations with training, which make them work more efficiently. All these accumulative facts support the possibility that the respiratory muscles can greatly benefit from specific training that could make them stronger, more efficient, less fatigable and subsequently more economical during heavy maximal exercise.

C. ASSESSMENT OF RESPIRATORY MUSCLE FUNCTION

The muscles of the respiratory system have two basic functions: to shorten and to generate force. Specialized measurements to quantify force generation and shortening of the respiratory muscles needed to be established. Respiratory muscle force can be estimated as pressure, and shortening of the respiratory muscles can be estimated as lung volume change or displacement of the chest wall structures. Thus, the quantification of respiratory muscle function include the measurement of volumes, displacements, pressures and rates of volume change over time (Green et al., 2002).

The following paragraphs describe the standardized methods to assess respiratory muscle function:

- (i) Pulmonary function (Flow-volume curve test) and maximum voluntary ventilation (MVV) to measure lung volumes and rates of lung volume change over time (assess respiratory muscle shortening).

- (ii) Pressure measurements during voluntary and involuntary maneuvers to assess respiratory muscle force (strength).
- (iii) Flow and pressure measurements to assess respiratory muscle endurance.

1. Pulmonary function and MVV

Dynamic pulmonary function is measured to determine lung volume and the ability of the lungs to move air rapidly in and out of the lungs (Beachey, 1998). It is also an indirect measurement of RM strength (forced vital capacity [FVC] maneuver) and RM endurance (maximum voluntary ventilation [MVV]). Factors that determine lung volume include stature, age, gender, body mass, posture, ethnic group and the amount of daily physical activity (Quanjer et al., 1993).

Volume measurements over time:

- (i) *Forced vital capacity (FVC)*: FVC is the maximum volume of air that a person can exhale as forcefully and rapidly as possible after a maximum inspiration up to total lung capacity (TLC). The FVC maneuver is effort-dependent.
- (ii) *Forced expiratory volume in 1 second (FEV_1)*: This is the volume of FVC exhaled in 1 second. Because FEV_1 is measured over time, it reflects the average flow rate over its time interval.
- (iii) *Peak expiratory flow rate (PEFR)*: PEFR is the highest instantaneous flow achieved during the FVC maneuver. The PEFR reflects initial flows coming from large airways at the beginning of the FVC maneuver. The PEFR is also effort-dependent which reflects whether the subject gave a maximal effort during the FVC maneuver or not.
- (iv) *Forced expiratory flow between 25% and 75% ($FEF_{25-75\%}$)*: This is the mean forced expiratory flow during the middle half of the FVC. It was also formerly called the maximal mid-expiratory flow (MMEF).
- (v) *Forced expiratory flow at 75% ($MEF_{75\%}$)*: This is the maximum instantaneous forced expiratory flow where 75% of FVC remains to be expired.
- (vi) *Maximum forced expiratory flow at 50% ($MEF_{50\%}$)*: This is the maximum instantaneous forced expiratory flow where 50% of FVC remains to be expired.

- (vii) *Maximum forced expiratory flow at 25% (MEF_{25%})*: This is the maximum instantaneous forced expiratory flow where 25% of FVC remains to be expired.
- (viii) *Maximum voluntary ventilation (MVV)*: This is the greatest volume of air that a person can move in and out of his/her lungs with maximal effort over 10 – 15 s. Results are expressed in litres per minute. MVV reflects airway function, lung compliance, neural control mechanisms and RM endurance (Beachy, 1998).

2. Pressure measurements

Pressure measurements can be used to assess respiratory muscle (RM) strength and respiratory muscle endurance. The assessment of RM strength can be divided into volitional and non-volitional maneuvers, as well as invasive and non-invasive methods (Green et al., 2002). RM endurance is usually assessed as a measurement of task failure during resistive threshold loading or target pressure generation (Clanton et al., 2002).

The following paragraphs briefly describe the different methods for pressure measurements to assess RM strength and endurance. The major implications for pressure measurements are also briefly mentioned. Maximum static inspiratory and expiratory pressure measurements and its implications are described more thoroughly than the other methods, since it was the chosen method for the assessment of respiratory muscle strength in the present study.

2.1. Volitional tests for RM strength assessment

Pressure measurements through volitional maneuvers include non-invasive methods, i.e. maximum static inspiratory and expiratory pressure, while invasive methods include maximum diaphragmatic pressure, sniff transdiaphragmatic pressure, sniff nasal pressure and maximum cough pressures (Green et al., 2002).

Maximum static inspiratory and expiratory mouth pressure measurements: The first authors to introduce a method to measure pressure at the mouth during maximum inspiratory

and expiratory maneuvers were Black & Hyatt (1969). This method is widely used, due to its simplicity and availability of reference values for the general population (children to the elderly). However, these tests are voluntary and therefore highly effort dependent, thus, a low result does not necessarily mean that the individual has poor RM strength. It could be that the individual did not perform a true maximal effort due to lack of motivation (Allen et al., 1995; Gandevia & McKenzie, 1985).

A true maximal effort is not the only concern when RM strength is assessed through mouth pressures. Other implications for maximum static inspiratory and expiratory mouth pressure measurements include the specific interpretation of the pressure measured, the initial lung volume from which the pressure is developed, the type of mouth piece, the small leak in the pressure measurement device, the specific calculation of pressure (definition of pressure) measured over time and the number of efforts utilized to determine the end result (outcome value).

There is a complex relationship between RM force generation and the pressure produced either in the thorax or mouth. During ventilation, the diaphragm (curved structure) acts as a piston and the pressure per unit area output is only indirectly related to muscle force or tension. In addition to the latter, the mechanical characteristics of the rib cage and the abdominal wall influence the muscle force development in the respiratory muscles which interacts with them (Chihara et al., 1996). The pressure developed does not reflect the specific RM force generated, due to the complex mechanical linkage between the respiratory muscles and wall structures. Therefore the pressure measurements should not be seen as a direct measure of RM tension (force), but rather global respiratory muscle “output” (Green et al., 2002).

The maximal static inspiratory and expiratory pressures measured are a reflection of pressure developed by the respiratory muscles, plus the pressure developed due to the passive elastic recoil of the lungs and the chest wall. Therefore, maximum inspiratory and expiratory pressure will vary at different lung volumes (Black & Hyatt, 1971). During maximum inhalation from residual volume (RV), the contribution of the passive elastic recoil of the

lung and chest wall to total pressure, can be as much as 30 cmH₂O. During maximum exhalation from total lung capacity (TLC), the contribution of passive elastic recoil pressure can be as much as 40 cmH₂O. This passive elastic recoil pressure is not conventionally subtracted from the total pressure reported, therefore it should be specifically stated at which lung volumes maximal inspiratory and expiratory pressures are measured (Green et al., 2002). The standardized method is to measure inspiratory pressure from RV and expiratory pressure from TLC, unless otherwise stated (Green et al., 2002).

It should also be specified what type of mouth piece was used for measurements, since a flanged mouth piece gives a lower value for pressure compared to a rubber tube mouth piece (Koulouris et al., 1988). Furthermore, a small leak should be incorporated in the device that measures maximum static pressure at the mouth to prevent glottic closure during maximal inhalation and the use of facial muscles for pressure development during maximal exhalation. However, the dimensions of the small leak should be specifically stated, since different air leaks will result in different pressure measurements (Mayos et al., 1991).

Another implication with maximum static mouth pressures is the definition of the pressure measured. Windisch et al. (2004) found that peak inspiratory pressure during a single effort is significantly higher than inspiratory pressure averaged over one second and/or pressure averaged over half a second. However, they found a strong correlation between peak inspiratory pressure and plateau pressures (pressure averaged over 1 second and pressure averaged over half a second). Windisch et al. (2004) clearly shows that the definition of the pressure measured are of utmost importance in order to compare pressure measurements from different studies.

The reported values for maximum inspiratory and expiratory mouth pressure, respectively, are usually given as the average value of a few efforts. The average value reported depends on the number of efforts allowed during an assessment. Volianitis et al. (2001a) found that a specific respiratory muscle warm-up session resulted in a significant (10%) increase in the outcome value of inspiratory muscle pressure (defined as the highest value of the first three efforts). Wen et al. (1997) reported a higher outcome value during 20 efforts compared to an

outcome value during 3 – 5 efforts (mean \pm SD difference, 9 ± 13 cmH₂O, $P < 0.02$). The outcome value was calculated as the average of the highest three values with $\leq 5\%$ variability from the recorded efforts (3 – 5 or 20 efforts). These findings show the importance of reporting the number of efforts in order to compare results from different studies. Another implication of the outcome value derived from a certain number of efforts is the limits of variability between efforts to calculate the outcome value. No specific standardization exists for this calculation; however, the ERS/ATS Statement of respiratory muscle testing recommends the maximum value of three efforts that vary less than 20% (Green et al., 2002).

Esophageal, gastric and transdiaphragmatic pressure measurements: Transdiaphragmatic pressure (Pdi) is defined as pleural pressure (Ppl) subtracted from abdominal (Pab) pressure (Agostoni & Rahn, 1960). Pleural pressure (Ppl) and Pab are usually measured with a double-balloon catheter system placed in the stomach (gastric pressure = Pga) and esophagus (esophageal pressure = Pes) and connected to a pressure transducer. Esophageal pressure reflects Ppl and Pga reflects Pab (Milic-Emili et al., 1964). Unlike maximum static mouth pressures, the elastic recoil pressure of the lung does not influence the measurement of Pes, but the chest wall pressure does. Maximum transdiaphragmatic pressure (Pga – Pes) is measured during a complex volitional maneuver where the individual has to “bear down” while superimposing a maximal inspiratory maneuver. A great advantage of this method is that the maximum Pdi measured provides information about the strength of the diaphragm, specifically. However, this method requires invasive procedures and excellent coordination of the individual to perform the specific maneuver correctly (Green et al., 2002). The measurement of Pdi during non-volitional procedures, i.e. phrenic nerve stimulation (PNS) is more often used in a laboratory setting.

Sniff pressure measurements: Pressure measurements during sniff maneuvers include: transdiaphragmatic pressure during sniff, esophageal pressure during sniff and sniff nasal pressure. Transdiaphragmatic pressure during sniff is measured with the balloon catheter system in the same manner as volitional Pdi during maximal inspiratory maneuvers through the mouth. Esophageal pressure during sniff is measured with an esophageal balloon. Sniff

nasal pressures are measured by inserting a catheter in one nostril. The subject sniffs through the other “open” nostril and the pressure measured is an indication of pressure in the nasopharynx, which reflects the pressure in the alveoli (Green et al., 2002). The main advantage of this method is that subjects achieve a greater Pdi with sniff maneuvers than with maximum static maneuvers through the mouth. Normal values for sniff pressure show less variability than for mouth pressures (Laroche et al., 1988).

2.2. Nonvolitional tests for RM strength assessment

The phrenic nerve is the only nerve that innervates the diaphragm; therefore, specific stimulation of this nerve can provide information on the strength of the diaphragm without the interference of other inspiratory muscles. Phrenic nerve stimulation (PNS) is either applied through an electric current or magnetic field currents. This stimulation elicits the activation of the motor units within the diaphragm and thus causes diaphragm muscle contraction.

There are four PNS techniques described in the literature. Needle stimulation (Mier & Brophy, 1991) and implanted wire stimulation (Hubmayr et al., 1989) are invasive techniques and not widely used by researchers due to high risk for muscle damage. The other two techniques are thoroughly studied: transcutaneous electrical PNS (McKenzie & Gandevia, 1985) and magnetic stimulation (Olney et al., 1990). All four measurements are concerned with diaphragm activation. Abdominal muscle stimulation is an indication of expiratory muscle activation and is briefly described under this section.

Transcutaneous electrical PNS and magnetic stimulation: Electrodes for electrical PNS are placed on the skin at the site of the phrenic nerve, which can be located below the sternocleidomastoid muscle on the side of the neck. This procedure is performed on both sides of the neck, to achieve bilateral electrical PNS. Magnetic stimulation creates rapidly changing magnetic fields which penetrates through skin easily and reaches deep nervous structures. The pressure response of the diaphragm to PNS, are known as twitches.

Transdiaphragmatic twitch pressure ($P_{di,tw}$) rapidly rises to its peak and then decreases exponentially to baseline values (Green et al., 2002).

Implications for transcutaneous electrical PNS and cervical magnetic stimulation (CMS):

In transcutaneous electrical PNS, it is difficult to find the correct site where the electrodes must be applied; however, it gives an isolated contraction of the diaphragm. Another concern is that the supramaximal stimulation can be uncomfortable for the individual. Repetitive electrical stimulation can increase twitch pressure by potentiation and cause the staircase phenomenon (Van Lunteren & Vafaie, 1993). CMS is not painful to the individual and the location of the nerves for CMS is easier to find than for electrical PNS. However, CMS is not that specific as electrical PNS, because the cervical roots innervate other muscles which also contract during CMS. CMS can also be influenced by other magnetic fields in the room, e.g. credit cards, computer disks etc. (Green et al., 2002).

Abdominal muscle stimulation: The abdominal muscles are active during expiration through its actions on the rib cage and its mechanical linkage with the diaphragm. Abdominal muscle strength can be assessed through volitional tests (maximum static expiratory mouth pressure), however it is difficult to tell if the individual can truly perform a maximal effort and master the maximal expiratory maneuver (Green et al., 2002). Nonvolitional tests, such as direct electrical stimulation can provide information on abdominal muscle action on the rib cage (Kyroussis et al., 1997); and magnetic stimulation at the eighth to tenth thoracic vertebra, can assess abdominal muscle strength and fatigue (Kyroussis et al., 1997).

3. Flow and pressure measurements to assess respiratory muscle endurance

Endurance is the ability of muscles to sustain a specific task over time. Respiratory muscle endurance can be defined as the ability to sustain a level of ventilation (ventilatory endurance) or the ability to sustain a level of inspiratory or expiratory pressure over time.

The following is a brief description of the different methods available to assess respiratory muscle endurance: Esophageal, Pdi, or mouth pressure can be measured over time. The results are the integration of inspiratory or expiratory pressure over time (Pressure-time-product) (Clanton et al., 1990; Bellemare & Grassino, 1982). The work of breathing can be measured against an external inspiratory or expiratory load, and provides information on endurance as a function of workload (Banner et al., 1994). Maximal sustainable ventilation, expressed as a percentage of maximum voluntary ventilation during 12 seconds, can also be measured to assess ventilatory endurance (Tenney & Reese, 1968).

Tests against external loads are widely used in the literature to assess respiratory muscle endurance: Maximum sustainable threshold loading is measured as the time the individual can sustain breathing against a constant resistive load (usually a percentage of measured maximum static pressure) (Nickerson & Keens, 1982). Another method using external loads is maximum incremental threshold loading. This assessment requires breathing against a resistive load to achieve a certain threshold pressure (percentage of maximum pressure). The threshold pressure increases over time and the outcome is the highest pressure that can be tolerated over a fixed time period (Morrison et al., 1989).

Other methods to assess respiratory muscle endurance include: repeated maximum inspiratory pressures (McKenzie & Gandevia, 1986) and maximal sustainable isoflow where the subject breathes against high impedance but a constant flow (Clanton & Ameredes, 1988). The endurance of the diaphragm specifically can be assessed by measuring maximum Pdi, thereafter, the subject has to inspire through a variable inspiratory flow resistance with a set breathing pattern and achieve a target Pdi (Bellemare & Grassino, 1982).

To specifically assess inspiratory muscle endurance, the incremental threshold loading test is recommended by the ERS/ATS. No recommendation for expiratory muscle endurance is mentioned (Clanton et al., 2002).

D. RESPIRATORY MUSCLE FATIGUE

Muscle fatigue is typically defined as the inability of muscles to maintain or produce a predetermined amount of work. Similarly, it is suggested that the inspiratory muscles also reach a maximal capacity to produce sufficient ventilation and gas exchange during exercise of increasing intensity. Thus, if exercise intensity increases even more, the respiratory muscles (RM) will fail to increase their capacity to contract, or may not be able to maintain this capacity, and this will result in RM fatigue.

The occurrence of RM fatigue during rest has significance in studying RM diseases, however, the occurrence of exercise-induced RM fatigue is of importance to the sport scientist to fully understand and investigate limitations to athletic performance.

1. Evidence of respiratory muscle fatigue

The quantification RM fatigue is not easily quantifiable as locomotor muscle fatigue, as the diaphragm and its innervated nerves are relatively inaccessible. Therefore, pressure differences across the diaphragm are used to estimate the force development of the muscle.

RM fatigue is quantified as a significant reduction in RM strength from baseline values, measured through global respiratory muscle strength or endurance, transdiaphragmatic pressure (Pdi) development by electric or magnetic stimulation, or gastric pressure (Pga) response to magnetic stimulation. RM fatigue of the diaphragm (Hamnegard et al., 1996) and abdominal muscles (Kyroussis et al., 1996) has been found under resting conditions during resistive breathing and voluntary hyperpnoea.

2. Exercise-induced respiratory muscle fatigue

A number of studies have demonstrated that whole-body endurance exercise elicits RM fatigue, either as a reduction in RM strength, or as a reduction in RM shortening (lung volume and air flow). Some researchers found a significant reduction in FVC (up to 0.5 L)

after exercise (O’Kroy et al., 1992; Hill et al., 1991; Buono et al., 1981; Mahler & Loke, 1981), and others reported a significant reduction in MVV after exercise (Bender & Martin, 1985; Loke et al., 1982). However, Romer & Polkey (2008) suggested that future studies are needed to fully determine the role of high velocities of RM shortening (i.e. high flow rates) in RM fatigue.

Inspiratory muscle fatigue: Since the diaphragm is the primary inspiratory muscle during both rest and exercise, many studies have investigated diaphragm fatigue during exercise. The fatigue of the diaphragm is recognized as a significant decrease in Pdi after a period of whole-body exercise (Babcock et al., 1998; Babcock et al., 1995; Mador et al., 1993; Bye et al., 1984).

Evidence of global inspiratory muscle fatigue measured as a reduction in static inspiratory mouth pressure and/or resistive breathing against an external load, are of greater interest in the present study, since global RM strength was used for the assessment of the respiratory muscles.

On separate occasions, Ker & Schultz (1996) and Loke et al. (1982) found significant decreases in maximum inspiratory mouth pressures (MIP) in marathon runners after a marathon event and an ultra-marathon event, respectively. Hill et al. (1991) tested triathletes during each stage of a triathlon and found that maximum inspiratory mouth pressure was significantly reduced after the cycle and run stages, respectively. McConnell et al. (1997) also found a reduction in MIP ($11 \pm 8.2\%$) in moderately trained men after a progressive shuttle run test. Perret et al. (2000) found a reduction in breathing endurance (time to task failure for breathing against a constant load) after low-to-moderate intensity (65% of VO_{2max}) and high intensity (95% of VO_{2max}) exercise to exhaustion in healthy, fit men. Romer et al. (2002c) found a significant reduction in maximum inspiratory mouth pressure in trained cyclists after a 20 km and 40 km cycling time trial, respectively. Lomax & McConnell (2003) found a significant reduction in MIP after a six minute swim event (200 m) in competitive swimmers. From all these studies it is clear that RM fatigue occurs during short-term, high-intensity exercise and during prolonged endurance exercise.

Furthermore, these findings are observed in moderately active individuals as well as in highly trained competitive athletes.

Expiratory muscle fatigue: Abdominal muscle fatigue measured as a reduction in gastric pressure (Pga) by magnetic nerve stimulation, is an indication of expiratory muscle fatigue. Although not so thoroughly studied as transdiaphragmatic fatigue, evidence exists of this phenomenon (Taylor et al., 2006; Verges et al., 2006; Kyroussis et al., 1996). Taylor and colleagues (2006) found a 25% reduction in Pga after a cycle test to exhaustion (> 90% of VO_{2max}) in physically active men. Verges and colleagues (2006) found a significant decrease in twitch Pga after a cycle test to exhaustion (85% of maximum workload achieved) in moderately to highly trained men.

Very little evidence of global expiratory muscle fatigue exists. Loke et al. (1982) found a significant decrease in maximum expiratory mouth pressure after a 42.2 km race in endurance runners.

3. Factors causing RM fatigue

There are mainly two factors which contribute to RM fatigue: Firstly, the RM cannot sustain the very high work rates throughout heavy exercise; and secondly, the RM need to share the available cardiac output with locomotor muscles, which leads to inadequate blood supply to the RM (Romer & Polkey, 2008).

Babcock et al. (2002) assisted diaphragmatic work during exercise by 50%, using a mechanical ventilator and demonstrated that this intervention prevented diaphragmatic fatigue. This study presented clear evidence that the reason for RM fatigue is the inability to sustain heavy workloads during heavy exercise.

Babcock et al. (2002) investigated the effect of mimicking the duration and magnitude of the diaphragm force output during exercise in a resting subject. Diaphragmatic fatigue only occurred after diaphragmatic pressures were voluntarily increased two-fold greater than the

diaphragmatic pressure required during whole-body exercise, to induce exercise-induced diaphragmatic fatigue. The latter demonstrated that during rest the diaphragm receives a large share of the cardiac output and receives adequate blood supply. However, during exercise the diaphragm has to compete with limb locomotor muscles for blood supply. Less blood supply to the diaphragm leads to inadequate oxygen transport for energy, and this contributes to fatigue. The less blood supply to the diaphragm, the less work is required for the diaphragm to fatigue. This supports the belief that diaphragmatic fatigue only occurs during exercise intensities of 80 to 85% of VO_{2max} and not at lower exercise intensities (Johnson et al., 1993).

4. Reasons for inconclusive evidence of RM fatigue in literature

The literature on RM fatigue is somewhat controversial, since some studies found no RM fatigue after exercise (Nava et al., 1992; Younes & Kivinen, 1984). However, a clear difference between these studies and others that did find RM fatigue, are the exercise intensity and specifically, exercise duration at maximal efforts. Younes & Kivinen (1984) found no change in baseline maximum static inspiratory pressure after an incremental test (3 min increment durations) in moderately trained men. However, all other studies that did find RM fatigue, used test protocols of time to exhaustion at intensities of 80 to 90% of VO_{2max} (longer than 3 min) or 80 to 85% of peak power output (Lomax & McConnell, 2003; Babcock et al., 1998; Babcock et al., 1995; Bye et al., 1984).

Nava et al. (1992) found no changes in MIP during and after a 17 km run in highly fit runners. They argued that the subjects in their study were better trained than subjects in the study by Loke et al. (1982), since they could sustain a higher running speed. However, their subjects only ran 17 km, compared to the marathon distance (42.2 km) in the study by Loke et al. (1982). It is obvious that endurance athletes would run at higher speeds in a race where the distance is much shorter compared to a marathon. Therefore, these findings show that not only exercise intensity, but also the duration of exercise at maximal efforts, must be taken into consideration in RM studies.

The other factor that can influence the occurrence of RM fatigue is the fitness levels of the subjects. Coast et al. (1990) found no significant changes in MIP in trained cross-country skiers compared to a significant decrease in MIP in untrained college students, after an incremental $\text{VO}_{2\text{max}}$ test to exhaustion.

The occurrence of RM fatigue under different air conditions also influenced the degree of RM fatigue. Bye et al. (1984) found a smaller decrease in transdiaphragmatic pressure (Pdi) after a cycle test to exhaustion (80% of maximal power output) while breathing 40% oxygen compared to normal air in active men. They contributed the delay of onset in diaphragmatic fatigue to a reduction in V_E during the test to exhaustion while breathing 40% oxygen. On the other hand, Williams et al. (2005) found a significant decrease in MIP from baseline values after a cycle ergometer test to exhaustion (60% of $\text{VO}_{2\text{max}}$) in a hot environment (± 37 °C) compared to no change after the same test in a thermo-neutral environment (± 22 °C), in untrained women. They suggested that RM fatigue in a hot environment at relatively low exercise intensities could be the result of lesser blood flow to the respiratory muscles, since an additional portion of the cardiac output had to be directed to the cutaneous circulation for thermoregulation.

Coast et al. (1998) investigated the possible mechanisms that could explain the decrease in FVC after heavy exercise, by studying the effect of changes in lower body pressures on thoracic blood volume. They found that an increase in lower body pressure, which happens during heavy exercise, caused a decrease of 0.18 L and 0.14 L from pre-lower body pressure application values in FVC and FEV_1 , respectively. Increased lower body pressure did not alter lung compliance. They suggested that a decrease in FVC after heavy exercise can not be attributed to the mechanical properties of the lung, but rather to an increase in thoracic blood volume.

In conclusion, many factors should be taken into consideration when studying RM function during exercise. Most importantly, exercise duration and intensity, and fitness levels of subjects in the study will influence the findings of RM fatigue. These factors explain the disagreements among previous studies with different outcomes. Furthermore, the precise

conditions (hot vs. neutral ambient temperatures, concentration of oxygen in the air) in which studies are undertaken can influence the outcome of the study. Researchers should also be careful when contributing changes in pulmonary function after heavy exercise to RM fatigue, because other factors, e.g. increased thoracic blood volume, can also cause a change in pulmonary function.

E. RESPIRATORY MUSCLE FATIGUE: EFFECT ON EXERCISE PERFORMANCE

Two approaches have been studied in the literature to illustrate the effect of RM fatigue on performance. One approach is partially unloading the RM muscles to prevent RM fatigue during exercise, and the other is loading of the RM muscles prior to exercise performance, thus inducing RM fatigue prior to performing exercise.

1. Partial unloading of the respiratory muscles

Studies investigated the effect of RM work on exercise performance by partially unloading the RM in two ways: either by breathing a gas mixture, called Heliox (79% helium, 21% oxygen) during the exercise test, or by breathing with an assist mechanical ventilator during exercise (Romer & Polkey, 2008).

Gas mixture: The gas mixture assists ventilation by reducing the turbulent component of air flow, and therefore lowering the expiratory flow limitation. The oxygen component also assists ventilation indirectly, by decreasing alveolar-arterial oxygen difference. Studies using this approach found that time-to-exhaustion at high exercise intensities (85 – 90% of VO_{2max}) was significantly increased, but not at lower exercise intensities (Johnson et al., 1996; Aaron et al., 1985).

Mechanical ventilator: Studies using a mechanical ventilator to partially unload the inspiratory muscles, found a significant increase in time-to-exhaustion during exercise intensities of > 90% of VO_{2max} (Harms et al., 2000). They also found a reduction in the rate

of increase in O_2 consumption and in the subjects' perceptions of limb and breathing discomfort.

2. Loading of the respiratory muscles

RM fatigue prior to exercise performance can be induced in two ways: by breathing against inspiratory or expiratory resistive loads to task failure, or voluntary hyperpnoea. Some studies that investigated the effect of prior RM fatigue on exercise performance, found significant decreases in exercise performance (Taylor & Romer, 2008; Verges et al., 2007b; Harms et al., 2000; Mador & Acevedo, 1991; Martin et al., 1982).

Taylor & Romer (2008) found a significant reduction in cycling exercise time to exhaustion at 90% of peak power output, after expiratory muscle fatigue was induced by flow resistive breathing (until task failure) at 40% of maximal expiratory gastric pressure.

Verges et al. (2007b) found a significant decrease in distances covered during a 12 minute running test, after expiratory muscle fatigue was induced by expiratory resistive breathing against 50% of maximum expiratory mouth pressure.

Harms et al. (2000) increased ventilatory work by inserting an external load in the inspiratory line during exercise at 90% of VO_{2max} on a cycle ergometer. Time to exhaustion decreased by 15%, compared to control conditions.

Mador & Acevedo (1991) induced RM fatigue in subjects by breathing against a threshold load (80% of predetermined maximum static inspiratory pressure) until task failure. Thereafter, time-to-exhaustion on a cycle ergometer at 90% of VO_{2max} was significantly reduced compared to control (no RM pre-fatigue) conditions.

Martin et al. (1982) induced ventilatory fatigue by means of a 150 min sustained maximum ventilation test. Short-term maximal running (5 – 9 min) at a constant speed, with 1%

elevation in incline every minute until volitional fatigue, was significantly reduced after ventilatory work.

3. Practical problems associated with performance studies

Loading of respiratory muscles to induce fatigue prior to exercise: The first problem with induction of RM fatigue prior to exercise, is the actual determination of RM fatigue in some studies (Martin et al., 1982), or a lack of objectively measuring RM fatigue using nerve stimulation techniques, as in the studies of Mador & Acevedo (1991) and Verges et al. (2007b). The lack of assessment of RM fatigue prior to exercise could overestimate the degree of fatigue associated with maximal exercise, or fail to induce adequate RM fatigue, or result in no RM fatigue at all.

McKenzie et al. (1997) found no change in transdiaphragmatic twitch pressure ($P_{di,tw}$) through bilateral phrenic nerve stimulation (PNS) after breathing against resistive loads to task failure, at 35, 50, 75 and 90% of MIP, respectively. MIP measurements before, during and after resistive breathing increased slightly, and no decrease in P_{di} during voluntary activation after resistive breathing, was observed. They found a significant increase in end-tidal partial pressure in CO_2 (PCO_2) in the trials, despite the absence of RM fatigue. They suggested that hypoventilation, increase in end-tidal PCO_2 , and task failure during resistive breathing are not simply dependent on the lack of diaphragm force-generation or the inability to voluntarily activate the diaphragm.

In agreement with the latter, Gorman et al. (1999) investigated the variation in certain ventilatory parameters and end-tidal PCO_2 , during different protocols of resistive breathing to a target pressure of 80% of MIP until task failure. Breathing discomfort was monitored using the modified Borg scale, during different protocols. Time to task failure increased in protocols where subjects were allowed to take an unloaded breath every 30 seconds, to prevent CO_2 accumulation. In the CO_2 rebreathing protocols, time to task failure decreased due to an increased sense of breathing discomfort. MIP did not decline during resistive breathing in any of the protocols. Gorman et al. (1999) suggested that time to task failure

may decrease due to increased sensations of breathing discomfort associated with CO₂ accumulation, and not due to inspiratory muscle fatigue.

The studies of Gorman et al. (1999) and of McKenzie et al. (1997) clearly show that RM fatigue is not necessarily the cause of task failure during resistive breathing, as a method to induce RM fatigue prior to exercise, therefore it is critical for researchers to include a thorough assessment of RM fatigue to prove that RM fatigue was the cause for task failure, if they want to investigate the effect of RM fatigue on exercise performance. It is suggested that ventilatory responses (end-tidal PCO₂, breathing discomfort) must also be measured in RM fatigue studies to fully explain the role of these factors in RM fatigue.

In conjunction with ventilatory responses during resistive breathing, the second problem with pre-loading of the RM, is a possible change in the breathing pattern of the subject. It is possible that the subject will change his/her breathing pattern during exercise after the induction of RM fatigue. The increased intensity of dyspnoea can cause a decrease in exercise tolerance, since the subject cannot sustain the high sensation of breathing discomfort and need to cease exercise (Romer & Polkey, 2008).

The third problem with pre-loading of the respiratory muscles is the possible influence of subject expectations to exercise performance. It is impossible to induce RM fatigue in subjects without them knowing it. Therefore, it is difficult to determine how much the subject's perception that he/she will perform worse during the test after the induction of RM fatigue, will affect the decrease in exercise tolerance, rather than RM fatigue itself (Romer & Polkey, 2008).

Unloading of the respiratory muscles during exercise: Some studies that investigated the effect of unloading of the RM on exercise performance, showed no increase in exercise performance compared to control conditions (Romer et al., 2007; Marciniuk et al., 1994; Gallagher & Younes, 1989). However, in both Gallagher & Younes (1989) and Marciniuk et al. (1994), exercise time to exhaustion was performed at 70 to 80% of VO_{2max} in less physically fit individuals. In Romer et al. (2007), moderately fit subjects performed an

incremental test to exhaustion during which they exercised at 90% of VO_{2max} for only 4 minutes. These findings show that the partial unloading of the RM only seems to affect exercise performance at very high exercise intensities ($> 90\%$ of VO_{2max}) and not during lower intensities. Not just the exercise intensity, but also the exercise duration, will affect the outcome of RM unloading studies on exercise performance.

The second concern with partial unloading studies, is the inclusion of a placebo group, where subjects still have to breathe through a ventilator, while undergoing sham unloading. To the researcher's knowledge only one study (Romer et al., 2007) incorporated a placebo cross-over design. Studies that did not include a placebo group can be criticized for weak internal validity and could be influenced by subject bias (Romer & Polkey, 2008).

A similar problem as with loading of the RM, is the difficulty to determine if the improvement in exercise performance is due to a relief in RM fatigue through RM unloading, or whether the subject's perception of less respiratory muscle work, is the reason for the improvement in performance.

There are two limitations to partial RM unloading studies: The pressures delivered by a mechanical ventilator during maximal exercise can be very disruptive and uncomfortable for subjects (Romer et al., 2007; Harms et al., 2000). Familiarization sessions did not relieve this discomfort. Furthermore, inspiratory muscle unloading caused a less negative intrathoracic pressure than during normal conditions. This, subsequently, caused a reduction in ventricular pre-load which in turn caused a reduction in stroke volume and cardiac output (Harms et al., 1998). This phenomenon might contribute to the finding of no improvement in exercise performance during RM unloading.

In conclusion, many factors should be taken into consideration when comparing studies that are concerned with loading and partially unloading of the RM. These factors include: the intensity and duration of exercise performance tests, the fitness level of subjects, subjects' perceptions of discomfort or relief in breathing sensations, changes in breathing patterns, true inductions of RM fatigue and less negative intrathoracic pressure development. More

investigations into these factors are needed to determine the exact influences of these factors on exercise performance.

4. Possible mechanisms by which the respiratory system affects exercise performance

The mechanisms by which the respiratory system can affect exercise performance are: inefficient alveolar-to-arterial oxygen exchange, an alteration in breathing mechanics, an increased sensation of dyspnoea, the respiratory muscle fatigue-induced metaboreflex and an increase in expiratory flow and expiratory time.

Inefficient alveolar-to-arterial oxygen exchange: Inefficiency in gas exchange during maximal exercise may lead to a widening in the alveolar-arterial oxygen pressure difference $P(A-a)O_2$ in highly trained individuals. This extreme $P(A-a) O_2$, together with limited hyperventilation and an acid pH-induced rightward shift in the hemoglobin-oxygen dissociation curve, can lead to arterial O_2 desaturation (80 – 90% SaO_2) (Dempsey et al., 2008). This subsequent arterial hypoxemia leads to a reduction in O_2 transport to the locomotor muscles. A reduction in O_2 transport to the locomotor muscles causes locomotor muscle fatigue. The widening of the $P(A-a) O_2$ occurs due to the diffusion limitation of O_2 over the respiratory membrane caused by very high pulmonary blood flow and increased pulmonary vascular pressures (Dempsey et al., 2008). However, it is evident that the diffusion limitation of the lungs and inefficient gas exchange happens during prolonged heavy-intensity exercise (Dempsey & Wagner, 1999), especially running (Hopkins et al., 1997), therefore one may assume that this type of limitation would be less relevant to repetitive sprint sport since these sports are characterized by high, short bouts of heavy intensity exercise and not prolonged periods of heavy-intensity exercise.

Breathing mechanics and dyspnoea: Diaphragmatic fatigue during exercise means that the diaphragm contributes less to total ventilation. The accessory muscles are recruited to sustain the heavy ventilatory demand during exercise. The progressive recruitment of the accessory muscles as exercise continues can distort the chest wall, reduce the mechanical

efficiency of breathing, increase the metabolic and blood flow demands of the RM, and increase sensory input to the central nervous system. The increase in sensory input to the central nervous system increases the intensity of dyspnoea (Romer & Polkey, 2008).

Other factors than the recruitment of accessory RM can increase the sensation of dyspnoea: i.e. alterations in the pattern of tension development within the RM, and the decrease in the operating length of the RM or an increase in the velocity of shortening of the RM. An increased sensation of dyspnoea can lead to exercise cessation, because the subject cannot sustain the sensation of breathing discomfort (McConnell & Romer, 2004a).

Respiratory muscle (RM) metaboreflex: The diaphragm and other respiratory muscles are richly innervated with metaboreceptors (Romer & Polkey, 2008). It is believed that an increase in metabolites during RM fatigue, causes the metaboreceptors to activate group IV phrenic afferents which in turn increases sympathetic vasoconstrictor activity via a supra-spinal reflex (Harms, 2007). This reflex is known as the RM metaboreflex. The increased sympathetic nerve activity cause a significant decrease in limb vascular conductance and limb blood flow during rest (Sheel et al., 2001) and during maximal exercise (Harms et al., 1997). The exact pathway through which sympathetic vasoconstrictor activity is increased, is still unknown. It could possibly be via a supra-spinal reflex or through the baroreflex (McIlveen et al., 2001). This area needs to be investigated in future studies.

Romer & Polkey (2008) suggested that RM fatigue induces the RM metaboreflex, which influence blood flow and oxygen transport to the locomotor muscles. This action directly affects locomotor muscle force output which can be seen as peripheral fatigue. The feedback effect of increased perceptions of effort reduced locomotor output to the limb muscles (i.e. central fatigue) (Romer & Polkey, 2008). However, it is still unclear whether the locomotor output in central fatigue happens because of “reflex inhibition” or an increased perception of effort (Dempsey et al., 2006). Thus, central and/or peripheral fatigue caused a cessation of exercise. Future studies are needed to investigate this area.

A critical point, in the occurrence of the RM metaboreflex, is that there seems to be a threshold for the activation of the metaboreflex (Harms, 2007). For instance, Wetter et al. (1999) found no change in leg vascular resistance or cardiac output to the legs during resistive load breathing at exercise intensities of 50 – 75% of $\text{VO}_{2\text{max}}$ in healthy competitive cyclists. However, it is unclear whether the metaboreflex will influence heavy exercise in the moderately trained athlete. Thus, it is unclear whether the athlete's fitness level plays a role in the activation of the metaboreflex. During whole-body exercise there is a strong vasodilator effect in the working muscles, while the RM metaboreflex causes a vasoconstriction effect in the working muscles. It is unclear how these two opposing effects interact with each other, and whether the metaboreflex can override the vasodilator effects to redistribute blood flow to the RM (Romer & Polkey, 2008).

Another concern is the presence of metaboreceptors in the limb muscles. It is unclear if the activation of the limb muscle metaboreflex will affect the blood distribution to the RM. It is also unclear whether the increase in global sympathetic outflow will cause vasoconstriction in the RM vasculature. It is suggested that the diaphragm is less responsive to vasoconstrictor influences, therefore, one will assume that global sympathetic outflow has a greater vasoconstriction effect in limb muscle vasculature (Romer & Polkey, 2008). However, evidence of such a phenomenon has not yet been investigated.

Expiratory flow and expiratory time: Evidence indicates that the abdominal muscles, responsible for forced expiration during heavy exercise, are fatigable (Verges et al., 2006) and that expiratory muscle fatigue elicits an increase in sympathetic nerve activity in resting limb muscles (Taylor & Romer, 2008; Derchak et al., 2002). Very high expiratory flows and increased expiratory time during prolonged heavy exercise causes a high intrathoracic pressure, which exceeds the dynamic pressure at which airway compression occurs (Johnson et al., 1992). In addition, positive intrathoracic pressures will reduce ventricular transmural pressure and decrease the time for ventricular filling. A decrease in ventricular filling time will reduce stroke volume and subsequently, cardiac output (Romer & Polkey, 2008). These additional expiratory effects can reduce systemic oxygen delivery and therefore, make the limb musculature more susceptible to fatigue (Aliverti et al., 2005). However, the effects of

extra changes in intrathoracic pressure (due to extra inspiration and expiration during exercise) on limb venous return and ventricular filling are still inconclusive and needs to be further investigated (Dempsey et al., 2006).

In conclusion to this section, the most important mechanism by which RM fatigue limits exercise performance, specifically during high-intensity repeated sprint activities, is the RM metaboreflex. Clear evidence exists (Harms et al., 1997) that the RM metaboreflex, caused by RM fatigue, leads to an increase in vasoconstrictor outflow to the working limb muscles and decreases the blood flow in limbs during heavy exercise, this leads to lower forces generated by the limb muscles and higher perceptions of limb discomfort in subjects (Romer et al., 2006), and ultimately, limb muscle fatigue and exercise cessation. However, many questions on the precise mechanism of the RM metaboreflex and how it affects performance are still unanswered.

F. CONCLUSION

Evidence shows that RM fatigue can limit exercise performance. The higher ventilatory demand during exercise requires a higher work rate from the RM. This leads to higher energy demands of the RM muscles during exercise (10 – 15% $\text{VO}_{2\text{max}}$ during maximal exercise). However the very high work rates and sharing of the available cardiac output with other working muscles, leads to RM fatigue. RM fatigue will subsequently limit exercise performance, this is evident through studies that investigated the effect of RM fatigue on performance by either inducing RM fatigue prior to exercise, or preventing RM fatigue during exercise (unloading).

Possible methods to overcome RM fatigue during whole-body exercise will serve as a solution to the limiting effect it has on exercise performance. In patients, RM fatigue can be relieved through breathing low density gas mixtures, supplemental oxygen or by means of a mechanical ventilator. However, these solutions are not suited for athletes to incorporate during exercise, therefore, specific respiratory muscle training (RMT) is suggested to overcome RM fatigue.

CHAPTER THREE

RESPIRATORY MUSCLE TRAINING AND DETRAINING

A. INTRODUCTION

The aim of physical training is to decrease the demands of exercise on the physiological systems and improve exercise performance. Skeletal muscle tissue has extraordinary plasticity and can reversibly change its functional characteristics and structural composition in accordance to the functional demands placed upon them. Training-induced skeletal muscle adaptations result in an increase in a muscle's tolerance to exercise. On the other hand, cessation or reduction in physical training will lead to a complete or partial reversal of training-induced adaptations, and therefore exercise performance will be compromised (Mujika & Padilla, 2001b).

It is clear from the previous chapter that widespread evidence exists of respiratory muscle (RM) fatigue during heavy exercise in well-trained athletes, and previous investigations showed that RM fatigue will affect exercise performance in well-trained athletes. The latter indicates that whole-body training is not adequate to prevent specific RM fatigue during heavy exercise. In recent years it has become apparent that the RM can also adapt to a specific RM training (RMT) stimulus (similar to limb muscles during whole-body training), which will abolish or delay the onset of RM fatigue and thus, indirectly enhance exercise performance. Thus, RMT could have an ergogenic effect on exercise performance, however, the outcomes of studies investigating this matter, are somewhat controversial.

It is suggested that the cessation or reduction in training will lead to a reversal of the training-induced adaptations to the RM and therefore diminish improvements in RM function and efficiency, and consequently diminish exercise performance. These suggestions are based on the reversal in adaptations to limb muscles after whole-body training cessation, which is widely documented in the literature. Only one study has been published so far on RM detraining (Romer & McConnell, 2003b). It is imperative to know

for how long the training adaptations to RM will last in order to maintain an ergogenic effect. One of the aims of the present study was to determine the existence of a time-frame for the reversal of RM adaptations after a RM training intervention.

This chapter describes the different types of RMT, the reasons for the contradictory study outcomes, the possible mechanisms of an ergogenic effect, the relevance of RMT in hockey players and the effect of detraining on RM function.

B. RESPIRATORY MUSCLE TRAINING

The direct goal of specific respiratory muscle training (RMT) is to improve respiratory muscle strength (RM) and endurance, and indirectly improve exercise performance. An improved RM strength and endurance could, theoretically, delay RM fatigue during whole body heavy exercise. This will have two major consequences: firstly, the more efficient and strong respiratory muscles will need a smaller fraction of the cardiac output, and the build up of metabolites as a result of high-intensity RM work will be delayed; this will directly prevent the possible reflex vasoconstrictive effects (RM metaboreflex) in the exercising limb muscles. Secondly, it can decrease the sensory input to the central nervous system which will cause a reduced perception of discomfort in breathing and limb muscle work (Romer & Polkey, 2008).

It is difficult to interpret existing literature on the effect of RMT on exercise performance, due to vastly different outcomes. As a consequence the ergogenic effect of RMT on exercise performance remains controversial. The differential outcomes among studies can be attributed to the large variations in the technique, intensity, frequency and duration of RMT and the different modes, intensities and durations of exercise performance tests.

The following paragraphs give a brief overview on: the different techniques of RMT, the controversial evidence, possible mechanisms of an ergogenic effect, and the possible explanations for the existing contradictions among studies in the literature.

1. Techniques of respiratory muscle training

There are three different techniques of RMT: voluntary isocapnic hyperpnoea, flow resistive loading and pressure threshold loading. The effectiveness of the RMT technique depends on whether the mode and intensity of the training will improve both the force generating capability and velocity of RM contraction.

1.1 Voluntary isocapnic hyperpnoea (VIH)

VIH training requires high levels of breathing to maintain a high target of ventilation for about 30 minutes. During the high level of ventilation, subjects are allowed to rebreathe through a dead space to maintain normal levels of PCO₂ and to prevent hypocapnia. The levels of ventilation usually correspond to 60 – 90% of maximum voluntary ventilation (MVV). VIH has been shown to improve indices of RM endurance in sedentary subjects (Markov et al., 2001; Stuessi et al., 2001; Boutellier & Piwko, 1992), moderately trained athletes (Verges et al., 2007a; McMahan et al., 2002; Spengler et al., 1999; Kohl et al., 1997; Morgan et al., 1987) and trained competitive athletes (Boutellier et al., 1992).

Although VIH training is the most specific type of training, since it mimics what happens during exercise, i.e. hyperventilation, a few shortcomings exist with this type of RMT. VIH does not improve the maximal pressure (force) generating capacity of the RM, which means that it only contributes to the velocity or shortening (flow) axis of the force–velocity relationship of the inspiratory muscles (McConnell & Romer, 2004b). Another concern is the long duration of sustained high levels of ventilation during VIH which is time consuming and physically demanding for the subject. The high degree of self-motivation to complete a training session might affect the quality of RM training. Lastly, high levels of ventilation can cause airway drying and lead to bronchoconstriction in subjects suffering from exercise-induced asthma (McConnell & Romer, 2004b).

1.2 Inspiratory flow resistive loading (IFRL)

IFRL requires breathing via an adjustable diameter orifice. At a given airflow, the resistive load against inspiration increase when the orifice size decreases. Evidence exists that this technique as mode of RMT showed a significant increase in inspiratory muscle strength (Hanel & Secher, 1991). A major limitation to this technique is that the resistive loads will change if inspiratory flow and breathing patterns change, and not only if the orifice size changes. It was shown that when ventilatory flow was not carefully monitored during IFRL, it failed to show an improvement in inspiratory muscle strength (Smith et al., 1992).

Chatham et al. (1999) used a modified technique of IFRL, called the test of incremental respiratory endurance (TIRE). A device with an orifice to provide a flow resistive load, an electronic pressure manometer and specially designed software were used to measure sustainable maximum inspiratory pressures. Breathing instructions were given by an audible signal. Subjects (active recreational athletes) had to achieve a pressure-time profile on the computer screen corresponding to 80% of maximal sustainable inspiratory efforts. Subjects had to complete six efforts with a specific resting time between efforts, at six different resistive loads with progressively less resting time per load set. The session was terminated when the subject completed all the levels or when efforts fell below 90% of the pressure-time profile. Both RM strength and endurance significantly improved after 8 weeks of TIRE (3 times a week). The authors suggested that this pressure/flow based training programme contributes to both the force (pressure) and velocity (flow) axis in the force-velocity relationship of the RM.

Enright et al. (2006) also used the TIRE technique and found a significant improvement in maximum inspiratory mouth pressure, sustainable maximum mouth pressure, total lung capacity and diaphragm thickness in moderately trained men and women.

Mickleborough et al. (2008) found significant improvement in maximum inspiratory and expiratory mouth pressure, pulmonary function and respiratory muscle endurance in elite swimmers.

However, in general, the relevance of the TIRE technique is in question, since sustainable maximal efforts are not related to the dynamic function of the RM during exercise (McConnell & Romer, 2004b, Romer & McConnell, 2003b). Other limitations to TIRE are the high costs, it is physically demanding for the subjects and it is time consuming (McConnell & Romer, 2004b).

1.3 Pressure threshold loading (PTL)

Inspiratory pressure threshold loading (IPTL) requires the generation of sufficient pressure to overcome a threshold load and thereby initiate inspiration. McConnell & Romer (2004b) reported that pressure threshold loading has been achieved by a weighted plunger (Clanton et al., 1995), a spring-loaded poppet valve (Caine & McConnell, 2000), a solenoid valve (Bardsley et al., 1993) and a constant negative pressure system (Chen et al., 1998). IPTL has shown significant increases in the maximal force generating capabilities of the RM in sedentary individuals (Edwards et al., 2008), moderately active individuals (Downey et al., 2007) and competitive athletes (Johnson et al., 2007; Romer & McConnell, 2003b; Romer et al., 2002a; Romer et al., 2002b; Romer et al., 2002c; Williams et al., 2002; Volianitis et al., 2001b). IPTL also resulted in significant increases in maximal rates of shortening (Romer & McConnell, 2003b; Romer et al., 2002a; Romer et al., 2002c) and the endurance of the respiratory muscles (Williams et al., 2002; Inbar et al., 2000) in competitive athletes. From the abovementioned studies, it is evident that IPTL improves both the force and velocity generating capabilities of the RM, therefore contributing to both ends of the force-velocity relationships (McConnell & Romer, 2004b).

Recent studies have also shown that expiratory pressure threshold loading (EPTL) can improve expiratory muscle strength (MEP) (Griffiths & McConnell, 2007; Wells et al., 2005; Weiner et al., 2003). Improvements in forced expiratory volume in 1 second (FEV₁) after a period of concurrent inspiratory and expiratory muscle training (CRMT) have also been observed. EPTL have not yet been thoroughly investigated, since the common belief exists that the negative effects of an additional expiratory load increases the intrathoracic pressure and the risks involved in this phenomenon might exceed the potential benefits

(McConnell & Romer, 2004b). A rise in intrathoracic pressure has negative influences on blood flow and stroke volume (see previous chapter).

A commercially available device (*PowerLung*®, *PowerLung*® Inc., Houston, TX, USA) can train both the inspiratory and expiratory muscles, simultaneously, and allows researchers to investigate the effect of EPTL on RM function and whole-body exercise performance. Threshold pressure loading is established in the same manner as inspiratory threshold loading by means of a spring-loaded poppet valve. The present study is one of only a few (Griffiths & McConnell, 2007) to investigate the effect of concurrent IPTL and EPTL training on RM function and exercise performance.

Romer & McConnell (2003b) confirmed the notion of inspiratory muscle training specificity. They found that high-pressure, low-flow RMT produced large improvements in pressure (force generating capacity), while high-flow, low-pressure RMT produced large improvements in flow (velocity of shortening), and intermediate pressure and flow RMT produced uniform improvements in pressure and flow. They found that a high-pressure RMT programme resulted in a 41% increase in maximum inspiratory mouth pressure, but a high-flow RMT programme only showed an 18% increase in flow. They concluded that pressure (force generating capacity) measurements are more responsive to training than flow (velocity of shortening) measurements. They concluded that the force-velocity specific adaptations to the inspiratory muscles due to training are the same as for skeletal muscles (Caiozzo et al., 1981). The easy-to-use, portable hand held devices for PTL training, the ability to quantify the intensity of RMT and the benefits of improving the force and velocity generating capacity of the RM, makes RMT by means of PTL most attractive. However, the technique chosen in a RMT study should be based on the desired type of RM adaptations (RMT specificity), cost, ease of use and time available to do RMT (McConnell & Romer, 2004b).

2. The effect of respiratory muscle training upon exercise performance

Studies that investigated the effect of RMT upon exercise performance have either found a significant improvement in exercise performance or no change in performance. Caution must be taken when summarizing the collective outcomes of these studies, since these studies differ widely with regards to the types of exercise tests used for the evaluation of performance and the fitness levels of the subjects.

Studies that have investigated the effect of RMT on fixed-workload or incremental, time-to-exhaustion performance tests found either significant improvements in sedentary subjects (Stuessi et al., 2001; Markov et al., 2001; Boutellier & Piwko, 1992) and moderately trained to well-trained athletes (Verges et al., 2008a; Verges et al., 2007a; Enright et al., 2006; Gething et al., 2004; McMahon et al., 2002; Spengler et al., 1999; Chatham et al., 1999; Boutellier et al., 1992); or found no changes in moderately trained to well-trained athletes (Downey et al., 2007; Williams et al., 2002; Inbar et al., 2000; Kohl et al., 1997; Fairbairn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987). The exercise performance tests in these studies do not mimic real world field exercise and therefore the relevance of these results is limited in an athletic setting.

In recent years, studies utilizing more relevant performance tests with definite end-points (time trial performances) have shown that RMT led to either an improvement in exercise performance (Johnson et al., 2007; Griffiths & McConnell, 2007; Holm et al., 2004; Romer et al., 2002a; Romer et al., 2002b; Romer et al., 2002c; Volianitis et al., 2001b) or no change in performance (Wells et al., 2005; Sonetti et al., 2001). Relevant to the present study, only one of the latter studies (Romer et al., 2002b) have shown that RMT has a positive effect upon high-intensity, repetitive sprint performance. All the other abovementioned studies investigated the effect of RMT upon endurance performance in endurance athletes or sedentary subjects.

Contrary to previous beliefs that the respiratory system are not a limiting factor to short, high-intensity exercise, evidence exists that respiratory muscle fatigue do develop after high-

intensity exercise (Perret et al., 2000). Additionally, lactic acidosis during high-intensity exercise drives pulmonary ventilation higher and increases the perception of breathlessness (Romer et al., 2002b). Romer et al. (2002b) suggests that intense breathlessness following a sprint activity will influence the recovery time needed for the next sprint. Studies have shown that RMT attenuated blood lactate responses during exercise tests (Spengler et al., 1999; Romer et al., 2002b, Kohl et al., 1997) and reduced perceptual effort sensations of breathing (Romer et al., 2002b; Williams et al., 2002; Volianitis et al., 2001b) during exercise. Evidence also exist that RMT reduces inspiratory muscle fatigue during exercise (Volianitis et al., 2001b). All these factors suggests that sports characterized by high-intensity, repetitive sprints will benefit from RMT. To the researcher's knowledge, only one study (Romer et al., 2002b) have addressed this question.

Of all the studies mentioned above, only two investigated the effect of inspiratory and expiratory muscle training on exercise performance. Griffiths & McConnell (2007) investigated the effect of a 4 week inspiratory or expiratory muscle training programme, as well as a subsequent concurrent inspiratory and expiratory muscle training programme (6 weeks), respectively, on rowing exercise performance. They found that only the inspiratory muscle training programme had a significant effect on a 6 minute all-out rowing ergometer test. Expiratory muscle training, and the subsequent combination of inspiratory and expiratory muscle training, did not elicit any significant changes in the 6 min all-out rowing effort. Wells et al. (2005) found no improvements in a 7×200 m swim test after 12 weeks of concurrent inspiratory and expiratory muscle training. A few reasons may explain the absence of an improvement in exercise performance. It could have been that RM fatigue did not occur during the 7×200 m swim test and therefore did not have an ergogenic effect. Although Lomax & McConnell (2003) reported RM fatigue after a 200 m swim in female swimmers, their swim protocol differed from the protocol used by Wells et al. (2005). In fact, Wells et al. (2005) reported that one of the limitations to the study was that the swimmers did not reach their maximum heart rate, as previously seen during competition, during the 7×200 m swim protocol. Another possible reason could be that the concurrent RMT (combination of inspiratory and expiratory training) did not elicit adequate

improvements in RM function to show an improvement in performance, as suggested by McConnell & Romer (2004b).

The purpose of the present study was to address two relatively unknown areas of RMT, namely the effect of concurrent inspiratory and expiratory muscle training on high intensity, repetitive sprint sport (field hockey).

3. Explanations of contradictory outcomes in the literature

The different outcomes among studies that investigated the effect of RMT upon exercise performance, can be attributed to poor methodological designs in earlier studies and the wide variation in methods among more recent studies.

The foremost explanation for the different outcomes among studies is the type of performance test used to evaluate the ergogenic effect of RMT. In order to find a true effect of RMT, a performance test must be highly reliable and truly simulate real competition situations within the sport. Studies that have used time-to-exhaustion tests at a fixed percentage of maximum work rate or VO_{2max} , failed to adhere to the latter requirements. Time-to-exhaustion at a fixed workload have volitional end-points which can be criticized as unreliable tests (Jeukendrup et al., 1996) and not reflecting real competition situations (Hopkins et al., 1999). Time-trial performances have definite end-points and have shown to be highly reliable (Bishop, 1997) and valid (Coyle et al., 1991) endurance performance tests. The unreliability and low external validity of time-to-exhaustion performance tests could explain why RMT had no ergogenic effect and question the relevance of the studies that did find ergogenic effects. In addition to the type of performance tests used within studies, the duration and intensity of exercise tests could also be reasons for different outcomes.

The variation in fitness levels of the subjects can also determine the outcome of the study. Studies that investigated the ergogenic effect of RMT in sedentary subjects, all showed significant improvements in performance (Stuessi et al., 2001; Markov et al., 2001; Boutellier & Piwko, 1992), while the studies that found no ergogenic effect were done in

well-trained, endurance athletes (Downey et al., 2007; Williams et al., 2002; Inbar et al., 2000; Kohl et al., 1997; Fairbairn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987). Already well-trained athletes will perform very well during a specific performance test, whether it is a time-to-exhaustion test or time-trial test, which will make the outcome of the performance test less sensitive to show an ergogenic effect.

Large inter-individual variance in exercise performance and respiratory muscle function outcomes, as well as small sample sizes, can explain why some studies did show an improvement in exercise performance, but did not reach statistical significance (Fairbairn et al., 1991; Sonetti et al., 2001).

Some studies included no control groups (Enright et al., 2006; McMahon et al., 2002; Stuessi et al., 2001; Markov et al., 2001; Chatham et al., 1999; Boutellier et al., 1992), while others included control groups, but they could not be classified as true placebo groups (Williams et al., 2002; Spengler et al., 1999; Kohl et al., 1997; Fairbairn et al., 1991). Ojaunen (1994) suggested that a placebo group should adhere to certain requirements to be classified as a true placebo: The placebo needs to be inert, and it should generate motivation, expectations and involvement to the same degree as for the experimental group. Another problem is that the intervention programmes were not assigned to subjects in a double-blinded manner (Stuessi et al., 2001; Sonetti et al., 2001; Markov et al., 2001; Inbar et al., 2000; Kohl et al., 1997; Morgan et al., 1987). No control groups, or no true placebo groups, and a lack of double-blinded experimental designs result in weak internal validity, and make them vulnerable to potential influences of subject and/or researcher bias.

Another common error among studies is the lack of quantifying adherence to the RMT programme, and to daily physical activities, respectively. These studies could not contribute changes in exercise performance to the RMT intervention, since they could not tell whether subjects increased the intensity, duration and/or frequency of daily physical activities.

An additional reason why studies could not find a significant improvement in exercise performance after RMT could be due to too little recovery time between cessation of RMT and post-RMT testing of endurance performance (Kohl et al., 1997).

Different RMT protocols and durations of RMT programmes could cause major differences in the outcome of RMT studies. Sonetti et al. (2001) found smaller improvements in inspiratory muscle function after a combination of RM strength (IPTL) and endurance (VIH) training. McConnell & Romer (2004b) suggested that a combination of two types of training could result in sub-optimal adaptations in the respiratory muscles.

Lastly, studies that did adhere to placebo-controlled experimental designs and used time-trial performance tests, failed to measure respiratory muscle fatigue (Wells et al., 2005; Holm et al., 2004; Romer et al., 2002b; Sonetti et al., 2001) before and after exercise performance tests. In these cases, the exercise intensity of the performance tests might not have been adequate enough to induce RM fatigue and therefore reveal no ergogenic effects.

4. Possible mechanisms for improved exercise performance

There is still uncertainty to the exact mechanisms governing the ergogenic effect of RMT. The reason for this is the lack of enough evidence that specifically investigates the mechanisms by which RMT can improve performance. However, some evidence show that the ergogenic effect of RMT is not related to central cardiovascular training effects, but rather related to the training adaptations within the respiratory muscles. These adaptations mediate a few mechanisms that cause an improvement in exercise performance.

An improvement in RM strength and endurance make them more fatigue resistant, therefore relieve or delay RM fatigue. A delay in RM fatigue will cause a relief in the RM metaboreflex vasoconstrictor effects in limb muscles. Adaptations within the RM will improve their mechanical efficiency which means they will demand a smaller fraction of cardiac output. If a smaller fraction of cardiac output goes to the RM and there is less vasoconstrictor activity in limb muscles, it will ensure that more blood flows to the limbs.

This suggests that limb muscle fatigue will be relieved or delayed. During heavy exercise these effects will reduce the perception of RM and limb discomfort and therefore increase exercise tolerance.

Studies on RMT widely supports the fact that RMT improve the force generating capability (Downey et al., 2007; Johnson et al., 2007; Romer & McConnell, 2003b; Romer et al., 2002a; Romer et al., 2002b; Romer et al., 2002c; Williams et al., 2002; Volianitis et al., 2001b), the maximal rates of shortening (Romer & McConnell, 2003b; Romer et al., 2002a; Romer et al., 2002c) and the endurance (Williams et al., 2002; Inbar et al., 2000) of the respiratory muscles. The improvement in RM function might prevent or delay the onset of exercise-induced diaphragmatic fatigue, during heavy exercise, following RMT in some individuals (Verges et al., 2007a). RMT might also prevent the onset of global inspiratory muscle fatigue, as smaller reductions in maximum inspiratory mouth pressure have been observed after heavy exercise following RMT (Griffiths & McConnell, 2007; Downey et al., 2007; Romer et al., 2002c; Volianitis et al., 2001b).

There is also evidence, via muscle biopsies, that RMT causes changes in RM fiber type. Ramirez-Sarmiento et al. (2002) found significant increases in the size of type II fibers and in the proportion of type I fibers of external intercostal muscles after 5 weeks of RMT in COPD patients. These changes were associated with the increase in both the strength and the endurance of the RM. These structural changes within the inspiratory muscles are consistent with increased fatigue resistance and increased mechanical efficiency of the RM (McConnell & Romer, 2004b).

A delay of RM fatigue after RMT may abolish or delay the RM metaboreflex. In turn this will result in more blood flow to the working limb muscles and a delay in limb muscle fatigue. Studies suggested that there is a definite threshold for the activation of the RM metaboreflex (Romer & Polkey, 2008; Wetter et al., 1999). McConnell & Lomax (2006) were the first authors to investigate the direct influence of RMT on the metaboreflex. They demonstrated that RM fatigue induced prior to a plantar flexion activity expedited plantar flexor fatigue. A 4-week specific RMT programme raised the threshold of inspiratory

muscle work needed to elicit the same effect. Thus, it provided evidence that the RM metaboreflex accelerated the rate of calf muscle fatigue during plantar flexion, and specific RMT attenuated this effect.

Witt et al. (2007) found significant increases in heart rate (HR) and mean arterial pressure (MAP), in both the experimental and sham groups, after a carefully controlled resistive breathing task (RBT). However, after a 5-week RMT intervention, the sham group showed similar HR and MAP responses to the RBT, while the experimental group failed to show the same rate of increase in HR and MAP as before. They attributed the attenuated cardiovascular responses to a reduced activity of chemosensitive afferents within the inspiratory muscles. They suggested that this may provide a mechanism for improved exercise performance associated with RMT. The outcome of the studies by Witt and colleagues (2007), and McConnell & Lomax (2006), confirm that RMT does delay or attenuate the RM metaboreflex and result in an increase in limb blood flow and limb muscle work.

Other mechanisms that possibly contribute to improved exercise performance are perceptual changes in RM and limb muscle discomfort. High levels of discomfort are experienced during high levels of fatigue in RM and limb muscles, respectively. However, attenuation of RM and limb muscle fatigue, attenuate the perception of discomfort and prolong performance (Romer & Polkey, 2008). Another suggestion is that the constant exposure to high accumulative metabolites within the RM during RMT, desensitize the responsiveness of the type III and IV afferents to metabolite accumulation. This causes a less pronounced sympathetically mediated vasoconstrictor outflow to limb muscles (Witt et al., 2007).

Previous studies investigating the RM metaboreflex were all concerned with inspiratory muscle training and its ergogenic effects. The two known studies that investigated expiratory muscle training (Griffiths & McConnell, 2007) and a combination of inspiratory and expiratory muscle training (Wells et al., 2005) on exercise performance found no significant improvements in performance. Therefore, the effect of expiratory muscle training on the expiratory muscle metaboreflex is still unknown.

C. RELEVANCE OF RESPIRATORY MUSCLE TRAINING IN TEAM SPORT PLAYERS

It is understandable that studies that investigated the ergogenic effect of RMT have looked, almost exclusively, at endurance sports. Most RMT research has been done in competitive cyclists (Johnson et al., 2007; Sonetti et al., 2007; Holm et al., 2004; Romer et al., 2002a; Romer et al., 2002c; McMahon et al., 2002; Fairbairn et al., 1991; Morgan et al., 1987). Some studies focused on rowers (Griffiths & McConnell, 2007; Volianitis et al., 2001b), one study looked at cross-country runners (Williams et al., 2002) and one involved competitive swimmers (Wells et al., 2005). Most other studies used subjects who were physically active (by means of running, swimming and cycling) and others participated in recreational sports (Downey et al., 2007; Verges et al., 2007a; Enright et al., 2006).

In this study, field hockey was used as the exercise model to study the effects of RMT in team sport athletes. Hockey can be characterized as a high-intensity, repeated sprint sport, requiring mostly high levels of anaerobic fitness (Reilly & Borrie, 1992). Aerobic fitness levels are not the most important contributor to success in hockey performance, and an optimal aerobic fitness is only needed for adequate recovery during short durations of rest between sprints. However, Boyle et al. (1994) found that the mean oxygen uptake during competition was $48 \pm 5 \text{ ml.kg}^{-1}.\text{min}^{-1}$ and the rate of energy expenditure during match play ranged from 61 to 83 kJ.min^{-1} in nine international hockey players. They concluded that competitive hockey matches place heavy demands on the aerobic system.

The competition schedule of major international hockey events (i.e. Olympic Games and World Cup) requires players to play seven to nine games within a fortnight. Therefore, quick recovery is needed for optimal performances in all games scheduled one or two days apart. It can be hypothesized that performance will decrease as whole body fatigue progressively sets in from match to match. Spencer et al. (2005) investigated the movement changes in hockey players during match play in three separate matches, played within 4 days. They found that the total amount of time spent standing increased significantly, and the amount of time running decreased significantly over the 4 days. They concluded that

repeated sprint ability is progressively reduced when hockey players compete in subsequent games with only 24 to 48 hours available for recovery.

Overall, whole-body fatigue may partially be attributed to RM fatigue. Studies have shown that RM fatigue occurred at high (80 – 85% of $\text{VO}_{2\text{max}}$) exercise intensities in endurance type exercise (Babcock et al., 2002; Johnson et al., 1993). The mean heart rate (HR) recorded during a match in eight national women hockey players (26 ± 3 years old) was 173 ± 8 bpm, which was equivalent to $89 \pm 4\%$ of their age predicted HR_{max} . The percentage of age predicted HR_{max} was converted to a predicted value of percentage $\text{VO}_{2\text{max}}$ by a regression equation determined by Swain et al. (1994). The calculations resulted in a predicted exercise intensity of $82 \pm 6\%$ of $\text{VO}_{2\text{max}}$ (personal communication with the fitness coach of hockey players participating in the study, 2007). Thus, the mean exercise intensity of international hockey matches correspond to the exercise intensity at which RM fatigue occurred. It is therefore likely that RM fatigue may occur during match play at international level. Lawrence & Polglaze (2000) also reported heart rate and lactate analysis of international competition in hockey players, indicates that players use on average 75 – 85% of their maximum oxygen uptake. This statement confirms that the exercise intensity during match play is high enough to induce RM fatigue. One can speculate that an improvement in RM strength, endurance and efficiency, through RMT, will partially attenuate whole-body fatigue with progressively more match play, due to the relief in exercise-induced RM fatigue. However, to date, no study has directly investigated this rationale.

Another rationale for RMT in high-intensity, repeated sprint sports is the evidence that RMT improves recovery between repeated sprints. To the researcher's knowledge, there is only one study available in the literature regarding this matter. Romer et al. (2002b) stated that optimal performance in repeated sprints require adequate recovery between sprints. High-intensity exercise causes high levels of lactic acidosis, which in turn causes an increase in hyperventilation. High levels of hyperventilation will also evoke perceptual sensations of breathlessness within the athlete. The intensity of breathlessness will make the athlete decide when to sprint again, and contribute to team performance. The study by Romer et al. (2002b) showed that recovery time during a repeated sprint test improved by 6%, following

RMT in the experimental group, while the sham training group showed no significant changes. It can be concluded that RMT reduced the perceptual sensations of breathlessness within the athletes which led to a shorter recovery time. Romer and colleagues (2002b) investigated this phenomenon by using a variety of repeated sprint athletes (soccer, rugby, basketball). Two subjects were field hockey players.

D. RESPIRATORY MUSCLE DETRAINING

Detraining can be defined as the complete or partial loss of the training-induced adaptations, in response to an insufficient training stimulus. The reversal of training adaptations differs between well-trained individuals (several months to years of training) and recently trained individuals (training ~ 3 months). VO_{2max} declines markedly within 12-21 days and will deteriorate further if detraining continues, but will stay above (50%) untrained levels in well-trained individuals (Coyle, 1998). However, in recently trained individuals, recently acquired VO_{2max} gains will be completely lost (Mujika & Padilla, 2000). Consequently, a reduction in endurance performance at maximal and submaximal levels is observed, due to marked reductions in cardiorespiratory functions, muscle metabolic activities (Mujika & Padilla, 2001a) and muscle morphology (Mujika & Padilla, 2000). A reduction in cardiorespiratory function is associated with a decrease in blood volume resulting in a decreased stroke volume and cardiac output (Coyle et al., 1985). Additionally, after longer periods of inactivity (4 weeks), muscle glycogen levels decline, carbohydrate utilization increases and lactate threshold is lowered. Muscle metabolic changes, due to inactivity, includes a decline in capillarisation, arterial-venous oxygen difference and oxidative enzyme activities. Muscle characteristic changes include muscle fiber distribution and fiber cross-sectional area changes (Mujika & Padilla, 2000).

Previous studies have shown that specific respiratory muscle training (RMT) does not elicit central circulatory adaptations as in the case during whole-body endurance training (Mier et al., 1997; Wolfe et al., 1979). Markov et al (2001) found that a 15-week RMT programme in sedentary subjects did not increase cardiac stroke volume, at a workload normally eliciting maximal values, during a constant-load exercise test. Therefore, it would be logical

to suggest that RMT detraining will not elicit any changes to the cardiovascular system as in the case of whole-body training. Instead, changes in endurance performance due to RMT can be attributed to the adaptations in the RM. These muscle adaptations provide an improvement in the force-generating capacity (Downey et al., 2007; Johnson et al., 2007), the maximal rate of shortening (Romer & McConnell, 2003b) and the endurance (Williams et al., 2002) of the respiratory muscles.

Therefore, the essence of RM detraining is the reversal of adaptations to the RM, induced by specific RMT. Only one study (Romer & McConnell, 2002b) have investigated the effect of detraining on RM function. The study only suggested possible mechanisms for the decrease in RM function, but did not specifically investigate or measure these mechanisms. Boutellier & Piwko (1992) briefly mentioned in their study that the improvements in respiratory muscle endurance, after a 4-week VIH training programme, returned to baseline values at 18 months after completion of the initial study in healthy sedentary subjects.

1. Detraining changes in limb muscles

In the majority of studies (including the present study), training of the RM in either well trained subjects or sedentary subjects was a novice task. Thus, these subjects could be classified as recently trained athletes. Therefore, the reversal of adaptations within recently trained athletes is described. The measurement of RM strength during RMT and RM detraining is similar to the measurement of limb muscle strength during whole-body endurance training and detraining.

Strength performance decreases only after long periods of detraining (> 4 weeks). Sysler & Stull (1970) found a 27% decrease in isokinetic strength endurance after 5 weeks of detraining. Shaver (1975) found significant losses of isometric strength performance of 2, 3 and 3.2% after 4, 6 and 8 weeks of detraining, respectively. The subjects in this study followed a 6-week high-intensity strength training programme.

Colliander & Tesch (1992) found that the strength gains during a 12-week concentric strength training programme were lost at a faster rate compared to the strength gains during a 12-week concurrent, concentric and eccentric strength training programme. They concluded that eccentric training is necessary to ensure that training-induced adaptations will last for a longer period of time during inactivity. Housh et al. (1996) agreed with the study of Colliander & Tesch (1992), by showing that strength gains achieved during an 8-week eccentric resistance training programme was retained at 100% after 8 weeks of training cessation. Hodikin (1982) showed that college students maintained their speed-strength after a 1.5 month training break, but it decreased by ~ 16% after 3 months of training cessation. Hodikin (1982) concluded that speed-strength was better maintained when the previous training programme included explosive strength training. Therefore, the maintenance of strength during periods of inactivity depends on the training method.

The reversal of muscle adaptations during detraining include a decrease in capillary density, arterial-venous oxygen difference, oxidative enzyme activity, type I fiber distribution, force production, eccentric force production and sport specific power. All these changes have a negative influence on endurance performance.

Moderate endurance training lasting several months will increase capillary density in the working muscles by 20 – 30% (Klausen et al., 1981; Ingjer, 1979). However, these improvements are fully or partially reversed after 8 weeks of detraining. Klausen et al. (1981) found a significant decrease in capillary density to 109% of pretrained values after 4 weeks of inactivity in recently trained subjects. Subjects followed an 8-week training programme which elicited an increase in capillary density to 122% of pretrained values, prior to the detraining period. The only available evidence on the decrease of arterial-venous oxygen difference during detraining was done on highly-trained athletes. However, it can be assumed that these changes also occur in recently trained athletes. Coyle et al. (1984) found no changes in arterial-venous oxygen difference after 21 days of detraining, but after 56 days and 84 days there was a 4% and 7% decrease in arterial-venous oxygen difference, respectively.

Moderate endurance training of 2 – 4 months causes an increase in mitochondrial enzyme activity of 20 – 40%. If training is stopped these improvements decrease rapidly and return to pretrained values within 28 – 56 days (Klausen et al., 1981; Coyle et al., 1984; Costill et al., 1985). Wibom et al. (1992) found a 4 – 17% decrease in mitochondrial enzyme activity after 3 weeks of inactivity. The values after 3 weeks of detraining were still 50% above pretrained values which was obtained prior to 6 weeks of training, in sedentary individuals. Moore et al. (1987) found a larger decrease of 25% in mitochondrial enzyme activity after the same period of detraining in recently trained (7-week training programme) subjects. The decreased value was still 19% above pretrained values.

Fiber distribution changes due to training and detraining depend on the type (endurance or strength) of exercise training (Mujika & Padilla, 2001b). In recently trained subjects (8 weeks endurance training), Klausen et al. (1981) found no change in fiber distribution after 4 weeks of detraining. However, the percentage of slow-twitch (ST) fibers decreased by 6% and the percentage of fast-twitch (FT type IIa and type IIb) increased by 3% after 8 weeks of detraining. Klausen et al. (1981) also reported changes in the cross-sectional area of the different fiber types after exercise cessation. Eight weeks of endurance training increased muscle fiber cross-sectional area of all fiber types with ~ 5%. Muscle fiber cross-sectional area decreased to 99, 98 and 96% of pretrained values in ST, FT type IIa and FT type IIb, respectively, after 4 weeks of detraining and decreased further to 93, 98 and 94% of pretrained values after 8 weeks of detraining, respectively.

2. Detraining of the respiratory muscles

It is already known that RMT increases RM strength (Downey et al., 2007; Romer & McConnell, 2003b; Romer et al., 2002a), RM endurance (Williams et al., 2002; Inbar et al., 2000) and it reduces the severity of RM fatigue after heavy exercise (Griffiths & McConnell, 2007; Volianitis et al., 2001b). It has been shown that RMT does not elicit changes to the central circulatory (cardiovascular) system (Markov et al., 2001), but it has an ergogenic effect on exercise performance (Griffiths & McConnell, 2007; Johnson et al., 2007). These findings, imply that RMT elicits training-induced adaptations to the RM. Additionally,

evidence exists of changes in fiber distribution after RMT (Ramirez-Sarmiento et al., 2002). However, limited information is available on the specific adaptations to the RM, therefore one can only speculate that these adaptations are similar to adaptations to limb muscles during specific whole-body training. It is speculated that the reversal of the RM adaptations correspond to the reversal of adaptations to limb muscles.

Romer & McConnell (2003b) investigated the effect of detraining on RM strength, inspiratory air flow and inspiratory power. Inspiratory air flow was the rate at which a certain volume of air could be inspired ($L \cdot s^{-1}$) by overcoming a specific inspiratory load. In this study, 24 healthy individuals were divided into three different inspiratory muscle training (IMT) groups and a control group. One group was assigned to a high-pressure, low-flow group; one to a high-flow, low-pressure group and one to an intermediate pressure-flow group (all groups, $n = 6$). All IMT groups showed a 9 – 41% increase in maximum inspiratory mouth pressure, 7 – 26% increase in inspiratory flow and 25 – 64% increase in inspiratory power. After 9 weeks of detraining, there was a significant decrease of 7, 9 and 16% in post-training values for maximum inspiratory mouth pressure, flow and inspiratory pressure, respectively. No further decreases occurred between 9 and 18 weeks of detraining.

These findings agree with studies on limb locomotor muscles. It is also in agreement with the loss of skeletal muscle strength of 8 – 12% after 8 – 31 weeks of detraining (Lemmer et al., 2000; Hakkinen et al., 1981). Inspiratory power suffered a 17% decrease after 9 weeks of detraining compared to only a 7% decrease in RM strength. This finding is similar to the finding of Neuffer et al. (1987), who found a 14% decrease in limb muscle power, but no change in muscle strength. In contrast to limb muscle detraining studies, the decrease in RM function do not revert to pretrained values after 3 and a half months of RM detraining.

A few shortcomings in the study by Romer & McConnell (2003b) leave critical questions about RM detraining unanswered. The study did not measure the specific adaptations to the RM responsible for the increase of RM strength, airflow and inspiratory power, and therefore did not determine the reversal of the specific adaptations to the RM during detraining. Secondly, the authors did not establish whether the adaptations to the three

different types of IMT responded differently to the period of inactivity, which is true for limb muscles (Hodikin, 1982). It is clear that thorough research is needed to reveal whether the assumptions of the adaptations elicited by RMT is reversible and to what extent.

E. CONCLUSION

The ergogenic effect of RMT is widely documented in the literature, with studies showing that specific RMT improves constant-load exercise and time-trial endurance performance, in sedentary and well-trained athletes. However, the findings of an ergogenic effect are still controversial. The reasons for the controversy among studies can be attributed to the differences in the type, intensity and duration of the exercise test to quantify exercise performance, the type, intensity and duration of the RMT programme, too small sample sizes, the lack of quantification of RMT adherence, and, either a lack of a control group and/or a true placebo group.

There are three types of RMT: voluntary isocapnic hyperpnoea (VIH), inspiratory flow resistive loading (IFRL) and pressure threshold loading (PTL). Pressure threshold loading seems to be the best type of RMT, since it contributes to both ends of the force-velocity relationship of the RM and this type of training is more responsive to elicit improvements in RM strength and endurance than high-flow RMT.

The specific mechanisms by which RMT improves performance still needs further investigation. However, it is clear that RMT elicits structural and functional improvements to the RM, which delay or relief respiratory muscle fatigue during exercise. This causes a delay in the RM metaboreflex, which allows more blood flow to the limb locomotor muscles and the delay of limb muscle fatigue. The delay or relief in RM fatigue also elicits less sensory input to the central nervous system (CNS) which lowers the perceptual sensation of breathing discomfort. RMT also causes a constant exposure to high concentrations of metabolites within the RM, which can desensitize the receptors of type III and IV afferents, which also elicits less sensory input to the CNS.

Two suggestions are made why hockey players may improve their performance by specific RMT. It has been shown that hockey players play matches at exercise intensities high enough to induce RM fatigue. RMT may relieve RM fatigue at these high exercise intensities. Improved RM function and efficiency can improve recovery time between repeated sprints, which is one of the most important contributors to improved performance.

RMT does not elicit improvements to the cardiovascular system (Markov et al., 2001), but it does elicit adaptations within the RM, which improves their strength, endurance and efficiency. Therefore, the essence of RM detraining falls upon the reversal of the specific adaptations to the RM. Only one study has investigated the effect of detraining on RM function (Romer & McConnell, 2003b) and found a significant reduction after 9 weeks of inactivity. However, the reduced values still remained above pretrained values and remained constant up to 18 weeks post training. The specific changes within the RM due to detraining have not yet been investigated. However, the reversal of adaptations in skeletal muscle detraining are well established. Since RMT elicits the same adaptations to RM as whole-body training to skeletal muscles, it is suggested that the reversal of adaptations to the RM are similar to that of skeletal muscle detraining.

CHAPTER FOUR

PREDICTORS OF RESPIRATORY MUSCLE STRENGTH AND ENDURANCE

A. INTRODUCTION

It is well established that anthropometry is an important predictor of lung function in the general healthy population. Pulmonary function prediction equations are generally based on gender, age and height regressions, while body mass usually has little or no association with pulmonary function (Ferris et al., 1965; Knudson et al., 1976). Lazarus et al. (1998) found that fat free mass (FFM) is positively correlated with FVC in 1235 subjects (men and women). It was speculated that extra fat mass, particularly in the trunk area, may cause a mechanical restriction on ventilatory function. The findings of Lazarus et al. (1998) suggested that prediction equations for pulmonary function could be improved by the inclusion of FFM in regression equations. Leech et al. (1983) studied 369 men between the ages of 15 to 35 years and 555 women between the ages of 13 to 35 years and found that for men and women, height was the major determinant of FVC and FEV₁ ($r = 0.45 - 0.46$ and $0.41 - 0.43$, respectively).

Apart from pulmonary function measurements, the assessment of respiratory muscle (RM) strength has become a quick, easy and non-invasive method to assess respiratory muscle weakness both in healthy subjects and in patients with COPD or neuromuscular diseases (Karvonen et al., 1991). Predicted values for respiratory muscle strength needed to be developed in order to establish a clear distinction between RM weakness and acceptable values for adequate and normal ventilation, and to successfully monitor changes in ventilatory status over time in these patients. Thus, a variety of studies have established predictive equations for RM strength in the general healthy population in order to set up reference values for normal RM strength (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992; Chen & Kuo, 1989; Wilson et al., 1984; Black & Hyatt, 1969). From these studies, it is evident that the same predictors for

lung volumes and airflow, i.e. gender, age, standing height and body mass, also predict RM strength in the general population.

The question arises whether these reference values for RM strength are also suitable for an athletic population. Crosbie et al. (1979) found that divers with five to ten years diving experience had significantly larger FVC values than those divers with less than one, and between one and five years, experience. Eastwood et al. (2001) also demonstrated that trained marathon runners had significantly higher lung functions, e.g. TLC, FVC and FEV₁ compared to sedentary subjects (athletes: TLC = 7.8 ± 0.6 L; FVC = 5.8 ± 0.6 L; FEV₁ = 4.7 ± 0.4 L.s⁻¹; non-athletes: TLC = 7.1 ± 0.7 L; FVC = 5.4 ± 0.6 L; FEV₁ = 4.4 ± 0.4 L.s⁻¹). Morrow et al. (1989) also found significantly higher lung volumes, inspiratory- and expiratory flows in athletes compared to sedentary subjects. These findings suggest that a long-term background of endurance training may lead to lung function adaptations and that athletes have superior lung volumes and air flows compared to non-athletes. However, the distinct differences in RM strength between athletes and non-athletes are less clear. Some studies showed no differences between marathon runners (Eastwood et al., 2001), skiers (Coast et al., 1990), long distance runners (Cordain et al., 1990), runners and swimmers (Armour et al., 1993) and sedentary subjects, respectively. On the contrary, Fuso et al. (1996) showed significant differences in maximum inspiratory mouth pressure between soccer players and sedentary subjects.

Superior athletic performance is a function of the complex interaction of various physiological, psychological, biomechanical and anthropometrical factors. Most system models that are used to analyze sport performance include these factors. Firstly it is known that athletes who compete at international level generally display distinctive body sizes, shapes and composition compared to the non-athletic population (Pitsiladis, 2005). Secondly, it is well accepted that there is a relationship between competitive sport performance and physical traits or anthropometry (Wyndham et al., 1971; Claessens et al., 1999; Slater et al., 2005). Thirdly, different sports command different anthropometry. Sports requiring abilities like running and jumping demand relatively low body fat, whereas strength and power activities demand larger muscle mass (Heyward & Wagner, 2004).

Among endurance athletes it has been shown that track event athletes are taller, because leg length is longer. A taller athlete has greater leverage of the muscles which contribute to the amplitude of movement compared to a shorter athlete. Swimmers need propulsive leverage and therefore the length of the arm and leg plays an important role. In rowers and canoeists all of the above, as well as sitting height, are considered important determinants of athletic success. Endurance athletes usually also have a medium body build, with a somatotype between mesomorph and ectomorph, while strength endurance athletes are usually mesomorphic (Heath & Carter, 1967). It is well known that if reference values are used to predict whether the observed values are up to standard or not for a specific subject group, that specific subject group should accurately reflect the status of the group in the reference study. Thus, the observed subjects and the reference subjects should be homogeneous in morphology (Ackland, 2006).

If athletes have superior RM function compared to the normal sedentary population, the use of reference values based on the normal population will be inadequate and will probably underestimate their RM function. In addition, the distinct differences in the morphology among athletes of different sports and between successful athletes and the general population, indicate that predicted values for RM function based on the general population (or based on different types of athletes) will not be suitable to use as reference values for a specific athletic group. A further question is whether there are other significant predictors for RM function in athletes that differ from the predictors in a non-athletic population.

Only a few studies have investigated the predictors of RM strength in an athletic population (Fuso et al., 1996; Cordain et al., 1990). Reference values for athletic populations are of importance to coaches and sport scientists in order to evaluate whether athletes have adequate RM function, and to monitor for improvements in RM fatigability and strength that will subsequently improve athletic performance.

B. PREDICTORS OF RESPIRATORY MUSCLE STRENGTH

It is evident from the literature that most anthropometric variables have a correlation with maximal respiratory pressures. However, these correlations can be considered weak to moderate. Very few studies have been done on the respiratory muscle function and its relationship with anthropometric parameters in trained individuals. Athletes have significantly different anthropometric characteristics and body composition compared to non-athletic and sedentary individuals, and distinct morphological differences also exist among athletes of different sports. Therefore, the question arises whether there are perhaps clearer relationships between the anthropometry and respiratory muscle function of trained individuals, and whether training itself is responsible for the variation in RM strength values among individuals.

1. Predictors of respiratory muscle strength in general populations

The relationship between physical characteristics and respiratory muscle strength has been the focus of a few studies. The regression equations that resulted from these descriptive studies usually incorporate age, height and/or body mass as independent variables, similar to the prediction equations for pulmonary functions. It can be suggested that if pulmonary function measurements strongly correlate with RM strength measurements, the same predictors for pulmonary function will account for the variance in RM strength. Hautmann et al. (2000) found significant but low correlations ($r = 0.39, 0.40, 0.36$) between FVC, PEFR, and FEV₁ and MIP, respectively. Leech et al. (1983) reported significant ($P < 0.01$), but low beta coefficients ranging from 0.142 to 0.313 between FVC, FEV₁, and PEFR and an index of RM strength (sum of MIP and MEP). In both studies, it is clear that there is a relationship between pulmonary function variables and RM strength. However, the reported relationships are not strong enough to confirm that the same predictors for pulmonary function will explain the variance in RM strength in a normal population.

Harik-Khan et al. (1998) pointed out that from all the previous studies, there is only agreement on the fact that gender is a significant determinant of RM strength, measured as

maximum inspiratory or expiratory mouth pressure. Other factors, such as age, height, body mass, physical activity level and smoking history have been investigated, but conflicting results have been reported.

1.1 Gender

In all previous studies it has been shown that there are significant differences in the respiratory muscle strength of men and women (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992; Wilson et al., 1984; Chen & Kuo, 1989; Leech et al., 1983; Black & Hyatt, 1969). Black and Hyatt (1969) suggested that MIP and MEP values for women are usually between 65 – 70% of those obtained in men. This was confirmed by Harik-Khan et al. (1998) when they reported that MIP values were on average 30% higher in men than in women across all age groups (20 – 90 years). Hautmann et al. (2000) reported that all women in their study obtained 25% lower MIP values compared to those of the men. Fiz et al. (1998) reported a significant correlation between gender and MIP values ($r = 0.37$, $P < 0.001$) and a higher correlation between gender and MEP ($r = 0.55$, $P < 0.001$).

On average, men (at age 18) have about 50% more muscle mass than women in the upper body, and 10 to 15% more muscle mass in the lower body. Men have larger branching bronchi and on average, about 30% greater lung volume than women (Glucksman, 1981). This explains the large differences between men and women in respiratory muscle function variables. The reason for the large differences in RM strength between men and women can be attributed exclusively to the larger RM mass in men enabling the RM to contract more forcefully to generate a higher pressure than those of women. It is therefore established that gender is one of the best predictors of the variance in MIP and MEP values. Therefore, most studies exclude gender from the regression analysis for MIP and MEP, and report regression equations for men and women, respectively.

1.2 Age

Age is included in all prediction equations reported in the literature (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992; Wilson et al., 1984; Chen & Kuo, 1989; Leech et al., 1983; Black & Hyatt, 1969), which makes it the most consistent and strongest predictor (to the same degree as gender) in normal, healthy populations. Most studies have found a significant negative correlation between age and RM strength in both men and women (Hautmann et al., 2000; Neder et al., 1999; McConnell & Copestake, 1999; Fiz et al., 1998; Enright et al., 1994; Bruschi et al., 1992). The studies of Hautmann et al. (2000), Neder et al. (1999), Fiz et al. (1998) and Bruschi et al. (1992) were done on subjects (large sample sizes of 99 – 625) ranging across wide age ranges (~18 to 70 years), while McConnell & Copestake (1999) and Enright et al. (1994) investigated the relationship restricted to the elderly population (> 59 years) in a small sample size (n = 39) and a large sample size (n > 4000), respectively.

Most studies (Wilson et al., 1984; Berry et al., 1996) have only noted significant effects of age on MIP in older men, or older women (Black & Hyatt, 1969). Bruschi et al. (1992) found that age only significantly influenced MEP in older women (> 55 years), but had no influence on MIP values across the wide age range in both men and women. Black & Hyatt (1969) found no significant relationship between MIP and MEP, and age, respectively, in subjects younger than 55 years, while MEP decreased with age in both men and women in subjects older than 55 years. From these studies it appears that the effect of age on RM strength is only manifested at later stages in life (> 55 years old).

It is known that skeletal muscles are affected by ageing. The force production ability of skeletal muscles decreases between 20 to 40% with increasing age. This is accompanied by a decrease in the size and muscle mass of the fibers. These changes occur because of muscle disuse related to the general reduction in physical activity. However, because the respiratory muscles are chronically active, even in the elderly, respiratory muscles might be differently affected by age than locomotor muscles. However, it is known that diaphragm strength is reduced with age (Polla et al., 2004), therefore the decrease in RM strength with increasing

age can mainly be attributed to the reduction in diaphragmatic and respiratory accessory muscle mass, mainly due to a reduction in type IIX muscle fibres (Polla et al., 2004), and a decline in the work output for the same level of neural stimulation (Enright et al., 1994). The pressure generated during forced expiration (MEP) can also be dissipated by the increased compliance of the abdominal compartment in older subjects (Neder et al., 1999). Ageing of the respiratory system is also associated with a decrease in lung elastic recoil and a decrease in the compliance of the chest wall and the respiratory system (Janssens, 2005).

1.3 Height

The findings of standing height as an important predictor for RM strength are somewhat controversial within the literature. Studies have shown that height correlates stronger with pulmonary function variables than RM strength variables, and height is therefore included in the majority of prediction equations that are used as reference values for normal pulmonary function. For instance, Leech et al. (1983) reported significant correlation coefficients of 0.41, 0.45 and 0.54 between height and FEV₁, FVC and TLC in men (n = 369). Johan et al. (1997) reported correlation coefficients of 0.63 and 0.53 between height and FVC in men (n = 277) and women (n = 175), respectively.

The relationship between height and RM strength variables has been less clear with studies reporting equivocal findings. Studies in which both men and women are included, either found only significant correlations between height and RM strength in women (McConnell & Copestake, 1999; Wilson et al., 1984), or only found significant correlations in men (Fiz et al., 1998; Johan et al., 1997). Overall, correlations of height with MIP ranged from 0.32 to 0.49 (Hautmann et al., 2000; Neder et al., 1999; Fiz et al., 1998; Bruschi et al., 1992) and correlations with MEP ranged from 0.49 to 0.59 (Neder et al., 1999; Bruschi et al., 1992).

Overall, it seems that correlation coefficients for height with inspiratory muscle strength are lower than with expiratory muscle strength. Harik-Khan et al. (1998) on the other hand found a significant negative correlation between height and inspiratory muscle strength. They suggested that smaller women usually have a more muscular build and this could

explain why height negatively correlated with MIP. However, the finding by Harik-Khan et al. (1998) completely disagrees with other studies who found only weak to moderate positive correlations between height and either MIP or MEP. The taller individual has a larger abdomen which probably indicates larger muscle mass and greater force generating capacity, especially with forced expiration, which may explain the stronger correlation of height with MEP compared to MIP.

Two studies found that height was a significant predictor for RM strength only in women (Harik-Khan et al., 1998; Wilson et al., 1984), while Johan et al. (1997) and McConnell & Copestake (1999) found that height were included in the regression equations for MIP and MEP in both men and women. Hautmann et al. (2000), Neder et al. (1999), Bruschi et al. (1992) and Black & Hyatt (1969) found that height did not significantly contribute to the variance in RM strength measurements.

1.4 Body mass and indices of body mass

Neder et al. (1999) and McConnell & Copestake (1999) found moderate to moderately-high positive correlations ($r = 0.49 - 0.69$) between body mass and RM strength measurements, however McConnell & Copestake (1999) only found this in women. Hautmann et al. (2000), Johan et al. (1997) and Bruschi et al. (1992) also found significant relationships between body mass and RM strength, however correlation coefficients were low ($r = 0.18 - 0.38$). Neder et al. (1999), Johan et al. (1997), Harik-Khan et al. (1998) and Enright et al. (1994) have included body mass as a co-variant in their prediction equations. The reason for the significant relationship between body mass and RM strength could be attributed to the fact that changes in body mass are associated with changes in diaphragm muscle mass (Arora & Rochester, 1982).

McConnell & Copestake (1999) found a positive correlation between BMI (body mass index) and RM strength measurements in women. Bruschi et al. (1992) also reported significant correlations between BMI and MIP and BMI and MEP. The regression equation of Hautmann et al. (2000) included BMI as a predictor of MIP only in male subjects. These

positive correlations are logical, since an increase in BMI is associated with an increase in muscle mass and subsequently an increase in strength.

Body surface area (BSA) significantly correlated with MIP ($r = 0.42$) and MEP ($r = 0.48$) in 625 subjects (Bruschi et al., 1992). McConnell & Copestake (1999) also reported moderately-high correlation coefficients between BSA and MIP ($r = 0.69$) and MEP ($r = 0.65$). Bruschi et al. (1992) included BSA as a co-variant in their prediction equations, together with gender and age. The positive relationship between BSA and RM strength can be explained by an overall larger muscle mass (only if fat mass falls within a normal healthy range), which indicates greater RM mass compared to subjects with a lower BSA.

1.5 Physical activity

McConnell & Copestake (1999) found strong positive correlations between physical activity (expressed as kcal/day) and MIP and MEP ($r = 0.87$ and 0.67 , respectively) in a small sample ($n = 10$) of healthy, elderly individuals. They suggested that this relationship may be explained by the fact that respiratory muscles respond to training in the same way as other skeletal muscles, and thus more physically active individuals should also have stronger respiratory muscles, and therefore higher MIP and MEP values. In a study by Neder et al. (1999), the physical activity score (calculated as an index of the amount of physical activity per individual per questionnaire) significantly correlated with MIP ($r = 0.47$) and MEP ($r = 0.46$) in 100 non-athletic, healthy subjects ($P < 0.01$). However, neither of the studies found that their index of physical activity was a co-variant in their prediction equations for RM strength and none of the subjects in these studies can be classified as athletes.

2. Predictors of respiratory muscle strength in athletic populations

In competitive runners, Cordain et al. (1987) reported no correlation of FVC with neither MIP ($r = -0.04$) nor MEP ($r = 0.09$). No relationships between MIP and MEP values and pulmonary function variables indicate that other factors than gender, age and height may be better predictors of RM strength in runners. This scenario might be true for other athletic

groups as well. Studies in the literature are in disagreement to whether physical activity qualifies as one of the other factors that influence RM strength measurements more than anthropometry. From all the above mentioned factors influencing RM strength variability in general populations, only gender and age seem to definitely account for the wide variability in measurements. However, the disagreement among studies about the influence of height, age, body mass, body mass indices and physical activity provokes the question: Why do some subjects (of same age and gender) have stronger respiratory muscles than others?

Fuso et al. (1996) found that competitive soccer players had significantly higher MIP values (15.7 cmH₂O) compared to sedentary individuals. However, the MIP measurements were obtained from functional residual capacity (FRC), not from residual volume (RV). MIP measurements from FRC are not recognized at the standardized volume from which MIP efforts are initiated. MIP measurements from RV can control for the initial length of the inspiratory muscles and it ensures that the highest pressures are recorded, which is not possible from FRC (Green et al., 2002).

Other studies found no significant differences in MIP or MEP measurements between athletes and non-athletes (Eastwood et al., 2001; Armour et al., 1993; Cordain et al., 1990; Coast et al., 1990). The athletes in these studies were long distance runners, swimmers and cross-country skiers. The majority of studies therefore suggest that physical training probably do not provide athletes with superior RM strength compared to non-athletes. However, what makes this finding confusing is the fact that a few studies have shown that endurance training significantly improves RM strength (Powers & Criswell, 1996; Clanton et al., 1987; Robinson & Kjeldgaard, 1982). On the other hand there are also studies that have found no changes in RM strength measurements after a period of endurance training (Thomas et al., 1998; Hanel et al., 1994; O’Kroy & Coast, 1993).

Fuso et al. (1996) investigated whether being a soccer player or not, significantly predicted MIP in 130 subjects (27 were soccer players and the rest were general, sedentary subjects). Predictive models for MIP measured from FRC and MIP measured from RV resulted in only weak predictions. This indicated that these factors, gender, age, and BMI only accounted for

38% of the variation in MIP from FRC and 36% of the variation in MIP from RV, respectively. The factor, being a soccer player or not, was also included in the predictive models for MIP, and it significantly contributed to the variation in MIP from FRC, but not from RV. However, Fuso et al. (1996) did not report the R^2 values for the predictive models which included the dichotomic variable (not a soccer player = 0, soccer player = 1). Other factors not measured or included in the predictive models accounted largely for the variation in MIP measurements.

Cordain et al. (1987) also investigated the factors that influence MIP and MEP measurements in male competitive runners (n =101). Age significantly correlated with MIP (r = -0.43) but did not correlate with MEP. Age was recognized as the only variant in the regression equations for predicted values for MIP and MEP. However, age only managed to explain 18% of the variance in MIP, and showed no influence on the variance in MEP. Thus, from the literature it is clear that anthropometric variables do not have a clear and strong influence on the variability in RM strength in an athletic population.

3. Prediction equations for respiratory muscle strength

Table 1 depicts some of the prediction equations for MIP and MEP, respectively, in normal, healthy populations. The table only includes those prediction equations based on populations with age ranges that included the age range of the present study (18 -32 years).

The only prediction equation for RM strength in an athletic population was that of Cordain et al. (1987) who reported a prediction equation for male competitive runners. They could only report a significant prediction equation for MIP and no equation for MEP, since none of the factors included in the regression analysis could explain the variation in MEP values. The prediction equation for MIP was as follows:

$$\text{MIP (cmH}_2\text{O)} = (0.99 \times \text{age in years}) - 161 \dots\dots\dots 4.1$$

($R^2 = 0.19$, SEE = 25.0)

Table 1: Prediction equations for RM strength in normal, healthy and sedentary populations.

<i>Authors</i>	<i>Gender</i>	<i>n</i>	<i>Age</i>	<i>MIP (cmH₂O)</i>	<i>MEP (cmH₂O)</i>
Hautmann et al. (2000)	Male	248	18-82	$[(0.158 \times \text{BMI}^*) - (0.051 \times \text{age}) + 8.22] \times 10.2$	
	Female	256	18-83	$[(-0.024 \times \text{age}) + 8.55] \times 10.2$	
Harik-Khan et al. (1998)	Male	139	23-90	$126 - (1.028 \times \text{age}) + (0.343 \times \text{weight})$	
	Female	128	20-83	$171 - (0.694 \times \text{age}) + (0.861 \times \text{weight}) - (0.743 \times \text{height})$	
Neder et al. (1999)	Brazilian males	50	20-80	$(-0.8 \times \text{age}) + (0.48 \times \text{weight}) + 119.7$	$(-0.81 \times \text{age}) + 165.3$
	Brazilian females	50	20-80	$(-0.49 \times \text{age}) + 110.5$	$(-0.62 \times \text{age}) + 115.7$
Johan et al. (1997)	Chinese Males	131	20-80	$37.24 - (0.67 \times \text{age}) + (0.15 \times \text{height}) + (0.85 \times \text{weight})$	$-106.17 - (0.52 \times \text{age}) + (1.05 \times \text{height}) + (1.03 \times \text{weight})$
	Chinese Females	90	20-80	$68.8 - (0.49 \times \text{age}) + (0.05 \times \text{height}) + (0.22 \times \text{weight})$	$112.14 - (0.59 \times \text{age}) - (0.11 \times \text{height}) - (0.07 \times \text{weight})$
Bruschi et al. (1992)	male or female	266 359	18-70	$4.02 - (0.26 \times \text{gender}^{**}) - (0.004 \times \text{age}) + (0.47 \times \text{BSA}^{\Psi})$	$4.48 - (0.18 \times \text{gender}^{**}) - (0.0004 \times \text{age}) - (0.003 \times \text{gender}^b \times \text{age}) + (0.25 \times \text{BSA}^{\Psi})$
Chen & Kuo (1989)	Chinese males	80	16-75	$149 - (1.00 \times \text{age})$	$157 - (0.55 \times \text{age})$
	Chinese females	80	16-75	$101 - (0.62 \times \text{age})$	$109 - (0.47 \times \text{age})$
Wilson et al. (1984)	Male	48	19-65	$142 - (1.03 \times \text{age})$	$180 - (0.91 \times \text{age})$
	Female	87	19-65	$-43 + (0.71 \times \text{height})$	$3.5 + (0.55 \times \text{height})$
Black & Hyatt (1969)	Male	60	20-80	$143 - (0.55 \times \text{age})$	$268 - (1.03 \times \text{age})$
	Female	60	20-86	$104 - (0.51 \times \text{age})$	$170 - (0.53 \times \text{age})$

Weight is measured in kg; height is measured in cm; age is measured in years; * BMI = body mass index calculated as $\text{weight [kg]} / (\text{height [m]}^2)$; ** gender: male = 0, female = 1; ^Ψ BSA = body surface area calculated as $(\{\text{weight [kg]} \times \text{height [cm]}\} / 3600)^{1/2}$

C. PREDICTORS OF RESPIRATORY MUSCLE ENDURANCE

1. Predictors of respiratory muscle endurance in general populations

Factors that seem to predict respiratory muscle endurance are age and height. According to Fiz et al. (1998), T_{lim} (the time to exhaustion at 80% of maximum tolerated load) correlated with height ($r = 0.42$; $P < 0.0001$) and age ($r = -0.59$; $P < 0.0001$) in 99 healthy subjects, while body mass had no effect on respiratory muscle endurance.

In a study by Chen & Kuo (1989) inspiratory muscle endurance also correlated with age. Eighty men and women volunteers were divided into four different age groups. Inspiratory muscle endurance was expressed as an inspiratory pressure-time index. The youngest group of women (aged 23 ± 0.6 years) had a significantly higher endurance time (difference between means = 14 sec) compared to the oldest group (aged 65.9 ± 1.0). The youngest group of men (aged = 24.2 ± 0.5 years) also had a significantly higher endurance time (difference between means = 16 sec) compared to the oldest group of men (aged 66.9 ± 1.0 years).

Neder et al. (1999) assessed respiratory muscle endurance by measuring maximum voluntary ventilation (MVV) over 15 sec in 100 healthy individuals. They also reported a significant negative correlation of age with MVV ($r = -0.56$) and a significant positive correlation of height with MVV ($r = 0.63$).

In the study by Neder et al. (1999) gender significantly predicted MVV together with age. Men showed a significantly higher MVV compared to women (167 ± 20 vs. 126 ± 13 L. min^{-1}), implying that men had better RM endurance than women. In contrast, Fiz et al. (1998) and Chen & Kuo (1989) found no significant differences between men and women in T_{lim} and pressure time indexes, respectively. However, a possible explanation for the different outcomes between Neder et al. (1999) and the latter two studies are the different methods used to assess respiratory muscle endurance. Both Fiz et al. (1998) and Chen & Kuo (1989) used a method where time was measured to sustain a specific intensity of work,

whilst Neder et al. (1999) used a test with a specific end point (maximum air ventilated in 15 sec). It is more likely to find specific differences between groups if a test is used that has a specific end point, since the inter-individual variability will be smaller compared to open-end (time to exhaustion) tests. Therefore, it is suggested that gender has an influence on RM endurance as seen in a test for RM endurance (Neder et al., 1999) with a definite end-point.

2. Respiratory muscle endurance in athletic populations

There is conflicting evidence as to whether whole-body endurance training causes an improvement in RM endurance. Chen & Kuo (1989) has found that RM endurance positively correlated ($r = 0.29$, $P < 0.01$) with physical activity in healthy men ($n = 80$). Neder et al. (1999) have also found a slightly stronger correlation ($r = 0.48$, $P < 0.01$) between physical activity and MVV in a healthy group of men and women ($n = 100$) compared to the correlation of Chen & Kuo (1989). Although these two studies did not investigate the effect of intense physical training on RM endurance, they did show that this phenomenon already existed in healthy, moderately active populations; therefore, one can assume that there will be large differences in RM endurance between trained athletes and sedentary individuals.

Pringle et al. (2005) found a significant negative correlation between MVV and the run time of a 10 km race ($r = -0.52$) in competitive runners. Further, stepwise regression analysis revealed that MVV could explain 27% of the variance in 10 km run time. They suggested that specific RMT can lead to an improvement in 10 km run time based on previous findings that MVV increased after specific RMT (Boutellier et al., 1992; Robinson & Kjeldgaard, 1981), and that MVV could explain a significant amount of variance in run time. This seems to be the only study to show significant correlation between MVV and actual race time.

If whole-body training improves RM endurance it should be evident in comparisons between athletes and non-athletes. Indeed, studies have shown significant differences in RM endurance between athletes and sedentary individuals (Eastwood et al., 2001; Martin & Chen, 1982; Martin & Stager, 1981). Eastwood et al. (2001) found that marathon runners

could achieve a significantly higher maximum threshold pressure, during a progressive threshold loading test, compared to sedentary subjects (90 ± 8 vs. $78 \pm 10\%$ of MIP). Martin & Chen (1982) found that distance runners could achieve a greater fraction of their 12-second MVV (75 vs. 62% of MVV) during an isocapnic hyperpnoea test compared to their untrained siblings. Martin & Stager (1981) found no difference in MVV between endurance athletes and non-athletes, but did find significant differences in a progressive ventilation threshold test (75 vs. 67% of MVV) and the ventilatory endurance time at 80% of MVV (11 vs. 3 min) between the two groups.

A number of studies have shown that RM endurance improved after a period of running (Robinson & Kjeldgaard, 1982), swimming (Clanton et al., 1987) and cycling training (O'Kroy & Coast, 1993). Robinson & Kjeldgaard (1982), Clanton et al. (1987) and O'Kroy & Coast (1993) found significant improvements in sustainable ventilatory endurance time at a specific percentage of MVV, while Robinson & Kjeldgaard (1982) also found a significant improvement in MVV after endurance training.

However, a more recent study by Thomas et al. (1998) showed no improvements in RM endurance after a period of cycling training. Thomas et al. (1998) found no significant difference in maximal ventilatory volume in 12 seconds (44 ± 2 vs. 48 ± 3 L in 12 seconds) after a 6-week incremental cycle endurance training programme compared to baseline. The previously sedentary subjects in the study by Robinson & Kjeldgaard (1982) ran 40 min per day, 3 times per week, at 80% of predicted heart rate maximum for 20 weeks. The subjects of Clanton et al. (1987) were competitive swimmers who ran 3 days per week for 40-60 min at a time, underwent isotonic conditioning (whole-body work out) 3 days per week, and progressively increased their swim training from 2285 m per day to 9140 m per day (5 days per week) in the first 4 weeks of the 12 week training programme. It could have been that the latter two studies found significant improvements in RM endurance after whole-body training, probably due to training at sufficient intensities to elicit adaptations to the respiratory muscles, and that this was not the case in the study by Thomas et al. (1998). The subjects in this study had to follow an incremental cycling programme for 30 min per day, 5 days per week, at 70% of VO_{2peak} . The cycling programme of the latter study can be

considered less intense than the exercise programmes by Robinson & Kjeldgaard (1982) and Clanton et al. (1987). Cycling is also a non-weight bearing exercise compared to running, and the cycling training intensity and programme duration was less compared to that of the other two studies, respectively.

Observed structural and functional adaptations to the RM confirmed that the RM improve their fatigue resistance, and therefore increase RM endurance after whole-body endurance training. A few studies have found a significant increase in the oxidative enzyme activity of the costal but not the crural diaphragm in rats (Powers et al., 1992a; Powers et al., 1992b; Uribe et al., 1992; Powers et al., 1990). In contrast, a few studies could not find an increase in diaphragmatic oxidative capacity after whole-body endurance training in rats (Green et al., 1989; Fregosi et al., 1987; Metzger & Fitts, 1986) and guinea pigs (Hoppeler et al., 1995). These studies investigated changes in the costal and crural diaphragm and considered these two regions as one. De Troyer et al. (1981) found that the costal and the crural diaphragm perform different functions in dogs. It seems that whole-body endurance training elicit improvements in the oxidative capacity of the costal diaphragm and not the crural diaphragm which explains the equivocal outcomes of the different studies.

Powers et al., (1992a; 1992b; 1990) and Uribe et al., (1992) found that the exercise intensity of whole-body training must be very high to induce structural adaptations in the diaphragm. The logical explanation for the demand of high exercise intensity is that the diaphragmatic mitochondrial content and capillary density are already so high that it surpasses that of locomotor muscles, which means the diaphragm is already highly fatigue resistant, and it is unlikely that further adaptations will take place due to whole-body training.

3. Prediction equations for respiratory muscle endurance

To researcher's knowledge no prediction equations have been reported in the literature for a specific athletic population. Recently, two studies have reported prediction equations for RM endurance for normal, healthy populations (Neder et al., 1999; Fiz et al., 1998). However, these two authors did not investigate the same index of RM endurance, and

therefore, are not comparable. Table 2 depicts the available prediction equations for RM endurance indices.

Table 2: Prediction equations for indices of respiratory muscle endurance

<i>Author</i>	<i>Gender</i>	<i>N</i>	<i>Age</i>	<i>Index</i>	<i>Prediction equation</i>
Neder et al. (1999)	males	50	20-80	MVV	$(-1.06 \times \text{age}) + (2.15 \times \text{height}) - 164.6$
	Females	50	20-80	MVV	$(-0.76 \times \text{age}) + 47.4$
Fiz et al. (1998)	males & females	99	20-70	T_{lim}^* (80% of max. threshold load)	$908.76 - (7.86 \times \text{age}) - (74.6 \times \text{gender}^{**})$

Age is measured in years; * T_{lim} = time to exhaustion at 80% of maximum threshold load achieved; ** gender: male = 0, female = 1.

D. IMPLICATIONS OF STUDY COMPARISONS IN THE LITERATURE

Respiratory muscle strength: The comparison of studies in which the factors are investigated that influence RM strength and endurance, are problematic. There are mainly two reasons for the great variability in the values reported for RM strength, measured as either maximum inspiratory mouth pressure (MIP) or maximum expiratory mouth pressure (MEP), which are the most used indexes of RM strength. Firstly, population differences can account for the different outcomes in RM strength and values among studies. Neder et al. (1999) reported that previous studies that reported reference values for MIP and MEP based on either Caucasian or non-Caucasian samples, underestimated the observed values for MIP in their population sample (mixed ethnic groups, all Brazilians).

Johan et al. (1997) reported lower mean values for MIP (74 ± 23 to 84 ± 2 cmH₂O) in Chinese, Malay and Indian men compared to mean MIP values (range: 105 ± 4 and 113 ± 2 cmH₂O) in Caucasian men (Vincken et al., 1987; Wilson et al., 1984; Leech et al., 1983). From these results, it is clear that ethnicity plays a role in the measurement of MIP; therefore, caution must be taken when reference values are compared among studies with different ethnic group selections.

In addition to ethnicity, studies who selected populations with different age ranges compared to other studies, resulted in different reference values. For example, Enright et al. (1994) and McConnell & Copestake (1999) reported MIP values based on elderly populations (> 59 years old) which were 20% and 5% lower than the values for MIP in men (age range = 18-80 years) by Hautmann et al. (2000), respectively. When reference values are compared among studies, the age selections of populations should fall within similar age ranges.

Another complication with population selections could be the specific criteria to which researchers selected a group of subjects that could be considered healthy. Studies do not report the same criteria or methods (e.g. specific questionnaires) according to which 'healthy' subjects were selected. Where no significant differences were found between athletes and non-athletes, an important explanation could be that studies used different criteria to consider subjects either as an athlete or a non-athlete. The differences in the specific level of fitness among subjects of different studies that were considered athletes could also have influenced the outcome of studies.

Secondly, the most profound reason for the wide variability in reference values among studies, are the differences in methodological procedures. These methodological differences include differences in pressure measurement devices, differences in type and number of efforts and differences in observer opinions and subject motivation. The response characteristics of the device will influence the pressure measured. Pressure measurement devices range from different pressure gauges (e.g. aneroid or mercury) to digital pressure transducers, all measuring pressure in either cmH₂O or kPa. The type of mouth piece used has a major influence on the outcome of the measurement. Koulouris et al. (1988) found that a rubber tube mouth piece elicited a higher value compared to plastic flanged mouth piece in the same individual. How the subject's mouth is placed over the mouth piece makes a difference in the measurement. Black & Hyatt (1969) instructed their subjects to place their lips against the mouth piece to prevent leaks, while others instructed their subjects to bite over the mouth piece.

A small leak in the device is necessary, to prevent generation of false high readings by closure of the glottis during MIP maneuvers and the use of the buccal muscles during MEP maneuvers (Black & Hyatt, 1969). Most studies did include a small leak; however, Smyth et al. (1984) did not incorporate a small leak in the pressure measuring device. The use of a nose clip will also influence the measurement of pressures. Most studies did incorporate the use of a nose clip to prevent air leaking through the nostrils, except for Wilson et al. (1984) who reported that their subjects conducted the maneuvers without a nose clip. This could have led to the measurement of lower pressures than with a nose clip.

Smyth et al. (1984) reported that sudden (high speed) RM strength maneuvers will elicit higher values than slow maneuvers. Besides the speed of the maneuver, the number of efforts allowed from which a final result is recorded, whether peak or sustained maximal pressure is measured, the duration of a single effort and the amount of rest between efforts, will all influence the pressure recorded. Most studies who have reported a rest period between efforts, reported a one minute rest period (Neder et al., 1999; Bruschi et al., 1992). Hautmann et al. (2000) reported 20-90 s rest between efforts. Studies also differ in the time maximal pressure had to be sustained. Some reported that efforts were held for one second (Johan et al., 1997; Wilson et al., 1984; Black & Hyatt, 1969), while others reported that efforts were held for 2-3 seconds (Hautmann et al., 2000; Harik-Khan et al., 1998; Fiz et al., 1998). Higher values are expected when efforts are held for shorter periods of time.

Studies recorded the highest value out of 3 efforts (Johan et al., 1997; Wilson et al., 1984; Leech et al., 1983), or 5 efforts (Neder et al., 1999; Bruschi et al., 1992), or 7 to 12 efforts (Hautmann et al., 2000). Wen and colleagues (1997) found that the average of the highest three values of only a few efforts underestimated the average value of the three highest values during twenty efforts in the same individual. This finding implies that higher values for MIP and MEP were probably recorded compared to the values recorded in studies where 2-3 efforts, were used.

Additionally, the lung volume at which either MIP or MEP maneuvers are initiated from, have an influence on the pressure achieved during the maneuver. For example, Fuso et al.

(1996) reported MIP values measured from functional residual capacity (FRC) and residual volume (RV), respectively. The mean values for MIP from RV were higher than that of MIP from FRC (113 ± 32 vs. 103 ± 30 cmH₂O). It is recommended that maximum mouth pressure is measured from either RV for MIP or total lung capacity (TLC) for MEP, since these lung volumes are easier to control for repeatability (Green et al., 2002). It is suggested that where possible, the specific lung volume should be measured and revealed to the subject in order to ensure that repeated efforts are initiated from the same lung volumes as previous efforts.

It is not feasible that studies with these wide ranges of methodological differences should be compared, since the above paragraphs explain why reference values of these particular studies will certainly differ. Both Neder et al. (1999) and Smyth et al. (1984) recommended that laboratories should develop their own reference values in the same laboratory with the same equipment and procedures, in order to rate an individual's RM strength by means of MIP and/or MEP. This will exclude all the discrepancies that may be the main reasons for MIP and/or MEP values to differ among studies, rather than weaker respiratory musculature.

Other influences on the measurement of RM strength and endurance are subject motivation and learning effect. The solution to a learning effect is to administer at least one or two familiarization sessions where subjects can get used to the maneuvers and produce repeatable results. Verbal encouragement at all times may be a tool to ensure that all subjects are equally motivated while performing the measurements.

Respiratory muscle endurance: The same problems are observed in studies on RM endurance, i.e. population and methodological differences. However, RM strength in all study comparisons measured specifically the same index of RM strength, i.e. maximum inspiratory and expiratory mouth pressure. However, studies who reported prediction equations for RM endurance (Neder et al., 1999; Fiz et al., 1998) used different indices of RM endurance, which makes it impractical to compare them with each other. Therefore, the large variability in the findings among studies can be attributed to the method of measuring RM endurance. Thomas et al. (1998), who found no significant improvements in RM

endurance after a 6-week incremental cycle training programme, also confirmed this statement. They reported that there appears to be no standardized protocol to assess RM endurance and that the absence of a universally accepted protocol among researchers complicates direct comparisons across studies.

Another problem with RM endurance tests are similar to whole-body endurance tests, i.e. open-end performance tests tend to be poorly repeatable and cannot be described as a valid test for endurance. Most studies have assessed RM endurance tests as the time ventilation could be sustained at a specific percentage of MVV. Open-end tests produce high inter-individual variability and can be the reason for non-significant improvements after intervention, and the reason for no differences among athletes and sedentary subjects, and differences in reference values among studies.

Another complication is that none of the studies that found a significant difference between athletes and non-athletes, could exclude the possibility that genetic predisposition for athleticism could be the reason for stronger and/or more fatigue resistant RM in athletes compared to sedentary individuals. Martin & Chen (1982) found significant differences between competitive runners and their untrained siblings in an incremental ventilatory endurance test and a time to exhaustion test at 80% of 12 second MVV. They concluded that the failure to demonstrate elevated RM endurance in the untrained siblings, suggests that whole-body training rather than genetic predispositions accounts for the improved RM endurance in athletes. No study has investigated this scenario in siblings for RM strength.

E. CONCLUSION

Gender, age and height, are the anthropometric factors which strongly contribute to the variation in pulmonary function measures. If there is a clear and strong relationship between pulmonary function measures and RM strength and/or endurance, these factors named above would also strongly contribute to the variation in RM strength and endurance measures. However, only two studies from the literature found significant but moderate relationships between pulmonary function variables and respiratory muscle strength (Hautmann et al.,

2000; Leech et al., 1983). Equivocal outcomes among studies make the determination of anthropometric predictors of RM strength and endurance problematic. From the literature, it seems that substantial agreement exist that gender and age are the first and the second most important predictors of RM strength in normal and athletic populations, respectively. Height, body mass, BMI, and BSA only showed minor contributions to the variability in RM strength. This was found in only a few studies, while other studies found no influence of these variables on RM strength or endurance. Studies investigating the influence of anthropometric variables on RM endurance found that age and height could be considered the main predictors of RM endurance. Gender did not seem to have a great influence on the variability in RM endurance (Fiz et al., 1998; Chen & Kuo, 1989).

It is clear that competitive athletes have superior morphology compared to sedentary individuals. It would be inappropriate to use reference values of RM strength and endurance based on sedentary populations for athletic populations; therefore, future research is needed to establish predicted values for athletic populations. Widespread evidence exist that whole-body endurance training does improve RM strength (Powers & Criswell, 1996; Clanton et al., 1987; Robinson & Kjeldgaard, 1982, Leith & Bradley, 1976), RM endurance (O’Kroy & Coast, 1993; Clanton et al., 1987; Robinson & Kjeldgaard, 1982) and RM fatigue resistance (Powers et al., 1992a; Powers et al., 1992b; Uribe et al., 1992; Powers et al., 1990).

Evidence also reveals that there are significant differences in RM strength (Fuso et al., 1996) and endurance (Eastwood et al., 2001; Martin & Chen, 1982, Martin & Stager, 1981) between athletes and sedentary subjects. However, there are some studies that contradict these findings, but can be explained by methodological differences or inadequate training intensities of the whole-body training programme. Thus, if whole-body endurance training improve RM strength and endurance and there are significant differences in these indices between athletes and sedentary individuals, one could speculate that physical activity has an influence on the variability in RM strength and endurance variables in an athletic or physically active population.

However, findings of physical activity or training indices which significantly predict RM strength and endurance lack in the literature. A few studies have shown that physical activity and/or fitness correlate significantly with RM strength (Neder et al., 1999; McConnell & Copestake, 1999) and RM endurance (Pringle et al., 2005; Neder et al., 1999; Chen & Kuo, 1989). However, none of the latter studies could show that indices of fitness or physical activity significantly predict RM strength or endurance in either normal populations or athletic populations. Fuso et al. (1996) found that being a soccer player or not, significantly contributed to the variation in MIP measured from FRC, however, they did not report R^2 value for the predictive models which included this dichotomic variable.

The controversial outcomes among studies, investigating the relationships between anthropometry and RM strength and endurance, and physical activity or training and RM strength and endurance, can be largely attributed to differences in methodological procedures and populations studied. Different ethnic groups, age range selections, and specific inclusion/exclusion criteria for selection of healthy subjects or athletes, reveal different outcomes in reference values reported, since these variables have a great influence on measurements. Methodological differences include the type of measurement device, type of mouth piece, mouth placement on mouth piece, a small leak in the device, high or low speed maneuvers, lung volumes from which maneuvers are initiated, duration of efforts, number of efforts, the units for quantification of physical activity or fitness, subject motivation and the observer's opinion of satisfactory efforts.

Additionally, the reason for the lack of, and conflicting outcomes in RM endurance studies can mainly be attributed to lack of a standardized, universally accepted test for RM endurance, as well as a highly repeatable and valid test which does not include inadequate open-end type tests as previously shown. Therefore, a great need exist for studies to develop a standardized test for RM endurance and a universally recognized method to quantify fitness for the use in stepwise regression analysis to reveal the predictors of RM strength and endurance in athletic populations. Some studies have suggested that research laboratories should develop their own reference values (Fuso et al., 1996, Smyth et al., 1984) with the same device and experimental procedures from which observed and comparable values will

be obtained in similar specific populations. This will avoid uncertainty over adequate reference values. This will also enable researchers and sport scientists to accurately rate an athlete's current RM strength and endurance status, and to whether intervention is needed to improve these indices which will ultimately improve athletic performance, as previously reported (Chapter 3).

CHAPTER FIVE

PROBLEM STATEMENT

A. SUMMARY OF THE LITERATURE REVIEW

Considering the literature in chapter two to chapter four, several issues on respiratory muscle (RM) function in athletes and its effect on performance emerge: It is evident that RM fatigue occurs during whole-body endurance exercise (Lomax & McConnell, 2003; Romer et al., 2002c; Perret et al., 2000; Babcock et al., 1998; McConnell et al., 1997; Babcock et al., 1995; Mador et al., 1993; Bye et al., 1984) and limits exercise performance. This has been shown in studies that investigated the effect of RM fatigue on performance by either inducing RM fatigue prior to exercise (Verges et al., 2007b; Harms et al., 2000; Mador & Acevedo, 1991; Martin et al., 1982), or preventing RM fatigue during exercise, through partial unloading of the RM (Harms et al., 2000; Johnson et al., 1996; Aaron et al., 1985).

It is suggested that specific respiratory muscle training (RMT) can overcome RM fatigue, or at least delay the onset of fatigue. The effect of RMT on athletic performance is widely documented in the literature, with studies showing that specific RMT can improve constant-load exercise performance (Verges et al., 2007a; Enright et al., 2006; Gething et al., 2004; McMahon et al., 2002; Stuessi et al., 2001; Markov et al., 2001) and time-trial endurance performance (Johnson et al., 2007; Griffiths & McConnell, 2007; Holm et al., 2004; Romer et al., 2002a; Romer et al., 2002b; Romer et al., 2002c; Volianitis et al., 2001b), in sedentary and well-trained athletes. However, the findings of a true ergogenic effect are still inconclusive.

The main reason for the improvement in performance is that, RMT elicits structural and functional improvements of the RM, which delay or relief respiratory muscle fatigue during exercise. This causes a delay in the RM metaboreflex, which allows more blood flow to the limb locomotor muscles and the delay of limb muscle fatigue (McConnell & Lomax, 2006). The delay or relief in RM fatigue also elicits less sensory input to the central nervous system

(CNS) which lowers the perceptual sensation of breathing discomfort (Romer & Polkey, 2008). RMT also causes a constant exposure to high concentrations of metabolites within the RM, which desensitize the receptors of type III and IV afferents, which also elicits less sensory input to the CNS (Witt et al., 2007).

Only one study has investigated the effect of detraining on RM function (Romer & McConnell, 2003b) and found a significant reduction after 9 weeks of inactivity. However, the detrained values still remained above pretrained values and remained constant up to 18 weeks post training. Boutellier and Piwko (1992) also just briefly mentioned in their study that the improvements in respiratory muscle endurance, after a 4-week VIH training programme, returned to baseline values 18 months after completion of the initial study in healthy sedentary subjects. RMT does not affect the cardiovascular responses to exercise (Markov et al., 2001), but it does elicit adaptations within the RM, that lead to improvement in their strength, endurance and efficiency (Downey et al., 2007; Johnson et al., 2007; Romer & McConnell, 2003b). Therefore, the essence of RM detraining falls upon the reversal of the specific adaptations to the RM. A thorough investigation into RM detraining and the specific reversal of training adaptations, is still lacking.

Studies on the predictors of RM strength and endurance in the general and specific athletic populations are controversial. There is substantial agreement that gender and age are the first and the second most important predictors of RM strength in general and athletic populations (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Fuso et al., 1996; Bruschi et al., 1992; Chen & Kuo, 1989; Wilson et al., 1984; Black & Hyatt, 1969). Height, weight, BMI, and BSA contributes to a lesser extent to the variability in RM strength. Studies investigating the influence of anthropometric variables on RM endurance found that age and height could be considered the main predictors of RM endurance. Gender did not seem to have a great influence on the variability in RM endurance (Fiz et al., 1998; Chen & Kuo, 1989).

It is clear that competitive athletes have superior morphology compared to sedentary individuals (Pitsiladis, 2005; Ackland, 2006; Heyward & Wagner, 2004; Heath & Carter,

1967). Widespread evidence exist that whole-body endurance training does improve RM strength (Powers & Criswell, 1996; Clanton et al., 1987; Robinson & Kjeldgaard, 1982, Leith & Bradley, 1976), RM endurance (O’Kroy & Coast, 1993; Clanton et al., 1987; Robinson & Kjeldgaard, 1982) and RM fatigue resistance (Powers et al., 1992a Powers et al., 1992b; Uribe et al., 1992; Powers et al., 1990). Evidence also reveals that there are significant differences in RM strength (Fuso et al., 1996) and endurance (Eastwood et al., 2001; Martin & Chen, 1982; Martin & Stager, 1981) between athletes and sedentary subjects. Thus, if athletes have superior anthropometry, and RM strength and endurance compared to the sedentary population, it would be irrelevant to use general reference values on an athletic population, since it will probably underestimate RM strength and endurance. However, it is not clear to what extent do physical activity or training indices predict RM strength and endurance.

B. AIMS OF THE STUDY

The study was divided into two sections, using two different subject groups. Section 1 consisted of the following two areas of investigation on RM function in a group of competitive women hockey players: (1) the effect of RMT on RM function and aerobic fitness, and (2) the effect of RM detraining on RM function. Competitive women hockey players were chosen as the subject group in order to investigate the effect of RMT in athletes participating in high-intensity, repetitive sprint sport. Section 2 involved the determination of predicted values for RM strength and endurance in an athletic population.

SECTION ONE

AIM ONE

To determine if a 12-week hockey training plus RMT (HT-RMT) intervention will improve the RM function and aerobic fitness of competitive women team sport (hockey) players.

Questions:

- (i) Is there an improvement in pulmonary function variables after the 12-week HT-RMT intervention?
- (ii) Is there an improvement in RM strength and endurance after the 12-week HT-RMT intervention?
- (iii) Will the changes (difference between pre- and post-RMT) in RM strength, endurance and pulmonary function significantly differ between the experimental and control group?
- (iv) Will the 12-week HT-RMT programme improve the fitness performance of competitive women hockey players to a greater extent than normal hockey training alone?
- (v) Are there any significant relationships between the change in RM strength, RM endurance and/or pulmonary function measures and the change in fitness during the 12-week HT-RMT programme?

AIM TWO

To determine the effect of a 20-week RM detraining period on the RM function in the same group of competitive women team sport players, following the RMT programme.

Questions:

- (i) Are there any significant decreases in RM strength, RM endurance and pulmonary function measures after a specific period of RM detraining?
- (ii) Will the changes in RM function in the experimental group differ from the changes in RM function in the control group over the period of RM detraining?
- (iii) If there is a reversal in RM function measures over the period of RM detraining, will the reversal manifest after 9 weeks of RM detraining or after 20 weeks of RM detraining?

SECTION TWO

AIM THREE

To determine the physical and physiological factors that predict RM strength and endurance in an athletic population.

Questions:

- (i) Are there significant relationships between certain kinanthropometric and pulmonary function measures, and RM strength and endurance, respectively?
- (ii) Are there significant differences in RM function measures between athletes and non-athletic individuals?
- (iii) Are there significant differences between RM function variables between men and women athletes?
- (iv) What are the significant predictors of RM strength and endurance, respectively, in an athletic population?

C. MOTIVATION AND POTENTIAL BENEFITS

A shortcoming in the literature is the investigation of the effect of RMT on high-intensity, intermittent exercise performance, since most previous studies have only investigated the effect of RMT on endurance exercise. Only one previous study investigated the effect RMT on high-intensity, repeated sprint performance (Romer et al., 2002b). Since there seem to be a specific threshold (80 – 85% $\text{VO}_{2\text{max}}$) at which RM fatigue takes place, it would be feasible to investigate this phenomenon in high-intensity, repeated sprint sport, since these sports are characterized by exercising at very high intensities. To the researcher's knowledge, the effect of RMT on the RM function and fitness performance in team sports has not yet been investigated. New insights into RMT in team sports will enable sport scientists and coaches to determine whether specific RMT will be beneficial to the fitness of team sport players and whether or not it needs to be incorporated into their daily training programme.

Specific RMT may specifically be beneficial to hockey players, since hockey players use their upper body muscles to breathe as well as to handle the ball with a hockey stick during play. Additionally, hockey players stay in a semi-crouched position during play, and this may alter and limit breathing patterns, and increase breathing effort sensation during exercise. These possible additional demands placed upon the RM of the hockey player, may accelerate the occurrence of RM fatigue that can limit performance. However, the latter has not been determined before and this is therefore not the primary reason for selecting field hockey as the exercise model in this study. The primary reasons for selecting field hockey players as subjects in this study, is because of the nature of the activity (short, repetitive sprints) and the availability of the whole team for this research project. It can further be argued that improved RM function and efficiency will improve recovery time between repeated sprints (Romer et al., 2002b), which is one of the most important contributors to improved performance in a high-intensity, repetitive sprint sport.

Very few studies have investigated the effect of either a concurrent (inspiratory and expiratory muscle training) RMT programme (Wells et al., 2005) or specific expiratory muscle training (Griffiths & McConnell, 2007) on RM function and exercise performance.

Investigation into expiratory muscle training will reveal the importance of this aspect in combination with inspiratory muscle training to improve overall RM strength and endurance.

The majority of studies that investigated the effect of RMT on athletic performance were done in men (Johnson et al., 2007; Griffiths & McConnell, 2007; Verges et al., 2007b; Romer et al., 2002a; Romer et al., 2002b; Romer et al., 2002c; McMahon et al., 2002; Sonetti et al., 2001; Spengler et al., 1999; Fairbairn et al., 1991; Morgan et al., 1991) or mixed groups consisting mostly out of men than women (Downey et al., 2007; Williams et al., 2002; Chatham et al., 1999; Markov et al., 2001; Stuessi et al., 2001). Only three studies investigated the effect of RMT on athletic performance in women (Wells et al., 2005; Enright et al., 2006; Volianitis 2001b). The present study will elaborate on the effect of RMT on exercise in women athletes specifically – an area limited in the literature.

Although the reversal of adaptations in skeletal muscle detraining are well established, the specific changes within the RM due to detraining have not yet been investigated. Since RMT elicits the same adaptations to RM as whole-body training to skeletal muscles, it is suggested that the reversal of adaptations to the RM are similar to that of skeletal muscle detraining. The study will provide new information on the RM detraining effect on RM function measures (RM strength, endurance and pulmonary function) and determine the time period in which adaptations to improved RM function measures may be lost. These findings will enable sport scientists and coaches to establish how often RMT needs to be incorporated into the athlete's training regime to maintain the improved adaptations to RM function.

The use of predicted values based on normal, non-athletic populations will underestimate the observed values in an athletic population, since clear evidence exist that athletes have superior RM strength and endurance compared to non-athletic or sedentary individuals, and that specific whole-body training improve RM strength and endurance. There are some studies that contradict the latter finding (Eastwood et al., 2001; Thomas et al., 1998; Armour et al., 1993), however their results could be explained by inadequate training intensities of the whole-body training programme. Thus, the lack of specific predicted values for athletic populations make it impossible to rate whether the athlete has adequate RM strength and

endurance, and whether it needs to be improved and not pose a possible limitation to his/her athletic success.

Some studies also suggested that research laboratories should develop their own reference values (Fuso et al., 1996; Smyth et al., 1984) with the device and experimental procedures that are used in the laboratory. This will eliminate uncertainty over adequate reference values, due to differences in population selections and methods used to obtain the reference values compared to that of the observed values. The development of adequate RM strength and endurance reference values for an athletic population will enable researchers and sport scientists to accurately rate an athlete's current RM strength and endurance status, and decisions can be made to whether a RM intervention is needed to improve these indices, which will ultimately improve athletic performance.

CHAPTER SIX

METHODOLOGY

The study was divided into two sections, investigating three different areas of respiratory muscle (RM) function. The following titles (Study 1 – 3) represent the three areas, with Study 1 and 2 described under **Section One** and Study 3 described under **Section Two**.

Section One:

- Study 1: An experimental study to determine the effect of hockey training plus additional RMT training (HT-RMT intervention) on RM function and aerobic fitness in women competitive field hockey players.
- Study 2: A descriptive study to determine the effect of RM detraining on RM function.

Section Two:

- Study 3: A descriptive study to determine the predictors of RM function.

In the following paragraphs the subjects, study designs, experimental procedures, measurements and tests and statistical analysis are discussed separately for the three different areas (Study 1 – 3) mentioned above.

SECTION ONE

A. STUDY ONE

An experimental study to determine the effect of hockey training plus additional RMT training (HT-RMT intervention) on RM function and aerobic fitness in competitive women team sport (hockey) players.

1. Subjects

Twenty five women field hockey players were recruited for this study. Players of the first and second field hockey teams of Stellenbosch University, who met the inclusion criteria, were included in the study. Three subjects showed abnormal pulmonary function and were excluded from the data analyses. Two of the three subjects showed an obstructive lung implication (percentage of predicted values for air flow was less than 80%) and the third subject showed a restrictive lung implication (percentage of predicted values for lung volumes was less than 80%). The remaining 22 subjects indicated normal pulmonary function and were included for data analysis for the intervention study. None of the subjects had experience in respiratory exercises prior to the study.

Inclusion criteria: Only the players who were available for both seasons, 2006 and 2007, were recruited for the study. Each subject had to meet the criteria of competitive hockey experience for at least five years and train at least four times a week. Subjects had to be free from a history of pulmonary function impairment (excluding asthma) and they had to be free of respiratory tract infection at least four weeks prior to baseline testing. It was decided to include subjects with asthma as long as they showed normal pulmonary function (percentage of predicted value for air flow was equal to or above 80%). Although, three subjects in the study were diagnosed with asthma, they all failed to show abnormal results for air flow or any of the other pulmonary outcome variables.

Exclusion criteria: A subject was excluded from the study if she showed abnormality in resting pulmonary function. A subject was further excluded from the study if she was

unable to continue with the RMT during the 12-week intervention due to illness, injury, other responsibilities and/or voluntarily withdrawal from intervention. Subjects were also excluded from the study if they did not adhere to 80% of the RMT programme (missed more than 29 sessions/14 days of training).

Characteristics: The 22 subjects had a mean \pm SD (range) age of 21 ± 2 (18 – 25) years, height 167 ± 7 (154 – 177) cm and weight 62 ± 9 (46 – 78) kg. Two subjects suffered from asthma and another subject suffered from exercise-induced asthma. All three subjects used an asthma pump (β_2 stimulant) as needed during the occurrence of asthma symptoms. However, all 3 asthma sufferers showed normal pulmonary function and their respiratory muscle function outcome variables did not differ significantly from the rest of the group. None of the subjects participated competitively in other sports. Their training regimes consisted of hockey training sessions, hockey fitness sessions and strength training in the gymnasium. The group trained on average 8 ± 3 (5 – 14) times per week during the hockey season (April to September). The subjects had an average of 12 ± 3 (8 – 15) years competitive experience in field hockey. Competitive experience ranged from provincial to junior national, national and international level. In 2006 eight players competed internationally and were selected for the national team. The fourteen remaining subjects all competed at provincial level. Eight of the fourteen provincial subjects were selected for the junior national team in previous years.

2. Experimental design and procedures

All subjects underwent a series of kinanthropometric measurements and respiratory muscle function measurements (FVC, FEV₁, PEF_R, MEF_{50%}, MVV, MIP and MEP) in the laboratory. The subjects were randomly assigned into an experimental group (n = 15) and a control group (n = 7) in a double-blinded study design. The players that were selected for the national team underwent extra training and played extra matches. It would have been ideal to equally divide these players into an experimental and control group and then add the rest of the subjects in randomized order. However, at the time of assigning subjects to the experimental and control group, it was unknown whom of the players in the total group

would have been selected for the national team. Subjects could also not be equally paired-off according to respiratory muscle function into two groups. The restricted availability of the subjects made it impossible to complete all baseline tests for the total group before the onset of the intervention. Subjects had to start the RMT at different times in order to fit everyone in for testing at the specific time points (baseline, 6 weeks, and 12 weeks). All baseline tests were completed within a time period of three weeks.

Subjects visited the laboratory on four separate occasions. During the first visit subjects completed a consent form and a short questionnaire regarding their health and general physical activity history. This was followed by the familiarization and first respiratory muscle function assessments. Assessments included pulmonary function, MVV, MIP, and MEP.

During the second visit the respiratory muscle function assessments were repeated to determine the reproducibility and/or exclude a possible learning effect that might be a potential problem during the study. The outcome variables of the second visit served as baseline values (BL). A minimum rest period of 48 hours and a maximum period of 7 days were allowed between the first and second visit. During the second visit a kinanthropometric assessment was conducted for data collection in STUDY 2.

The respiratory muscle function assessments were repeated at six weeks (reported as: 6wk) of the 12-week intervention. Post-intervention assessments (reported as: 12wk) took place at least 5 days after cessation of RMT, to avoid any possible influence of respiratory muscle fatigue on respiratory performance. For each visit subjects were instructed not to exercise on the day prior to testing to minimize any type of fatigue that could influence a maximal effort during testing. Subjects were also instructed to avoid eating and drinking fluids, except water (*ad libitum*), two hours before testing, to ensure the correct measurement of body weight. Subjects were also requested to refrain from alcohol and caffeine intake 24 hours before testing.

Intervention: All subjects underwent a 12-week specific respiratory muscle training programme (RMTP) in addition to their normal hockey training. The experimental group underwent hockey training plus RMT (HT-RMT) with a true RMT device. The control group underwent hockey training plus sham training (HT-ST) with a sham RMT device that could only produce 15% of the resistance of the true RMT device. Subjects were told that the differences in resistance among the *PowerLung*® devices, was to compare RM strength training with RM endurance training. The intervention started for each subject on the day after the second visit to the laboratory. Training was done with a commercial, hand-held RMT device, the *PowerLung*®-Sport (*PowerLung*® Inc., Houston, Texas, USA) (see Appendix A). The RMT programme consisted of 30 breaths twice daily, 6 days a week for a period of 12 weeks. The first two weeks of training consisted of 3 sets of 10 consecutive breaths (repetitions) in the mornings and evenings, respectively. During the rest of the intervention the RMT programme consisted of 2 sets of 15 consecutive breaths (repetitions) in the mornings and evenings, respectively.

The first RMT session was completed in the laboratory under supervision. Thereafter, subjects were trusted to perform the RMT sessions at home, without supervision of the researcher. Subjects were required to keep a diary of both their RMT sessions and physical activity throughout the intervention. Subjects had to specify the number of hours they did specific hockey training, hockey fitness sessions, training sessions in the gymnasium and the number of hockey matches played throughout the twelve weeks.

The PowerLung®: The *PowerLung*® consisted of two resistance cells and threshold resistance controls for inhalation and exhalation, respectively. The cells and controls for exhalation are situated at the bottom (proximal end) of the device, and for inhalation at the distal end of the device (*PowerLung*® User Guide, 2007). At the opposite end of the device a flange mouth piece was adapted and the device was covered with a hand grip to comfortably hold the device during training. Each resistance cell consisted of a spring valve. The spring could either be compressed or released with the threshold resistance control to change the resistance against air flow. The inhalation threshold resistance control had 6 settings and the exhalation threshold resistance control had 3 settings to increase or decrease

resistance against air flow. The springs (for inspiration and expiration) of the sham *PowerLung*® device were weaker compared to the “true” devices, which required only 15% of the resistance of the “true” device needed to let air through the valve to breathe.

Specific procedure for PowerLung® usage: During the second visit subjects underwent familiarization and their first RMT session to ensure the correct use of the *PowerLung*®. Subjects had to do the training in a seated position wearing a nose clip. Subjects were instructed to breathe forcefully through the device at a slower rate than normal breathing (3 seconds for inhalation and 3 seconds for exhalation). During inhalation subjects were advised to count slowly to 3, while trying to fill their lungs and hear a “whoosing” sound through the *PowerLung*®. Subjects were also instructed not to raise their shoulders or jerk their heads back during inhalation.

After inhalation, subjects had to exhale forcefully and maximally, while slowly counting to 3 and trying to empty their lungs. During exhalation a “whoosing” sound had to be heard and subjects had to make sure not to tense their shoulders or backs. Subjects were also instructed to inhale and exhale adequately enough to repeat 10 – 15 consecutive breaths through the device. The researcher chose the settings for inhalation and exhalation for the first session under supervision, thereafter, subjects had to progressively increase the resistance (~ 0.5 to 1 full turn every fortnight) of the *PowerLung*®, as the intervention continued. The resistance had to be set difficult enough to just complete either the 10 breaths (during the first week) or the 15 breaths in a set, before resting for one minute. The last two breaths of each set had to be very difficult to complete. If subjects in the control group could easily complete 15 breaths consecutively, subjects were instructed to continue training at the first setting (set by researcher) for two weeks, and then increase settings (for inhalation and exhalation) by 1 full turn every fortnight thereafter.

Fitness tests: Subjects performed the 20 m multistage shuttle run test to assess aerobic fitness on four separate occasions. The test was conducted once during the beginning of the hockey season and at the end of the hockey season during 2006 and 2007, respectively. Eight of the subjects in the experimental group and five subjects in the control group

performed all 4 fitness tests during 2006 and 2007. The changes between the end season and pre-season results in 2006 were compared to the changes between the same tests in 2007 in order to determine whether the RMT programme had an effect on the fitness level of the subjects.

3. Measurements and tests

3.1 Anthropometric measurements

All anthropometric measurements were taken with subjects bare footed and dressed in minimum and tight clothing. All measurements were taken according to the International Society of Assessment of Kinanthropometry (ISAK, Australia) recommendations for the standardization of measurements, unless otherwise indicated.

Stretched stature: Stature was measured with a measuring tape mounted vertically to a wall perpendicular to the floor. The subject stood with his/her back and heels against the wall. The midline of the body was positioned in-line with the measuring tape behind the subject. A perspex board (32 x 23 cm) was placed firmly on the vertex of the head and, while the head was in the *Frankfort* plane and the subject took a deep breath, the measurement was taken. The measurement was taken to the nearest 0.1 cm.

Sitting height: A chair with an exact height of 45 cm was placed against the wall with the midpoint of the chair width in-line with the mounted measuring tape. The subject was instructed to sit upright with buttocks against the wall, while the feet were placed on the floor and the lower legs were at right angles with the thighs. The subject was also instructed to avoid an arched back. The measurement was taken following the exact procedure as for stretched stature. The sitting height was then calculated by subtracting the chair height from the measured value to the nearest 0.1 cm.

Arm span: A modified method of Hahn (1990) was used to measure arm span. A steel ruler (200 cm in length, standardized equipment) was mounted to a steel fitting that was

mounted to a wall more or less at shoulder height of a person of average height. A steel plate (60 cm in length, 2.5 cm wide and 2.5 cm thick) was mounted perpendicular to the floor at the zero cm mark of the ruler. At the other end of the ruler a vertical steel plate (60 cm in length, 2.5 cm wide, and 2.5 cm thick) was mounted over the fitting of the steel ruler so that it could slide back and forth over the ruler. The subject was instructed to stand with his feet 3 – 4 cm apart with his right third fingertip just touching the vertical steel plate (at the zero mark side of the ruler). The subject was instructed to stretch out, with his torso pressed against the wall and his head turned sideways, to obtain the greatest possible span. The adjustable steel plate was pushed to the left until it touched the subject's third fingertip. The span was read to the nearest 0.1 cm where the inside of the adjustable steel plate crossed the ruler.

Body mass: Body mass was measured with an electronic scale (*UWE BW-150* freeweight, 1997 model, Brisbane, Australia) to the nearest 0.1 kg.

Sum of skinfolds: Seven skinfolds were taken at the specific landmark sites as described by Norton et al. (2000) with a skinfold caliper (*Harpender*, British Indicators, West Sussex, England). Each measurement was read 2 seconds after the release of the caliper trigger to the nearest 0.5 mm. The seven skinfold measurements were: triceps, subscapular, biceps, supraspinale, abdominal, front thigh and mid-calf. The sum of the seven skinfolds was reported as the outcome variable for body fatness. No calculation of body fat percentage to quantify body fatness was included in the study. The main reason for this decision is the questionable validity and accuracy of body fat prediction from skinfold measurements in highly trained athletes. Body fat percentage is derived from body density (Siri et al, 1961). Firstly, in order to accurately predict an athlete's body density, the equation chosen for the calculation of body density should accurately resemble the population in the prediction equation study. There are over 100 equations available in the literature (Martin et al, 1986).

Only a few of these equations have been cross-validated and accepted as generalized equations for body density prediction. To the researcher's knowledge, only one study, Withers et al. (1987a; 1987b), reported a prediction equation for male and female Australian

college athletes. However, the equations of Withers and colleagues cannot be effectively validated in the independent sample of the present study. All the hockey players in the present study were competitive on a provincial level and some of the subjects were also competitive on a national level. These competitive levels can be classified as a higher level of competition compared to college level. The difference among the two populations studied might lead to either an overestimation or underestimation caused by difference in fitness levels. Secondly, prediction equations are based on the theory that subcutaneous fat contribute to 50% of total body fat, however, athletes may carry a lower portion of total body fat subcutaneously (Boileau & Horshill, 2000). This can lead to overestimation of total body fat in highly trained athletes. Therefore, the direct measurement of seven skinfolds was used as the quantification of body fatness, eliminating the questionable accuracy of the prediction of body fat percentage in an athletic population.

Girths: Five girths were taken with a flexible steel tape (Rosscraft, Surrey, Canada) at the specific standardized sites. Measurements were taken to the nearest 0.1 cm. The girths were: upper arm (relaxed at the mid-acromiale-radiale point), upper arm (tensed at maximum girth), chest (at meso-sternal landmark) and calf (maximum girth).

Breadths: The breadths measured included the humerus width, femur width, biacromial breadth and bi-iliac breadth. These measurements were taken to the nearest 0.1 cm. Humerus and femur width were taken with a small-sliding (Tommy) caliper (Rosscraft, Surrey, Canada) and all breadths were taken at specific landmarks with a large sliding caliper (Rosscraft, Surrey, Canada). These measurements were taken to the nearest 0.1 cm.

Somatotype: Each subject's specific somatotype ratio was calculated from the skinfolds, girth and diameter measurements according to the Heath-Carter method (1967). Somatotyping is the quantification of the present shape of the human body and is expressed as a three-digit value; each digit represents one of three components always in the same order: endomorphy (first component) – describes the relative degree of fatness of the body, mesomorphy (second component) – describes the relative musculoskeletal development of

the body relative to the individual's standing height, and ectomorphy (third component) describes the relative slenderness or leanness of the body (Heath & Carter, 1967).

Two consecutive scores within 0.2 cm for length and girth measures, 1 mm for skinfold measures and 0.2 kg for weight measures were required. All measurements complied with ISAK standardization (Marfell-Jones et al., 2006). The average of these two measurements was calculated as the final score. All the other measurements were taken once. Relative arm length, arm span and sitting height to standing height were calculated. All measurements were taken by the researcher who is a qualified ISAK anthropometrist (level I in 2006 and level II in 2007).

3.2 Respiratory muscle function assessments

The physiological assessment of respiratory muscle function included four major respiratory function tests, namely: flow-volume curve test, maximum voluntary ventilation (MVV) in twelve seconds, maximum inspiratory mouth pressure (MIP) and maximum expiratory mouth pressure (MEP). All four tests were thoroughly demonstrated by the researcher to familiarize the subject with the procedures for each test.

3.2.1. Flow-volume curve test

Forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁) and peak expiratory flow rate (PEFR) were measured by means of a flow-volume curve test. This test was conducted to measure functional lung capacity, as well as to assess normal pulmonary function. The spirometric variables were measured with a *Cosmed Quark b²* unit (Rome, Italy) using a turbine flow meter for volume and flow measurements and the *Spirometry reader 2000 (Cosmed Quark b², Rome, Italy)*. Calculations were made with the *Spirometry* computer software according to the spirometry standards of the American Thoracic Society (ATS) and the European Respiratory Society (ERS).

The flow meter: The flow meter consisted of a sampling plug that fitted into a sampling adapter; this in turn connected to a turbine inside an optoelectronic reader (*Spirometry*

reader 2000 [Quark b², Rome, Italy]). The flow meter was connected to a spirometry mouth piece adapter with a hand holder that slides over the spirometry mouth piece adapter for the subject to comfortably hold the device during the test. Subjects closed their mouths over a disposable carton mouth piece that was placed firmly into the mouth piece adapter (see Appendix B). Inside the flow meter is a bidirectional digital turbine with helical conveyors. When the air passes through the conveyors, it turns in a spiral motion which in turn rotates the turbine rotor. The rolling blade of the turbine rotor interrupts an infrared light which is send out by three diodes of the optoelectronic reader. One interruption represents 1/6 turn of the rotor. There is a constant ratio between the air that passes through the turbine and the number of turns, which allows an accurate measure of volume and flows (*Cosmed Quark b² User Manual, 2000*).

The flow meter was calibrated for volume with a 3 L calibration syringe (*Cosmed, Italy*) on a daily basis and prior to the start of assessments. During the experimental period (Oct 2006 – Oct 2007), the average calibration values showed an error of 0.03% and 0.06% for expiration and inspiration, respectively (average ambient temperature of 18 °C, barometric pressure of 747 mmHg and relative humidity of 54%).

Additionally to the daily calibration, calibration checks recommended by the ERS/ATS (Miller et al., 2005), were done on a regular basis (once every two weeks). Calibration checks were done with the 3 L calibration syringe (*Cosmed, Italy*). A patient file was opened in the spirometry software and the 3 L syringe was connected to the flow meter. A single discharge of the syringe was administrated. The average value of three consecutive discharges was taken as the final outcome variable. The average value of the recorded values over a year (Oct 2006 – Oct 2007), was 3.04 L, which resulted in a +1.3% error of the true volume (3 L). The percentage error was within the recommended percentage error according to the ERS/ATS ($\pm 3.5\%$) (Miller et al., 2005).

Each subject performed the maneuvers while seated upright (Stewart & Basson, 1991), and wearing a nose clip to prevent air leaks through the nostrils. The subject had to seal his/her mouth around a carton mouth piece connected to the flow meter, take three normal breaths,

then inhale to total lung capacity (TLC) and immediately exhale maximally, as rapidly and as forcefully as possible. The subject was encouraged to inhale rapidly to full inspiration and only pause for 1 second before beginning their forceful and rapid exhalation. The reason for this encouragement was that previous studies found reductions in PEF_R (Tzelepis et al., 1997) and FEV₁ when inspiration was slow and/or there was a 4-6 second pause at full inspiration (Miller et al., 2005). Each subject performed a minimum of three and a maximum of eight manoeuvres. A one minute rest period was allowed between maneuvers. At least two flow-volume curves, of which the FVC and FEV₁ did not differ by more than 5%, were required from each subject. The curve with the greatest sum of FVC and FEV₁ was selected as the final measurement. Both FVC and FEV₁ were measured in litres at BTPS.

Peak expiratory flow rate (PEFR) and maximum instantaneous forced expiratory flow at 50% of FVC (MEF_{50%}) were also obtained from the flow-volume curve test. All flow-over-time variables were measured in L.s⁻¹. According to the recommendations of the ERS/ATS (Miller et al. 2005) the highest values from at least two flow-volume curves, of which these flows did not differ by more than 10% or 0.3 L.s⁻¹, were selected as the final measurement.

Romer & McConnell (2003a) assessed pulmonary function in 46 healthy, physically active individuals on two separate occasions (> 2 days but < 3 weeks between sessions). The 95% ratio limits of agreement for FVC and PEF_R according to Bland & Altman (1986) were calculated and proved to be acceptable. The random error component of the agreement ratios was 1.047 and 1.149 for FVC and PEF_R, respectively. FEV₁ was not reported in the study.

3.2.2. Maximum voluntary ventilation (MVV)

MVV in 12 seconds was measured to assess respiratory muscle endurance. The test was administered using the same equipment and software as the flow-volume curve test. The subject was instructed to inhale and exhale through the mouth piece as maximally and as rapidly as possible for 12 s. MVV was performed at least 3 times and the best measurement

of two efforts within 10% or 15 L.min⁻¹ of variance was recorded as the final score. Ventilated air was measured in litres per minute (L.min⁻¹).

During both manoeuvres, subjects received verbal encouragement and motivational coaching using phrases, such as “force out the air”, “fast”, “keep on breathing out” to ensure a maximal effort by the subject. The inter-test reproducibility of MVV seems to be high. Romer & McConnell (2003a) found no significant differences in MVV (187 ± 28 vs. 188 ± 27 L.min⁻¹, $P > 0.05$) between the baseline and repeated measurements.

3.2.3. Respiratory muscle strength

Maximal inspiratory and expiratory mouth pressures (MIP and MEP) were measured with a portable, hand-held respiratory pressure meter (*Micro RPM* meter, Micro Medical Ltd., England) to determine maximal inspiratory muscle strength and maximal expiratory muscle strength, respectively (see Appendix C). The meter consisted of a microcomputer unit, a valve assembly, a mouth pressure bacterial filter and a flanged rubber mouth piece. Portable, hand-held devices have been demonstrated to measure inspiratory efforts accurately and reliably (Hamnegard et al., 1994).

Two valve assemblies were used. The expiration valve allowed the subject to inhale fully through the valve, but was closed during expiration. The inspiration valve worked in the opposite manner. During manufacturing a small controlled leak (2 mm orifice) was inserted in the micro-computer unit of the device to prevent the generation of false high readings generated by closure of the glottis during the MIP maneuver, and the use of the buccal muscles during the MEP maneuver (Black & Hyatt, 1969). All the parts of the meter were connected to the microcomputer which was connected via a 9 way serial cable to the serial port of the computer in order to use specialized computer software (*Puma*, Micromedical Ltd., England) for analysis. The sampling frequency of mouth pressure was 100 Hz.

Both maximum mouth pressure tests are volitional and greatly effort dependant, therefore, strong verbal encouragement and clear instructions were given during each effort to ensure

that the subject provided a maximal effort. Subjects received real-time visual feedback on a pressure-over-time area graph displayed on the computer monitor, as well as the value of the average pressure generated over one second. The unit of measurement for all maximum mouth pressure variables was cmH₂O. All pressure values for MIP were negative pressures, however, the minus was not added to the value for easier reading purposes.

Maximum inspiratory mouth pressure (MIP): The inspiration valve was connected to the device for the assessment of MIP. Subjects performed the test in a standing position, wearing a nose clip to prevent air leaks through the nostrils (Romer & McConnell, 2003a; Volianitis et al., 2001a). The subject inserted the mouth piece into the mouth with the flange positioned over the gums and inside the lips, with the bite blocks between the teeth.

The subject had to inhale normally, then insert the mouth piece and exhale to residual volume (RV) followed by an inhalation through the device as forcefully and rapidly as possible. Each maximal effort was produced from RV; because this method can control for the initial length of the inspiratory muscles and it ensures that the highest pressures are recorded. This brief, quasi-static contraction of the inspiratory muscles are also known as the Mueller maneuver (Volianitis et al, 2001a). The subject started the effort at the sound of a “beep”, while the test administrator clicked “start” on the test screen. Subjects were instructed to sustain the maximal inspiratory effort for 1.5 seconds. Subjects were given three efforts to familiarize them with the procedure. A total of twelve technically, satisfactory maneuvers (4 sets of 3 consecutive efforts with 60 seconds rest between sets) were performed.

Volianitis et al. (2001a) reported the coefficient of variation (CV) to assess the within-session reproducibility of MIP. Fourteen healthy subjects performed 18 maximal efforts within a session. The mean \pm SD CV for MIP was 5.3 ± 3.7 kPa (54 ± 37.7 cmH₂O). Volianitis et al. (2001a) also reported the coefficient of repeatability (CR) for the maximum values of MIP between two occasions. They found a similar CR (25 cmH₂O) compared to Maillard et al. (1998) (28 cmH₂O).

Maximum expiratory mouth pressure (MEP): The same procedures were followed for MEP assessment after the expiration valve was connected to the device. The subject had to exhale, then insert the mouth piece and inhale to total lung capacity (TLC) followed by an exhalation as forcefully and as rapidly as possible (Valsalva maneuver). Subjects were encouraged to ensure that their lips did not separate from the mouth piece during the exhalation. A total of twelve technically, satisfactory maneuvers (4 sets of 3 consecutive efforts with 60 seconds rest between sets) were performed.

Romer & McConnell (2003a) reported excellent inter-test reproducibility for MEP (average value over one second). Forty six healthy, physically active individuals performed MEP measurements on two separate occasions (> 2 days but < 3 weeks between sessions). The 95% ratio limits of agreement for MEP according to the method by Bland & Altman (1986) were calculated and proved to be high. The random error component of the agreement ratio was 1.045.

Selection of end result for RM strength measurements: Twelve efforts were selected to assess MIP and MEP to ensure enough readings which truly represents a maximum. Wen and colleagues (1997) found that the average of the highest three values of only a few efforts underestimated the average value of the three highest values during twenty efforts.

Previous studies mostly reported peak inspiratory pressure, which is the highest value out of the total number of efforts during a single assessment. In the present study the CV (mean \pm SD) for 180 subjects was $8.1 \pm 4.2\%$ and $8.2 \pm 4.9\%$ for the twelve consecutive MIP and MEP measurements, respectively. Thus, due to the substantial intra-individual variability of mouth pressure measurements, the average value of the highest three measurements, within 5% variability, was defined as maximum inspiratory mouth pressure (MIP) and maximum expiratory mouth pressure (MEP). The highest three measurements for MIP and MEP were selected as follows: the highest 2 values measured between the 1st and the 9th inspiratory effort, and the highest value measured between the 10th and the 12th effort. Thus, MIP and MEP represented mean maximum inspiratory muscle strength and mean maximum expiratory muscle strength, respectively. This calculation of MIP and MEP (average of the

highest three values) was considered a valid estimation of inspiratory and expiratory muscle strength.

However, peak maximum inspiratory mouth pressure calculated as the highest value of the highest three values for MIP (within 5% variability) showed an almost perfect correlation ($r = 0.9995$, $P < 0.001$) with the average of the highest three MIP values. Similar results were obtained for MEP ($r = 0.9994$, $P < 0.0001$). To simplify the results, MIP and MEP (average of highest three values) were used as the only representations of inspiratory and expiratory muscle strength, respectively. These values were also used to compare RM strength to that of other studies which used either peak and/or mean MIP and MEP values, respectively.

Reproducibility: The inter-test reproducibility of all the outcome variables of the respiratory muscle function assessments was calculated through interclass correlation coefficients (ICC). The ICC for all lung volume and flow-over-time variables ranged from 0.88 to 0.96 and for the pressure variables from 0.89 to 0.94. A detailed table of all these results is depicted in Appendix D.

Twenty metre multistage shuttle run test: The test was conducted to assess aerobic fitness. The 20 m multistage shuttle run test (Australian Sports Commission, 2000) is a progressive time test where the subject has to run between two beacons placed 20 metres apart. A single run between the beacons represented one shuttle. A “beep” sound on a compact disk recorder indicated the start of the next shuttle. The time between two beeps progressively decreased with every level to increase the sprinting speed with every level. Each level consisted of a number of shuttles. The goal was to reach the beacon before the next “beep” sound. If the subject failed to reach the beacon before the “beep” on two consecutive occasions, the test was terminated. The final score is the last level and shuttle that the subject could complete.

Previous studies have shown that the 20 m multistage shuttle run test is a reliable method to assess fitness in groups. Leger and Lambert (1982) reported a correlation coefficient of $r =$

0.975 (SEE = 2 ml O₂.kg⁻¹.min⁻¹) for test-retest reproducibility. Cooper et al. (2005) found a mean ± SD difference of 0.4 ± 1.4 ml O₂.kg⁻¹.min⁻¹ (*P* = 0.19) between predicted VO_{2max} values obtained from the 20 m multistage shuttle run on two separate occasions. The random error component of the agreement ratio was 1.050 for predicted VO_{2max}.

B. STUDY TWO

A descriptive study to determine the effect of RM detraining on RM function.

1. Subjects

Twenty of the 22 women field hockey players who participated in STUDY ONE, continued to take part in STUDY TWO. Only two subjects from the experimental group had to withdraw from STUDY TWO to play club hockey overseas for one quarter of the year in 2007. No recruitment method was needed for this study and written informed consent was given during STUDY ONE.

Inclusion criteria: Only the subjects who participated and completed the intervention were allowed to take part in STUDY 2. Subjects had to be free from respiratory tract infection at least one week prior to testing.

Exclusion criteria: A subject was excluded from the study if she showed abnormality in resting pulmonary function, or if she was unable to continue with the RMT during the 12-week intervention due to illness, injury, or the quitting hockey. Subjects were excluded from the study if they participated in any other competitive sport during the hockey off-season. None of the subjects participated in other sports during the hockey off-season. The three subjects who showed abnormal pulmonary function (original group of 25) were also excluded from STUDY 2.

Characteristics: The mean physical characteristics did not change significantly, with the exclusion of the two subjects who could not continue with STUDY 2. The 20 subjects had a mean ± SD (range) age of 21 ± 2 (18 – 25) years, height 166 ± 7 (154 – 177) cm and weight

61 ± 9 (46 – 78) kg. One subject suffered from exercise-induced asthma. The subject used an asthma inhaler as needed during the occurrence of asthma symptoms. However, the subject showed normal pulmonary function and her respiratory muscle function outcome variables did not differ significantly from the rest of the group.

The subjects had an average of 11 ± 3 (8 – 15) years competitive experience in hockey. At 9 week post-intervention the subjects trained on average 4 ± 1.5 times per week and at 20 weeks post-intervention, subjects trained on average 5 ± 2.4 times per week. Competitive experience ranged from provincial to junior national, national and international level. During the detraining study (end of 2006 to March 2007), eight players competed internationally and were selected for the national team. The twelve remaining subjects all competed at provincial level. Six out of the twelve provincial subjects were selected for the junior national team in previous years.

2. Experimental design and procedures

All subjects underwent respiratory muscle function measurements (FVC, FEV₁, PEF_R, MVV, MIP and MEP) in the laboratory. Subjects visited the laboratory on two separate occasions. Respiratory muscle function assessments were repeated at 9 weeks after the completion of the 12 week intervention (reported as: 21wk), and at 20 weeks after the completion of the 12 week intervention (32 weeks post BL, reported as: 32wk). Subjects were required to keep a diary of their physical activity throughout the 20 weeks. Subjects had to indicate, separately, the number of hours they did specific hockey training, hockey fitness sessions, training sessions in the gymnasium and any other form of physical activity throughout the 20 weeks.

3. Measurements and tests

All subjects underwent the same physiological assessments as described in STUDY ONE (see 3.2 under section A. STUDY ONE, pp.95 – 101).

SECTION TWO

C. STUDY THREE

A descriptive study to determine the predictors of RM function.

1. Subjects

One hundred and sixty subjects from eight different sports codes volunteered to participate in the study. Table 1 depicts the groups and sports codes represented by the total number of subjects. Twenty two normal, non-athletic subjects were included as a control group in the study to establish possible differences between athletes and non-athletes with regard to kinanthropometric and respiratory muscle function characteristics. In this section, the word “athlete” refers to a subject who is a healthy sports person and competitive in a specific sport and a “non-athlete” refers to a non-competitive, healthy subject. Only subjects who met the inclusion criteria were selected for the study. None of the subjects had any experience in respiratory exercises prior to testing.

Selected Sports: Athletes from eight different sport clubs were recruited for the study. Competitive athletes from the following sports were included: Endurance sports, namely cycling, running, rowing and swimming. Team sports included rugby, field hockey, netball and underwater hockey.

Table 3: Groups and gender description of subjects

<i>Competitive sport</i>	<i>Men</i>	<i>Women</i>	<i>Total</i>
Field hockey		27	27
Cycling	23		23
Rugby	16		16
Netball		14	14
Swimming	12	8	20
Running	12	8	20
Underwater hockey	20		20
Rowing	20		20
Non-athletic individuals	12	10	22
Total	115	67	182

Inclusion criteria for competitive athletes: Each subject had to meet the criteria of competitive experience at club, provincial, or national level in their specific sport. Each subject had to competitively engage in their sport for at least three years and engage in their specific sport at least four times a week during the competitive season and engage in fitness sessions (e.g. gym, jogging, cross-training) at least twice a week. Competitive athletes had to be between the ages of 17 to 34 years old.

Inclusion criteria for non-athletic subjects: Each subject had to meet the criteria of no participation in competitive sport and no history of participation in competitive sport. Subjects were included in the study if they were either sedentary, or engage in moderate physical activity less than four times per week. The intensity of a single exercise session could not exceed a perceived exertion of 60% of maximal perceived effort and could not exceed duration of more than one hour. Physical activities included: light jogging, walking, swimming and exercise in a gymnasium.

All subjects (athletes and non-athletes) should have been free from a history of pulmonary function impairment (excluding asthma) and free of respiratory tract infection for at least

four weeks prior to testing. It was decided to include individuals with asthma or a childhood history of asthma, provided that the subject showed normal pulmonary function during testing. For the same reason smokers and/or ex-smokers were allowed to participate in the study. However, if the subject showed abnormal pulmonary function, he/she was excluded from the study.

Exclusion criteria: A subject was excluded from the study if he/she showed abnormality in resting pulmonary function. The latter was classified as abnormal when the measured forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁) and/or peak expiratory flow rate (PEFR) were less than 80% of the age- and gender specific predicted value.

2. Experimental design and procedures

All subjects underwent a series of kinanthropometric and pulmonary measurements in the laboratory. Subjects visited the laboratory once, during which they completed a consent form, a short questionnaire regarding their health and general physical activity history, as well as the physical and physiological tests and measurements. Tests were conducted in the following order: questionnaire, kinanthropometry, lung volumes and flow rates (pulmonary function), maximum voluntary ventilation (MVV), maximum inspiratory mouth pressure (MIP) and maximum expiratory mouth pressure (MEP). Subjects were requested to refrain from exercise, alcohol and caffeine 24 hours before testing. Subjects were also instructed to avoid eating and drinking fluids, except water (*ad libitum*), two hours before testing, to ensure the correct measurement of body weight.

Subjects were asked to perform these tests during the competition phase of their specific sport season, therefore the assessments for rugby, field hockey and netball players were during winter (mid-year); swimmers, rowers and underwater hockey players during summer (end or beginning of the year). The cyclists were assessed during the first quarter of the year, which may be classified as their major competitive season. The runners were tested near the end of the year, since their competitive season lasted throughout the year.

Assessment of athletes during the competition season for each sport, ensured that subjects had a reasonable level of fitness for their specific sport during their evaluation.

3. Measurements and tests

All subjects underwent the same kinanthropometric and respiratory muscle function assessments as described in STUDY ONE (see 3.1 and 3.2 under section A. STUDY ONE, pp.92 – 101).

Intra-individual variability: The CV (coefficient of variation) for the twelve MIP and MEP measurements during a single assessment of all the subjects participating in the study (n = 182) was calculated to determine the intra-individual variability of these measurements. The mean CV (\pm SD) for the average of 12 MIP and MEP measurements was $8.1 \pm 4.2\%$ and $8.2 \pm 4.9\%$, respectively. The mean CV (\pm SD) for the average of three highest MIP and MEP measurements was $1.6 \pm 0.8\%$ and $1.5 \pm 0.8\%$, respectively. The mean of the three highest efforts was used as the final outcome variable of MIP and MEP.

Ethics and consent: The testing procedures and protocols within the project were approved by the Division of Research Development, Stellenbosch University (Ref: 79/2008). All testing procedures, the requirements of the intervention and the risks involved with participation, as stated in the consent forms (Appendix E and F), were explained to each subject and the opportunity was given for questions. The subject agreed to all testing requirements and procedures by giving his/her written consent.

D. STATISTICAL ANALYSIS

All descriptive statistics were presented as mean \pm SD (standard deviation) and the graphical representations of data as mean \pm SEM (standard error of the mean).

Section one: Baseline kinanthropometric and respiratory muscle function differences between the experimental (EXP) and control (CON) groups were determined by unpaired Student t-tests. The data for each of the dependent variables of respiratory muscle function (FVC, FEV₁, PEFR, MEF_{50%}, MVV, MIP and MEP) and aerobic fitness (number of shuttles during the bleep test) was analyzed with a two factor (intervention \times time) repeated measures analysis of variance (ANOVA) with repeated measures over the second factor (time). Fisher LSD (*least significant difference*) tests were used for all *post hoc* analysis of the main effects. The same statistical tests (two factor ANOVA's and Fisher LSD) were used to analyze the differences in the percentage changes in the respiratory muscle function and aerobic fitness variables over time between the EXP and CON groups. Coefficients of variation (CV) were calculated to illustrate intra-individual variations in MIP and MEP measurements during baseline assessments ($[SD/mean] \times 100$). Pearson product moment correlations were calculated to determine relationships between changes in respiratory muscle function variables and changes in aerobic fitness. The reproducibility of the respiratory muscle function variables was determined using intra-class correlation coefficients (ICC between pre-baseline and baseline measurements, Appendix D).

Section two: Kinanthropometric and respiratory muscle function outcome differences between athletes and non-athletes (men and women respectively), and between men athletes and women athletes, were determined by unpaired Student t-tests. Box-and-whisker plots were used to illustrate respiratory muscle function comparisons between athletes and non-athletes (men and women respectively). Pearson product moment correlations were used to determine significant relationships between kinanthropometric variables and respiratory muscle function variables, and MIP, MEP and MVV, respectively. Best subsets multiple regression analyses were performed to determine the predictors of respiratory muscle strength (MIP and MEP) and endurance (MVV). Regression equations were generated to

obtain the lowest possible standard error of prediction. Multiple regression analysis results were reported in table format as the Beta coefficients or factors, SE (standard error) of Beta, B, SE of B and t-value. For each dependent variables (MIP, MEP, MVV) the R^2 (multiple correlation coefficient) and R^2_{adjusted} (adjusted coefficient of determination) and F-value were reported. The error of prediction was calculated as the standard error of the estimate (SEE).

All statistical analyses were performed using Statistica 8 software (data analysis software system, version 8.0, 2008). Level of statistical significance for all statistical analyses in section one and two (ANOVA, Fisher LSD, Student t-tests, Pearson product moment correlations, stepwise multiple regression analyses) were chosen *a priori* as $P < 0.05$.

CHAPTER SEVEN

RESULTS

A. INTRODUCTION

This chapter is divided into two sections, reporting the results of Study 1 and 2 under **Section One** and Study 3 under **Section Two**. With the exception of two subjects, the same group participated in Study 1 and Study 2. Two subjects had to withdraw from Study 2 due to hockey commitments overseas. The results from Study 1 will be referred to as the respiratory muscle training (RMTP) study and the results of Study 2 will be referred to as the respiratory muscle detraining (DT) study. The results of these two studies were reported together in order to clearly show the overall changes from baseline values in the respective variables due to RMTP and DT. **Section Two** shows the results of the predictors for RM strength (MIP and MEP) and endurance (MVV), respectively, from data in Study 3. Comparisons are made between men and women athletes with regards to RM strength and endurance. The data from Study 3 also describe the kinanthropometric and respiratory muscle function differences between athletes and non-athletes for men and women, respectively. Prediction equations for RM strength and endurance from the literature (discussed in Chapter 4) were used to calculate and compare the various predicted values for RM strength and endurance with the observed values in this study.

B. SECTION ONE

1. Subject characteristics

The twenty two women hockey players who participated in the study were randomly assigned to the experimental (n = 15) and control (n = 7) groups. There were no significant differences between the two groups for all the physical and fitness characteristics. Predicted VO_{2max} values were derived from the final scores of the baseline 20 m multistage shuttle run test according to Leger & Lambert (1982).

Table 4: Physical and fitness characteristics (mean \pm SD, range) of the experimental (EXP) and control (CON) groups.

<i>Variable:</i>	<i>EXP (n = 15)</i>		<i>CON (n = 7)</i>	
	<i>mean \pm SD</i>	<i>Range</i>	<i>mean \pm SD</i>	<i>range</i>
Age (years)	22 \pm 2.1	19 – 25	20 \pm 1.8	18 – 23
Height (cm)	166 \pm 6.5	154 – 177	168 \pm 6.8	157 – 175
Weight (kg)	61 \pm 9	46 – 78	63 \pm 8.8	53 – 76
BMI (kg per m²)	22 \pm 2.7	18 – 27	22 \pm 2.3	19 – 26
Sum of 7 skinfolds (mm) *	92 \pm 32	56 – 149	90 \pm 20	70 – 129
Predicted VO_{2max} (ml.kg⁻¹.min⁻¹)	51 \pm 5	40 – 59	50 \pm 4	42 – 55
Years of hockey participation	12 \pm 3	8 – 15	11 \pm 2	8 – 14
Exercise sessions per week	8 \pm 3	5 – 14	8 \pm 3	5 – 12

* sum of 7 skinfolds = Σ (triceps, biceps, subscapular, supraspinale, abdominal, front thigh, medial calf)

There were no significant differences in the baseline pulmonary function and respiratory muscle (RM) endurance (MVV) between the EXP and CON groups (Table 5). Maximum expiratory mouth pressure (MEP) showed no significant difference between the EXP and CON groups, however, maximum inspiratory mouth pressure (MIP) was significantly higher in the CON group compared to the EXP group ($P = 0.01$). The latter finding may be attributed to one very high measurement (MIP = 185 cmH₂O) of a subject in the CON group and very low measurements (≤ 80 cmH₂O) of two subjects in the EXP group. Since, the effect of RMTP and DT were quantified as changes in values from baseline measurements for each subject, these outlier values were included in the subsequent analysis.

Table 5: Respiratory muscle function (mean \pm SD, range) measured at baseline for the experimental (EXP) and control (CON) groups.

<i>Variable:</i>	<i>EXP (n = 15)</i>		<i>CON (n = 7)</i>	
	<i>mean \pm SD</i>	<i>Range</i>	<i>mean \pm SD</i>	<i>Range</i>
FVC (L)	4.07 \pm 0.6	3.20 – 4.93	4.26 \pm 0.5	3.68 – 4.77
FEV₁ (L)	3.40 \pm 0.4	2.91 – 4.21	3.62 \pm 0.3	3.27 – 4.06
PEFR (L.s⁻¹)	7.66 \pm 1.1	6.48 – 10.70	7.96 \pm 1.1	6.79 – 9.79
MEF_{50%} (L.s⁻¹)	3.95 \pm 0.7	2.43 – 4.90	4.38 \pm 0.9	3.12 – 5.98
MVV (L.min⁻¹)	141.0 \pm 19.1	117.1 – 183.2	149.7 \pm 19.8	127.5 – 181.6
MIP (cmH₂O)	115 \pm 24 *	73 – 155	144 \pm 22	120 – 185
MEP (cmH₂O)	130 \pm 27	93 – 178	138 \pm 31	91 – 195

* significantly different from control group (CON).

2. Adherence to respiratory muscle training programme and detraining period

Training adherence to the RMTP was similar between groups ($P = 0.09$). The EXP group completed a total of 133 ± 8 sessions out of the prescribed 144 sessions ($92 \pm 6\%$) and the CON group completed a total of 139 ± 8 sessions out of the prescribed 144 sessions ($96 \pm 6\%$). The EXP and CON group missed 6 ± 4 and 3 ± 4 days respectively, out of a total of 72 days of training due to illness. The maximum number of days missed by a subject was 10 days (three subjects in the EXP group and one subject in the CON group). None of the subjects ($n = 20$) continued with RMT during the DT period. Subjects followed their prescribed fitness programme for the hockey off-season and there were no significant differences between the training volume of the EXP and CON groups during the DT period (EXP: 5 ± 3 vs. CON: 4 ± 2 training sessions per week, $P = 0.55$).

3. Changes in variables over time between experimental and control groups

Figure 1 illustrates the time line of the assessments made during RMTP and DT. The measurements taken at these different time points were used to determine a change over time due to the intervention (RMTP) and due to detraining (DT). Mid-intervention assessments are reported as **6wk**, post-intervention assessments as **12wk**, 9 weeks into DT as **21wk** and 20 weeks into DT as **32wk**. Post-intervention assessments at 12wk also served as the baseline assessments for DT (onset of the detraining period).

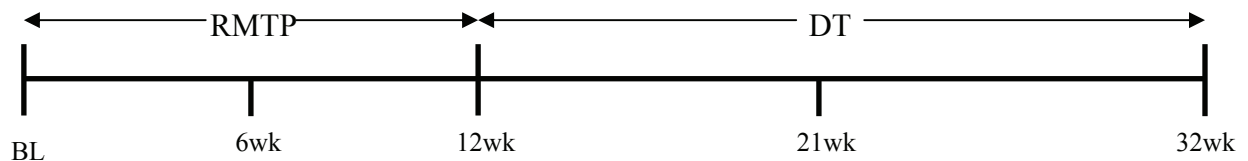


Figure 1: Schematic diagram of the timeline of assessments during RMTP and DT. Time points (*bottom line*) for respiratory muscle function assessments during RMTP and DT (*top of line*).

The results of the outcome variables during RMTP are reported as percentage changes from baseline values at 6wk (mid-intervention) and at 12wk (end of intervention). The results of

the outcome variables during DT are reported as percentage changes from 12wk (baseline values for the DT period), at 21wk (9 weeks after the termination of RMTP) and at 32wk (20 weeks after the termination of RMTP). The absolute values of all the measured variables (pulmonary function, respiratory muscle strength and endurance, and 20 m multistage shuttle run tests) are reported in table format in Appendix G.

3.1 Pulmonary function changes over time

Lung volumes:

FVC: FVC (Figure 2a) changed significantly over time in both groups, however the group – time interaction was not significant ($P = 0.996$). **RMTP:** After 6 weeks of specific RMT, the EXP group showed a significant increase in FVC. At the end of the RMTP (12wk) both groups showed a significant increase of 5 ± 4.5 ($P = 0.001$) and $5 \pm 2.9\%$ ($P = 0.02$) respectively. **DT:** FVC decreased to below baseline values at 21wk (9 weeks after cessation of intervention) in both groups and then increased slightly to above baseline values at the end of the study.

FEV₁: Figure 2b illustrates the changes over time for FEV₁ in both groups. There was a significant change over time ($P < 0.001$) in both groups, but no significant group – time interaction ($P = 0.306$). **RMTP:** There was a 5 ± 4 and $6 \pm 5\%$ increase from baseline at 12wk in the EXP and CON groups, respectively. **DT:** Although there was no significant difference in the percentage changes over time between the EXP and the CON groups during the detraining period, the CON group showed a higher percentage decrease ($7 \pm 4.1\%$) at 21wk compared to the percentage decrease in the EXP group ($4 \pm 3.6\%$). Similarly to FVC, there was a slight but non-significant improvement in FEV₁ at 32wk.

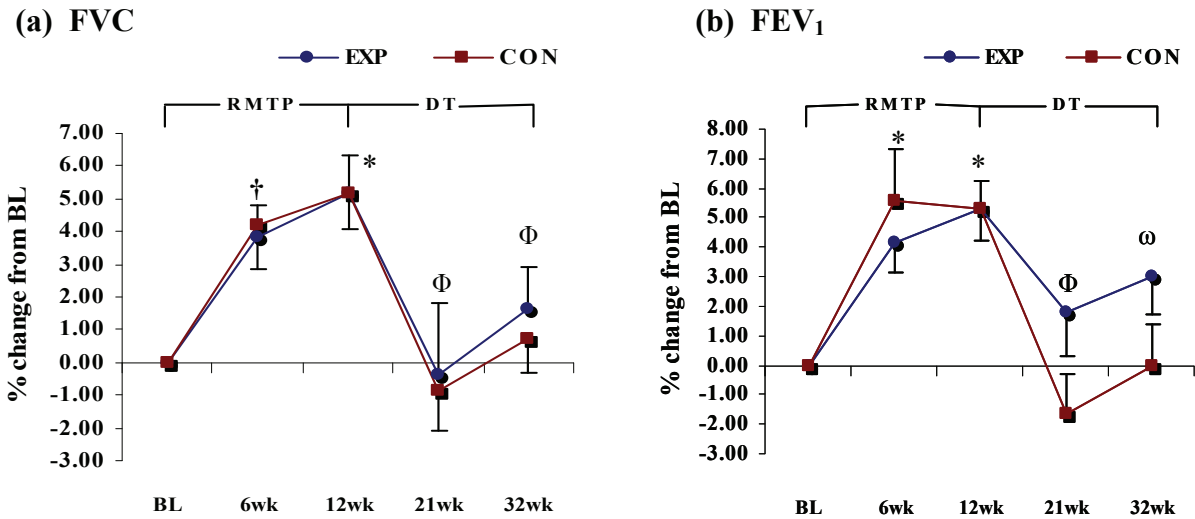


Figure 2: Percentage changes (*error bars*: SEM) in lung volumes over time during RMTP and DT for the EXP (*blue*) and the CON (*red*) group. (a) Forced vital capacity (FVC) in L, (b) Forced expiratory volume in one second (FEV₁) in L. † Significantly different from baseline (BL) in EXP (n = 15) group, * significantly different from baseline (BL) in both EXP and CON (n = 7) groups, Φ significantly different from 12wk in both EXP (n = 13) and CON (n = 7) groups, ω significantly different from 12wk in CON group.

Air flow:

PEFR: There was an interaction over time (Figure 3a) within groups ($P = 0.03$), but no interaction between groups over time ($P = 0.99$). **RMTP:** The EXP group showed a significant increase ($4 \pm 5.2\%$) in PEFR after 6 weeks ($P = 0.02$) and after 12 weeks ($5 \pm 6.3\%$ increase from BL, $P = 0.01$) of specific RMT. The increase in PEFR in the CON group over the same time was not significant. **DT:** Nine weeks after the cessation of RMT, PEFR remained unchanged in the EXP group, but dropped by 1% in the CON group. At 32wk, PEFR decreased further in the CON group. However, none of the changes in PEFR were statistically significant over the DT period.

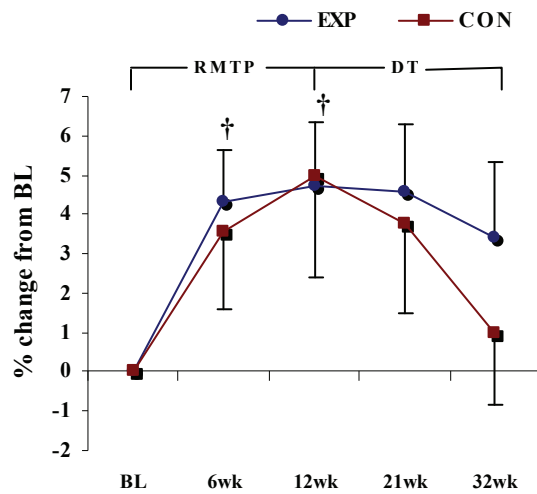
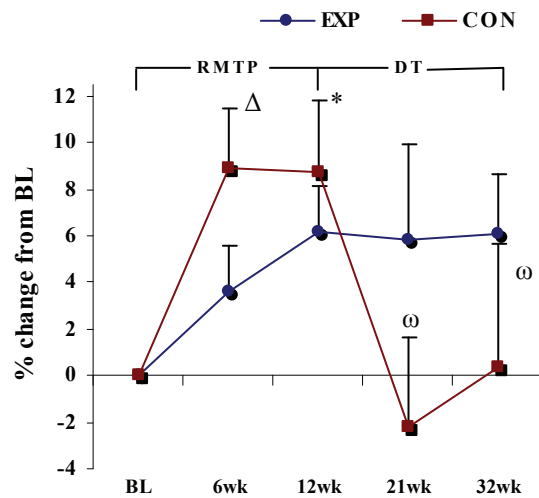
(a) PEF_R**(b) MEF_{50%}**

Figure 3: Percentage changes (*error bars*: SEM) in air flow variables over time during RMTP and DT for the EXP (*blue*) and the CON (*red*) group. (a) Peak expiratory flow rate (PEFR) in $L \cdot s^{-1}$, (b) maximal instantaneous forced expiratory flow at 50% FVC (MEF_{50%}) in $L \cdot s^{-1}$. † Significantly different from baseline (BL) in EXP ($n = 15$) group during RMTP, Δ significantly different from baseline (BL) in the CON ($n = 7$) during RMTP, * significantly different from baseline (BL) in both (EXP and CON) groups during RMTP, ω significantly different from 12wk in CON group during DT.

MEF_{50%}: There was an interaction over time (Figure 3b) within both groups ($P = 0.003$), and a significant interaction between groups over time ($P = 0.05$). **RMTP:** The CON group showed a significant increase ($9 \pm 6.8\%$, $P = 0.02$) after 6 weeks of placebo training, which remained constant after 12 weeks ($P = 0.01$). The EXP group showed a non-significant (4%) increase after 6 weeks of RMT and this increased further to a significant $6 \pm 7.6\%$ after 12wk ($P = 0.02$). **DT:** MEF_{50%} in the EXP group remained unchanged at 21wk ($0 \pm 15.5\%$ decrease) and at 32wk ($0 \pm 7.7\%$ decrease) compared to the 12wk values (onset of detraining period). However, in the CON group there was a significant decrease of $10 \pm 4.9\%$ ($P = 0.002$) at 21wk to a mean value below the baseline value. At 32wk, the decrease in MEF_{50%} stayed significantly lower compared to 12wk ($8 \pm 7.3\%$, $P = 0.02$), although there was a slight increase from 21wk to 32wk. The percentage decreases in MEF_{50%} did not differ significantly between the two groups at 21wk, however at 32wk the percentage decrease from 12wk was significantly higher in the CON group compared to the percentage decrease in the EXP group (0 ± 7.7 vs. $8 \pm 7.3\%$, $P = 0.04$).

3.2 Respiratory muscle strength changes over time

Global inspiratory and expiratory muscle strength was assessed by measuring maximum inspiratory mouth pressure (MIP) and maximum expiratory mouth pressure (MEP), respectively, at each of the time points during the RMTP and DT period.

Maximum inspiratory and expiratory mouth pressures (MIP and MEP):

MIP: There was a significant interaction over time within groups during RMTP and DT period ($P = 0.04$), but no significant group – time interaction (Figure 4a). **RMTP:** There was no significant change in MIP in both groups after 6 weeks of RMT. However, after 12 weeks the EXP group showed a significant increase in MIP ($13 \pm 15\%$, $P = 0.003$), while MIP in the CON group remained relatively unchanged ($2 \pm 11\%$). **DT:** There was a slight ($4 \pm 12\%$), but non-significant decrease in MIP after 9 weeks of detraining and these values remained constant at 32wk in the EXP group. MIP values remained relatively unchanged during the detraining period in the CON group.

MEP: There was a borderline significant interaction over time within groups ($P = 0.045$) during the RMTP and DT period, but no significant interaction between the two groups over time (Figure 4b). **RMTP:** There was a significant increase in MEP in the EXP group after 6 weeks ($9 \pm 13\%$, $P = 0.01$), but no further increase after 12 weeks. The CON group only improved their MEP values with $4 \pm 9\%$ after 12 weeks; this change, however, was not significant ($P = 0.109$). **DT:** MEP in the CON group returned to baseline values after 9 weeks of detraining and remained at baseline values until the end of the detraining period (32wk). Although there was a decrease in MEP in the EXP group during the detraining period, these changes were not statistically significant.

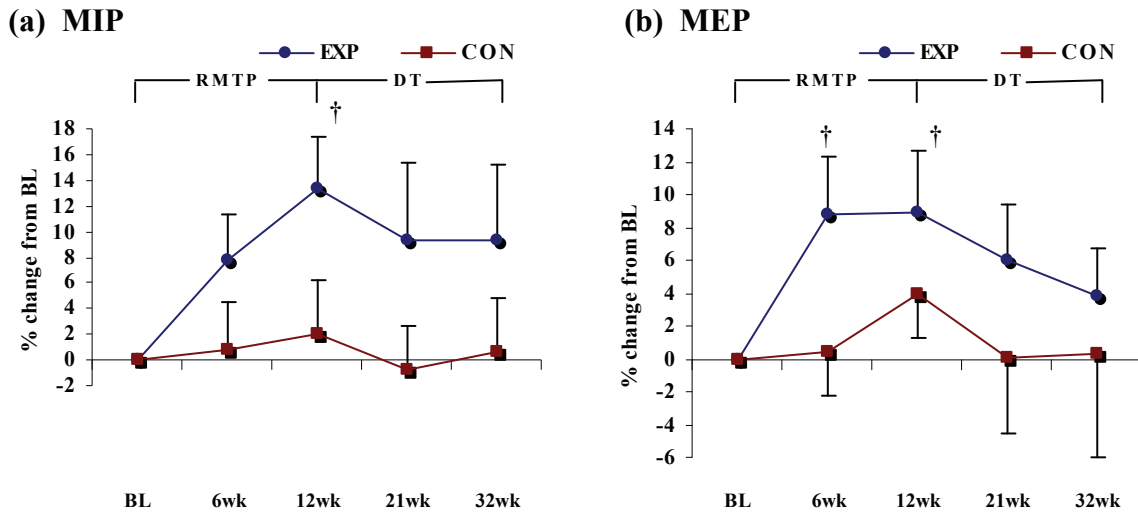


Figure 4: Percentage changes (*error bars: SEM*) in maximum respiratory mouth pressure variables over time during RMTP and DT for the EXP (*blue*) and the CON (*red*) group. (a) Maximum inspiratory mouth pressure (MIP) in cmH₂O, (b) maximum expiratory mouth pressure (MEP) in cmH₂O. † Significantly different from baseline (BL) in EXP (n =15) group during RMTP.

3.3 Respiratory muscle endurance changes over time

Respiratory muscle endurance was assessed by measuring maximum voluntary ventilation (MVV) in 12 seconds (L.min⁻¹) at each of the time points during the RMTP and DT period.

Maximum voluntary ventilation (MVV):

MVV: There was an interaction over time within groups ($P < 0.001$), but no interaction between the two groups over the RMTP and DT period (Figure 5). **RMTP:** After six weeks of RMT training, there was a 3% increase in MVV values in the EXP group and no change in the CON group. At 12wk both groups showed a significant increase in MVV (*EXP:* 8 ± 4.9 , $P < 0.001$; *CON:* $6 \pm 6.4\%$, $P = 0.02$). **DT:** MVV in the EXP and CON group showed no significant decrease at 21wk from the 12wk values, however at 32wk there was a significant decrease ($4 \pm 6.5\%$, $P = 0.03$) from the 12wk values in the EXP group, and no significant decrease in the CON group.

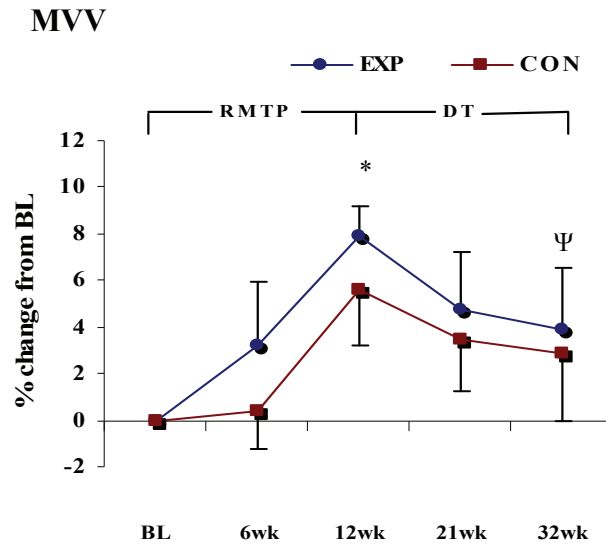


Figure 5: Percentage changes (*error bars: SEM*) in maximum voluntary ventilation (MVV in L.min⁻¹) over time during RMTP and DT for the EXP (*blue*) and CON (*red*) group. * significantly different from baseline (BL) in both EXP and CON (n = 7) groups, Ψ significantly different from 12wk in EXP group.

4. Aerobic fitness

4.1. Changes over time

The twenty meter multistage shuttle run tests were conducted on four different occasions. Pre- and post-hockey season tests to assess aerobic fitness changes were conducted during the hockey season with additional RMT and during the following hockey season without RMT. Eight subjects in the EXP group and five subjects in the CON group completed these tests. Figure 6 illustrates the time line of fitness tests over two hockey seasons.

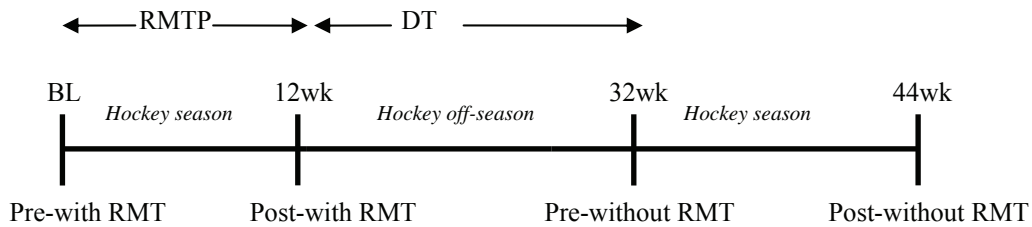


Figure 6: Schematic diagram of the timeline for the 20 m multistage shuttle run tests. *Bottom of line:* Illustration of time points for shuttle run test assessments, (Pre-with RMT, Post-with RMT, Pre-without RMT, Post-without RMT) and how they correspond with the time points for respiratory muscle function assessments during RMTp and DT (*top of line*) and the respective hockey competitive or off-seasons (*above line in italics*).

Hockey season with RMT: There was a significant improvement in the number of shuttles achieved by the EXP group (increase by $8 \pm 16\%$, $P = 0.045$) and the CON group (increase by $12 \pm 11\%$, $P = 0.02$) during the post-season test (figure 7, *blue*). However, these changes were not statistically significantly different between the two groups.

Hockey season without RMT: There was a significant decrease in the number of shuttles achieved by the EXP group (decrease by $4 \pm 11\%$, $P = 0.03$) and the CON group (decrease by $6 \pm 7\%$, $P = 0.01$) during the post-season tests (figure 7, *maroon*). However, the declines in fitness performance were not significantly different between the two groups.

Changes in fitness between seasons: There was a significant difference between the changes in the number of shuttles achieved during the season with RMT and during the season without RMT, in the EXP ($8 \pm 16\%$ vs. $-4 \pm 11\%$, $P = 0.02$) and CON ($12 \pm 11\%$ vs. $-6 \pm 7\%$, $P = 0.006$) groups, respectively.

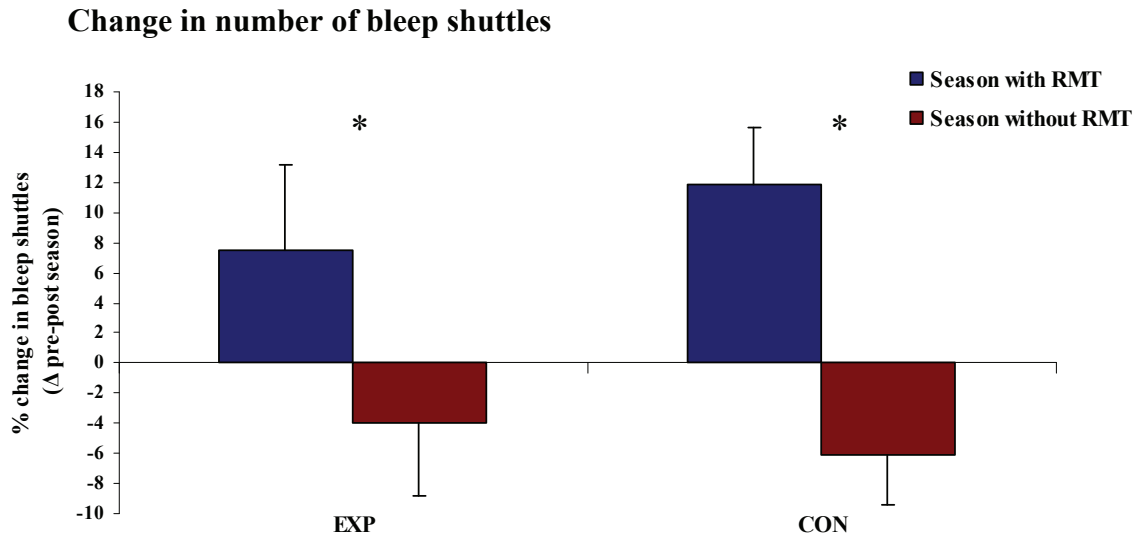


Figure 7: Percentage changes (*error bars*: SEM) in the number of bleep shuttles between the pre and post 20 m multistage shuttle run tests during the hockey season with RMT (*blue*) and the hockey season without RMT (*red*), for the EXP ($n = 8$) and the CON group ($n = 5$), respectively. * Significantly different between hockey seasons.

4.2. Correlations with changes in aerobic fitness

Correlations between the changes in the different respiratory muscle function variables and the changes in aerobic fitness over the RMTP and DT period were calculated, respectively, to determine whether there were significant relationships between the improvement in RM function variables and the improvement in aerobic fitness during RMTP, and between the decline in RM function variables and the decline in aerobic fitness during DT (Table 6). These results may explain whether aerobic fitness due to specific hockey training is one of the reasons for the improvement or decline in RM function and/or vice versa.

The only significant correlation was found in the EXP group between the percentage change in MEP and the percentage change in aerobic fitness during RMTP ($r = 0.78$, $P < 0.05$). Since most of the respiratory muscle function (except for $MEF_{50\%}$) and aerobic fitness variables revealed the same percentage changes over the RMTP and DT period, the two groups (EXP and CON) were combined (total group, $n = 13$) for further analysis. The percentage changes in MEP and PEF_R (Table 6) correlated significantly with the percentage changes in aerobic fitness (number of shuttles). No significant correlations were found

between the percentage changes in RM function and percentage changes in aerobic fitness during the 20 week RM detraining period (DT).

Table 6: Pearson’s product moment correlations between the percentage changes in respiratory muscle function variables and the percentage changes in aerobic fitness (number of shuttles) in the different groups.

% Change in variable:	% Change in number of shuttles during RMTP			% Change in number of shuttles during DT		
	EXP (n = 8)	CON (n = 5)	Total group	EXP (n = 8)	CON (n = 5)	Total group
FVC	0.51	-	-	-	-	-
FEV ₁	-	-	-	-	-	-
PEFR	0.55	0.62	0.56 *	0.51	-	-
MVV	0.60	-	-	-	-	-
MIP	0.57	-	-	-	-	-
MEP	0.78 *	-	0.65 *	0.51	-	-
Exercise per week (≥ 4 times per week)	-	-	-	-	-	-

only correlations $r = 0.50$ are reported

* significant correlations ($P < 0.05$)

C. SECTION TWO

A hundred and sixty athletes (103 men) were recruited from the first teams of eight different sports codes (± 20 subjects per sport) from Stellenbosch University sports clubs to participate in Study 3. Athletes from eight different sports codes were selected to obtain predicted values for RM strength and endurance that would be valid for a general athletic population. The athletic population was compared to healthy, non-athletic individuals to assess whether athletes have superior RM function compared to the non-athletic population. Therefore, twenty two healthy subjects who met the inclusion criteria (12 men, 10 women) were recruited to serve as non-athletes.

1. Kinanthropometric differences between athletes and non-athletes

There were no significant differences between men athletes and men non-athletes with regards to age, height, weight, BMI and body fatness (sum of 7 skinfolds). However, men athletes trained 2.3 times more than men non-athletes ($P < 0.001$). There were no significant

differences between women athletes and non-athletes with regards to age, height and weight. Although statistical analysis showed no difference in weight, women non-athletes weighed 8% more compared to women athletes ($P = 0.07$), which led to a significant difference in BMI between the two groups (Table 7). The women athletes trained three and half times more per week than women non-athletes ($P < 0.001$).

Table 7: Physical characteristics (mean \pm SD) of athletes and non-athletes.

Variable:	Men athletes (n = 103)	Men non-athletes (n = 12)	Women athletes (n = 57)	Women non-athletes (n = 10)
Age (years)	22 \pm 4.0	24 \pm 3.5	21 \pm 2.6	23 \pm 3.7
Height (cm)	182.6 \pm 6.6	179.5 \pm 5.1	168.6 \pm 6.3	167.5 \pm 5.0
Weight (kg)	81.1 \pm 12.4	78.0 \pm 9.0	62.6 \pm 8.5	68.1 \pm 11.5
BMI (kg per m²)	24.3 \pm 3.0	24.2 \pm 2.4	22.0 \pm 2.4 *	24.3 \pm 4.0
Sum of 7 skinfolds (mm)	57.4 \pm 20.6	64.5 \pm 27.7	87.9 \pm 29.3	92.9 \pm 11.3
Exercise sessions per week	7 \pm 3 *	3 \pm 2	7 \pm 3.8 *	2 \pm 1.2

* significantly different from non-athletes of the same gender, $P < 0.05$.

Men athletes had a significantly lower relative sitting height compared to non-athletes ($P = 0.01$) and a significantly smaller bi-iliac breadth compared to non-athletes ($P = 0.006$). The women athletes also had a significantly smaller bi-iliac breadth compared to non-athletes ($P = 0.007$). Body type components (Table 8) were similar between the athletes and non-athletes for both men and women.

Table 8: Body dimension and body type variables (mean \pm SD) of athletes and non-athletes.

<i>Variable:</i>	<i>Men athletes (n = 103)</i>	<i>Men non-athletes (n = 12)</i>	<i>Women athletes (n = 57)</i>	<i>Women non-athletes (n = 10)</i>
<i>Body dimensions:</i>				
Relative sitting height (%) †	52 \pm 2.0 *	54.3 \pm 7.7	52.8 \pm 1.5	52.8 \pm 1.1
Relative arm span (%) †	103 \pm 2.8	102.7 \pm 3.5	101.5 \pm 2.5	101.3 \pm 1.8
Bi-acromial breadth (cm)	41.4 \pm 2.3	42.1 \pm 3.4	36.8 \pm 3.1	38.0 \pm 3.2
Bi-iliac breadth (cm)	28.9 \pm 2.8 *	31.3 \pm 3.2	27.2 \pm 3.5 *	30.7 \pm 4.4
Chest girth (cm)	100.2 \pm 6.7	98.0 \pm 5.1	86.5 \pm 4.9	84.9 \pm 7.0
<i>Body type:</i>				
Endomorphic component	2.4 \pm 1.3	2.5 \pm 1.3	3.6 \pm 1.4	3.5 \pm 1.2
Mesomorphic component	4.8 \pm 1.3	5.2 \pm 1.2	3.8 \pm 1.0	4.3 \pm 1.5
Ectomorphic component	2.5 \pm 1.1	2.3 \pm 0.8	2.7 \pm 1.1	2.5 \pm 1.5

* significantly different from non-athletes of the same gender, $P < 0.05$.

† relative sitting height (%) and relative arm span (%) are the measured sitting height (cm) and the measured arm span (cm) divided by the standing height (cm) times 100, respectively.

2. Respiratory muscle function differences between athletes and non-athletes

Men athletes vs. men non-athletes:

Men athletes had significantly higher FVC (6.16 ± 0.9 vs. 5.55 ± 0.6 L, $P = 0.03$) and FEV₁ (4.96 ± 0.7 vs. 4.37 ± 0.6 L, $P = 0.005$) compared to non-athletes (Figure 8a & b). Respiratory muscle endurance, measured as MVV (Figure 8d), was 11% higher in men athletes compared to the mean value achieved by men non-athletes (203 ± 28 vs. 183 ± 30 L.min⁻¹, $P = 0.02$). MIP (Figure 8e), assessing inspiratory muscle strength, was 19% greater in men athletes compared to men non-athletes (147 ± 27 vs. 124 ± 32 cmH₂O, $P = 0.007$).

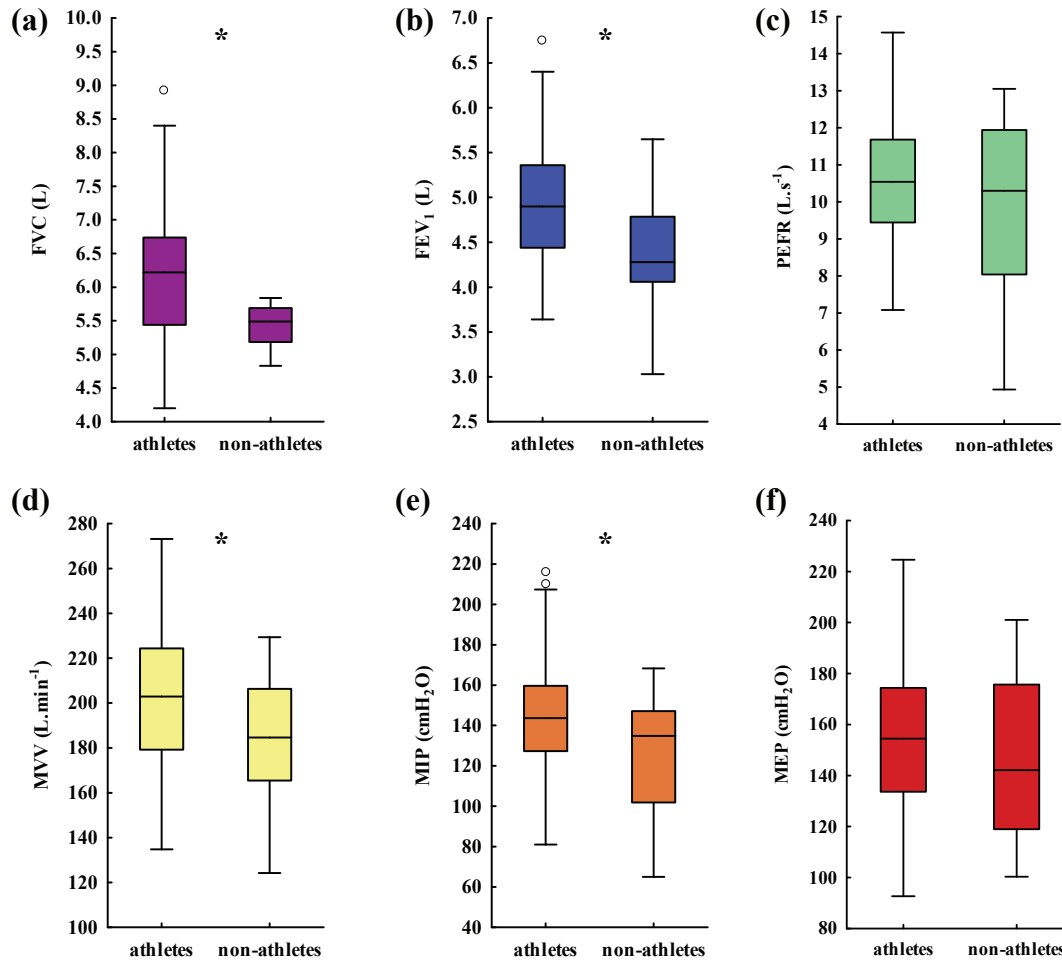


Figure 8: Comparisons of respiratory muscle function variables between **men** athletes and non-athletes. (a) FVC, (b) FEV₁, (c) PEFr, (d) MVV, (e) MIP, (f) MEP. * Significant difference between athletes and non-athletes. (° outliers)

Women athletes vs. women non-athletes:

Figure 9 illustrates the different respiratory muscle function variables between women athletes and non-athletes. Most of the outcome variables (FVC, FEV₁, PEFr and MVV) were similar between the two groups (Figure 9a-d). However, women athletes achieved a 26% higher MIP (110 ± 29 vs. 87 ± 18 , $P = 0.02$) and 24% higher MEP (125 ± 26 vs. 101 ± 29 , $P = 0.01$) compared to non-athletes.

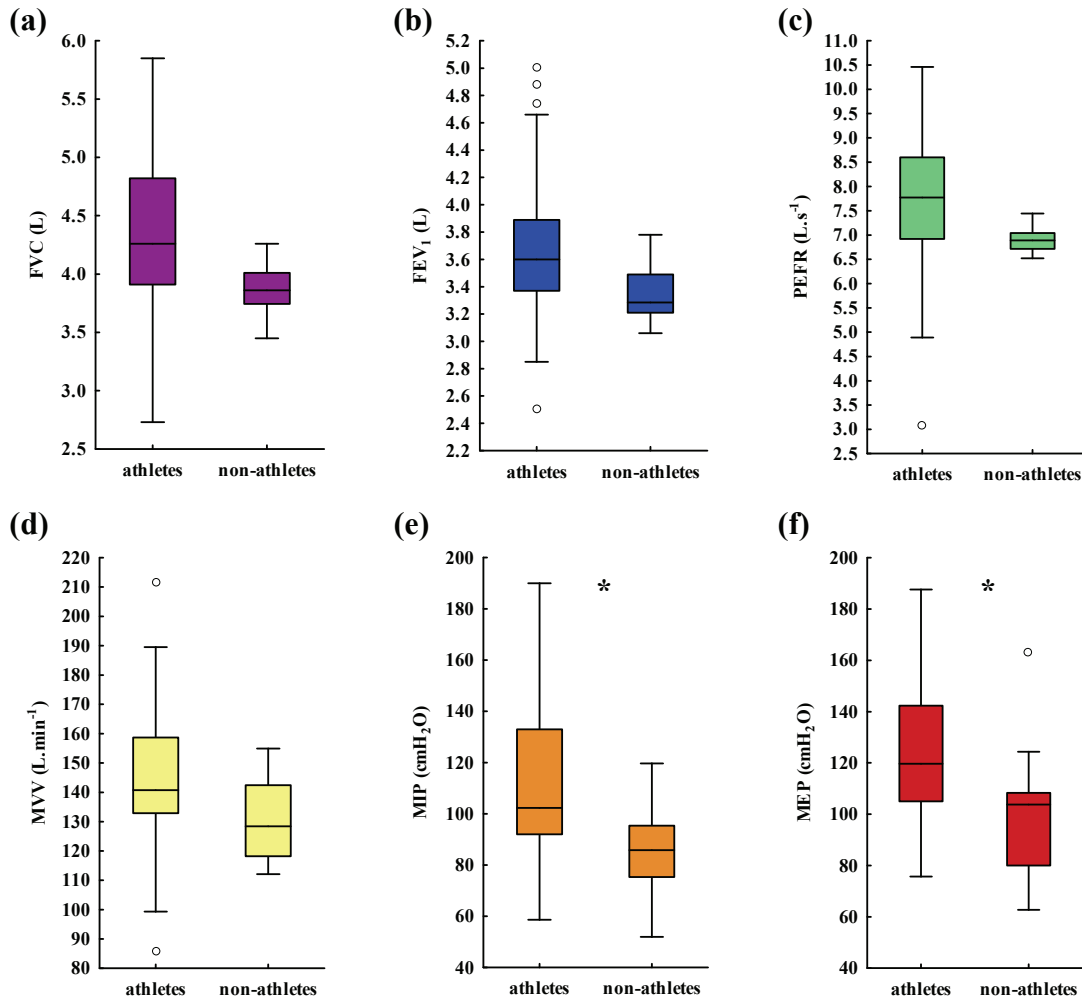


Figure 9: Comparisons of respiratory muscle function variables between **women** athletes and non-athletes. (a) FVC, (b) FEV₁, (c) PEFr, (d) MVV, (e) MIP, (f) MEP. * Significant difference between athletes and non-athletes. (° outliers)

3. Kinanthropometric and respiratory muscle function differences between men and women athletes

There was a significant difference in age ($P = 0.02$) between men and women athletes (Table 7); men athletes were slightly older (2 years) than women athletes. BMI (Table 7) was significantly higher in men athletes compared to women athletes ($P < 0.001$). Women athletes had significantly higher relative sitting height (sitting height/standing height $\times 100$) compared to men athletes ($P = 0.009$, Table 8), while relative arm span was significantly greater in men athletes compared to women athletes ($P = 0.002$, Table 8).

Table 9 depicts the mean \pm SD values for pulmonary function, RM strength and RM endurance outcome variables for men ($n = 103$) and women ($n = 57$) athletes. Men athletes achieved significantly higher FVC, FEV₁, PEFR, MVV, MIP and MEP values compared to women athletes ($P < 0.001$). However, when the pulmonary function variables (FVC, FEV₁, PEFR and MVV) were expressed as a percentage of predicted values (corrected for gender, age and height), only MVV (126 ± 17 vs. $119 \pm 16\%$ of MVV, $P = 0.006$) was significantly higher in men athletes compared to women athletes. Women athletes achieved on average 75% and 80% of the MIP and MEP values achieved by men athletes.

Table 9: Respiratory muscle function variables (mean \pm SD) of men athletes compared to women athletes.

<i>Variable:</i>	<i>Men athletes (n = 103)</i>	<i>Women athletes (n = 57)</i>
FVC (L)	$6.16 \pm 0.9 *$	4.32 ± 0.6
FEV₁ (L)	$4.96 \pm 0.7 *$	3.67 ± 0.5
PEFR (L.s⁻¹)	$10.6 \pm 1.7 *$	7.7 ± 1.3
MVV (L.min⁻¹)	$203.1 \pm 28.0 *$	145 ± 22.0
MIP (cmH₂O)	$147 \pm 26.7 *$	110 ± 29
MEP (cmH₂O)	$156 \pm 30 *$	125 ± 26

* significantly different from women athletes, $P < 0.001$.

4. Relationships between measured variables and RM strength and endurance

4.1 Kinanthropometric correlations with RM strength and endurance

All the kinanthropometric outcome variables were correlated with MIP, MEP and MVV, respectively, in order to determine relationships between kinanthropometry and respiratory muscle strength and endurance (Table 10). Significant relationships will reveal which of the kinanthropometric outcome variables may influence RM strength and endurance.

Correlations with MIP: Chest girth and transverse chest breadth revealed the highest correlations (both, $r = 0.56$), followed by weight ($r = 0.44$) and mesomorphic component ($r = 0.40$). Sitting height, arm span and height revealed statistically significant, but low correlations. The sum of 7 skinfolds showed a significantly negative relationship with MIP. Age did not correlate significantly with MIP.

Correlations with MEP: Anthropometric variables revealed significant correlations with MEP (Table 10). Similar to MIP, chest girth and transverse chest breadth revealed the highest correlations ($r = 0.43$ and $r = 0.41$, respectively). Height and weight revealed the second highest correlations, while mesomorphic component, sitting height and arm span revealed significant but low correlations with MEP. Sum of 7 skinfolds, which was an indication of body fatness, showed a significantly negative correlation with MEP ($r = -0.21$). Age did not correlate significantly with MEP.

Correlations with MVV: Height revealed the highest significant correlation with MVV (Table 10) in the total group of 160 subjects. The second highest correlation with MVV was arm span ($r = 0.68$). This was followed by chest girth, transverse chest breadth, weight and sitting height which also revealed significant relationships with MVV. There was a negative correlation between sum of 7 skinfolds and MVV, while age and the mesomorphic component revealed a statistically significant but low correlation with MVV.

Table 10: Pearson product moment correlations of kinanthropometric variables with MIP, MEP and MVV.

<i>Variable:</i>	<i>RM strength</i>		<i>RM endurance</i>
	<i>MIP</i>	<i>MEP</i>	<i>MVV</i>
Age	-	-	0.26
Height	0.33	0.37	0.72
Weight	0.44	0.39	0.57
Chest girth	0.56	0.43	0.64
Transverse chest breadth	0.56	0.41	0.63
Sitting height	0.36	0.28	0.56
Arm span	0.35	0.33	0.68
Sum of 7 skinfolds	-0.18	-0.21	-0.41
Mesomorphic component	0.40	0.31	0.25

All correlations, $P < 0.05$

4.2 Pulmonary function correlations with RM strength and endurance

There were high correlations between pulmonary function variables and MVV (Table 11). All three pulmonary function variables (FVC, FEV₁, PEF_R) revealed statistically significant

correlations with MIP ($r = 0.42 - 0.46$) and MEP ($r = 0.32 - 0.41$), respectively. MEP also showed a significant correlation with MIP ($r = 0.66$, Table 11).

Table 11: Pearson product moment correlations of respiratory muscle function variables with MIP, MEP and MVV.

<i>Variable:</i>	<i>RM strength</i>		<i>RM endurance</i>
	<i>MIP</i>	<i>MEP</i>	<i>MVV</i>
FVC	0.46	0.41	0.72
FEV₁	0.42	0.34	0.80
PEFR	0.42	0.32	0.80
MIP	-	0.66	0.44
MEP	-	-	0.37

All correlations significant, $P < 0.05$

5. Predictors of respiratory muscle strength and endurance

The main aim of the study was to determine the kinanthropometric and RM function predictors of RM strength and endurance. The following paragraphs describe those factors that influenced the variance in each of the outcome variables for RM strength and endurance.

5.1 Predictors of maximum inspiratory mouth pressure (MIP)

Best subsets multiple regression analysis revealed a significant but moderate multiple correlation coefficient of $R = 0.59$ and a standard error of estimate of 26.6 cmH₂O to predict MIP (Figure 10). Gender ($P < 0.001$) and mesomorphic component ($P = 0.003$) significantly predicted MIP, while exercise sessions per week also contributed to the prediction with borderline significance ($P = 0.051$). These three predictors (gender, mesomorphic component, exercise sessions per week) accounted for only 35% ($R^2_{\text{adjusted}} = 34\%$) of the variance in MIP (cmH₂O). No other outcome variables contributed to the prediction of MIP.

Table 12: Predictive linear models for **MIP**.

<i>Predictive variable:</i>	<i>Beta</i>	<i>SE of Beta</i>	<i>B</i>	<i>SE of B</i>	<i>t – value</i>	<i>P – value</i>
<i>Intercept</i>			111.6	9.97	11.9	0.0000
Gender *	-0.48	0.07	-32.3	4.72	-6.85	0.0000
Meso	0.21	0.07	5.4	1.77	3.05	0.003
EX	0.13	0.07	1.4	0.69	1.96	0.051

SE = standard error of Beta and B, * gender: male = 0, female = 1, meso = mesomorphic component, EX = exercise sessions per week. Multiple regression analysis: R = 0.59 (P < 0.001), R² = 0.35, Adjusted R² = 0.34, F = 28.15, SEE = 26.6 cmH₂O

The resulting regression equation to predict MIP from gender, mesomorphic component and exercise sessions per week (Table 12) was:

$$\mathbf{MIP (cmH_2O)} = 111.6 - (32.3 \times \text{gender}) + (5.4 \times \text{meso}) + (1.4 \times \text{EX}) \dots \dots \dots \text{eq. 7.1,}$$

where MIP is the maximum inspiratory mouth pressure in cmH₂O, gender is represented by a dummy variable – male = 0, female = 1, meso is the mesomorphic component (numeric value) calculated from anthropometric variables according the Heath & Carter (1967) method and EX is exercise sessions per week (number of exercise sessions per week).

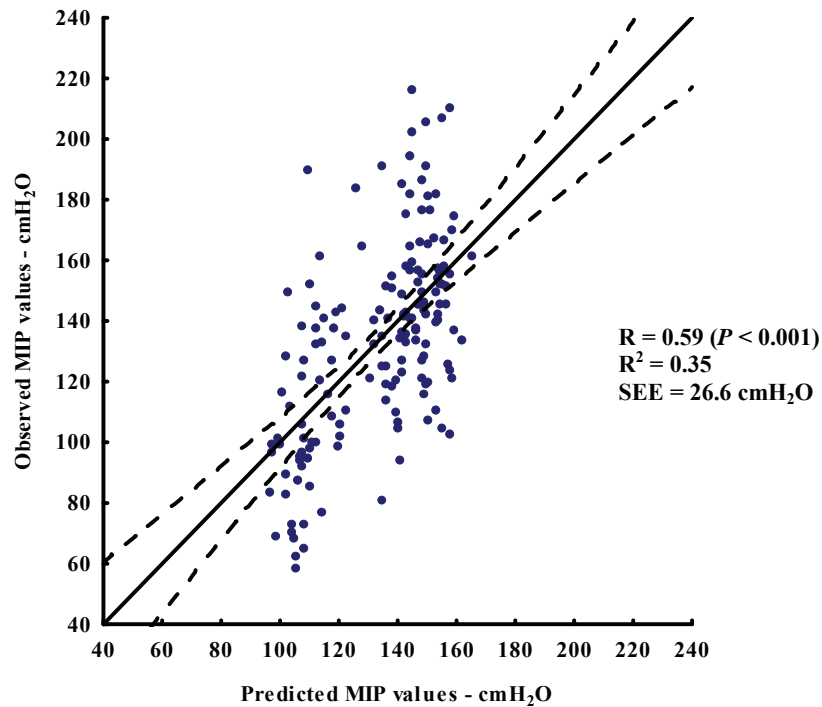


Figure 10: Scatter plot (*blue*) of the observed vs. the predicted **MIP** values (solid line: regression line, dotted line: 95% confidence intervals, R: multiple correlation coefficient, R^2 : coefficient of determination, SEE: standard error of estimate, P value: level of significance).

5.2 Predictors of maximum expiratory mouth pressure (MEP)

Best subsets multiple regression analysis revealed a significant but a fairly low multiple correlation coefficient of $R = 0.49$ and a standard error of estimate of $28.3 \text{ cmH}_2\text{O}$ to predict MEP (Figure 11). Gender was the only variable that significantly predicted MEP ($P < 0.001$), while mesomorphic component also contributed to the prediction with borderline significance ($P = 0.05$). These two predictors (gender, mesomorphic component) accounted for only 24% ($R^2_{\text{adjusted}} = 23\%$) of the variance in MEP (cmH_2O). No other outcome variables contributed to the prediction of MEP.

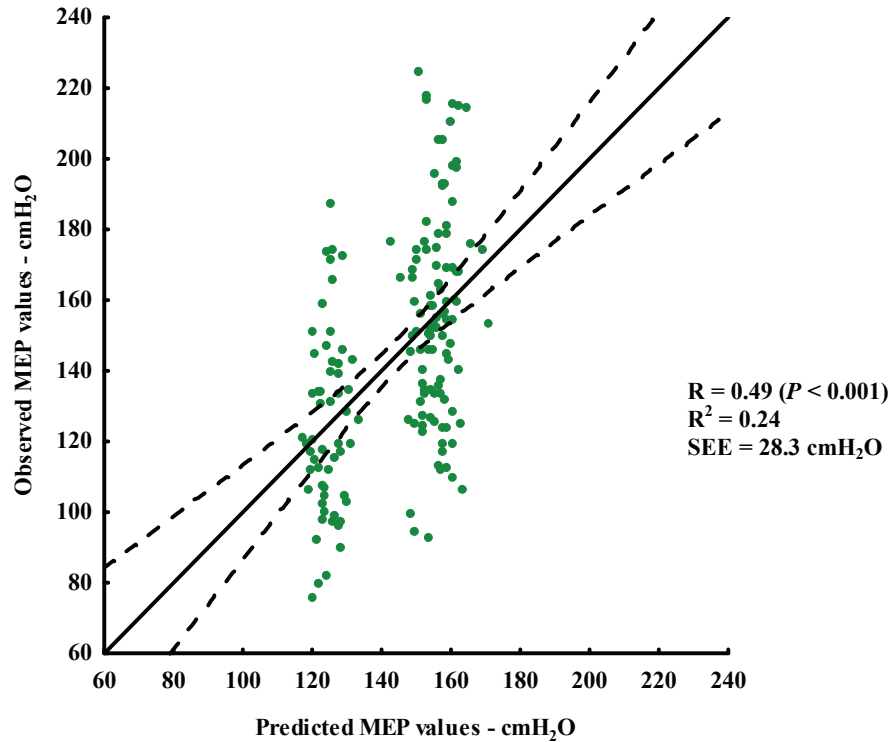


Figure 11: Scatter plot (*green*) of the observed vs. the predicted **MEP** values (solid line: regression line, dotted line: 95% confidence intervals, R: multiple correlation coefficient, R²: coefficient of determination, SEE: standard error of estimate, P value: level of significance).

Table 13: Predictive linear models for **MEP**.

<i>Predictive variable:</i>	<i>Beta</i>	<i>SE of Beta</i>	<i>B</i>	<i>SE of B</i>	<i>t – value</i>	<i>P – value</i>
<i>Intercept</i>			138.2	9.47	14.6	0.0000
Gender *	-0.41	0.07	-27.78	5.02	-5.53	0.0000
Meso	0.15	0.07	3.71	1.88	1.97	0.0506

SE = standard error of Beta and B, * gender: male = 0, female = 1, meso = mesomorphic component. Multiple regression analysis: R = 0.49 (P < 0.001), R² = 0.24, Adjusted R² = 0.23, F = 24.4, SEE = 28.3 cmH₂O

The resulting regression equation to predict MEP from gender and the mesomorphic component (Table 13) was:

$$\text{MEP (cmH}_2\text{O)} = 138.2 - (27.78 \times \text{gender}) + (3.71 \times \text{meso}) \dots \dots \dots \text{eq. 7.2,}$$

where MEP is the maximum expiratory mouth pressure in cmH₂O, gender is represented by a dummy variable – male = 0, female = 1 and meso is the mesomorphic component (numeric

value) calculated from anthropometric variables according to the Heath & Carter (1967) method.

5.3 Predictors of maximum voluntary ventilation (MVV)

Best subsets multiple regression analysis revealed a significantly high multiple correlation coefficient of $R = 0.88$ and a standard error of estimate of $18.2 \text{ L}\cdot\text{min}^{-1}$ to predict MVV (Figure 12). Gender ($P = 0.02$), relative sitting height ($P = 0.006$), FEV_1 ($P < 0.001$) and PEFR ($P < 0.001$) were the four variables which significantly predicted MVV ($\text{L}\cdot\text{min}^{-1}$). These four variables accounted for 78% ($R^2_{\text{adjusted}} = 77\%$) of the variance in MVV.

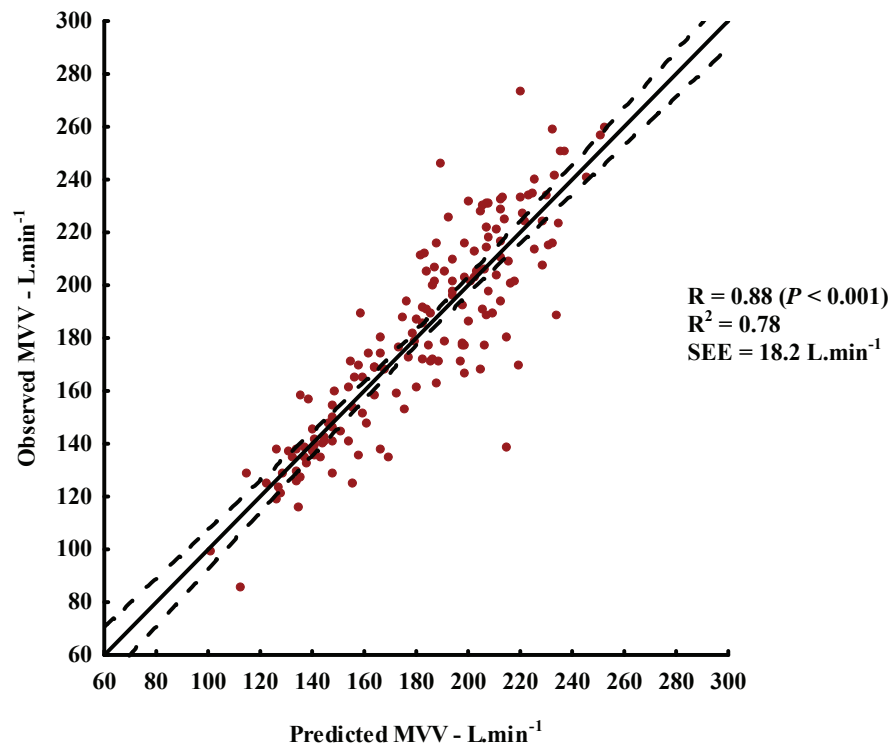


Figure 12: Scatter plot (red) of the observed vs. the predicted MVV values (solid line: regression line, dotted line: 95% confidence intervals, R: multiple correlation coefficient, R^2 : coefficient of determination, SEE: standard error of estimate, P value: level of significance).

FEV_1 (Table 14) contributed the most to the variance in MVV with a Beta factor of 0.45, followed by PEFR , gender and lastly, relative sitting height. No other outcome variables

from the anthropometric and respiratory muscle function assessments contributed significantly to the prediction of MVV.

Table 14: Predictive linear models for MVV.

<i>Predictive variable:</i>	<i>Beta</i>	<i>SE of Beta</i>	<i>B</i>	<i>SE of B</i>	<i>t - value</i>	<i>P - value</i>
<i>Intercept</i>			150.34	42.69	3.52	0.0005
Gender	-0.14	0.06	-10.84	4.73	-2.29	0.02
Relative sitting height	0.11	0.04	-2.26	0.80	-2.82	0.006
FEV₁	0.45	0.06	19.59	2.53	7.73	0.0000
PEFR	0.37	0.06	6.88	1.08	6.38	0.0000

SE = standard error of Beta and B, * gender: male = 0, female = 1, relative sitting height = (sitting height /standing height) × 100. Multiple regression analysis: R = 0.88 (P < 0.001), R² = 0.78, Adjusted R² = 0.77, F = 135.2, SEE = 18.2 L.min⁻¹

The resulting regression equation to predict MVV from gender, relative sitting height, FEV₁ and PEFR (Table 14) was:

$$\text{MVV (L.min}^{-1}\text{)} = 150.34 - (10.84 \times \text{gender}) - (2.26 \times \text{relative sitting height}) + (19.59 \times \text{FEV}_1) + (6.88 \times \text{PEFR}) \dots \dots \dots \text{eq. 7.3}$$

where MVV is the maximum voluntary ventilation in L.min⁻¹, gender is substituted by a dummy variable – male = 0, female = 1, relative sitting height is calculated as [sitting height (cm) divided by standing height (cm)] × 100, FEV₁ is the forced expiratory volume (L) in one second during the flow-volume curve test and PEFR is the peak expiratory flow rate in L.s⁻¹ during the flow-volume curve test.

6. Observed values compared to predicted values

The mean observed values of MIP and MEP in 103 men athletes and 57 women athletes were compared to the predicted values, derived from the prediction equations of several studies on normal populations. The comparisons were made to determine how accurately these predicted values based on normal, non-athletic populations compare to the observed values in the group of athletes in the present study. The predicted MIP and MEP, and MVV, for each individual in the present study were calculated from the prediction equations

reported in each of the studies in Table 15, Table 16 and Table 17, respectively. The mean value of these predicted values were reported next to the corresponding study from which the predicted values were derived, for men and women, respectively. The mean observed value (directly measured in the present study) for the group of athletes (men and women, respectively) is reported in the column next to the mean predictive values. The mean observed value (measured value) was subtracted from the mean predicted value (calculations from prediction equations) for MIP, MEP and MVV, respectively to indicate the difference between predicted and observed values. A difference (cmH₂O) of more than ± 10 values were considered as a true underestimation (-) or overestimation (+) of the observed value.

All studies involved normal, non-athletic populations, except for Cordian et al. (1987) who tested male competitive runners. The predicted MIP values for men from five different studies underestimated the observed values in the men athletes (n = 103) of the present study. Neder et al. (1999) and Cordian et al. (1987) showed similar values to the present study with a difference of less than 10 cmH₂O from the observed mean values (Table 15). Most of the predicted MIP values for normal, non-athletic women also underestimated the observed values in the group of women athletes (n = 57). The predicted values from the study of Wilson et al. (1984) and Neder et al. (1999) were similar to the observed values in the women athletes of the present study.

Table 15: Predicted mean values from MIP prediction equations compared to the observed mean MIP values of the present study.

<i>Authors:</i>	<i>MIP (cmH₂O) for men</i>			<i>MIP (cmH₂O) for women</i>		
	<i>Predicted:</i>	<i>Observed:</i>	<i>Predicted – Observed</i>	<i>Predicted:</i>	<i>Observed:</i>	<i>Predicted – Observed</i>
Hautmann et al. (2000) *	111	147	-36	82	110	-28
Harik-Khan et al. (1998) **	131	147	-16	85	110	-25
Neder et al. (1999) †	141	147	-6	100	110	-10
Brushi et al. (1992) ^Δ	132	147	-15	88	110	-22
Cordian et al. (1987) ^Ω	139	147	-8	-	110	-
Wilson et al. (1984) ^Φ	119	147	-28	119	110	+9
Black & Hyatt (1969) ^Ψ	131	147	-16	93	110	-17

Predicted (cmH₂O): is the mean predicted MIP value of the corresponding study, *Observed (cmH₂O)*: is the measured mean MIP value of the present study. *Predicted – Observed* is the mean predicted MIP value of the corresponding study – the mean observed MIP value of the present study. *Sample size, age group of each study*: * 248 men, 256 women, age 18-82. ** 50 men, 50 women, age 20-80. † 139 men, 128 women, age 20-90. ^Δ 266 men, 359 women, age 18-70. ^Ω 101 men, age 16-58, ^Φ 48 men, 87 women, age 19-65. ^Ψ 60 men, 60 women, age 20-86.

Two studies (Neder et al., 1999; Wilson et al., 1984) reported slightly lower and higher predicted mean values for MEP in men, compared to the observed values, however these predicted values did not differ much from the observed mean MEP value in the group of men athletes. The mean predicted value of Bruschi et al. (1992) underestimated the observed value in men athletes. On the other hand, the predicted value for MEP by Black & Hyatt (1969) largely overestimated the mean observed value in the group of men athletes (Table 16). On average, the mean MEP value obtained in men athletes was only 64% of the predicted value of Black & Hyatt (1969). The same was true for the women; the mean observed value obtained by the group of women athletes was only 79% of the predicted value derived from the prediction equation by Black & Hyatt (1969). However, all the other studies (Neder et al., 1999; Brushi et al., 1992; Wilson et al., 1984) underestimated the observed mean value in the group of women athletes.

Table 16: Predicted mean values from MEP prediction equations compared to the observed mean MEP values of the present study.

<i>Authors:</i>	<i>MEP (cmH₂O) for men</i>			<i>MEP (cmH₂O) for women</i>		
	<i>Predicted:</i>	<i>Observed:</i>	<i>Predicted – Observed</i>	<i>Predicted:</i>	<i>Observed:</i>	<i>Predicted – Observed</i>
Neder et al. (1999)[†]	147	156	-9	103	125	-22
Brushi et al. (1992)^Δ	145	156	-11	105	125	-20
Wilson et al. (1984)^Φ	160	156	+4	96	125	-29
Black & Hyatt (1969)^Ψ	245	156	+89	159	125	+34

Predicted (cmH₂O): is the mean predicted MEP value of the corresponding study, *Observed (cmH₂O)*: is the measured mean MEP value of the present study. *Predicted – Observed* is the mean predicted MEP value of the corresponding study – the mean observed MEP value of the present study. *Sample size, age group of each study*: [†] 139 men, 128 women, age 20-90. ^Δ 266 men, 359 women, age 18-70. ^Φ 48 men, 87 women, age 19-65. ^Ψ 60 men, 60 women, age 20-86. *Present study*: 103 men, 57 women, age 17-34.

The mean predicted value for MVV by Neder et al. (1999) was almost identical to the observed mean value in the group of men athletes (Table 17). The mean predicted value for women by Neder et al. (1999) underestimated the mean observed value in the group of women athletes by 14 cmH₂O.

Table 17: Predicted mean values from MVV prediction equations compared to the observed mean MVV values of the present study.

<i>Authors:</i>	<i>Mean predicted MVV:</i>		<i>Predicted MVV - Observed MVV (L.min⁻¹)</i>	
	<i>Men</i>	<i>Women</i>	<i>Men</i>	<i>Women</i>
Neder et al. (1999)[*]	204	131	+1	-14

Predicted MVV - Observed MVV (L.min⁻¹) is the predicted value of the corresponding study – the observed value of the present study (**MVV = 203 L.min⁻¹ in men and 145 L.min⁻¹ in women**). *Sample size, age group of each study*: ^{*} 139 men, 128 women, age 20-90. *Present study*: 103 men, 57 women, age 17-34.

In summary, six out of the twelve mean predicted values depicted in Tables 15, 16 and 17 underestimated the observed values in the group of men athletes, while five predicted values were similar to the mean observed value and one prediction largely overestimated the mean observed value. In the women athletes, eight out of the eleven mean predicted values for RM strength (MIP and MEP) and endurance (MVV) underestimated the mean observed value, and two revealed similar predicted values to the observed values, while one prediction largely overestimated the values obtained by the women athletes.

CHAPTER EIGHT

DISCUSSION: SECTION ONE

RESPIRATORY MUSCLE TRAINING AND DETRAINING

A. INTRODUCTION

The results of the RMTP were mostly compared to two studies (Griffiths & McConnell, 2007; Wells et al., 2005) that incorporated similar concurrent inspiratory and expiratory muscle training. To the researcher's knowledge, these two studies are the only studies available in the literature on concurrent RMT. The present study was similar to the study by Wells et al. (2005) in that (a) both included a sham training group as control, (b) used women athletes as a subject group, (c) used the *PowerLung*© as a training device, (d) implemented a 12 week concurrent respiratory muscle training programme, (e) increased training intensities once every few weeks throughout the intervention to ensure a dose response relationship, (f) both groups continued with regular training for their specific sport during the intervention and (g) used a sport specific field test to evaluate the effect of concurrent RMT on exercise performance. Where possible, the results of the women group in the study by Wells et al. (2005) were compared to the present study to avoid any discrepancies that might be caused by gender differences. The concurrent RMT in the study by Griffiths & McConnell (2007) lasted six weeks and was preceded by either a four-week IMT phase or an EMT phase. The protocol resulted in the comparison of two groups, namely IMT and EMT, but no sham training group as control was included in the study.

The present study was also compared to studies that used voluntary isocapnic hypernoea as RMT mode (Verges et al., 2007a; McMahon et al., 2002; Markov et al., 2001; Stuessi et al., 2001; Sonetti et al., 2001; Spengler et al., 1999; Boutellier et al., 1992; Boutellier & Piwko, 1992). Even though these two methods (VIH and concurrent pressure threshold loading) differ in mode, they both apply a training load to inspiratory and expiratory respiratory muscles, respectively, which makes these studies comparable. The RMTP results were also compared to studies that incorporated inspiratory muscle training (IMT) only (Johnson et al.,

2007; Downey et al., 2007; Williams et al., 2002; Inbar et al., 2002; Romer et al., 2002a, Romer et al., 2002b; Romer et al., 2002c; Volianitis et al., 2001b) and the TIRE (test for incremental respiratory endurance) technique (Mickleborough et al., 2008; Enright et al., 2006; Chatham et al., 1999) as RMT interventions, therefore differences in outcomes were expected.

The relevance of the RM detraining part of the study was to determine the time period over which an athlete may experience a significant or complete reversal in his/her improved RM function due to the cessation of RMT. This information will indicate how often RMT should be incorporated in the daily training regime of athletes to maintain the adaptations which were gained during a RMT programme.

B. MAIN FINDINGS

HT-RMT intervention: The purpose of this study was to determine the effect of the HT-RMT intervention (12-week hockey training plus additional RMT) on pulmonary function and respiratory muscle strength and endurance, and to determine whether the specific respiratory muscle training (RMT) will affect exercise performance (aerobic fitness) and RM function of competitive women hockey players.

The HT-RMT intervention elicited significant improvements in pulmonary function (FVC, FEV₁, PEFR, and MEF_{50%}), inspiratory and expiratory muscle strength, and MVV in the experimental (EXP) group. The HT-ST intervention also elicited significant improvements in the three pulmonary function variables (FVC, FEV₁, MEF_{50%}) and MVV in the control (CON) group. Improvements of 5 – 13% in the respiratory muscle (RM) function variables of the EXP group and improvements of 5 – 9% in the CON group were found after the intervention. There was no difference in the percentage changes in any of the RM function variables between the EXP and CON groups over time. From these results it is evident that the sham training device provided a strong enough stimulus to improve the respiratory muscle endurance of the CON group, but not respiratory muscle strength.

There was a significant improvement in aerobic fitness, measured as the number of shuttles ran during the 20 m multistage shuttle run test in the EXP (8%) and the CON (12%) groups after the 12-week HT-RMT and HT-ST interventions, respectively. There was no difference in the percentage changes in the 20 m multistage shuttle run test between the EXP and CON groups over time. It is proposed that the improvement in number of shuttles achieved in both groups can be attributed to the improvement in RM endurance elicited by the RMT and placebo training programme. This improvement in RM function variables reflected an improvement in their efficiency during exercise which delayed the onset of RM fatigue and a delay in triggering the RM metaboreflex.

RM detraining: From all the RM function variable changes, it is clear that the reduction in RM function occurs mostly at or before nine weeks of inactivity and that these reductions reach a plateau after nine weeks of inactivity. It also shows that reductions in strength do not return to pre-training values during a long period of inactivity. The latter two conclusions are confirmed by other studies (Romer & McConnell, 2003b; Lemmer et al., 2000; Hakkinen et al., 1981).

To the researcher's knowledge, this study is the first to demonstrate changes in RM function during a prolonged period of inactivity (four and a half months) after the completion of a concurrent RMT programme; and to demonstrate the differences in the changes in these variables between the RMT and placebo training groups during the RM detraining period. Except for MIP and RM endurance, the study is also the first study to reveal the reductions in other RM function variables which emphasize the importance that regular RMT should be incorporated in the athlete's training regime to maintain improved RM function and subsequently improve athletic performance. From the present study, it is evident that after nine weeks of RM detraining there will be significant losses in the improvements elicited by the preceding RMT programme. Therefore, it is advised that RMT should not be terminated for nine weeks or more at a time, since this will lead to a loss in RM functionality.

C. SUBJECT CHARACTERISTICS

All the physical and respiratory muscle function characteristics, except for MIP, were similar between the EXP and CON groups at the start of the study. The high mean value of MIP in the CON group was influenced by one very high measurement (185 cmH₂O), while the other MIP measurements in the CON group ranged from 120 to 158 cmH₂O. The significantly lower mean value of MIP in the EXP group was influenced by two very low measurements (< 80 cmH₂O), while the other MIP measurements in the EXP group ranged from 93 to 155 cmH₂O.

Very few published data on the physical and fitness characteristics of women hockey players are available in order to compare the subject group with other national and provincial players around the world. However, the Australian Sports Commission (2000) did report some limited data on elite Australian women players. The mean \pm SD values for height (166 \pm 7 and 168 \pm 7 cm) and weight (61 \pm 9 and 63 \pm 9 kg) in the EXP and CON groups, respectively, were very similar to the height (167 \pm 5 cm) and weight (61 \pm 6 kg) of the senior elite Australian women players (n = 385). The participants in this study reached levels ranging from level 8 to level 13 during the 20 m multistage shuttle run test. The Australian group's results were slightly higher, ranging from level 9 to level 13, which indicated that the Australian players had a higher aerobic endurance capacity compared to the present group. The range of predicted VO_{2max} values derived from the 20 m multistage shuttle run test was 40 to 59 ml.kg⁻¹.min⁻¹ in the present group, while the direct measurement of VO_{2max} in Australian players ranged from 39 to 64 ml.kg⁻¹.min⁻¹ (Lawrence & Polglaze, 2000). Thus, physically the players are of same height and weight, but the Australians have better endurance capacities.

D. RESPIRATORY MUSCLE TRAINING ADHERENCE

The subjects maintained a high percentage of compliance to the prescribed RMTP over the twelve week intervention. The EXP group reported a 92% adherence to the RMTP and the CON group a 96% adherence to placebo training over the twelve week intervention period.

The high percentage of adherence to the RMT programme agreed with the majority of studies (92 – 97% adherence) (Downey et al., 2007; Johnson et al., 2007; Wells et al., 2005; Romer et al., 2002a; Romer et al., 2002c; Volianitis et al., 2001b).

Romer et al. (2002b) and Griffiths & McConnell (2007) reported lower RMT compliance. Romer et al. (2002b) reported that their IMT group and control group completed 82% and 88% of the prescribed RMT sessions. However, the lower percentage of completed RMT sessions still caused a significant improvement in RM strength and sprint recovery performance. Griffiths & McConnell (2007) reported that during the IMT or EMT phase only, the IMT group completed 76% of its prescribed RMT sessions and the EMT group completed 78% of its prescribed RMT sessions. During the concurrent training phase the IMT group maintained their adherence of 75% of prescribed training sessions, however the EMT group's adherence declined to 60% during the combined IMT/EMT phase. The IMT group showed a significant improvement in rowing time trial performance after the IMT phase, showing that RMT compliance of 76% is an adequate stimulus to cause changes in whole-body exercise performance. However, the EMT group showed no significant improvement in time trial rowing performance after neither the EMT training nor the concurrent training phase. This suggests that the completion of 60% or less of the prescribed training sessions could have been too little to cause the necessary adaptations for an improvement in performance. The lower adherence of the combined training phase in the EMT group was attributed to two subjects contracting upper respiratory infections during this phase.

E. EFFECTS ON PULMONARY FUNCTION

1. Effects on lung volumes

There were no significant differences in baseline FVC (4.07 ± 0.6 vs. 4.26 ± 0.5 L) and FEV₁ (3.40 ± 0.4 vs. 3.62 ± 0.3 L) between the EXP and CON groups. After 6 weeks of specific RMT there was a significant 4% increase in FVC in EXP group. There was only a slight further increase during the second half of the 12-week intervention to an overall

increase of 5% in FVC. The CON group also showed a significant increase (5%) in FVC after 12 weeks of placebo training. There were no significant differences between the two groups over time. The changes in FVC agreed with the findings by Wells et al. (2005) who found a 21% and 18% increase in the experimental and sham training group after a specific RMT intervention, respectively. Although they found a larger percentage of improvement in FVC compared to the present study, the findings were very similar since they also found significant improvements in FVC over twelve weeks of concurrent RMT, and also found the improvement in both the experimental and sham training groups. A possible explanation for these results are the fact that both training regimes, concurrent RMT and placebo training, elicited an adequate stimulus to cause an increase in the velocity or shortening capacity of the expiratory muscles. A greater rate of shortening of these muscles caused a greater displacement of the lung and chest wall cavity resulting in a greater volume of air that was expired during a forceful effort (Green et al., 2002). The present study also agrees with Mickleborough et al. (2008) who found significant improvements in FVC (12%) in all three groups participating in their study. One group underwent swim training and IMT by means of the TIRE technique, another group performed swim training and sham training, and the third group only performed the swim training programme. The changes in FVC among these three groups were not statistically significantly different.

Sonetti et al. (2001) reported a significant 4% increase in FVC after a five week combined RMT (voluntary isocapnic hypernoea and IMT), and no significant changes in FVC of the sham training group. An explanation for this finding is that placebo training did not elicit training adaptations to the RM and therefore did not result in significant changes in lung volume. Vice versa, it can also be stated that the experimental group underwent a combination of VIH and inspiratory threshold loading training compared to the sham training group which only followed a sham inspiratory threshold loading protocol and no VIH, therefore the much higher training load of the experimental group may have caused the significant difference in the changes in FVC between the two groups.

Enright et al. (2006) and McMahon et al. (2002) reported a significant 7% and 3% increase in vital capacity (VC), and no change in their respective control groups, who did no training

during the respective RMT interventions. Verges et al. (2008a) found a modest but significant increase in FVC of 1.5% compared to a non-significant change in the control group. In contrast, Griffiths & McConnell (2007) found a significant decrease in FVC after their concurrent (IMT and EMT) training phase (six weeks) in the IMT group. The EMT group did not experience a decrease in FVC after either the EMT phase or the combined RMT phase. The reasons for this particular finding are unclear. A limitation to the study is that they did not include a control or placebo group.

After 6 weeks of specific RMT there was a significant 4% increase in FEV₁ in the EXP group in the present study. There was only a slight further increase during the second half of the 12-week RMTP to an overall increase of 6% in FEV₁. In the CON group, FEV₁ also showed a significant increase (6%) after six weeks of placebo training, but remained the same until the end of the 12-week intervention. Very few studies reported a change in FEV₁. Wells et al. (2005) reported a mean 31% and 19% increase in FEV₁ in the women experimental and sham training groups, respectively. Mickleborough et al. (2008) also reported similar changes when the three groups in their study were combined - the increase in FEV₁ was 13%.

The present study and the study by Mickleborough et al. (2008) and Wells et al. (2005) found similar improvements in FVC and FEV₁. It is expected that if FVC increases, there will be an increase in FEV₁, since airway resistance decreases with increasing lung volumes. An increase in FVC means that FEV₁ starts at a higher lung volume and will result in a higher FEV₁. One can also expect an increase in the elastic recoil of the lung at higher volumes which may further contribute to an increase in FEV₁ (Wells et al., 2005). In contrast to this statement, Sonetti et al. (2001) and Verges et al. (2008a) found a significant increase in FVC, but no significant improvement in FEV₁. A possible explanation for this could be that they only found an improvement of 3% and 1.5% in FVC, respectively, which is only 50% of the improvement in the present study and eight to twenty times less than the 19-31% in the study by Wells et al. (2005). It could be that a 3% change in FVC was too small to elicit an adequate higher lung volume to significantly improve FEV₁.

2. Effects on air flow variables

There were no significant differences in baseline PEFR (7.66 ± 1.1 vs. 7.96 ± 1.1 L.s⁻¹) and MEF_{50%} (3.95 ± 0.7 vs. 4.38 ± 0.9 L.s⁻¹) between the EXP and CON groups. After 6 weeks of specific RMT there was a significant 4% increase in PEFR in the EXP group followed by a slight further increase during the second half of the RMT period to an overall 5% increase at the end of the intervention. The CON group also showed a 4% increase in PEFR at 6 weeks and a 5% increase at the end of the intervention, however, these improvements were not significant. There were no significant differences in the changes in PEFR between the EXP and CON groups. Verges et al. (2008a) found similar changes in PEFR compared to the present study. They reported a significant change in PEFR of 4% compared to the non-significant change in PEFR in the control group of 1.1%. No other study reported a significant change in PEFR. However, some of the studies that investigated IMT did report a significant improvement in peak inspiratory flow rate (PIFR). Griffiths & McConnell (2007) found a significant increase in PIFR after the IMT phase in the IMT group and Romer et al. (2002c) found a 17% increase in maximum inspiratory flow rate after six weeks of IMT. Romer et al. (2002b) found a significant 20% increase in PIFR after six weeks of IMT.

After 6 weeks of HT-ST, there was a significant change (9%) in MEF_{50%} in the CON group and this remained the same until the end of the 12-week intervention. The EXP group only showed a significant improvement (6%) in MEF_{50%} after 12 weeks of HT-RMT. No other study reported a change in MEF_{50%} after specific RMT, however, the substantial increase in especially the CON group, made it feasible to report the change in this particular air flow variable. There was a substantial greater increase in MEF_{50%} after placebo training compared to RMT of the experimental group, although the changes between groups were not significantly different.

The significant changes in air flow variables (PEFR and MEF_{50%}) indicate that the concurrent RMT contribute to the velocity or shortening (flow) axis of the force-velocity relationship of the RM (McConnell & Romer, 2004b). This means that the generation of

higher air flow to overcome the resistance in order to breathe, was adequate enough in the EXP and CON group to elicit a strong enough stimulus for the improvement of the rate of shortening of the expiratory muscles. One can speculate that it is specifically the training of the expiratory muscles which indirectly caused an increase in PEFR and MEF_{50%}, since your expiratory muscles become active and need to contract forcefully during forced expiration.

One can speculate that the greater increase in MEF_{50%} in the CON group compared to the EXP group can be attributed to the low pressure threshold load at which placebo training took place. A lesser resistance to overcome to breathe through the sham training device gave subjects the opportunity to breathe deeper during their 3 seconds inspiration and 3 seconds expiration. Thus, continuous breathing at deeper levels and more efficient breathing at a lower pressure threshold (placebo) increased air flow at this specific lung volume.

F. EFFECTS ON RESPIRATORY MUSCLE STRENGTH

1. Effects on inspiratory muscle strength

There was no significant increase in MIP after 6 weeks of HT-RMT or HT-ST. However, after 12 weeks of training there was a significant 13% increase in MIP in the EXP group and no significant increase in the CON group. However, the changes in MIP did not significantly differ between the EXP and CON groups after 12 weeks of intervention. Four studies showed more or less the same increase in MIP after specific RMT.

The results of the present study mostly agree with Wells et al. (2005) who found a 14% improvement in MIP after a 12 week concurrent RMT programme in their experimental group. They also found a significant increase in the sham training group, and similar to the present study, no significant differences in the changes in MIP between the experimental and control group. Edwards et al. (2008) also found no significant difference between the experimental and control group improvements of 15% and 8%, respectively. Griffiths & McConnell (2007) found the same increase in MIP than the present study (13%) after six weeks of concurrent RMT in the EMT group who underwent EMT for four weeks prior to

the start of concurrent RMT. Johnson et al. (2007) found a 17% increase in MIP, very similar to the present study, although the subjects followed a six week IMT programme. Sonetti et al. (2001) who also incorporated a combined programme of IMT and VIH only showed a 7% increase in MIP. The increase in MIP in the experimental groups in the latter two studies was significantly greater than the change in the respective sham training groups.

The increases in MIP in the present study are less than those reported by the majority of RMT studies, especially those studies that incorporated an IMT programme, who found between 20 and 40% increases in MIP. Romer et al. (2002b) and Williams et al. (2002) found a 31% increase in MIP, while Chatham et al. (1999) found the same amount of increase after eight weeks of RMT training using the TIRE technique. Romer et al. (2002c), Inbar et al. (2000), and Downey et al. (2007) found 28, 25 and 24% increases in MIP after six, ten and four weeks of IMT, respectively. Griffiths & McConnell (2007) also found a similar increase in MIP (26%) compared to the latter three studies, after four weeks of IMT.

Greater than 40% increases in MIP, due to specific IMT have also been reported which is more than three times the increases found in the present study. Volianitis et al. (2001b) found a 44% increase in MIP after 11 weeks of IMT in women competitive rowers, however most of the changes in MIP occurred in the first four weeks of IMT, since MIP increased from 104 ± 8 to 144 ± 10 cmH₂O after four weeks, and a further 4 cmH₂O to 148 ± 10 cmH₂O after completion of the eleven week IMT programme. Enright et al. (2006) found a 41% increase (90 ± 16 to 127 ± 10 cmH₂O) in MIP after 8 weeks of RMT using the TIRE technique. Although Mickleborough et al. (2008) found significant improvements in MIP in the sham and control groups, they also found a 48% increase in MIP after a combined twelve week swimming and RMT intervention. This suggests that intensive swim training itself must have created a strong training load for an adaptation in the force-generating capacity of the RM.

The huge differences in the percentage increases in MIP between the present study and those studies showing a 24 to 48% increase in MIP can mostly be attributed to the differences in the RMT mode and the initial baseline values of MIP. It seems that studies using combined

RMT programmes, such as concurrent IMT and EMT training (present study; Griffiths & McConnell, 2007; Wells et al., 2005) and combined VIH and IMT (Sonetti et al., 2001), revealed a smaller increase in MIP (7 – 14%) compared to studies using only IMT or the TIRE technique as training mode (Mickleborough et al., 2008; Downey et al., 2007; Romer et al., 2002b; Romer et al., 2002c; Williams et al., 2002; Inbar et al., 2000; Chatham et al., 1999).

Differences in initial baseline values of the present study (115 ± 24 in EXP and 144 ± 22 cmH₂O in the CON group), firstly explains why there was a significant change in the EXP group but no significant change in the CON group, and no significant differences in the changes in MIP between the two groups. Baseline MIP in the CON group was 25% higher compared to the EXP group ($P < 0.05$). The difference is attributed to outliers with exceptionally high MIP values in the CON group and exceptionally low values in the EXP group.

Astrand (2003) stated that the degree of physiological adaptation of a system depends on its baseline status. If baseline status is already in a superior condition, it leaves a small window for adaptation to an even better condition. Thus, the CON group showed only a small change compared to the 13% increase in the lower baseline values of the EXP group. Thus, the 44% increase in MIP in a group of women rowers (Volianitis et al., 2001b) compared to the 13% increase in MIP in women hockey players of the present study can be attributed to the lower baseline values of the women rowers compared to the baseline values of the women hockey players (104 vs. 115 cmH₂O). In fact, the two studies that showed increases in MIP over 40% reported some of the lowest mean baseline values (Enright et al., 2006: BL = 90 ± 16 cmH₂O and Volianitis et al., 2001b: BL = 104 ± 8 cmH₂O).

Johnson et al. (2007) also found only a 17% increase in MIP compared to higher increases in MIP of similar IMT studies. This phenomenon can also be attributed to the fact that the subjects in the study by Johnson et al. (2007) achieved baseline values of 150 ± 29 to 153 ± 32 cmH₂O in men, which can be considered high MIP values. The other studies with high

percentage increases in MIP (28 – 31%) revealed mean baseline values of 102 to 130 cmH₂O in men.

Other reasons that could explain the differences in the percentage changes in MIP, are the duration, frequency and intensity of the RMT programme. Most of the studies which showed large increases in MIP, used a training frequency of 30 breaths twice daily (Griffiths & McConnell, 2007; Romer et al., 2002b; Romer et al., 2002c; Volianitis et al., 2001b), whereas the present study used a training frequency of 2 sets of 15 breaths each. Therefore, modality of the RMT programme in the EXP and CON groups of the present study can be recognized as a typical strength training programme for the inspiratory and expiratory muscles, simultaneously. The fact that the resistance of the placebo was only 15% of that of the experimental device, one would expect the CON group to show more endurance type adaptations compared to the EXP group, since they breathed more freely and deeper during each effort.

Most studies that found large increases in MIP, found these changes within four to six weeks of RMT, where the present study only found a significant increase after 12 weeks. There may be two possible explanations for this: firstly, concurrent IMT and EMT took longer (12 weeks) to elicit significant changes in MIP, which is also suggested by Wells et al. (2005). Secondly, the intensity of RMT was increased on a more frequent basis in other studies compared to the present study. The majority of studies increased the resistances on the devices once a week, or even more frequently to maintain a 30 repetition maximum breaths at a time (Griffiths & McConnell, 2007; Downey et al., 2007; Romer et al., 2002b; Romer et al., 2002c; Williams et al., 2002; Inbar et al., 2000). In the present study subjects were instructed to increase the resistance on the training device once every two weeks.

2. Effects on expiratory muscle strength

There were no significant differences in baseline values of MEP (130 ± 27 vs. 138 ± 31 cmH₂O) between the EXP and CON group. MEP showed a significant 9% increase after 6 weeks of HT-RMT, but did not increase any further during the second half of the 12-week

intervention. MEP in the CON group remained at baseline values after 6 weeks of HT-ST and, although there was a slight improvement (4%), it showed no significant improvement at the end of the intervention. However, these changes in MEP were not significantly different between the EXP and CON groups at either 6 weeks or at the end of the intervention. The 9% improvement after six weeks observed in the study differed from the findings of other studies, who found more substantial increases in MEP during either EMT or concurrent IMT and EMT (Griffiths & McConnell, 2007; Wells et al., 2005; Romer et al., 2002b; Suzuki et al., 1995).

Griffiths & McConnell (2007) found no significant increases in MEP in the IMT group after four weeks of IMT. During the concurrent training phase, MEP did improve by 23%, however the increase was not statistically significant ($P = 0.06$). The EMT group showed 18% increase in MEP after the EMT phase, but the change was not significant. After the concurrent training phase MEP increased further to a significant 31%. In agreement with Griffiths & McConnell (2007), Romer et al. (2002b) also measured MEP before and after a six week IMT programme, and found no significant increases in baseline MEP values. Wells et al. (2005) found a significant improvement of 12% and 15% in the experimental and sham training group, respectively. The percentage increases between the two groups were not significantly different. Suzuki et al. (1995) found a significant 25% increase in MEP after four weeks of EMT in healthy subjects. Mickleborough et al. (2008) found a 40% improvement in MEP in the joined group which consisted of swim, IMT and sham training sub-groups after 12 weeks of intense, either swim training, or swim and IMT training, or swim and sham training.

Although other studies showed greater increases in MEP, the present study mostly agrees with the changes in MEP (9% vs. 12%) by Wells et al. (2005). This can be attributed to the almost identical type, frequency and duration of the RMT programme. These two studies also revealed similar baseline values for MEP (130 ± 27 to 138 ± 34 cmH₂O). However, one reason for the slightly better result in MEP in the study by Wells et al. (2005), is that they increased the resistance of the training device by 10% every 3 weeks and completed 30 consecutive breaths per set. Subjects in the present study were instructed to increase the

resistance every fortnight by just enough to manage 15 breaths per set, repeated twice. Griffiths & McConnell (2007) used the same training frequency as Wells et al. (2005); however, they found a larger increase in MEP after the concurrent training regime compared to the present study and Wells et al. (2005). The main reason for the larger increase can be attributed to the fact that the concurrent RMT phase was preceded by four weeks of EMT only. The EMT programme only elicited greater changes in MEP compared to concurrent IMT and EMT (Griffiths & McConnell, 2007; Suzuki et al., 1995). The only exception is Mickleborough et al. (2008) who found an improvement in MEP after flow-resistive loading training (TIRE technique).

The current study revealed a significant increase in MIP of 13% and a significant increase in MEP of 9%; however these changes seemed to be modest since they did not differ significantly from the non-significant changes in the sham training group. Wells et al. (2005) also revealed a 14% and 12% increase in MIP and MEP, respectively, following concurrent RMT, also not significantly different from the changes in their sham training group. It seems that, as previously mentioned, a combined training programme do not elicit substantial to large increases in MIP and MEP, as IMT (24 – 44%) or EMT (18 – 25%) programmes separately. This statement is confirmed by Griffiths & McConnell (2007) who showed that IMT and EMT separately caused a 26% and 18% change in MIP and MEP, respectively. However, a combined training phase thereafter could only elicit a further 4% and 13% increase in MIP and MEP, respectively. Therefore, it can be concluded that a combination of inspiratory and expiratory muscle training resulted in sub-optimal adaptations to the inspiratory and expiratory muscles in response to both resistances simultaneously (McConnell & Romer, 2004b).

The finding that concurrent RMT elicit smaller percentage improvements in RM strength parameters compared to RM strength training alone, is also evident in concurrent training regimes of locomotor muscles. The majority of studies found that concurrent strength and endurance training elicited smaller increases in limb muscle strength compared to strength training alone (Bell et al., 2000; Leveritt et al., 1999; Kraemer et al., 1995; Hennessy & Watson, 1994; Dudley & Fleck, 1987).

For example, Bell et al. (2000) compared the training effects of strength training, endurance training and concurrent training programmes on muscle strength and whole-body endurance in a group of female university students. The strength training group increased their one repetition knee extension strength by 58% compared to the 41% increase in the concurrent training group. After six weeks of training the strength training groups experienced a significant 15% and 17% increase in type I and type II muscle fiber area compared to no changes in the concurrent training group. After 12 weeks of training the strength training group showed a further 28% (type I) and 27% (type II) increase in muscle fiber area compared to a significant 14% increase in type II muscle fiber area only in the concurrent training group. However, they found a significantly greater increase in VO_{2max} in the endurance (12%) and concurrent training groups (11%) compared to the strength training group (-6%) after the respective training programmes.

The study concluded that concurrent training do not necessarily interfere with the improvements in central circulatory adaptations to improve whole-body endurance, since both endurance training and concurrent training elicited similar changes in VO_{2max} . However, optimal strength gains are reduced during concurrent training compared to a strength training programme alone. Thus, the finding of this study and the study by Sonetti et al. (2001) completely agrees that concurrent strength and endurance training elicit smaller improvements in strength compared to strength training alone. The same argument can be applied to inspiratory and expiratory muscle training. Although, it is not a combination of strength and endurance training, but the combination of two different muscle groups strength training regimes, it would most likely also cause a reduction in the improvements in muscle adaptations compared to inspiratory or expiratory training, separately.

A more subjective explanation for the modest changes in respiratory muscle strength indices after concurrent IMT and EMT is the fact that subjects in the present study, and the study by Griffiths & McConnell (2007), found this type of training challenging. Almost all subjects in the experimental group experienced difficulty with the breathing effort against a resistance during inspiration and especially expiration during one respiratory cycle, therefore the

intensity of training that could be tolerated was less compared to the intensity that could have been tolerated if only inspiration or expiration were loaded.

It is also clear from the above mentioned results, that specific IMT causes greater adaptations to the inspiratory muscles compared to that of EMT to the expiratory muscles, since increases in MIP due to IMT always seem to be greater than increases in MEP due to EMT.

G. EFFECTS ON RESPIRATORY MUSCLE ENDURANCE

There were no significant changes in MVV after 6 weeks of HT-RMT in the EXP group and MVV remained at baseline values for the CON group after 6 weeks of HT-ST. However, at the end of the intervention there was a significant 8% and 6% increase in the EXP and CON groups, respectively. The improvement in MVV after the 12-week intervention was similar for the EXP and the CON group. The significant changes in both groups mean that the concurrent RMT and placebo training caused the same adaptations to the inspiratory and expiratory muscles to improve their endurance capacity.

The improvement of 8% in MVV agrees with the improvement in MVV after concurrent training in both the experimental and sham training group of Wells et al. (2005). Wells et al. (2005) also found no significant differences in the changes in MVV between the experimental and control groups. The only difference between the present study and Wells et al. (2005) is that they found a higher percentage of change (20 and 21%) in MVV compared to the present study. VIH training also elicited significant changes of 11 – 14% in MVV (McMahon et al., 2002; Morgan et al., 1987), however, the control groups, who refrained from sham training, showed no significant increase in MVV. A number of studies found no significant increases in MVV after specific RMT (IMT – Romer et al., 2002b; Inbar et al., 2000; VIH and IMT – Sonetti et al., 2001; VIH – Boutellier & Piwko, 1992).

The argument exist that MVV is not the best method to assess respiratory muscle endurance. One can argue that a test of 12 to 15 seconds is not long enough to test endurance, since it

does not measure the time to sustain a required amount of work or force until there is a definite decrease in the sustainable work (Clanton et al., 2002). This measurement also does not mimic the level of hyperventilation needed during exercise, since exercise time will continue longer than 12 to 15 seconds. Therefore, maximum sustained ventilation (MSVC) at a percentage of MVV can be used to measure time to maintain a required level of ventilation, which can be seen as a true test of endurance. A number of studies which incorporated VIH as RMT mode (which is known to elicit significant changes in RM endurance rather than RM strength) found a 250 to 770% increase in MSVC (at 60 – 74% MVV) after following a VIH programme (Verges et al., 2008a; Verges et al., 2007a; McMahon et al., 2002; Markov et al., 2001; Stuessi et al., 2001; Spengler et al., 1999; Boutellier & Piwko, 1992). Morgan et al. (1987) found an even greater percentage of change in MSVC (at 100% MVV) of 17 times more than baseline values. Boutellier et al. (1992) and Fairbairn et al. (1991) showed a more modest but significant change of 38% and 12% in MSVC. Studies that investigated the effect of IMT on breathing endurance showed no significant changes in the time that subjects could tolerate breathing at 90% MVV (Downey et al., 2007; Sonetti et al., 2001). However, Williams et al. (2002) conducted a breathing endurance test (BET) at 60% of MVV, and found an improvement of 128%.

The huge range of increases in the time that a particular level of ventilation could be sustained (BET), indicate that the BET has large inter-test variability. The majority of VIH studies found that these huge increases in BET are due to the poor reliability of this type of test (Sonetti et al., 2001). Even though breathing endurance tests differ from whole-body endurance tests, the principles are the same. Jeukendrup et al. (1996) found that time to exhaustion tests which are open-end tests have low reproducibility and do not mimic the endurance demands placed upon the muscle during real life situations. Thus, changes of 250% to 770% in breathing endurance and even a change of 17 times greater than baseline values seem highly unlikely to be a true training effect. However, a learning effect and a change in effort perception during the second test seem to be the reasons for these very large changes in breathing endurance time. Therefore, due to the high risk of a learning effect and open-end nature of a BET, this type of test for RM endurance was not included in the study.

The ERS/ATS recommends an incremental threshold loading test as a method to assess inspiratory muscle endurance (Clanton et al., 2002). Inbar et al. (2000) incorporated an incremental threshold loading test where subjects had to inspire through a two-way Hans-Rudolph valve. The inspiratory port of the valve was connected to a chamber with a plunger to which weights could be added in order to increase the resistance to overcome during inhalation. This method was first introduced by Nickerson & Keens (1982). Subjects had to complete two minute intervals of progressive resistance (adding 25 – 100 g weights with each increment) until subjects could no longer inhale. The pressure achieved at the highest external load and tolerated for at least 60 seconds was defined as the final score. A ten week IMT programme elicited a significant 27% increase (from 122 ± 14 to 154 ± 22 cmH₂O, $P < 0.005$) in RM endurance. This type of RM endurance test revealed better outcomes than the 12 second-MVV test (8% improvement) of the present study. Two limitations of this recommended method for testing RM endurance, is that the incremental threshold loading test is also an open-end test, where the subject's perception of the end of the test is still unknown. The test also only measures inspiratory muscle endurance and not inspiratory and expiratory muscle endurance simultaneously.

In summary, concurrent inspiratory and expiratory muscle training elicited significant improvements in lung volumes, air flow and RM endurance in both the EXP and CON groups. It also elicited modest improvements in RM strength in the EXP group, since these improvements did not significantly differ from the non-significant change in the CON group. The main explanation for this finding can be attributed to a type II error. The small sample size (EXP: $n = 15$; CON: $n = 7$) and large inter-individual variability in MIP and MEP in both groups could account for the small changes in RM strength. The CON group experienced significant improvements in lung volumes, air flow and RM endurance to the same extent as the EXP group, which indicates that the placebo training stimulus was sufficient to elicit identical adaptations to the velocity or shortening of the inspiratory and expiratory muscles. The higher pressure threshold training in the EXP group tends to elicit changes in the force generation capacity of the inspiratory and expiratory muscles as was evident in the significant improvements in MIP and MEP.

H. EFFECTS ON EXERCISE PERFORMANCE

1. Choice of performance test

The 20 m multistage shuttle run (bleep) test was chosen as the exercise performance test to evaluate the effect of concurrent RMT on aerobic performance. There are a number of reasons why this particular test was chosen. Firstly, the bleep test was the only field test which closely mimics the type of movement required by hockey players during match play. Hockey players run a few metres in one direction and then turn to run in the opposite direction. A closed-end laboratory test would have been more reliable and could have been used to evaluate other physiological changes as well; however, the aim of the study was to assess the effect of RMT in a real competition situation, which closely mimics hockey match play.

Secondly, the bleep test was the only field test to evaluate endurance performance that the subjects were very familiar with. All subjects who participated in the study have completed the bleep tests at least five times prior to the onset of the study. This familiarity with the bleep test greatly minimized the possibility that the changes in performance could be attributed to learning effects. Thirdly, due to the very strict regulations with regards to availability for testing; laboratory testing of exercise performance was not allowed, however, bleep tests were part of the regular test battery for the players' fitness evaluations. Lastly, the 20 m multistage shuttle run test has been shown to be a valid and reliable test to predict a subject's VO_{2max} (Cooper et al., 2005; Leger et al., 1988; Ramsbottom et al., 1987; Leger & Lambert, 1982).

Although respiratory muscle fatigue has not been measured after the performance test in the present study, evidence of inspiratory muscle fatigue during the 20 m multistage shuttle run test do exist. McConnell et al. (1997) found a significant $11 \pm 8\%$ decrease in MIP after the bleep test in moderately trained men. The evidence of RM fatigue by McConnell et al. (1997) confirms the importance of RMT to possibly have an ergogenic effect in hockey players. If RM fatigue occurs during the bleep, one can argue that the activation of the RM

metaboreflex causes whole-body fatigue during the bleep test. Numerous studies have shown that RM fatigue was reduced and exercise performance was improved after specific RMT (Griffiths & McConnell, 2007; Romer et al., 2002c; Volianitis et al., 2001b). Thus, RMT in the present study could have improved the severity of RM fatigue during exercise, a delay in the RM metaboreflex and an improvement in performance.

2. Effects of RMT on the 20 m multistage shuttle run test

There was a significant 8% and 12% improvement in the number of shuttles ran during the 20 m multistage shuttle run test, in the EXP and CON groups, respectively. Since both the EXP and CON group showed significant improvements in aerobic fitness, the improvement may be attributed to improved aerobic fitness due to regular hockey training during the competitive season. However, over the same time period during the subsequent hockey season (and without RMT), the same group of subjects showed a significant 4% and 6% decrease in the number of shuttles ran. During the two hockey seasons (with and without RMT) the subjects followed the same hockey training and fitness programme, almost identical in frequency, duration and intensity. The fact that both the EXP and CON groups, showed an improvement in respiratory muscle endurance, and both groups showed a significant improvement in 20 m multistage shuttle run performance, it is suggested that the improved performance could have been the result of an improved respiratory muscle function due to the additional RMT and sham training.

Since the EXP and CON groups both showed significant changes in aerobic performance and there were no significant differences in number of hours playing hockey or training between the two groups, these two groups were combined for further data analysis. The total number of hours spent either playing hockey or training during the hockey season with RMT did not significantly differ from the total number of hours during the hockey season without RMT (113 ± 40 vs. 112 ± 41 hours over 12 weeks, $P = 0.969$). Of all the hockey training variables (hockey training, fitness sessions, gymnasium sessions and match play, all measured in hours), fitness hours correlated negatively with the change in shuttle run performance during the season with RMT ($r = -0.56$).

This indicates that players who engaged in fewer fitness sessions actually experienced a higher percentage improvement in aerobic performance, or players who engaged in more fitness sessions actually experienced a decrease in aerobic performance. Evidence of a decrease in exercise capacity has been found in athletes after increased training volume. Jeukendrup et al. (1992) found an 8% decrease in VO_{2max} (4.8 vs. 4.4 L.min⁻¹) after fourteen days of intensified training in competitive cyclists. Snyder et al. (1995) also found a decrease in VO_{2max} (4.94 vs. 4.65 L.min⁻¹) after 15 days of increased high-intensity training. It is therefore possible that the players were fatigued at the time when the shuttle run was administered after completion of the hockey season.

During the season without RMT there was no significant correlation between hockey training variables and changes in shuttle run performance. Since there was no difference in hockey training between the two seasons, the increase in performance during one season and decrease during the next season must be explained by other factors. Clearly, these findings show that the additional RMT (“true” training and sham training) could be one of the reasons for the improvement in aerobic fitness during the hockey season with RMT.

The finding that both groups (EXP and CON) improved their aerobic fitness during the season with RMT, but performed worse after the next season without RMT, was an unexpected finding. The question arises whether aerobic fitness may actually be lost during a competitive season. Koutedakis (1995) suggested that if specialized training during the competitive season is based on technique and competition tactics and less on fitness training it will not be enough to maintain or improve aerobic fitness that was first attained during the pre-season fitness period. Thus, it is quite possible that these players lost their level of aerobic fitness during the hockey competitive season. Evidence of a decrease in fitness over a competitive season has been found in other sports. Koutedakis et al. (1993) found a significant decrease in aerobic fitness (58 ± 2.6 vs. 55 ± 3.8 ml.kg⁻¹.min⁻¹ between mid-off season and mid-competitive season) after 4 – 5 months of competition in seven competitive fencers. Koutedakis et al. (1992) also found significant decreases in VO_{2max} (60.9 ± 3.6 vs. 54.9 ± 3.8 ml.kg⁻¹.min⁻¹), isokinetic flexion strength (peak torque: 162 ± 28 vs. 136 ± 29

N.m; $P < 0.05$) and extension strength (peak torque: 296 ± 53 vs. 256 ± 42 N.m; $P < 0.05$) after seven months of the competitive season in downhill and free-style skiers.

Thus, a possible explanation for the decrease in fitness during the season without RMT, is that the training programme which the players followed during the competitive season was not designed to maintain aerobic fitness or even improve it to the same extent as the training programme during the pre-season period. However, during the season with RMT, fitness was improved due to the extra training elicited by the RMT programme.

Another possible explanation for the decrease in performance at the end of the hockey season could have been overtraining, although no methods were incorporated to evaluate whether this was actually the case. The effect of overtraining on performance is very seldom studied, since it is not in any athlete's best interest to participate in a study where overtraining is induced (Halson & Jeukendrup, 2004). Some studies reported a decrease in maximum oxygen uptake and power output during maximal exercise after a period of increasing training volume (Jeukendrup et al., 1992; Urhausen et al., 1998; Lehmann et al., 1991). Halson & Jeukendrup (2004) suggested that the declines in physiological measures can be attributed to a reduction in exercise time during incremental testing due to fatigue. The increased training volume caused subjects to feel constantly fatigued and prevented them from achieving maximal efforts.

Besides the possible influence of concurrent RMT, the following factors could have influenced performance outcomes: (1) the bleep test is an open-end test, which means that the subject do not know when or at what stage the test will stop. The test is terminated after two consecutive warnings that the subject did not reach the beacon at the 20 m markers before the "bleep" sound. The final score is the last level and shuttle number that the subject could successfully complete. The individual's perception of effort and intrinsic motivation plays an important role in the outcome of the test. Therefore, this type of test is highly effort dependent, and it could have been that the subjects were more self-motivated to give a maximal effort during the one season compared to the following season.

It should be mentioned that the subjects were blinded to the purpose of the multistage shuttle run tests. They were informed that the baseline tests (pre-season tests) were to evaluate the effectiveness of the pre-season fitness programme and the follow-up tests (post-season tests) were to determine the intensity of the off-season fitness programme. (2) It is also possible that the slightly higher baseline values during the season without RMT compared to the season with RMT, could have influenced the margin for changes in performance. Especially in the CON group, the difference between baseline tests (with RMT vs. without RMT) was marginally not significant (102 ± 6 shuttles with RMT vs. 108 ± 4 shuttles without RMT), and could have been the reason why the fitness parameter did not improve during the following season.

(3) An important club competition tournament was played just after the completion of the post-season tests during both seasons. However, during the season with RMT the names of the players that were selected for the team were not yet announced. Some players were highly motivated to do well during this particular bleep tests, since they thought the purpose of the bleep test was to select the players with the highest fitness levels; however, this was just an assumption by some players and not part of the instructions to the players. During the season without RMT, the players already knew who were selected for the team to participate in the important inter-club competition tournament, which could have influenced their perception of the purpose of the test and created a lack of motivation to perform at their best.

(4) Weather conditions could have influenced the outcome of the performance tests, since the tests were all performed outside on the hockey field (astro-turf). No direct monitoring (temperature, humidity) of the weather conditions was done during the four separate multistage shuttle run tests. However, both pre-season tests (with and without RMT) were conducted during April (autumn) and both post-season tests were conducted during September (spring) and the researcher did make a note on weather conditions in three categories: clear sky/overcast, warm/cold/mild and humid/not humid. Observations during both pre-season tests (clear sky, mild, not humid) and both post-season tests (overcast but no rain, not humid) were similar, except for the observation that during the post-season without

RMT test it was slightly colder compared to the post-season test with RMT. All four bleep tests were conducted under the hockey arena lights. (5) Fatigue due to previous physical activity could have influenced the outcome of each test. Although all tests were scheduled on the same time of day and more or less on the same date during both hockey seasons (within 5 days of previous date for pre- and post-tests, respectively), control over the level of fatigue within each subject was impossible, since players had to adhere to their hockey training and fitness programmes. (6) Players were exposed to the unforeseen incidence of illness and personal reasons for missed training sessions, which could have been one of the reasons for the decrease in fitness during the season without RMT. (7) Players could have been over-trained during the season without RMT caused by the possible increase in the intensity of training, not the duration or frequency of training.

The comparison of the effects of RMT on exercise performance between the present study and other studies was done with caution, since studies differ in the mode of RMT, mode of exercise performance tests and fitness levels of subjects. These differences have shown to elicit equivocal outcomes among studies, as discussed in Chapter 3 (p.40). Therefore, the performance outcomes of the present study were compared with, firstly, studies that also used: (1) highly trained athletes as a subject group, (2) multistage shuttle run tests or tests of progressive intensities to fatigue and (3) a concurrent inspiratory and expiratory training programme as intervention. Secondly, the performance outcomes are compared to those studies that agree to at least two of the abovementioned three requirements, and lastly a comparison to studies that agree to only one of the three requirements.

Wells et al. (2005) and Sonetti et al. (2001) complied to all three requirements for comparison. The 8% increase in aerobic performance agrees mostly with the increase in aerobic performance by Sonetti et al. (2001), who found a significant 9% (experimental group) and 6% (sham group) increase in the maximum work rate achieved on a cycle VO_{2max} test in trained cyclists, after a five week concurrent VIH and IMT programme. Similar to the present study they also found significant changes in both the experimental and sham training group with no significant differences in the changes between groups.

The subjects in the study by Wells et al. (2005) completed a 7×200 m incremental swim test. The time to complete the 200 m progressively decreased during each of the 7 increments, starting at a time equal to the subject's best 200 m time plus 35 seconds. With each increment the time to finish the 200 m decreased with 5 seconds, with 3 minutes rest between increments. Subjects were instructed to swim the seventh 200 m as fast as possible. The 20 m multistage shuttle run test is similar to the 7×200 m, since it also consists of progressive increments (levels) and each increment consists of several lengths of a specific distance (shuttles). Wells et al. (2005) found no significant improvement in swimming performance in the total group of men and women swimmers. However, the women showed a significant 4% improvement in performance after twelve weeks of concurrent RMT (IMT and EMT). Thus, the effect on performance of the present study and the study by Wells et al. (2005) were very similar (8% and 4%), except for the fact that the present study also found a significant effect on performance in the control group which was not the case in the study by Wells et al. (2005).

Griffiths & McConnell (2007), Romer et al. (2002b) and Inbar et al. (2000) complied with two of the three requirements for comparison. While Griffiths & McConnell (2007) selected trained athletes as subject group and incorporated concurrent RMT, the other two studies selected trained athletes as subject groups and used incremental exercise tests to evaluate exercise performance. Griffiths & McConnell (2007) found a significant 2.7% increase in six minute all-out rowing performance in competitive rowers and no significant changes in the performance of the control group. However, six weeks later they found no further improvements in performance in either the experimental or the control groups.

Romer et al. (2002b) found a significant decrease of 6.9% in total recovery time accumulated during 15 repetitions of 20 m sprints, with self-selected recovery periods between repetitions (not more than 30 seconds of rest), in well-trained sprint athletes (soccer, hockey, rugby and basketball players) after six weeks of IMT. The control group showed a 0.7% non-significant improvement in total recovery time. This result and the result of the present study are of great importance to the specific sport of hockey, since both the improvement in endurance to run more shuttles at a faster pace and the decrease in time needed for recovery

between sprints, are precisely the adaptations required for hockey players to improve performance during match play.

In contrast with the present study, Inbar et al. (2000) found no significant changes in an incremental treadmill VO_{2max} test in well-trained endurance athletes after ten weeks of IMT. Reasons for the lack of improvement in an incremental exercise test after IMT are evident in the literature. Firstly, Markov et al. (2001) found no significant increase in stroke volume at an exercise workload that normally elicited maximal values during a constant load exercise test, after a 15-week RMT in sedentary subjects. In agreement with Markov et al. (2001), Romer et al. (2002a) found no significant change (mean of 4%, $P > 0.05$) in estimated stroke volume (and therefore, cardiac output) across all stages during a cycle maximal incremental exercise test. These findings show that RMT does not induce central circulatory adaptations to whole-body endurance exercise, which may explain why Inbar et al. (2001) found no significant changes in the incremental treadmill VO_{2max} test.

Secondly, other RMT studies also found no significant changes in maximum workload and VO_{2max} achieved during incremental testing (Romer et al., 2002a; Romer et al., 2002b) or modest changes (6 – 9%) in both experimental and control groups. Only one of the studies found a significant change in peak power output during a cycling incremental test after RMT (Enright et al., 2006). Thus, the majority of studies found that RMT had no ergogenic effect on incremental exercise performance (open-end tests). This finding can be attributed to the poor reliability and highly effort-dependent nature of open-end exercise tests (Jeukendrup et al., 1996).

Sonetti et al. (2001) included two additional performance tests to investigate the effect of concurrent RMT (VIH and IMT) on different modes of performance tests. They found a significant 26% and 16% increase in cycling exercise time to exhaustion at 80 – 85% of maximum work rate achieved, during the incremental test, in the experimental and sham training groups, respectively. Although, the percentage of improvement in performance was higher than in the present study, they also found significant improvements in the sham training group and these changes did not differ from the experimental group. They also

found a significant 1.8% improvement in an 8 km cycling time-trial performance and by no significant changes in the sham training group. Although it was only a modest improvement compared to the other two performance tests by the same group of subjects, it is of greater practical relevance, since the time trial performance test mimics real cycling competition and this type of test is not influenced by a placebo effect. The authors cannot conclude that the improvements in performances in the other two performance tests were caused by specific voluntary isocapnic hyperpnoea and inspiratory resistive loading training, since the sham training group also showed improvements in performances. The sham training group was instructed to undergo “hypoxic training” – the inspiratory ports of the sham training devices were loosely packed with aquarium gravel, which gave subjects the impression that the gravel absorbed the oxygen content of the air. Thus the improvements in these two performance tests could have been caused by other factors, e.g. motivation and/or expectation of “hypoxic training”. This means that the improvement in performance in two of the three performance tests by Sonetti et al., (2001) were influenced by subject expectations and that the 8 km time trial performance was the only test to which the improvement could truly be attributed to the RMT programme.

Enright et al. (2006) and Chatham et al. (1999) complied to one of the three requirements for comparison with the present study, i.e. the mode of performance test. Chatham et al. (1999) found a 9.8% improvement in the 20 m multistage shuttle run test which closely resembles the 8% improvement in the EXP group of the present study. A limitation of Chatham’s study is that their control group was not a true placebo group, as they did not engage in sham training and subsequently did not show any change during the 20 m multistage shuttle run test. Enright et al. (2006) found a higher percentage of change (23%) in peak power output during a cycle incremental test after eight weeks of RMT, and no significant improvement in the control group who did no training during the intervention. The main reason for this greater percentage improvement in performance compared to the present study can be attributed to the fitness levels of the subjects. They selected recreational athletes compared to the highly trained athletes of the present study; it is expected to see a greater improvement in less fit subjects due to the greater margin left for improvement in aerobic performance.

Johnson et al. (2007), Romer et al. (2002c) and Volianitis et al. (2001b) also used highly trained athletes as subject groups. Johnson et al. (2007) and Romer et al. (2002c) found significant 2.7%, 3.4% and 3.5% improvements in a 25 km cycle time trial, a 20km and 40 km time trial, respectively, in well-trained cyclists after six weeks of IMT. These two studies found no significant changes in their respective sham training groups. Volianitis et al. (2001b) found similar improvements in performance (3.5%) during a six minute all-out rowing effort after eleven weeks of IMT. The sham training group also experienced a significant improvement (1.6%) in rowing performance; however, this improvement was significantly less than the change in the IMT group.

The remaining studies did not comply with any of the three requirements for comparison to the results of the present study. These studies used an exercise time to exhaustion test as a measurement of performance. As previously described (Chapter 3), this type of performance test is not as valid and reproducible as time trial or field test performance, since it does not mimic real life competition in sport.

Downey et al. (2007) and Williams et al. (2002) found no improvements in treadmill exercise to exhaustion at 85% of VO_{2max} after four weeks of IMT in moderately trained athletes. In studies using VIH as a RMT method and a cycling endurance test at 70 – 95% of VO_{2max} or maximum work rate, Verges et al. (2007a), Fairbairn et al. (1991) and Morgan et al. (1987) found no significant improvements in performance after four weeks of VIH training in moderately trained to well-trained subjects. McMahon et al. (2002) and Boutellier et al. (1992) found a 38% and a 3.3 minute improvement in exercise time to exhaustion at an intensity corresponding to the anaerobic threshold and 85% of maximum work rate, respectively. Markov et al. (2001), Stuessi et al. (2001), Spengler et al. (1999) and Boutellier & Piwko (1992) found 23 – 50% improvements in cycling exercise time to exhaustion at a specific percentage of maximum work loads. Almost all the studies, using VIH as training method, except for Verges et al. (2007a), incorporated no control group or included control groups that did no training during the time of the intervention. The main reason for these high percentages of improvements can be attributed to the fact that these studies used subjects who were either moderately trained or sedentary.

In general it seems that RMT elicit small but significant changes (2.67 – 3.5%) in time trial performance (Griffiths & McConnell, 2007; Johnson et al., 2007; Romer et al. 2002c; Volianitis et al., 2001b), with no effect of placebo training on performance in the CON group. Studies that used incremental or exercise time to exhaustion tests revealed higher percentage changes in performance and used either no control group or a control group that could not engage in training, and could not be considered as true placebo groups. These higher percentage changes in performance may be attributed to larger intra-individual variability found in the performance outcomes of these open-end performance tests, compared to that of time trial tests (Jeukendrup et al., 1996).

An interesting finding is that studies that investigated the effect of EMT on performance in either athletes or healthy sedentary subjects, found no significant changes in performance (Griffiths & McConnell, 2007; Suzuki et al., 1995). Studies that incorporated concurrent IMT and EMT also found no changes in performance (Griffiths & McConnell, 2007; Wells et al., 2005), or significant improvements in performance due to a placebo effect (present study). However, the majority of studies incorporating IMT only, found significant improvements in performance in the IMT groups but not in the sham training groups (Johnson et al., 2007; Griffiths & McConnell, 2007; Romer et al., 2002b; Romer et al., 2002c; Volianitis et al., 2001b). A possible explanation for the significant placebo effect on performance in the present study, using concurrent IMT and EMT, is that the resistance against inspiration and expiration simultaneously, provided by the sham training device, causes a stimulus strong enough to elicit an improvement in the efficiency of the respiratory muscles during exercise and a subsequent improvement in exercise performance.

3. Evidence of possible mechanisms for the effect on exercise performance

One of the possible reasons for the improvement in exercise performance during the 20 m multistage shuttle run tests could have been due to the improvement in the efficiency of the RM during exercise. This is evident from the significant improvements in pulmonary function (5 – 6%), RM endurance (6 – 8%) and improvements in RM strength (9 – 13%).

One can assume that if an improvement in RM function leads to an improvement in performance, there will be significant relationships between the change in RM function and the change in performance. The present study only found significant correlations between the change in PEFr and the change in bleep performance ($r = 0.56$) and the change in MEP and the change in bleep performance ($r = 0.65$). Thus, the present study does not support the above statement, with regard to all RM function improvements. In fact very few studies have found significant relationships between the change in RM function and the change in performance. Romer et al. (2002c) found significant correlations of $r = 0.62$, while Johnson et al. (2007) found no correlation between change in MIP and change in time trial performance, however, these studies have shown a RMT ergogenic effect. This can be explained by the fact that respiratory muscle assessments are concerned with maximal efforts, while the improvement lies within the efficiency of respiratory muscle work during submaximal exercise, thus not evident in maximal RM muscle assessments.

The question arises as to how the improved efficiency of the RM caused an increase in the number of shuttles that a subject could complete during the 20 m multistage shuttle run test? It is believed that greater efficiency makes the RM more fatigue resistant, and therefore will delay or relieve the onset of exercise-induced RM fatigue. A delay in RM fatigue will also postpone the RM metaboreflex (Chapter 2). These adaptations will lead to an improvement in performance. RM fatigue was not measured in the present study, however quite a few studies have shown that RM fatigue was attenuated after specific RMT (Griffiths & McConnell, 2007; Romer et al., 2002c; Volianitis et al., 2001b).

Griffiths & McConnell (2007) found that MIP declined by 11.8% (baseline) during a six minute all-out rowing effort, prior to a 4-week IMT and 6-week concurrent IMT and EMT training. After the RMT programme MIP increased by 1% after the same performance test. This result shows that RMT completely abolished inspiratory muscle fatigue during a six minute all-out rowing effort. Similar to this result, Volianitis et al. (2001b) reported a decrease in MIP of 11.2% during a six minute all-out rowing effort, prior to IMT, and only a modest 3% decrease post-IMT. Romer et al. (2002c) reported a 17% decline in MIP after a 20 km cycling time-trial prior to IMT, but only a 10% fall in MIP after six weeks of IMT.

Similarly, during the 40 km time-trial, there was a 13% decrease in MIP prior to IMT, but only a 7% decrease after six weeks of IMT. All the abovementioned studies found a significant improvement in exercise performance after RMT, probably due to the lower degree of RM fatigue.

In contrast to these findings, Johnson et al. (2007) found a significant improvement in a 25 km cycling time-trial, but no improvement in inspiratory muscle fatigue. It could have been that RM fatigue, only happened later during the 25 km time trial post-IMT compared to pre-IMT, but elicited the same degree of reduction in MIP at the end of the time-trial. However, this is only assumed since they did not measure MIP during the 25 km time-trial. This may explain why they still found a significant improvement in time-trial performance.

Two studies in the literature presented evidence of the relief or delay in the RM metaboreflex after specific RMT. McConnell & Lomax (2006) investigated the effect of prior respiratory work on the fatigue characteristics of the plantar flexors. The time to fatigue (T_{lim}) of isometric plantar flexion exercise set at 85% of maximum voluntary contractions was measured under different conditions. T_{lim} was measured as the time of isometric plantar flexion until supramaximal twitches dropped below 50% of baseline maximum voluntary contraction. T_{lim} decreased significantly from 9.93 min (control) to 4.89 min during plantar flexion exercise with a thigh cuff inflated to 140 mmHg. T_{lim} also significantly decreased from control T_{lim} when inspiratory muscle fatigue was induced prior to the plantar flexion (9.93 to 6.28 min).

After four weeks of IMT, T_{lim} remained similar to the control T_{lim} when inspiratory muscle work prior to plantar flexion was performed at the same absolute intensity (60% of pre-training MIP). T_{lim} was significantly decreased compared to control plantar flexion (9.93 to 6.33 min) when it was preceded by inducing inspiratory muscle fatigue at the same relative intensity (60% of post-training MIP).

MIP was measured before and after the breathing fatigue task and revealed a significant improvement in the fall of MIP from 26% (pre-IMT) to 19% (post-IMT). Resting MIP

significantly improved by 21% after the four week IMT. From the study it is evident that the IMT improved RM strength and decreased the extent of RM fatigue, after a resistive breathing task. It showed that T_{lim} was reduced after inducing RM fatigue prior to the plantar flexion exercise test and that IMT abolished this reduction. It revealed that RM fatigue accelerated fatigue of the plantar flexors in the same manner as when limb blood flow was restricted by means of an inflated thigh pressure cuff. This finding provided evidence that the RM metaboreflex was activated during plantar flexion exercise. These results also show that a reduction in inspiratory muscle fatigue during the same resistive breathing task, abolished plantar flexor fatigue, which indicates that the RM metaboreflex was not triggered during the same intensity of exercise after specific IMT. However, when respiratory work was increased during the resistive breathing task to exhaustion, plantar flexion fatigue was retained. This finding suggests that the RM metaboreflex has a specific threshold at which it is activated. More efficient RM, i.e. due to RMT, means the threshold required to evoke the RM metaboreflex is only reached at a later stage during exercise, thus permitting exercise to continue for longer.

Witt et al. (2007) measured sympathetically mediated heart rate (HR) and mean arterial pressure (MAP) during a resistive breathing task before and after a 5-week IMT programme. Subjects were randomly assigned to an experimental or sham training control group. An increase in MIP indicated an improvement in RM strength, while a change in the rise of HR and MAP indicated a change in the activation of the RM metaboreflex. Resting MIP increased significantly by 17% after five weeks of IMT. After the intervention, the mean rise in HR decreased significantly from 41% (baseline) to 27% and the mean rise in MAP decreased from 17% (baseline) to 4% during the resistive breathing task in the experimental group. The sham group experienced no significant changes in the rise of HR or MAP after the intervention. An explanation for this finding is that the improved strength of the RM caused them to work at a lower relative intensity during the resistive breathing task and therefore delayed the activation of the RM metaboreflex, causing a smaller rise in HR and MAP.

The findings of McConnell & Lomax (2006) and Witt et al. (2007), thus confirm the existence of a RM metaboreflex. McConnell & Lomax (2006) showed that the improved efficiency of the RM after IMT causes a delay in RM fatigue and a delay in RM metaboreflex activation. Witt et al. (2005) showed that an improvement in RM strength caused a reduction in the rise of HR and MAP during a resistive breathing task evident of a delay in the activation of the RM metaboreflex.

Another reason for the positive effect of RMT on exercise performance is that the improved efficiency of the RM during exercise leads to a decrease in the subject's perception of fatigue. Romer et al. (2002a) found a significant reduction (16% and 7.9%, respectively) in respiratory RPE across all workloads during a cycling incremental exercise test, and during a constant speed shuttle run test post-IMT (Romer et al., 2002b). They also found a significant relationship ($r = -0.80$) between the change in respiratory RPE and change in MIP before and after the IMT programme. Volianitis et al. (2001b) also reported a decrease in dyspnoea during a rowing incremental exercise test, following an RMT programme.

In addition to the decrease in respiratory RPE, some studies also reported a decrease in limb RPE, which also confirms that there was a delay in the onset of the RM metaboreflex. Romer et al. (2002b) found a significant reduction in leg RPE (7.2%) during a constant speed modified shuttle run test and an 18% reduction in leg RPE across all workloads during a cycling incremental exercise test.

In summary, although the RM metaboreflex is a relatively new area of investigation, some evidence exist that specific RMT induce the relief or delay in RM fatigue. The more efficient respiratory muscle work during exercise causes a delay in reaching the threshold that triggers the RM metaboreflex. In addition, the more efficient RM work during exercise causes a decrease in the perceptual effort of RM and limb muscle fatigue during exercise, resulting in improved exercise performance.

I. EFFECTS OF RESPIRATORY MUSCLE DETRAINING

The relevance of this part of the study was to determine the time period over which an athlete may experience a significant or complete reversal in his/her improved RM function due to the cessation of RMT. This information will indicate how often RMT should be incorporated in the daily training regime of athletes to maintain the adaptations which were gained during a RMT programme.

1. Reductions in lung volumes

The changes in lung volumes (FVC and FEV₁) over the detraining period were identical between the EXP and CON groups. FVC decreased by 6% after nine weeks of RM detraining. The decrease in FEV₁ was 4% in EXP, and 7% in the CON group after nine weeks of RM detraining, but not significantly different between the two groups. This means that there was a complete reversal in the adaptations to lung elastic recoil and chest wall dynamics nine weeks after the cessation of the RMTP. The decrease could be linked to the slight decrease in expiratory muscle strength (MEP) in the EXP group and the complete reversal in the original small, non-significant increase (4%) in the CON group. These small non-significant decreases in MEP could have caused a less forceful expiratory muscle contraction during a forceful expiratory effort, which implied a smaller volume of air (FVC) that could be expired during the FVC maneuver. The small decrease in MIP could also have led to a reduction in inspiratory capacity, which will directly affect expiratory capacity. As mentioned before, FEV₁ increased as a consequence of an increase in FVC during RMT. This happened due to a decrease in airway resistance at high lung volumes (Wells et al., 2005). It is expected that a decrease in FVC will lead to an increase in airway resistance and subsequently a decrease in FEV₁.

Two other reasons could also have caused the decrease in FVC and FEV₁. Firstly, both interventions (RMT and sham training) were novel tasks for the subjects in the present study; therefore, for this type of training the subjects were classified as recently trained athletes. It is well known that recently trained athletes will very quickly improve strength

and endurance indices during the time of exposure to a training load, but will also lose those adaptations to the muscles very quickly after cessation of training (Mujika & Padilla, 2000).

Secondly, it could be that the decrease in whole-body training caused decreases in lung volume variables. There were no correlations between the changes in lung volume variables and the changes in the number of training sessions per week, neither during the RMT intervention nor during the two different time periods during the RM detraining periods. However, there was a significant decrease in the number of exercise sessions in a subgroup of 13 subjects from the onset of the RM detraining period (8 ± 3 sessions per week) to nine weeks (4 ± 1.6 sessions per week), and 20 weeks of the RM detraining period (5 ± 2.4 sessions per week), respectively.

It could be speculated that the adaptations to the RM due to whole-body training (constant exposure to hypernoea) was already manifested during baseline testing, since the hockey players ended their fitness season and started with the competition season at the onset of the 12-week intervention. The number of training sessions per week did not change during the RMT period, and there was no correlation between change in fitness and changes in lung volumes. The latter two results support the speculation that adaptations to the RM due to whole-body training were already manifested at the start of the intervention. This whole-body exercise induced adaptations are only a speculation, since no clear evidence in the literature exist to whether athletes have superior RM function compared to non-athletes (Armour et al., 2003; Eastwood et al., 2001; Cordian et al, 1991). However, when there was a significant decrease in training frequency, the reversal of possible whole-body exercise induced adaptations to the RM could have been abolished. This could also explain why lung volumes decreased after nine weeks of detraining below the original baseline values (pre-RMT intervention).

Interestingly, both lung volume variables increased to slightly above pre-RMT values, although this changes was not statistically significant. This may be the effect of whole-body training since exercise sessions per week increased from 4 ± 1.6 times to 5 ± 2.4 times per week, between nine and twenty weeks of RM detraining. The increase in the number of

exercise sessions per week was not significant, but could have been just enough to cause a very slight improvement in lung volumes.

None of the previous studies on RM detraining (Romer & McConnell, 2003b; Boutellier & Piwko, 1992) reported changes in lung volume variables during RM detraining. Romer & McConnell (2003b) only reported baseline values in lung volumes before the start of the RMT intervention and Boutellier & Piwko (1992) mentioned that vital capacity and FEV₁ were not influenced by the RMT or a RM detraining period (18 months).

2. Reductions in air flow variables

The main finding of the results from the detraining period is the significant difference in the changes over time in MEF_{50%} between the EXP and CON groups. MEF_{50%} in the EXP group remained unchanged after nine and twenty weeks of RM detraining. The CON group showed a significant 10% decrease in MEF_{50%} after nine weeks of RM detraining and improved slightly, but not significantly, after twenty weeks of RM detraining (8% decrease). The change in MEF_{50%} after twenty weeks of RM detraining in the CON group was significantly different from zero percent change in MEF_{50%} in the EXP group.

The other air flow variable, PEF_R, showed more or less the same changes over time in the EXP group, but in the CON group PEF_R changed less drastic, but still significantly after nine weeks (1% decrease) and twenty weeks (4% decrease) of RM detraining. PEF_R in the EXP group remained unchanged from baseline detraining values after nine weeks (0% decrease) and twenty weeks (1% decrease, $P > 0.05$) of RM detraining, respectively. Both airflow variables in the EXP group did not show a significant decrease after nine or twenty weeks of RM detraining. Therefore one can conclude that the HT-RMT intervention caused an adaptation to the expiratory muscles and the dynamics of the lung to maintain the improved instantaneous flow rate capacity for longer than twenty weeks after cessation of RMT. However, the HT-ST intervention did not result in the same adaptations to maintain the improvement in MEF_{50%} during the 12-week intervention, since this value returned to baseline values after nine weeks of RM detraining. This is also evident in the fact that the

original 12-week training programme induced a significant improvement in expiratory muscle strength (MEP) in the EXP, but not in the CON group. Although, the changes in MEP was not significantly different between the two groups, the 9% increase in the EXP group could have been just enough to maintain the forceful contraction to elicit high instantaneous flows compared to the more modest 4% increase in MEP in the CON group.

Although the changes in PEFR between the two groups were not significantly different over time, it showed more or less the same tendency as $MEF_{50\%}$, with the CON group showing a significant decrease in PEFR after nine weeks of RM detraining. This finding show that if an individual lost his/her ability to force the air out quickly, he/she will probably also decrease his/her ability to force the air out quickly at a lower lung volume (from the smaller bronchi and bronchioles). To the researcher's knowledge, this is the only study that reported a significant group-time interaction in $MEF_{50\%}$ during the RM detraining period.

Romer & McConnell (2003b) measured inspiratory flow rate (V_{max}) during a single maximum inspiratory pressure maneuver by extrapolation on the inspiratory pressure-flow curve during a 9-week IMT programme followed by a period of RM detraining. They did not find the same effect in maximal inspiratory flow compared to the effect on expiratory flow rate of the present study. V_{max} decreased by 9% after nine weeks of RM detraining and remained at this decreased percentage after 18 weeks of RM detraining. The original gain in V_{max} was 16% during a 9-week IMT programme.

3. Reductions in RM strength variables

The EXP group showed modest, but no significant changes in any of the RM strength variables (MIP and MEP) after nine weeks of RM detraining, respectively. MIP decreased 4% (9 weeks and 20 weeks), and MEP decreased 3% after nine weeks and a further 2% after twenty weeks of RM detraining ($P > 0.05$). However, despite these lower values during detraining, it was still above the original pre-RMT values (MIP: 115 ± 24 vs. 120 ± 23 cmH₂O; MEP: 130 ± 27 vs. 132 ± 31 cmH₂O).

MIP and MEP showed no significant improvements during the 12-week intervention in the CON group. After nine weeks of detraining, values returned to the baseline values before the onset of the RMT study. After twenty weeks of RM detraining, MIP slightly increased to almost reach post intervention values (144 ± 22 vs. 147 ± 22 cmH₂O, $P > 0.05$). MEP in the CON group also returned to pre-intervention values after nine weeks of RM detraining and remained unchanged until twenty weeks of RM detraining. Thus, neither the intervention nor the RM detraining period elicited true changes in MIP and MEP in the CON group.

It would therefore seem that the improvement in MIP and MEP in the EXP group during the HT-RMT intervention (13% and 9%) was enough to prevent a complete reversal in the adaptations to the RM. However, the changes in the CON group were too small and resulted in complete reversal after 9 weeks of detraining. This finding may be related to the training load of the additional RMT programme in the EXP group while sham training was not done at the same high resistance load and could therefore not elicit the same degree of adaptations to the RM.

Furthermore, the EXP group was informed that their specific RMT devices “trained” RM strength compared to RM endurance of the CON group. Psychologically, this could have motivated them to perform well in the RM strength tests during the RM detraining period, since they were aware of the fact that MIP and MEP measured RM strength.

Romer & McConnell (2003b) agreed with the present study that MIP did not return to original pre-RMT values. They found that the original 29% increase in MIP, decreased significantly by 7% after nine weeks of RM detraining and then plateaued between nine and eighteen weeks of RM detraining. This finding is in accordance with studies on the effect of long periods of detraining on peripheral skeletal muscle. Lemmer et al. (2000) and Hakkinen et al. (1981) found that prolonged periods of inactivity reveal more pronounced declines in muscle strength, however, the loss is still only between 8-12% over 8-31 weeks of inactivity.

4. Reduction in RM endurance

There was a significant decrease (4%) in MVV in the EXP group during the RM detraining period; however, MVV remained 4% above the original baseline values measured at the onset of the RMT intervention, in both groups. In the CON group the original improvement of 6% in RM endurance was more or less maintained after twenty weeks of RM detraining, while RM endurance in the EXP group suffered a significant reduction after twenty weeks. The more pronounced reduction in MVV after twenty weeks in the EXP group could be attributed to the more pronounced improvement (8%) during RMTP.

The difference in the dynamics of RM training could possibly have influenced the type of adaptations to the RM which caused a smaller reduction in MVV in the CON group compared to the EXP group during detraining. The smaller resistance against inspiration and expiration during placebo training could have caused more endurance type adaptations to the RM and not significant adaptations to maximal strength such as in the EXP group. This is evident from the non-significant increases in MIP and MEP after HT-ST compared to the significant increases in these strength variables in the EXP group after HT-RMT. Therefore, the different dynamics of each breath during placebo training “trained” RM endurance more than RM strength, which could have caused the maintenance of the improved RM endurance over a longer period of inactivity in the CON group.

On the other hand, this finding could have been due to psychological factors. Subjects in the CON group were told specifically that they followed a RM endurance training programme compared to the strength training programme of the EXP group. They were aware that the MVV test evaluated RM endurance. Although the reproducibility of MVV during baseline tests were very high (ICC = 0.95), MVV is recognized as a highly effort-dependable test. Therefore, the subjects’ perceptions that they did RM endurance training could have motivated them more during the RM detraining period to still perform well in this specific test.

The only other study that reported RM endurance during periods of RM detraining was Boutellier & Piwko (1992). They found that BET returned to pre-training values (BET = time to exhaustion test at a specific tidal volume and breathing frequency) after 18 months of inactivity. The original increase in BET during a four week VIH intervention was from 4.2 ± 1.9 minutes to 15.3 ± 3.9 minutes which returned to 6 ± 3 minutes after 18 months.

J. PRACTICAL PROBLEMS AND LIMITATIONS OF THE STUDY

1. Complications with study design and experimental procedures

The large inter-individual variability in the percentage changes in MIP and MEP outcomes between baseline values and post-intervention were problematic and could be one of the reasons for the non-significant difference in the changes in MIP (EXP: $13 \pm 15.4\%$; CON: $2 \pm 10.8\%$) and MEP (EXP: 9 ± 15 ; CON: $4 \pm 9\%$) between the EXP and CON groups. From the mean values it is evident that a 13% improvement versus 2% improvement is a large difference, as well as 9% and 4%, however the CV for the percentage change between these time points were 118% (EXP) and 540% (CON) for changes in MIP and 167% (EXP) and 225% (CON) for changes in MEP.

The lack of a resistive breathing test as another method (additional to MVV) to assess RM endurance, limited the study to report the effect of the specific RMT programme on RM endurance. The inclusion of a test assessing breathing power (inspiratory and expiratory power) could have shown the effect of RMT on the force-velocity relationship of the RM. A measuring device that could simultaneously measure pressure and flow during a single inspiratory or expiratory effort, were needed for this type of assessment. The largest product of mean pressure and flow can be calculated to reveal either the inspiratory or expiratory power. Some studies used this assessment by using the following equation, $W = P \times V$, where W is the power in Watts, P is the pressure (cmH₂O) generated and V is the air flow (L.s⁻¹). Romer et al. (2002a) showed a 39% improvement in maximal inspiratory muscle power (237 ± 23 vs. 329 ± 22 cmH₂O.L⁻¹.s⁻¹) after IMT. Wells et al. (2005) calculated breathing-power as the product of the inspiratory or expiratory volume and the average

mouth pressure during inspiration and expiration, respectively, and divided it by the respective inspiratory and expiratory times to reveal power in watts. They found a 70% and 66% improvement in inspiratory and expiratory power, respectively after concurrent RMT. Both studies showed the significant influence of RMT in the improvement of the force-velocity relationship of the RM. However, the unavailability of a spirometer that could be connected to the pressure measuring device, made it impossible to include this important assessment in this study.

The lack of measurement of lung volume during inspiratory and expiratory efforts made it impossible to control whether each effort was successfully performed from either residual volume (MIP) or total lung capacity (MEP). Although subjects were instructed and closely observed to exhale to RV and inhale to TLC before the start of the respective maneuvers, it could have been that these efforts were not successfully achieved during each effort and could have influenced the measurement. Romer et al. (2002a) reported they ensured that each inspiratory effort started from RV by measuring changes in vital capacity with a spirometer connected in series to the expiratory port of the pressure measuring device.

The inclusion of a second control group, consisting of hockey players with the same fitness status and who followed the same hockey and fitness training regime, but without RMT would have been beneficial to the outcome of the study. This control group could have shown the effect of the hockey and fitness training on the 20 m multistage performance test and RM function over the exact time period as the RMT period. The evaluation of this effect in the following year made the outcome of the results susceptible to other factors that could have influenced the result, such as injury, more matches played, illness and lack of motivation. However, only a limited number of players played at the same level (provincial level) and followed the same hockey and fitness training regime. The division of the total number of available subjects ($n = 22$) into three groups would have resulted in very small sample sizes, increasing the possibility of type II errors.

A shortcoming of the study was that the researcher was unable to control for the potential hormonal effects of the menstrual cycle in subjects. The changes in RM function and

performance tests were conducted over periods of 32 weeks and 44 weeks, respectively. Thus, any observed changes in the measured parameters might have been influenced by the changes in the levels of circulating hormones and not due to the RMTP and/or hockey training. There is evidence in the literature that certain phases of the menstrual cycle might negatively influence performance. Lebrun et al. (1995) found a slightly lower VO_{2max} during the mid-luteal phase compared to the early follicular phase of the menstrual cycle in endurance athletes. The same study found no significant differences between the two phases of the menstrual cycle in maximal heart rate, maximum ventilation, RER, anaerobic performance and exercise time to exhaustion at 90% of VO_{2max} . Reilly (2000) suggested that feelings of anxiety, irritability, confusion and aggression during the premenstrual tension syndrome may influence athletic performance. However, there is no conclusive evidence that exercise performance will be impaired during any phase of the menstrual cycle, since Olympic gold medals have been won at all stages of the menstrual cycle.

2. Complications with the concurrent RMT programme

The present study, as well as other studies using combined training methods (VIH and IMT: Sonetti et al., 2001; IMT and EMT: Griffiths & McConnell, 2007; Wells et al., 2005) revealed very small improvements in pulmonary function, RM strength and RM endurance in the experimental group and no changes in exercise performance; or similar improvements in respiratory muscle function and no change or similar changes in exercise performance. From these findings, it is clear that the combined RM training method in the present study, is problematic since it leads to sub-optimal adaptations in response to both stimuli (inspiratory and expiratory load). Therefore, it probably would have been more advantageous to conduct an IMT and EMT study separately.

The researcher suggests that breathing through an inspiratory and expiratory resistance, simultaneously, led to the sub-optimal adaptations in response to the two different loads. In locomotor muscle research, this finding is also evident. Sale et al. (1990) compared two different concurrent strength and endurance training programmes – they found that concurrent strength and endurance training, each on alternated days (2×2 sessions per

week), caused a significantly greater improvement (25%) in a one repetition maximum leg press test compared to a concurrent training regime (13%) of strength and endurance training (twice a week). Therefore, it could have been of greater benefit for strength adaptations, to incorporate an inspiratory and expiratory muscle training programme, concurrently, but divide the training programme into 2 sets of 30 breaths of IMT and 2 sets of 30 breaths of EMT on alternated days, or conduct both inspiratory and expiratory muscle training on one day separately (morning – IMT and evening – EMT).

The rationale behind the implementation of concurrent inspiratory and expiratory muscle training was due to evidence that training both the agonist and antagonist muscle groups improved neuromuscular co-ordination and movement efficiency in locomotor muscles (Sale, 1988). The hypothesis was tested whether this phenomenon exists for the RM muscles. From the present study and those of Wells et al. (2005) and Griffiths & McConnell (2007), it is clear that this is not the case.

Although not specifically investigated, McConnell & Romer (2004b) stated that expiratory pressure threshold training raises intra-thoracic pressure. Romer & Polkey (2008) stated that positive intra-thoracic pressure reduces ventricular transmural pressure and decrease the time for ventricular filling which subsequently will reduce stroke volume. It will also impede femoral venous return, thus compromising oxygen delivery to the working muscles during exercise and lead to fatigue. Thus, the benefit of a more forceful effort to exhale during exercise could possibly have a negative influence on exercise performance. Griffiths & McConnell (2007) were the only study that investigated the effect of EMT on exercise performance and found no significant change in a 6 minute all-out rowing effort. Suzuki et al. (1995) did not investigate an improvement in performance since they terminated the incremental treadmill test after 10 stages. All subjects were able to complete the 10 stages before and after the EMT programme.

The sham training devices elicited only 15% of the resistance than that of the true device in the present study. Subjects in the sham training group were told that their devices permitted endurance training compared to the experimental group who underwent strength training.

Both groups followed the same number of sets and repetitions. Other studies (Romer et al., 2002a; Romer et al., 2002b) also instructed the sham groups that they completed an endurance training programme versus a strength training programme (experimental group); however they instructed the sham training groups to complete 60 breaths during one set at a resistance equal to 15% of MIP. The sham device of the present study also consisted of spring-poppet valves within the inspiratory and expiratory ports similar to the true device. However, these springs were weaker than the springs of the true device to elicit only 15% of the resistance of the springs of the true device. Johnson et al. (2007) and Sonetti et al. (2001) packed aquarium gravel in the inspiratory ports of their sham training devices and made the sham training group believe that the substance reduced the oxygen content of each breath, mimicking hypoxic training. The fact that the sham training subjects did not directly receive completely different instructions similar to that of the studies above, could have influenced the results. The resistance of the springs in the sham devices of the present study could have been an adequate training stimulus to elicit adaptations to the RM, unlike the sham devices in other studies who removed the springs or filled the port with aquarium gravel.

Although Wells et al. (2005) found very similar results compared to the present study, they determined the flow-pressure characteristics of each resistance setting of the *PowerLung*® device, which enabled them to let each subject start at a specific (50%) percentage of their own baseline MIP value. The present study did not have the means to quantify the pressure-flow characteristics of the *PowerLung*® devices and therefore could not determine the different settings for each subject to start at a resistance equal to 50% of MIP. Subjects in the present study were instructed to start at a setting that allowed only 15 breaths per set. Thus, subjectively, subjects could have under-trained their RM in the present study, at least in the beginning of the intervention period.

Romer et al. (2002a; 2002b) monitored the number of inspiratory efforts completed by the subjects through a thermistor placed inside the training device. The thermistor sensed acute drops in temperature associated with changes in air flow. The decreases in temperature (sum of changes) were stored on a microchip attached to the outside of the training device. Data

on the microchip were retrieved to investigate whether it corresponded with the requirement of the prescribed total number of breaths. In this study, the researcher was dependent on the subject's integrity to report adherence to the prescribed training programme.

Another problem was the quantification of specific whole-body training during the intervention. Whole-body exercise was quantified as the number of hours spent playing hockey, undergoing fitness sessions, playing hockey matches, or strength training in the gymnasium. This method did not include the quantification of the intensity of training together with the duration of training. Romer et al. (2002b) measured training impulse (TRIMP), which quantified training using training volume and intensity. Volume (measured as training in minutes) was multiplied by intensity (delta heart rate ratio = [exercise heart rate – resting heart rate]/[maximum heart rate – resting heart rate]). The equation was multiplied by a factor determined by the modality of the exercise session (e.g. cycling = 1, swimming = 1, running = 0.9). Using this type of quantification (TRIMP), which was corrected for exercise intensity and modality, would have been a more specific way to quantify whole-body training and its relationship with the change in exercise performance.

3. Complications with the performance test

The problem with the 20 m multistage shuttle run tests was the open-end nature of the test. The test, except for the increasing increments, agrees with constant-load time to exhaustion tests. These tests are terminated at fatigue, thus until the subject is no longer able to maintain a certain exercise intensity which is highly effort-dependable. Evidence of the poor reliability of this type of exercise performance tests was investigated by Jeukendrup et al. (1996).

Jeukendrup et al. (1996) divided thirty well-trained cyclists into three groups. Each group performed one of three different endurance performance tests (A, B or C). Protocol A was a 'time to exhaustion' test at 75% of VO_{2max} . In protocol B subjects performed as much work as possible in 15 min after a preload of 45 min at 70% of maximal workload. In protocol C subjects had to complete a preset amount of work as fast as possible. Each protocol was

repeated 5 times. The coefficient of variation (CV) for each protocol was $CV(A) = 26.6\%$, $CV(B) = 3.5\%$ and $CV(C) = 3.4\%$. Therefore, time to fatigue was a less reliable performance test than a time trial protocol in trained cyclists. Jeukendrup et al. (1996) suggested that endurance performance tests with a known "endpoint" (a certain time or amount of work) appear to be highly reproducible. They also suggested that performance tests with an "open end" (time to exhaustion) can be influenced by psychological factors, such as motivation and boredom. The poor reliability of "time to exhaustion" is further due to the unusual type of exercise, because this test does not simulate the actual competition conditions in endurance events. Although the multistage shuttle run test was chosen as performance test due to the subject's familiarity with the test and the similar movement during play, too many other factors could also have influenced the outcome of the tests, e.g. lack of motivation or boredom and competitiveness.

Another shortcoming in the present study, was the lack of a constant-load test to assess physiological variables, i.e. V_E , f_b , VO_2 , HR and lactate accumulation. Changes in these variables could have added to the evidence that RMT contributed to the efficiency of breathing during exercise.

One of the greatest shortcomings in the study was the quantification of respiratory RPE. The possible improvement in respiratory RPE during the bleep test or any other suitable performance test would also have added to the significance of the effect of RMT on performance. However, the bleep required subjects to run back and forth between beacons twenty metres apart, which made it unlikely to successfully determine perceptual effort of breathing fatigue from the subject, since they did not perform exercise in a stationary position. It was also not possible to measure exercise-induced RM fatigue, since 10 – 12 subjects completed the bleep test simultaneously, and MIP could not be measured in all subjects, simultaneously. Subjects completed the bleep test together, rather than individually, due to the limited availability of the hockey field and the limited availability of the subjects for testing.

4. Practical problems and limitations to RM detraining study

The same practical problems with the experimental procedures in the RMT study were also limitations to the RM detraining study. The measurement of inspiratory and expiratory power as described above would have revealed important information on the reduction of breathing power during the RM detraining period. Romer & McConnell (2003b) found a 17% reduction in maximal inspiratory power after nine weeks of detraining. They found that the reduction in power was substantially higher compared to the reduction in RM strength (7%).

The measurement of this RM function variable (power) is of great relevance to athletes, since having strong RM contractions reveal information about the force-generating capacity of the RM, but no information on the rate of shortening of the RM which determines the rate of air flow. High flow rates of volumes of air determine the efficiency of hypernoea during exercise. Thus, it is important to know the relationship between these two characteristics of the RM, since both needs to be improved to improve the efficiency of breathing during exercise. It is therefore important to know when there will be a reduction in the improved breathing power during detraining, to establish how often RMT is needed to maintain breathing power.

The lack of measuring training impulse (TRIMP) as the quantification of whole-body training volume was also a limitation to the RM detraining study. This measurement would have been more accurate and might have revealed significant correlations between the change in whole-body training and reductions in RM function variables. This finding might have demonstrated the influence of a decrease in training volume on the reduction in RM function variables.

The evaluation of fitness after nine weeks of RM detraining also limited the study to whether or not whole-body fitness had an impact on the RM function changes. However, the players were not available for fitness testing during this time, since they started with their intensive pre-season fitness sessions. On the other hand, it was expected that exercise performance

would have decreased during the hockey off-season, since exercise sessions per week significantly decreased from end-intervention to nine weeks after RM detraining, and from end-intervention to twenty weeks of RM detraining. This was evident in the CON group, they still showed a significant reduction in the fitness (114 ± 8 vs. 108 ± 4 , $P = 0.04$) from post-season with RMT, to twenty weeks of RM detraining (pre-season without RMT assessment).

Hockey is a winter sport, therefore training volumes changes (pre-season, competitive season, off-season) throughout the year. This meant that the hockey players reduced their training volume significantly during the RM detraining period. The RMT study had to be conducted over the competitive season, since this was the only twelve weeks throughout the year that the training volume remained the same. By keeping the whole-body training volume constant, one could exclude the fact that an increase in whole-body training load caused the significant improvements in RM function and exercise performance. However, this meant that the RM detraining study was conducted during the hockey off-season. Thus, the question that remains unanswered is: to what degree could the changes in RM function be attributed to the significant reduction in whole-body training volume, and to what degree could it be attributed to the reversal of RM adaptations due to cessation of RMT?

As mentioned before, since the changes in RM function and fitness performance were similar between the RMT group and the sham training group, it would have been beneficial to include a second control group in the RM detraining study, who would have engaged in hockey training only. This would have answered the question mentioned above. However, the limited number of subjects available for the study made division of subjects into three groups not feasible.

The study did not include a sub-group who maintained at least a fraction of the RMT programme to determine whether this will maintain the original adaptations to RM function. Romer & McConnell (2003b) investigated the effect of the continuation of RMT at two-thirds of the frequency, but at the same intensity of the RMT intervention. They found very small and non-significant changes in MIP (0% and -2%) and inspiratory power (-1 and -4%)

after nine and 18 weeks of RM detraining, respectively. This finding shows that if RMT is maintained at the same intensity, but at only two-thirds of the frequency of a 9-week IMT programme, it maintains the adaptations to the RM and the improvements in RM strength and power.

K. FUTURE STUDIES

It is suggested that the measurement of diaphragm thickness is included in future studies investigating the effect of RMT on RM strength. Enright et al. (2006) and Downey et al. (2007) found a significant increase of 12% at total lung capacity and an increase of 8% during the MIP maneuver in diaphragm thickness after RMT, respectively. Enright et al. (2006) suggested that the diaphragm thickening ratio should be determined, which is the ratio between diaphragm thickness during the MIP maneuver at FRC and the mean diaphragm thickness while relaxing at FRC. This measurement is more reliable than the measurement of diaphragm thickness during the MIP maneuver, since vital capacity and TLC could also improve due to RMT and influence the measurement of diaphragm thickness (McCool et al., 1997).

The investigation into physiological changes during exercise needs further investigation. The majority of studies showed no significant improvements in V_E , VO_2 , f_b , VCO_2 or HR during incremental tests (Griffiths & McConnell, 2007; Inbar et al., 2000), during a time trial test (Johnson et al., 2007) and a steady-state time to exhaustion test (Williams et al., 2002), except for Griffiths & McConnell (2007) who found a significant reduction in peak HR after IMT. However, more thorough investigations are needed to determine whether physiological responses of the same intensity of exercise as before RMT, changes as a result of RMT.

A great need exists to study the effect of expiratory muscle training and the possible negative effects that it may have on intra-thoracic pressure. Thus, changes in stroke volume, cardiac output and femoral venous return should be measured to determine whether the benefits of EMT are over-shadowed by the possible negative physiological effects.

Evidence of increased ventilation during exercise, while respiratory RPE and/or breathlessness decreased has been found (Verges et al., 2008a; Verges et al., 2008b). This means that even though respiratory drive was increased, the subjects perceived the effort as less fatiguing or less intense. More studies are needed to specifically investigate the mechanism through which an individual will perceive more respiratory work as a less fatiguing effort compared to before RMT.

The measurement of the energy cost of breathing, the occurrence of RM fatigue and the restriction placed upon breathing during exercise in a semi-crouched position need to be investigated in order to establish whether this specific body position during hockey play, place extra demands on the RM. New insights into whether hockey players experience additional demands on the RM to perform work during exercise, will confirm the necessity of specific RMT in these groups of athletes to improve athletic performance.

Investigations into the specific mechanism of the RM metaboreflex are lacking in the literature, thus this relatively new area of investigation is still widely open and many questions with regards to the mechanism, how it limits exercise performance and how RMT causes a delay in RM metaboreflex are still unclear. It seems that there is a specific threshold for the activation of the RM metaboreflex, and that RMT causes a delay in the achievement of this threshold to activate the RM metaboreflex. Further investigations are needed to answer the questions surrounding the specific mechanism of the RM metaboreflex. McConnell & Lomax (2006) found an increase in T_{lim} during plantar flexion after performing the same inspiratory task prior to plantar flexion compared to before the IMT period. This showed that the metaboreflex has threshold qualities. It is proposed that the accumulation of metabolites (less after RMT) in the more efficient trained RM was insufficient after IMT to reach this threshold and activate the RM metaboreflex. However, the specific mechanism (stimuli) that activates the RM metaboreflex to cause an increase in sympathetic vasoconstrictor activity is still unknown. During whole-body exercise there is a strong vasodilator effect in the working muscles while the RM metaboreflex causes a vasoconstrictive effect in the working muscles. Does the vasoconstrictor effect override the vasodilator effect? *In vitro* studies have shown that isolated diaphragm arterioles are less

responsive to vasoconstrictive influences compared to limb muscles (Aaker & Laughlin, 2002), however *in vivo* is required to determine the vasoconstrictive influence on the limb and respiratory muscles during exercise. If fatiguing RM activates the RM metaboreflex, do the fatiguing locomotor muscles have a metaboreflex effect upon the RM (Romer & Polkey, 2008)?

Limited data available on RM detraining (present study, Romer & McConnell, 2003b) emphasize the necessity of the in-depth research needed on RM detraining. Numerous studies have confirmed that the improvement in the efficiency of the RM during exercise, cause a significant improvement in athletic performance. Thus, it is important to specifically investigate all factors that can cause a decrease in the efficiency of the RM, in order to ensure that the RM work remains at optimal levels to have an ergogenic effect on performance.

Romer & McConnell (2003b) investigated the improvements of three different modes of RMT programmes on RM function; however, during the detraining period they did not distinguish between these three sub-groups which made it impossible to conclude whether the RM function changes in the three different groups responded differently to RM detraining. Future studies should determine if different modes of RMT (concurrent RMT, IMT, EMT or VIH) will cause different rates of reductions in RM function indices during RM detraining. It has been shown that the training mode influence the maintenance of strength during periods of inactivity in locomotor muscles (Hodikin, 1982). This will allow researchers to determine which mode of RMT will elicit the improvements in the efficiency of the RM and maintain it for longer compared to other modes of RMT during periods of inactivity.

Future studies should also determine whether the duration of the RMT programme have an effect on the time period over which improvements in RM function will be maintained during RM detraining. This will enable researchers and athletes to choose the best mode and duration of RMT that will elicit the least reductions in improved RM function for the longest period of time during RM detraining.

Future studies should determine the optimal frequency and intensity of RMT to maintain a plateau in RM function, efficiency and indirectly exercise performance. The present study and the study by Romer & McConnell (2003b) found decreases in RM function during the first nine weeks of detraining and further, up to 18-20 weeks, only a plateau in these original decreases in RM function. The question remains for how long will this plateau continue before a complete reversal, if any, occurs? It is also recommended to decrease the time window between assessments during the RM detraining period, for instance every three weeks, since the present study could only show changes after nine weeks, however the significant reductions could have manifested after three or six weeks during RM detraining.

The specific adaptations due to whole-body training and the specific reversal of adaptations due to whole-body detraining have been investigated by several studies in limb muscles (Mujika & Padilla, 2001b; Moore et al., 1987; Coyle et al., 1984; Klausen et al., 1981). However, the investigation into specific muscle adaptations and reversal of adaptations were easy accessible through muscle biopsies in one of the muscles of the quadriceps muscle group. Adaptations to RM due to specific RMT and the reversal of adaptations due to RM detraining are practically impossible due to the inaccessibility of the RM for muscle biopsies. Evidence of fiber distribution changes in the RM exist in animal studies after a period of RMT (Ramirez-Sarmiento et al., 2002). It is therefore suggested that the reversal of adaptations to the RM due to RM detraining should be investigated in animals to provide information on the exact reversal and time period for the reversal in adaptations after the cessation of RMT.

CHAPTER NINE

DISCUSSION: SECTION TWO PREDICTORS OF RESPIRATORY MUSCLE STRENGTH AND ENDURANCE

A. INTRODUCTION

The purpose of Study 3 was to determine the physical and physiological factors that predict respiratory muscle (RM) strength and endurance in an athletic population. Previous studies have determined reference values for pulmonary function and these values are primarily based on gender, age and height (Falaschetti, 2004; Hankinson et al., 1999; Quanjer et al., 1993; Knudson, 1976, Morris et al., 1971). The development of hand-held, non-invasive techniques to measure RM strength made it necessary to establish reference values in order to evaluate RM weakness. Thus, reference values for the general population have been determined by a few studies (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992) and it seems that the same factors (gender, age, height and weight) determined the variation in RM strength variables.

B. DIFFERENCES BETWEEN ATHLETES AND NON-ATHLETES

1. Kinanthropometric differences

Both men and women athletes showed significantly smaller bi-iliac breadth measurements compared to non-athletes of the same gender. The mean bi-iliac measurements for men and women athletes were below 30 cm (men: 28.9 ± 2.8 ; women: 27.2 ± 3.5) compared to values above 30 cm for non-athletes (men: 31.3 ± 3.2 ; women: 30.7 ± 4.4). The results are in agreement with the study by Ruff (2000) who also reported bi-iliac measurements between 27.1 and 30 cm groups among track-and-field athletes. Evidence of smaller bi-iliac breadths in athletes compared to non-athletes has been found in adolescent girls (12 -13 years old).

Telford et al. (1986) reported a mean bi-iliac breadth of 22.9 cm in athletic girls compared to 24.3 cm in non-athletic girls ($P < 0.05$). Broekhoff et al. (1986) found that the hip width (i.e. bi-iliac breadth) in gymnasts were significantly smaller in gymnasts (23.6 ± 2.3 cm) compared to non-athletic girls (26.1 ± 2.7 cm).

The smaller bi-iliac breadth measurement in athletes can possibly be attributed to the fact that athletes tend to be taller than non-athletes. Although the present group of athletes was not significantly taller than the non-athletes, men athletes were on average 3 cm taller than men non-athletes and women athletes were 1.1 cm taller than women non-athletes. It is possible that these small differences in height could have contributed to a more “stretched out” morphological profile of athletes compared to non-athletes. In fact, previous studies have shown that athletes are significantly taller than their same age non-athletic counterparts in adults (Coast et al., 1990) and adolescents (Broekhoff et al., 1986; Cacciari et al., 1990). Martin & Stager (1981) found a mean height of 172 ± 3 cm in women endurance athletes compared to a mean height of 167 ± 3 cm in women non-athletes. This difference was, however, not statistically significant.

Women athletes had significantly lower body mass index (BMI) compared to women non-athletes. The lower BMI in women athletes can be attributed to the 8% lower body mass found in this group compared to the women non-athletes. Broekhoff et al. (1986) also found a significantly lower body mass in adolescent female gymnasts compared to their non-active age-matched counterparts.

Men athletes had a lower relative sitting height (52 ± 2.0 vs. $54.3 \pm 7.7\%$) compared to men non-athletes. Since there were no significant difference in height between these two groups, one may conclude that if the athletes had shorter upper bodies in relation to the same height than non-athletes, they probably had longer legs in relation to height than the non-athletic group. Telford et al. (1986) found a relatively longer thigh length in young male athletes compared to non-athletes (39.4 vs. 38.2) and a longer thigh length in athletic girls compared to non-active girls (38.3 vs. 36.0 cm). Leg length, in relation to height, also vary among different sports, with higher relative lower limb length in distance runners, jumpers,

basketball players and volleyball players compared to sprinters, gymnasts, wrestlers and weight lifters (Ackland, 2006).

The results of the present study cannot convincingly state that athletes have superior morphology, but they do have a different morphology, compared to non-athletes. Whether the difference in morphology is caused by physical activity itself, or whether it is due to fast-maturing youngsters who excel in their sport and continue with playing sport during adulthood, remains unknown.

2. Respiratory muscle function differences

The only differences between athletes and non-athletes concerning the pulmonary outcomes were significant higher lung volume measurements in men athletes compared to non-athletes. Men athletes achieved on average 11% and 14% higher FVC and FEV₁ than non-athletes, respectively. No differences were found between women athletes and non-athletes with regards to pulmonary function assessments. This finding of superior lung capacities in athletes is in agreement with Armour et al. (1993). They compared national level swimmers and runners to each other and to a control group (non-athletes). The mean values for FVC among groups showed that swimmers (7.12 ± 1.2 L) achieved a significant 21% and 28% higher value compared to the runners (5.89 ± 0.8 L) and the control group (5.55 ± 0.6 L), respectively. Swimmers also achieved a significantly higher FEV₁ compared to the control group (5.98 ± 1.0 vs. 4.55 ± 0.6 L). Similar results were reported by Cordian et al. (1990). Conversely, Eastwood et al. (2001) found no significant differences in FVC (7.18 ± 0.6 vs. 7.1 ± 0.7 L) or FEV₁ (4.7 ± 0.4 vs. 4.4 ± 0.4 L) between endurance athletes and sedentary subjects.

Inspiratory muscle strength, measured as MIP, was significantly higher in athletes compared to non-athletes for both genders. Men and women athletes achieved 19% and 26% higher MIP measurements compared to non-athletic men and women, respectively. Women athletes also had 24% higher MEP values compared to non-athletic women, while no difference was observed between men athletes and non-athletes. Only a few studies agree

with this finding. Cordain et al. (1990) found that women competitive runners achieved a significantly higher MEP (170 ± 10 cmH₂O) compared to competitive swimmers (147 ± 13 cmH₂O) and compared to a control group (135 ± 10 cmH₂O). Fuso et al. (1996) found a significantly higher MIP (from FRC) in competitive soccer players compared to non-athletic subjects (103 ± 29.6 vs. 70 ± 29.6 cmH₂O). However, the main reason for the large difference (47% between mean values) can be attributed to the fact that the group of soccer players consisted of only men, whilst the group of non-athletes consisted of 77 men and 26 women. Previous findings show that women have significantly lower MIP values compared to men due to larger RM mass found in men (Glucksman, 1981).

The majority of studies found no significant differences in RM strength measurements between athletes and non-athletes (Eastwood et al., 2001; Armour et al., 1993; Coast et al., 1990; Cordain et al., 1990). Eastwood et al. (2001) reported similar MIP values in endurance athletes (152 ± 41 cmH₂O) compared to sedentary individuals (141 ± 25 cmH₂O). Cordain et al. (1990) and Armour et al. (1993) found similar values for MIP and MEP among a group of swimmers, runners and controls, respectively. MIP appeared to be higher in skiers compared to the sedentary individuals (116 ± 5.7 vs. 101 ± 11.1 mmHg) in the study by Coast et al. (1990), but the difference was not significant.

In the present study women athletes had superior inspiratory and expiratory muscle strength compared to non-athletes, but men athletes only had better inspiratory muscle strength compared to non-athletes. The fact that Fuso et al. (1996) compared a group of men athletes to a mixed group of sedentary men and women make their findings irrelevant to use since differences between men and women, regardless of their fitness status, are expected. Cordain et al. (1990) also found only a difference in expiratory muscle strength, but similar values for inspiratory muscle strength in women athletes and non-athletes. Most studies, however, reported no differences in RM strength between athletes and non-athletes. Therefore, it is concluded that there are no conclusive evidence that athletes have superior RM strength compared to non-athletes.

Men athletes showed a significantly 11% higher MVV compared to non-athletic men which is possibly evidence that whole-body training affects RM endurance. No significant difference was found in RM endurance between women athletes and non-athletes. This finding is agreement with Martin & Stager (1981) who found similar MVV values between women endurance athletes and non-athletes. The finding in men support a similar finding by Martin & Chen (1982) who found significantly higher 12-second MVV values in competitive athletes compared to their non-active siblings (172 ± 11 vs. 107 ± 9 L.min⁻¹). This large difference in MVV values between athletes and non-athletes can be attributed to the fact that the athlete group in the study consisted of seven men and one woman, but these individuals were not compared with a same-gender sibling. The group of non-athletic siblings consisted of three men and four women. A greater number of women within a group of subjects will lower the average group value of MVV compared to a group of subjects consisting of mostly men. Therefore, the validity of the large difference in MVV in the study by Martin & Chen (1982) is questionable. The present study and Neder et al. (1999) found significantly lower MVV values in women compared to men and can be attributed to the smaller RM mass in women compared to men.

Very few studies investigated the differences in MVV between athletes and non-athletes; however, differences in specific breathing endurance tests (longer RM endurance tests) between these two groups have been reported. Eastwood et al. (2001) reported that athletes were able to achieve a higher percentage of their maximum MIP during an incremental BET compared to sedentary individuals (90 ± 8 vs. $78 \pm 10\%$ of MIP). Martin & Stager (1981) also found superior RM endurance in athletes compared to non-athletes during an incremental BET (75% vs. 67% of MVV) and a breathing to exhaustion test (T_{lim} : 11 vs. 3 min). From these findings, it is evident that superior RM endurance in athletes compared to non-athletes is easier detectable in longer RM endurance tests than short RM endurance tests (MVV).

In summary, men athletes had superior lung volumes, inspiratory muscle strength and RM endurance compared to men non-athletes, whilst women athletes only revealed superior inspiratory and expiratory muscle strength compared to women non-athletes. The finding of

more RM function differences between men athletes and non-athletes can possibly be related to the larger sample size in men athletes ($n = 103$) compared to women athletes ($n = 57$).

The present study reveals new evidence that lung capacities, RM strength and RM endurance are advanced compared to their non-athletic same – age and – gender counterparts. None of the physical factors which influence RM function could have caused the differences in RM function measurement between the two groups, since there were no differences in age, height or weight between the two groups in men and women, respectively. Two reasons may account for the significant differences in RM function between athletes and non-athletes. It could be that athletes with a genetic predisposition to have superior RM function are the athletes who continue and excel in their sport. Or it could be that whole-body training improves RM function.

Whole-body endurance training: Evidence in the literature exist that a whole-body training intervention causes significant changes in RM function. However, the outcomes of the studies investigating this phenomenon have been contradictory. The main reason for the positive outcomes in some studies and contradicting outcomes of other studies is the differences in the intensity and duration of the whole-body interventions among the studies. For instance, the subjects in the study by Robinson & Kjeldgaard (1982) ran at 80% of their predicted maximum heart rate for 40 min per day, three times per week for a total of twenty weeks. After the twenty-week intervention they found a significant 14.4%, 13.5% and 15.8% increase in MEP, MVV and maximum sustainable ventilatory capacity for 15 min, respectively. The subjects in the study by Clanton et al. (1987) underwent three types of training during one intervention. They ran three times per week for 40 – 60 min, underwent isotonic training in the gymnasium three times per week and progressively increased their swim training from ~ 2 km to ~ 9 km during the 12-week intervention. From the mean pre- and post-values for MIP (estimated from graphical data), it seems that the group of subjects who underwent the abovementioned training without additional inspiratory muscle training experienced a 19% increase in MIP and a 112% increase (from ~ 3.9 min to ~ 8.3 min) in time to exhaustion (T_{lim}) at 65% of MIP during a breathing endurance test.

In contrast, the subjects in Thomas et al. (1998) underwent incremental exercise training for 30 min per day at 70% of baseline $\text{VO}_{2\text{max}}$, 5 days a week for 6 weeks. They found no significant changes in MIP (112 ± 9 vs. 107 ± 9 cmH₂O) or maximal voluntary volume (44 ± 2 vs. 48 ± 3 L in 12 seconds) after the whole-body training period. Similarly, O’Kroy & Coast (1993) found no significant changes in MEP and MVV after subjects underwent whole-body cycle endurance training for 20 min per day at 80% of heart rate reserve, four days per week for four weeks.

It is clear that the latter two studies found no significant changes in RM function due to substantial differences in the whole-body training regimes compared to the previous two studies (Robinson & Kjeldgaard, 1982; Clanton et al. 1987) which incorporated longer and more intensive whole-body training regimes. It can be concluded that whole-body training improves RM function; however, the whole-body training programme needs to incorporate exercise intensities higher than 70% of $\text{VO}_{2\text{max}}$ and should be of longer duration than six weeks.

Whole-body endurance training elicits physiological adaptations to locomotor muscles which imply that the same effect manifests in RM. In animal studies it is evident that whole-body endurance training elicits increases in the oxidative enzyme activity of the costal diaphragm (Powers et al., 1992a; Powers et al., 1992b; Uribe et al., 1992; Powers et al., 1990). Some studies did contradict these findings; however, they investigated the changes in the costal and crural diaphragm and considered these two regions as one region (Green et al., 1989; Fregosi et al., 1987; Metzger & Fitts, 1986). However, it has been shown that whole-body endurance training only improves the oxidative capacity of the costal diaphragm and not the crural diaphragm (De Troyer et al., 1981).

Endurance training causes the same type of changes in the diaphragm and the locomotor muscles, although the adaptations are not of the same magnitude. Changes between 20 and 30% in the oxidative capacity of the costal diaphragm after 8 – 10 weeks of treadmill training have been reported (Powers & Criswell, 1994; Gosselin et al., 1992; Uribe et al., 1992), while locomotor muscles with the same fiber type composition (e.g. plantaris)

experience between 40 and 80% increase in oxidative enzyme capacity after similar whole-body training regimes (Powers et al., 1992b; Laughlin et al., 1990).

The positive influence of physical activity or athletic training on RM strength and endurance outcomes have been demonstrated by the significant correlation between physical activity and indices of RM strength and endurance. Neder et al. (1999) found significant correlation coefficients of 0.47, 0.49 and 0.48 between physical activity (measured as the physical activity score) and MIP, MEP and MVV, respectively. Pringle et al. (2005) found that MVV could explain 27% of the variance in 10 km race time in active runners. However, the present study failed to reveal a significant relationship between physical activity (exercise sessions per week) and any of the RM strength and endurance variables ($r = 0.10$ to -0.14 , $P > 0.05$).

Two scenarios may explain the differences in adaptations between locomotor muscles and the diaphragm. Firstly, the metabolic stress on the diaphragm during locomotion is less than that placed upon the locomotor muscles, and the energy demands during exercise determine the increases in muscle oxidative enzyme capacity (Dudley et al., 1982). Secondly, the initial mitochondrial enzyme activity in the costal diaphragm is significantly higher than in locomotor muscle (e.g. plantaris); citrate synthase activity equals $\sim 28\%$ in the plantaris muscle compared to $\sim 50\%$ in the untrained diaphragm of rats (Powers et al., 1992b). Logically this is due to the fact that the diaphragm is chronically active throughout life compared to very little activation of the locomotor muscles of caged rats. One can conclude from the latter two explanations that the RM do experience whole-body exercise-induced adaptations, however, they require much higher exercise intensities to significantly experience an improvement in function compared to locomotor muscles. Therefore, studies that did not report an improvement in RM strength and endurance after whole-body endurance training incorporated exercise programmes of lower intensities and duration that is required to elicit adaptations.

Genetic predisposition: Another reason for the differences in RM function variables between athletes and non-athletes could be due to athletes possessing a genetic

predisposition for superior lung capacities and RM function. To investigate this phenomenon, a comparison was made between competitive and non-athletic siblings. Martin & Chen (1982) found a significantly higher 12 second MVV (172 vs. 107 L.min⁻¹) and a significantly higher progressive breathing endurance test result (75% vs. 62% of MVV) in the athletic subjects compared to their non-athletic siblings. It appears from this finding that genetic make-up can, at least in part explain the differences in RM function between athletes and non-athletes.

Cordain et al. (1990) suggested that the superior static lung volumes of swimmers compared to runners may be due to the significantly younger age at which swimmers started their swim training compared to the runners (9.4 ± 2.8 vs. 14.8 ± 2.4 years). In fact, Fu (1976) found an increased alveolar density in rats that had to swim early in their lives compared to their non-swimming controls. Thus, the exposure to a training stimulus at a very early age in life may have caused the superior RM function in athletes. However, the need exist for longitudinal studies in humans to thoroughly investigate this phenomenon.

In conclusion, the researcher found that the significant and substantial differences in lung volumes, RM strength and RM endurance between athletes and non-athletes can be attributed whole-body training of sufficient intensity and duration. Therefore, specific RMT may assist athletes to improve their RM function during periods when their training volume and intensity is low.

C. DIFFERENCES BETWEEN MEN ATHLETES AND WOMEN ATHLETES

Kinanthropometric differences between men athletes and women athletes revealed that men were significantly older (2 years) and had a higher BMI and relative arm span compared to women. Women had significantly higher relative sitting height compared to men. The lower relative sitting height and higher relative arm span reveal that men athletes had longer upper and lower body extremities in relation to their standing height compared to women. The higher BMI values in men, indicate that men were heavier relative to their standing height compared to women athletes. This finding can be attributed to the fact that men had

more muscle mass compared to the women (73 ± 9 vs. $51 \pm 6\%$, $P < 0.001$), which made them heavier in relation to standing height. Glucksman (1981) showed that at the age of eighteen, men have on average 50% and 15% more fat free mass compared to women in the upper and lower body, respectively.

It was expected that the absolute values for RM function variables will be considerably higher in men compared to women due to the morphological differences between men and women. Thus it was not surprising for FVC, FEV₁, PEF_R and MVV were significantly higher in men compared to women. However, it was expected that these differences will not be reflected when these values were corrected for gender differences (percentage of predicted values). The percentage of predicted values of FVC, FEV₁ and PEF_R revealed no difference between men and women, however, the percentage of predicted values for MVV (126 ± 17 vs. $119 \pm 16\%$) was significantly higher in men athletes compared to women athletes.

This finding suggests that other factors, rather than the morphological differences between men and women, are responsible for the higher percentage of MVV values in men. There were no difference in the exercise frequency per week between men athletes and women athletes, which indicated that the difference could not be attributed to activity levels. Cordain et al. (1990) suggested that swimmers had superior lung function compared to runners since they started competing in their sport at a very young age, however, the opposite were true for the present study. Women athletes started competing in their sport at a younger age compared to men (years of competitive experience in men: 7.7 ± 4.8 vs. 10.6 ± 3 years in women).

There are three possible explanations for the higher percentage of predicted MVV in men: (1) Physical training in men causes greater muscle endurance adaptations in the RM of men compared to women which permitted the men to achieve a higher percentage of their predicted values during the MVV test. (2) The very short duration (12 seconds) MVV test carries a large strength component. Therefore, men would perform better in all strength tests, since they had significantly stronger inspiratory and expiratory muscles compared to

women. (3) The higher percentage of predicted MVV in men compared to women athletes may be due to the larger absolute lung volumes in men. Airway resistance decreases with increasing lung volumes (Wells et al., 2005). Therefore, men will have less airway resistance during maximal voluntary ventilation compared to women and subsequently ventilate more air in 12 seconds.

Inspiratory and expiratory muscle strength revealed on average a 25% and 20% higher value in men athletes compared to women athletes. These differences in RM strength can mainly be attributed to the larger muscle mass in men compared to women that can contract more forcefully during the inspiratory and expiratory maneuvers and generate a higher pressure difference between the atmospheric air and the lungs. The average differences in MIP between men and women agree with the majority of other RM strength studies. Most studies found that men had 25 – 30% higher MIP and/or MEP values compared to women (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Black & Hyatt, 1969).

Reiter et al. (2006) found a slightly smaller difference (14%) between healthy men and women with regards to MIP (109 – 147 vs. 93 – 106 cmH₂O). However, all studies that included men and women in their subject group reported significantly higher values in RM strength for healthy men compared to healthy women.

D. CORRELATIONS BETWEEN ANTHROPOMETRY AND RESPIRATORY MUSCLE FUNCTION

1. Correlations with inspiratory muscle strength

Chest girth and transverse chest breadth revealed the highest correlations with MIP (both $r = 0.56$). This was followed by body mass and the mesomorphic component ($r = 0.44$ and 0.40 , respectively). The present study agrees with the majority of studies that found a significant but weak to moderate relationship ($r = 0.20$ to 0.39) between body mass and inspiratory muscle strength (Reiter et al., 2006; Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992). In contrast to these findings, Fiz et al.

(1998) found no significant relationship between body mass and MIP in healthy subjects. Fuso et al. (1996) was the only study who reported a significant relationship between body mass and MIP ($r = 0.33$) where a number of subjects ($n = 27$) out of the total group ($n = 130$) were competitive athletes (soccer players).

The association between in inspiratory muscle strength and body mass can be attributed to the fact that an increase in muscle mass leads to an increase in body mass. Schoenberg et al. (1978) called this phenomenon the “muscularity effect”. This statement was confirmed by Neder et al. (1999) who found a positive correlation between body mass and leg strength ($r = 0.49$) and body mass and RM strength ($r = 0.36$), which indicates that an increase in body mass must be associated with an increase in muscle mass that not only causes an increase in limb muscle strength, but also RM strength. Furthermore, Arora & Rochester (1982) provided evidence that an alteration in body mass caused alterations in diaphragm muscle mass.

To the researcher’s knowledge no previous study investigated the relationship between somatotype and RM strength. However, Neder et al. (1999) reported a significant relationship between lean body mass and inspiratory muscle strength ($r = 0.66$) and mesomorphy and lean body mass are indicators of muscularity. The significant relationship between the mesomorphic component and MIP indicates that individuals with a more muscular build (more lean body mass) have stronger inspiratory muscles.

Standing height, sitting height and arm span revealed correlations between $r = 0.33$ to 0.36 . The sum of seven skinfolds which is an indication of body fatness showed a weak negative correlation with MIP. To the researcher’s knowledge, no other study reported the relationships of these specific upper body dimensions (chest girth, transverse chest breadth, sitting height and arm span) and the sum of skinfolds with inspiratory muscle strength. Only Harik-Khan et al. (1998) reported a significant correlation between an upper body extremity, i.e. forearm girth (men: $r = 0.39$; women: $r = 0.43$) and MIP.

There was a moderately strong correlation between MIP and MEP ($r = 0.66$). This finding is in agreement with Neder et al. (1999) who also found a significant and strong relationship between inspiratory and expiratory muscle strength ($r = 0.85$). This implies that if a person has strong inspiratory muscles they also have strong expiratory muscles. This finding confirms the previous statement that all indices of muscular strength, i.e. RM strength, limb muscle strength and indices of muscle mass, are interrelated.

Lung volumes (FVC and FEV_1), peak flow (PEFR) and RM endurance (MVV) revealed similar correlations with MIP ($r = 0.42 - 0.46$) which confirmed findings of previous studies (Hautmann et al., 2000; Johan et al., 1997). Harik-Khan et al. (1998) reported a stronger relationship between FVC and MIP in men ($r = 0.51$) compared to women ($r = 0.37$). Fiz et al. (1998) also reported stronger correlations between FVC and MIP ($r = 0.59$) as well as FEV_1 and MIP ($r = 0.65$). The similar correlations of FVC and FEV_1 with MIP can be explained by the fact that higher lung volumes cause a decrease in airway resistance that resulted in higher values for FEV_1 .

Hautmann et al. (2000) reported the same relationship between PEFR and MIP ($r = 0.40$) compared to the present study, while Harik-Kahn et al. (1998) found a slightly stronger relationship between PEFR and MIP ($r = 0.51$). Leech et al. (1983) confirmed the relationship between pulmonary function and RM pressure. They found that an index of RM strength (sum of MIP and MEP) significantly accounted for the variation in different pulmonary function variables (beta coefficients), i.e. FVC (0.173), FEV_1 (0.156) and PEFR (0.249). They concluded that RM muscularity influence the result of pulmonary function variables. Logically, one may speculate that if an individual can contract his/her RM more forcefully, it will generate a higher pressure difference between the atmosphere and the lungs which will result in more air flow. This same phenomenon will cause a greater TLC which will lead to greater FVC and FEV_1 during forceful expiration. A higher TLC will result in a greater volume of air that can pass through the lungs, and less airway resistance at high lung volumes.

Conversely to the present study, Reiter et al. (2006) found no significant relationship between RM endurance and RM strength. RM endurance was measured as the time to exhaustion (T_{lim}) during resistive breathing test at 80% of MIP. T_{lim} did not correlate with MIP or 12 second MVV.

2. Correlations with expiratory muscle strength

Chest girth and transverse chest breadth revealed the highest correlations with MEP ($r = 0.43$ and $r = 0.41$), similar to the kinanthropometric relationships with MIP. This was followed by body mass, standing height, arm span, mesomorphic component and sitting height ($r = 0.28$ to 0.39). To the researcher's knowledge no other study investigated the relationship between chest dimensions and expiratory muscle strength. However, larger upper body dimensions must be related to larger chest cavities. Thus, if an individual has a larger chest cavity, he/she will proportionally have a larger RM mass and therefore stronger RM.

Other studies found that correlations of body mass and standing height with MEP revealed slightly stronger relationships (Neder et al., 1999; Bruschi et al., 1992). Neder et al. (1999) reported correlation coefficients of 0.49 and 0.59 between body mass, standing height and MEP. Bruschi et al. (1992) found correlations of 0.42 and 0.49 between body mass, and standing height and MEP. Fiz et al. (1998) found a stronger relationship between standing height and expiratory muscle strength ($r = 0.50$).

Although other studies did not investigate the relationship between somatotype and expiratory muscle strength, the measurement of lean body mass is similar to that of mesomorphy (one somatotype component), since both is an indication of the muscularity of the body. Neder et al. (1999) reported a substantial relationship between muscularity (lean body mass) and expiratory muscle strength ($r = 0.46$). These findings explain that a larger muscle mass resulted in stronger expiratory muscles. The sum of seven skinfold measurements (body fatness) revealed a weak negative correlation with MEP ($r = -0.21$). This finding confirmed that fat mass has a negative impact on both inspiratory and expiratory muscle strength.

All the kinanthropometric measurements, except standing height, revealed slightly stronger relationships with inspiratory muscle strength compared to expiratory muscle strength.

The positive correlations between lung volumes (FVC, FEV₁) and expiratory muscle strength indicated that those individuals with larger lung volumes also had stronger expiratory muscles. Fiz et al. (1998) reported moderately strong correlations between lung volumes and expiratory muscle strength (FVC: $r = 0.51$; FEV₁: $r = 0.51$), while Johan et al. (1997) reported similar correlations between FVC and MEP ($r = 0.39$ in men and 0.32 in women) as in the present study.

3. Correlations with RM endurance

The comparison of studies on the association between anthropometric and pulmonary function variables, and RM endurance is difficult, since different methods were used to assess RM endurance. The present study and Neder et al. (1999) measured MVV, while Fiz et al. (1998) and Chen & Kuo (1989) measured time to exhaustion during a resistive breathing test (T_{lim}). Standing height correlated strongly with MVV ($r = 0.72$). One other study also found a substantial relationship ($r = 0.63$) between standing height and RM endurance (Neder et al., 1999), while other studies reported weaker relationships ($r = 0.35 - 0.42$) between these two variables.

Arm span, chest girth and transverse chest breadth revealed correlations between 0.63 and 0.68 . To the researcher's knowledge no other study reported significant relationships of upper body dimensions with RM endurance measurements. Body mass and sitting height revealed similar correlations with MVV ($r = 0.57$ and 0.56). Neder et al. (1999) found a weaker correlation between body mass and MVV ($r = 0.38$), while Fiz et al. (1998) found no significant relationship between these two variables. One may conclude that the difference between the findings of the present study and Neder et al. (1999) compared to Fiz et al. (1998) is the different type of RM endurance assessments (MVV vs. BET). This finding may explain that muscle mass plays a role in the outcome of MVV. Greater body mass is

associated with greater muscle mass and therefore the outcome of the MVV test is related to the strength of the RM.

The sum of seven skinfolds (body fatness) revealed a significant negative relationship with MVV ($r = -0.41$), while age and the mesomorphic component only revealed a weak correlation with MVV (both $r = 0.25$). Similar to the present study (age range: 17 – 34 years old), Leech et al. (1983) reported that age had a positive influence on all pulmonary function measurements. Thus it is concluded that up to age 35, RM endurance will increase before it will decline with increasing age. This finding is consistent with the finding of Black & Hyatt (1969) who found that age significantly influenced (negative) MIP after the age of 55 years.

Neder et al. (1999) reported a substantial relationship between lean body mass and MVV ($r = 0.67$), which is in disagreement with the present study who found a weak but significant relationship between muscularity (mesomorphy) and RM endurance.

Pulmonary function variables ($r = 0.72 - 0.80$) revealed strong relationships with MVV, but RM strength variables revealed weaker relationships with MVV ($r = 0.37 - 0.44$). Fiz et al. (1998) reported weaker relationships between FVC ($r = 0.54$) and FEV₁ ($r = 0.64$), and RM endurance, measured as time (T_{lim}) during BET. Neder et al. (1999) reported stronger relationships between RM strength variables and MVV (MIP: $r = 0.67$; MEP: $r = 0.72$). The significant relationships between RM strength and RM endurance measured as MVV are clearly related to the nature of the MVV test. The test is very short in duration and therefore strength dependant, whereas Reiter et al (2006) found no relationship between a BET and RM strength measurements. However, in contrast to the finding of Reiter et al. (2006), Fiz et al. (1998) found correlation coefficients of 0.52 and 0.43 between MIP and MEP, and BET, respectively. This finding cannot be attributed to nature of the test, since this test is continued until exhaustion, and not related to RM strength. Thus it can be concluded that other factors influenced the relationship between RM strength and RM endurance.

4. Summary

The correlations between kinanthropometric and pulmonary function variables revealed stronger relationships with RM endurance ($r = 0.25 - 0.80$) compared to inspiratory ($r = -0.18 - 0.56$) and expiratory muscle strength ($r = -0.21 - 0.43$). On average, the relationships between these variables and inspiratory muscle strength were also stronger compared to that of expiratory muscle strength.

Age did not correlate with inspiratory or expiratory muscle strength, but it correlated significantly with RM endurance. This finding can be attributed to the small age range within the group of subjects. Previous studies have shown that age is one of the strongest factors, after gender, which influenced RM strength and endurance in the general population. All previous prediction equations for RM strength and endurance included age as a covariant (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Bruschi et al., 1992; Wilson et al., 1984; Black & Hyatt, 1969). Since this relationship has been well established, the aim of the study was to determine which other factors will influence the variance in RM strength and endurance measurements within a specific population. Thus, the ages of the individuals participating in the present study ranged from 18 – 34 years in men athletes and 17 – 30 years in women athletes. Significant relationships between age and RM strength and endurance measurements were therefore not expected, since the age range within the selected populations was small. However, the significant positive correlation of age with MVV (over the small age range) indicates that individuals may improve the RM endurance between ages 17 to 34 years.

Interestingly, other studies did not investigate the effect of body fatness on either RM strength or endurance measurements, however, these comparisons are widely reported for locomotor muscle strength and endurance tests. The present study found that relative fatness had a negative influence and muscularity (mesomorphic component) had a positive influence on RM strength and endurance measurements. This finding add to the notion that athletes have superior RM strength and endurance compared to non-athletes, due to greater muscle mass as a result of physical training.

E. PREDICTORS OF RESPIRATORY MUSCLE STRENGTH AND ENDURANCE

1. Predictors of inspiratory muscle strength

The best subset of predictors for inspiratory muscle strength was gender, mesomorphic component and exercise sessions per week. Gender and mesomorphy significantly predicted MIP, whereas exercise sessions per week reached borderline significance in predicting MIP. However, these variables were responsible for only 35% of the variation in MIP measurements. Thus, the majority of variance in MIP in this specific population remains unexplained. This finding that anthropometric variables could only account for a small percentage of variance in MIP is in agreement with other studies that also investigated the predictors for RM strength. Most studies reported predictors of MIP for men and women separately, due to the large differences in MIP between men and women (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Wilson et al., 1984; Black & Hyatt, 1969). Fuso et al. (1996) and Bruschi et al. (1992) included men and women in their multiple regression analysis, similar to the present study. Fuso et al. (1996) reported that gender, BMI and age were significant predictors of MIP and accounted for 38% of the variance in MIP in 130 subjects (27 subjects were competitive soccer players). Bruschi et al. (1992) found gender, age and BSA to be the significant predictors of MIP. These variables accounted for 27% of the variance in MIP in 625 healthy subjects. These studies agreed with the present study, that gender is the strongest predictor of inspiratory muscle strength.

In addition to Fuso et al. (1996), Cordain et al. (1987) also determined the predictors for MIP in an athletic population (101 male runners). Age was the only factor that could account for the variance in MIP up to 43%; the rest of the variance was due to unknown factors. The prediction of MIP by other studies that predicted MIP separately for men and women, found that either age or height, age and height, or age, height and weight contributed to the variance in MIP (Hautmann et al., 2000; Neder et al., 1999; Harik-Khan et al., 1998; Johan et al., 1997; Wilson et al., 1984). In all the above studies, age was the best predictor of MIP. However, age, height and weight could only explain a small percentage of the variance in

MIP. Neder et al. (1999) reported the highest R^2 –values of 0.48 for men and 0.47 for women. The rest of the studies reported R^2 –values between 0.05 and 0.41 (Hautmann et al., 2000; Harik-Khan et al., 1998; Johan et al., 1997; Wilson et al., 1984).

The literature is in consensus that age, height and weight can not solely explain the variance in MIP measurement in healthy and physically active individuals. In addition, the present study revealed new evidence that other kinanthropometric measurements, i.e. mesomorphy and activity level, i.e. exercise sessions per week, do significantly predict MIP, but together with other co-variants, could still only explain less than half of the variance in MIP. Therefore, other factors, not measured yet account for more or less 60% of the variance in MIP.

2. Predictors of expiratory muscle strength

The present study revealed that only gender significantly predicted MEP ($P < 0.001$). Mesomorphic component was also included in the prediction equation, since it was marginally significant ($P = 0.051$). Still, these two variables only accounted for 24% of the variance in MEP, which revealed that kinanthropometric variables and gender influence MEP measurements only to a small degree. The only study which agreed with the present finding is Johan et al. (1997) who found that age, height and weight accounted for 24 – 29% of the variance in MEP in three different female subject groups (Chinese, Malay and Indian). The same predictors for men in Johan et al. (1997) could explain 33-42% of the variance in MEP. Bruschi et al. (1992) found that gender; age and BSA significantly predicted MEP in their subjects and accounted for 46% of the variance in MEP. In agreement with Bruschi et al. (1992), Neder et al. (1999) reported that age (only significant predictor) accounted for 48% in the variance in MEP for men and women, respectively.

The present study shows that the measured variables included in the regression analysis could predict MIP to a better extent than MEP. It could be speculated that the MEP maneuver were a more novel test for individuals to perform compared to the MIP maneuver and that resulted in a higher inter-individual variability. This speculation is based on the fact

that expiration is usually passive and only becomes active during hyperventilation, however, inspiratory muscle contractions happens during resting inspiration and during hyperventilation. However, the present study revealed coefficients of variation (%) of 18% and 26% in MIP for men and women, respectively; and 19% and 21% in MEP for women and men, respectively. Therefore, it is unlikely that inter-individual variability was responsible for the better predictors for MIP, compared to MEP.

Bruschi et al. (1992) suggested that the lower R^2 -value for MIP compared to MEP in their study might be attributed to the greater difficulty in coordination and activation of the inspiratory muscles compared to coordination and activation of the expiratory muscles. The expiratory muscles are mainly the abdominal muscles and contraction of these muscles is a familiar muscle contraction (same as sit-ups) for most individuals.

However, the researcher speculates that the novelty of the maneuver may cause weaker coordination of muscle contraction during the MEP maneuver, since the end result of the muscle contraction is not the same. Contraction of abdominal muscles to sit upright is a familiar type of movement to subjects, but the contraction of abdominal muscles to exhale forcefully is a novel task for subjects. The researcher, therefore, speculate that the novelty of the maneuver may be the reason for poorer coordination of muscle contraction during the MEP rather than the MIP maneuver. Subjects are more aware of inspiratory muscle contractions, since these muscles are active during rest and exercise.

The question remains unanswered: Which factors are responsible for the variance in RM strength measurements? The researcher agrees with Bruschi et al. (1992), that the measured kinanthropometric variables can only partially explain for the variation in respiratory pressures. The degree of coordination of RM contraction may play a role in the inter-individual variability of these measurements. The cooperation of subjects also plays an important role, since the measurements of respiratory pressures are highly effort-dependable. The present study also agrees with the suggestions of Bruschi et al. (1992) that the muscular work capability, elastic recoil of the respiratory system, genetic and environmental factors collectively play a role in the variance in respiratory pressure measurements. Future studies

need to find means to measure these factors and include them in regression models for MIP and MEP.

3. Predictors of respiratory muscle endurance

The present study revealed that the kinanthropometric variables (gender, relative sitting height) and pulmonary function variables (FEV₁, PEFR) included in the regression model for MVV could explain 78% of the variance in MVV. This R² -value for MVV was substantially higher compared to Neder et al. (1999) who reported that age and height could account for 57% of variance in MVV in men and age accounted for 43% of the variance in MVV in women.

Fiz et al. (1998) measured RM endurance in two ways: (1) C_{max} was the highest possible pressure that could be maintained for 2 minutes during an incremental resistive breathing test. (2) T_{lim} was the time duration of a breathing endurance test at 80% of C_{max}. Age and gender could explain 38% of variance in T_{lim} and 43% of the variance in C_{max}. Although the study by Fiz et al. (1998) is not directly comparable to the present study, the present study found that a combination of kinanthropometric and pulmonary function variables explained the variation in RM endurance to a larger extent than kinanthropometric variables alone (Neder et al., 1999; Fiz et al., 1998).

The prediction of MVV by FEV₁ and PEFR confirms the fact that higher lung volumes decreases airway resistance (Wells et al., 2005), and subsequently causes greater air flow. Greater air flow at low airway resistance would explain higher voluntary ventilation.

4. Observed values compared to predicted values

The observed values for men and women athletes of the present study were compared to the predicted values for MIP, MEP and MVV. The predicted values were derived from the available reference equations determined by previous studies. Only studies that resembled or consisted of subjects within the same age group of the present study were used for

comparison. The reference equations of McConnell & Copestake (1999) and Enright et al. (1994) were not included, since these two equations were derived from a subject population of 59 years of age and older. The mean observed values of the present study were subtracted from the mean predicted value as calculated from the prediction equation of the respective studies. A difference of more than 10 between the two values was considered an overestimation or underestimation of the observed value. These comparisons were made to establish whether prediction equations derived from the general population will be valid and representative of values found in an athletic populations.

MIP values: The predicted values calculated from the prediction equations of Harik-Khan et al. (1998); Bruschi et al. (1992) and Black & Hyatt (1969) just underestimated the observed values for MIP in men. The difference between the mean predicted MIP and the observed MIP were -16; -15 and -16 cmH₂O. Hautmann et al. (2000) and Wilson et al. (1984) largely underestimated the observed values (-28 and -36 cmH₂O, respectively). Neder et al. (1999) and Cordain et al. (1987) predicted similar values compared to the observed values in the present study, although their values were still slightly lower (-6% and -8%, respectively). Both these studies included physically active people, which could explain the comparative values.

In women, the mean predicted values from Neder et al. (1999) and Black & Hyatt (1969) only marginally underestimated the mean observed values (-10 and -17 cmH₂O, respectively). Hautmann et al. (2000), Harik-Khan et al. (1998) and Bruschi et al. (1992) largely underestimated the observed values by -25, -28 and -22 cmH₂O, respectively. The predicted value of Wilson et al. (1984) revealed a similar, but a slightly higher value (9 cmH₂O) compared to the observed values of MIP.

MEP values: In men the predicted value for MEP from the reference equation of Bruschi et al. (1992) showed a slight underestimation of the observed MEP values. The reference equations by Neder et al. (1999) and Wilson et al. (1984) resulted in similar values to the observed values. The exception was the predicted value derived from the equation by Black & Hyatt (1969). This predicted value largely overestimated the mean observed value for

MEP in men (245 vs. 159 cmH₂O). The same was true for MEP values in women (+34 cmH₂O). The large overestimation can be attributed to methodological differences between the present study and Black & Hyatt (1969) for the assessment of MEP.

Black & Hyatt (1969) instructed subjects to press their lips against a rubber mouthpiece, which created the advantage to produce higher pressures compared to the present study where subjects used a flanged mouth piece and had to place their lips over the mouth piece. This seems to be the most logical explanation, since predicted MIP values from the equation derived by Black & Hyatt (1969) did not overestimate the observed MIP values, when the subjects their study also placed their mouths around the mouth piece rather than against the mouth piece.

Cordain et al. (1987) was the only other study who compared their observed values from 100 male competitive runners with the predicted value derived from Black & Hyatt (1969). They reported that the predicted MIP did not significantly differ from the observed MIP (-4 cmH₂O), but the observed MEP only reached 86% of predicted MEP by the equation of Black & Hyatt (1969). They followed the same method (lips against mouth piece) compared to Black & Hyatt (1969) to measure MEP, however Cordain et al. (1987) reported other explanations for the overestimation of observed values. They suggested since Black & Hyatt (1969) included smokers in their subject group – smokers might have increased the mean expiratory muscle strength since smoking induced higher expiratory flow resistance and/or chronic coughing which trained the expiratory muscles.

Pringle et al. (2005) also reported that male and female competitive runners achieved 56% and 58% of the predicted values for MEP (men: 198 cmH₂O; women: 152 cmH₂O), respectively. They suggested that the lower observed values for MEP is related to breathing high lung volumes at rest and also during exercise for long periods of time, which could have caused a reduction in airway resistance. Reduction in airway resistance causes lower pressure development during the expiratory mouth pressure maneuver. The rest of the predicted values according to Neder et al. (1999), Bruschi et al. (1992) and Wilson et al.

(1984) underestimated (-22 to -29 cmH₂O) the observed MEP values in women of the present study.

MVV values: The only recent study that reported prediction equations for MVV was Neder et al. (1999). In men, the observed MVV were almost identical (203 vs. 204 L.min⁻¹) to the predicted MVV derived from the equation by Neder et al. (1999). However, the mean observed MVV in women athletes was underestimated by the predicted MVV. The difference between the observed and predicted values in women, but not in men, can be attributed to the fact that RM endurance could have increased more from a sedentary or moderately active state to a highly trained state in women athletes, since women engaged in competitive sport for a significantly longer period of time than men athletes (11 ± 3 vs. 8 ± 5 years).

F. MAIN FINDINGS AND CONCLUSION

Morphologically, athletes tend to have longer legs and narrower hips compared to non-athletes. Whether these morphological differences are the result of physical training, or whether this superior morphology causes children to excel in the sport which in return, motivates them to participate until adulthood, is unknown.

In addition to morphological differences, the present study found RM function differences between athletes and non-athletes. Athletes (men) revealed greater lung volumes compared to non-athletes and this is supported by the literature (Armour et al., 1993; Cordain et al., 1990). Athletes also showed greater RM strength (supported by Fuso et al., 1996) and greater RM endurance (supported by Martin & Chen et al., 1982) compared to non-athletes.

The differences between athletes and non-athletes can be attributed to either genetic predisposition, or whole-body endurance training. Greater RM strength in athletes compared to non-athletes is supported by physiological adaptations to the RM muscles after endurance training observed in animal studies (Powers et al., 1992a; Powers et al., 1992b; Uribe et al., 1992; Powers et al., 1990). However, Martin & Chen (1982) found significant differences in

RM function between competitive athletes and their non-athletic siblings. The latter two findings support the suggestion that whole-body endurance training causes an improvement in RM function.

Men athletes had relatively longer legs and a more bulky morphology than women athletes. Men achieved a greater percentage of predicted MVV and achieved 20-25% higher MIP and MEP values compared to women athletes. These differences can be attributed to the greater muscle mass in the men athletes compared to the women athletes. The present study revealed a significant correlation between MVV and MIP, and MVV and MEP. This finding was also supported by Neder et al. (1999). These findings support the conclusion that MVV is strongly influenced by muscular strength.

The most important finding among the relationships of anthropometry and pulmonary function with MIP, MEP and MVV, are that chest girth and transverse chest breadth revealed the strongest correlation with RM strength indices and that pulmonary variables (FEV_1 and PEFR) revealed the strongest correlations with RM endurance (MVV). Body mass and mesomorphy had significant positive correlations, while body fatness (sum of seven skinfolds) had significant negative correlations with MIP, MEP and MVV, respectively. These findings suggest that the higher body mass in individuals are attributed to larger muscle mass and have a positive influence on RM strength and endurance.

Overall, the correlations with MIP were stronger compared to the correlations with MEP and the correlations with MVV were higher compared to those with MIP and MEP, respectively. The higher correlations between pulmonary function and MVV can be attributed to the influence of a decrease in airway resistance on FEV_1 , PEFR and MVV. The stronger correlations with MIP compared to MEP can be attributed to the fact that the MEP maneuver is a more novel activity compared to MIP, which might have been strongly influenced by muscle coordination. However muscle coordination were not measured in the present study.

In men, about 50% and in women about 72% of reference equations in the literature underestimated the observed values for MIP and MEP in the present athletic population.

This means that the majority of reference equations are not suitable to use for the prediction of MIP and MEP in athletic populations.

The anthropometric and pulmonary function variables that influenced RM strength, could only explain 35% and 24% of the variance in MIP and MEP, respectively. The predictors could explain the variance in MIP to a larger extent than for MEP. It is unknown why this is the case since the inter-individual variability in MEP was smaller compared to that of MIP. Other unknown factors possibly play a more important role in the prediction of RM strength, such as coordination of RM contraction, subject motivation, the elastic recoil of the lungs and RM work capacity. Although the predictive value of the reference equation derived in this study is still not adequate to predict RM strength in athletes, it does provide alternative equations that are more suitable for athletic populations, than previously reported equations.

The pulmonary function and anthropometric variables that significantly predicted MVV could explain 78% of the variance in MVV, and FEV₁ and PEFR made the largest contribution to this reference equation. This prediction equation for MVV can therefore be used in laboratory settings to calculate the predicted values of MVV for athletes within the age range of 17 to 34 years.

G. FUTURE STUDIES

The need exist for the standardization of RM strength and endurance measurements. This will make comparisons among studies less complicated. It is recommended that whilst there are no standardized methods to evaluate MIP, MEP and RM endurance, laboratories should develop their own reference values with the same device and experimental procedures to obtain comparable observed and predicted values for similar populations. This will eliminate the underestimation or overestimation of observed values.

Future studies should investigate the ideal mode, intensity, duration and frequency of whole-body training that will cause optimal improvements in RM strength and endurance. An

increase in RM strength and endurance may indirectly – through the delay of the activation of RM metaboreflex – improve athletic performance.

Longitudinal studies are needed to investigate the relationship between the change in fitness level and physical conditioning of an individual and the change in RM strength. Cordain et al. (1990) mentioned that swimmers showed larger static lung volumes since they started at a younger age with competitive swimming compared to runners who started at a later stage in their lives with competition. The need exist to investigate whether this phenomenon would also be true for RM strength indices.

Future studies are needed to determine the exact factors which influence the variance in MIP and MEP, since anthropometric variables only explain a small percentage of variance in MIP and MEP. Future studies should find reliable methods to quantify the degree of coordination of RM contraction, subject motivation, the elastic recoil of lungs and muscular work capability of the RM to determine the possible influences of these indices on RM strength measurements.

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APPENDIX A



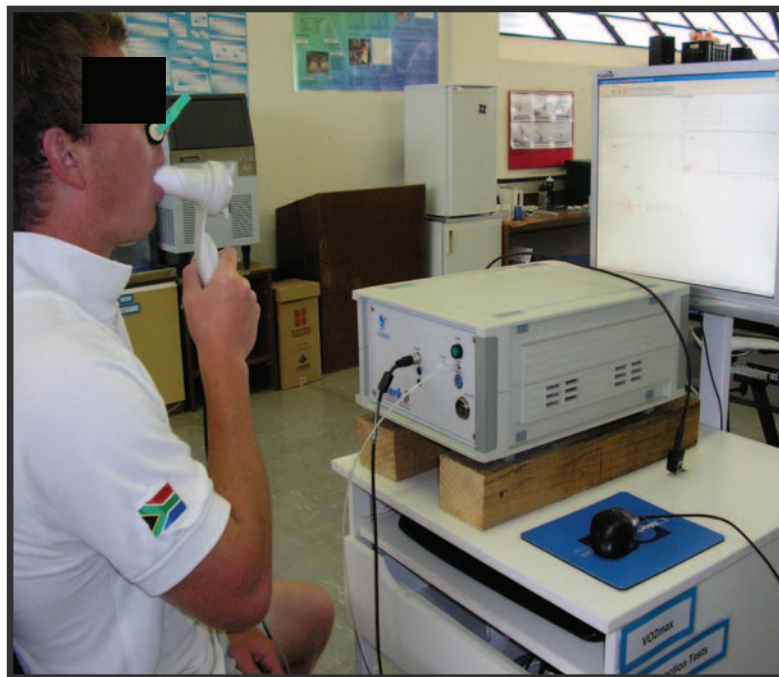
Illustrations of the *PowerLung*® (*PowerLung*® Inc. Houston, Texas, USA) respiratory muscle training devices (*top*) and the use of the device during a respiratory muscle training session (*bottom*).



APPENDIX B



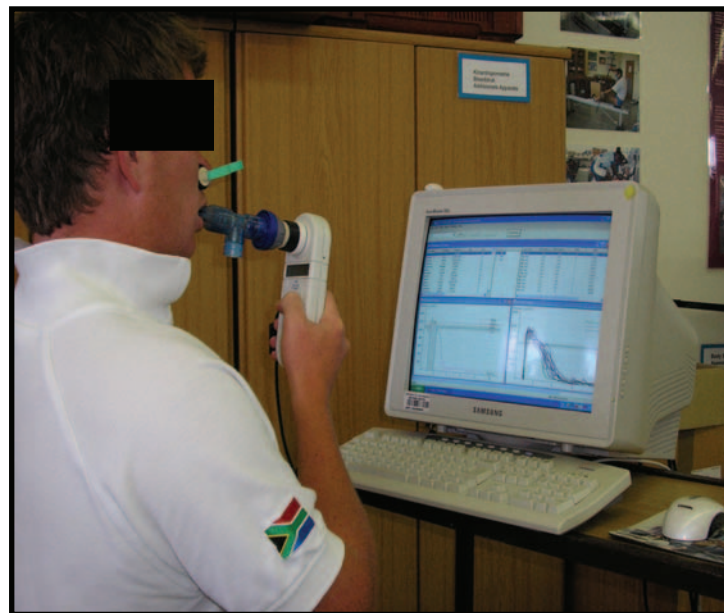
Illustrations of the flow meter with a disposable carton mouth piece (*top left*), the specific placement of the mouth over the mouth piece (*top right*), and a subject performing a flow-volume curve test, using the *Cosmed Quark b²* spirometer (Rome, Italy) (*bottom*)



APPENDIX C



Illustrations of the portable, hand-held respiratory pressure meter (*Micro RPM* meter, Micro Medical Ltd., England) (*top*) and a subject performing a maximum mouth pressure test recorded by the *PUMA* software (Micromedical Ltd., England) (*bottom*).



APPENDIX D

Table A: Reproducibility results between first and second baseline tests for all respiratory muscle function variables during the respiratory muscle training study

<i>Variable</i>	<i>Baseline 1</i>	<i>Baseline 2</i>	<i>ICC</i>	<i>Lower limit 95% CI</i>	<i>Upper limit 95% CI</i>	<i>SEM</i>
FVC (L)	4.23 ± 0.6	4.14 ± 0.6	0.915	0.790	0.965	0.16
FEV₁ (L)	3.55 ± 0.5	3.49 ± 0.4	0.889	0.745	0.953	0.13
PEFR (L.s⁻¹)	7.9 ± 1.1	7.8 ± 1.1	0.915	0.805	0.964	0.30
MVV (L.min⁻¹)	142.7 ± 18.4	145.3 ± 18.4	0.955	0.897	0.981	4.01
MEF_{50%} (L.s⁻¹)	4.28 ± 1.1	4.14 ± 0.7	0.858	0.682	0.939	0.34
MIP (cmH₂O)	121 ± 29	123 ± 27	0.895	0.766	0.955	8.91
MEP (cmH₂O)	131 ± 29	130 ± 26	0.935	0.852	0.972	7.28

ICC: Interclass correlation coefficient; CI: confidence intervals; SEM: standard error of the mean. Variables: FVC – forced vital capacity; FEV₁ – forced expiratory volume in one second; PEFR – peak expiratory flow rate; MVV – maximum voluntary ventilation; MEF_{50%} - maximum instantaneous flow at 50% FVC; MIP – maximum inspiratory mouth pressure; MEP – maximum expiratory mouth pressure.

APPENDIX E

INFORMED CONSENT

Title of the research project:
New Insights into Respiratory Muscle Function in an Athletic Population”
Respiratory Muscle Training and Detraining

Reference number: _____

STATEMENT BY SUBJECT:

I, the undersigned,
_____, [ID _____],
of (address) _____

confirm that:

1. I was invited to participate in the abovementioned research project that is undertaken by the Sport Science Department at Stellenbosch University.
2. It was explained to me that
 - 2.1 the aim of the study is to determine the effect of a 12-week lung muscle training programme on lung muscle function and subsequently exercise performance.
 - 2.2 from the project I will receive interesting information on my general fitness level and lung functions that can be beneficial to improving my sport performance.
 - 2.3 I will undergo a 12-week lung muscle training programme. During this intervention I will receive a lung muscle trainer (*Powerlung*) through which I will be required to breathe, 30 breaths, twice daily for six days a week over a period of 12 weeks.
 - 2.4 I will record a logbook of my daily activities and hand it in to the researcher on a weekly basis.
 - 2.5 I will visit the Sport physiology laboratory on four different occasions. During these visits, the following tests will be done:
 - 2.5.1 during the first visit I will complete a questionnaire regarding my health status and activity level, and undertake the first baseline lung function and lung muscle strength tests.

- 2.5.2 during the second visit I will repeat the baseline lung function and lung muscle strength tests. During this visit I will also receive the lung muscle trainer (*Powerlung*) to start with the 12-week intervention.
- 2.5.3 the first and second visit will be separated by at least 48 hours but not more than 7 days.
- 2.5.4 I will visit the laboratory on the third occasion after 6 weeks during the intervention. During the visit I will undertake the 6-week lung function and lung muscle strength follow up tests. During this visit my skinfolds, limb circumferences, diameters and limb lengths will be measured to determine my fat percentage and body shape/size.
- 2.5.5 I will complete the 20-m multi-stage shuttle run test before and after the intervention in order to determine the effect of lung muscle training on endurance performance.
- 2.6 I can/may be selected to take part in the follow up study of this study where the detraining effect of lung muscle training will be determined. This study will consist of a repeat of the follow up tests (as in the fourth visit), at 9 weeks and at 20 weeks after the lung muscle intervention.
3. I was warned that there is a possibility that I may experience one or more symptoms during the lung function and lung muscle strength tests. These symptoms include dizziness and an irritated throat. I understand that I may stop the tests at any time when I experience any of these symptoms. I was also informed that a medical doctor (Dr. P.L. Viviers or Dr. A. Williams) will be present at all times at Coetzenburg to take care of medical emergencies.
4. I understand that the researcher/test observers and/or Stellenbosch University cannot be kept responsible for any injuries that might occur during any of the tests included in the project.
5. I was informed that the information which will be obtained through this project will be handled confidentially, but that the results will be published in research journals. However, the results within the publications will be done anonymously.
6. The above information was explained to me in English by Jacolene Kroff. I was given the opportunity to ask questions and all the questions were satisfactorily answered. If I have any questions, I can contact: Me. J. Kroff (Tel. 021 808 2818 of 082 448 3778; e-mail: jkroff@sun.ac.za) or Prof. E. Terblanche (Tel. 021 808 2742 of 082 707 6501; e-mail: et2@sun.ac.za).
7. It was explained to me that my participation in this project is voluntary and that I may withdraw from the study at any time. I also understand that the researcher or medical doctor may withdraw me from the study if deemed necessary for medical purposes.
8. I was informed that there are no costs involved for my participation in this project. I was also informed that I will receive no payment for participation in the research.
9. I was informed that I can contact Me. Hunter-Husselman (tel. 021 808 4623; e-mail: mh3@sun.ac.za) if I have any questions regarding my rights as a subject in a research project.

I voluntarily agree to participate in the abovementioned project.

I take the responsibility to endeavour to complete all (4 visits to the laboratory and fitness tests) tests.

I realise that the outcome of each test depends on how well motivated I am to take part in the intervention and I undertake to thoroughly follow the lung muscle training programme

I take the responsibility to be and stay **highly motivated** during the **12 week lung muscle training programme** and to complete my **daily logbook** and hand it in to the researcher on a weekly basis.

Signed at _____ on _____ 20 _____.

Subject

Witness

STATEMENT BY RESEARCHER

I, _____, declare that I:

1. Explained the information in this document to _____;
2. requested her to ask questions if anything was unclear;
3. that this conversation took place in English/Afrikaans.

Signed at _____ on _____ 20 _____

Researcher

Witness

APPENDIX F

INFORMED CONSENT

Title of research project:
New Insights into Respiratory Muscle Function in an Athletic Population:
Predictors of Lung Muscle Function in Competitive Athletes

Reference number: _____

CONSENT OF SUBJECT:

I, _____ [ID: _____]
from (address) _____

confirm that:

1. I was invited to participate in the above-mentioned project conducted by the Department of Sport Science of the University of Stellenbosch.
2. It was explained to me that:
 - 2.1 the aim of this project is to determine what the relationship is between body composition and respiratory muscle strength.
 - 2.2 I will participate in various experimental tests during a single visit to the exercise laboratory. During the visit: my skinfolds, limb circumferences, diameters and limb lengths will be measured to determine my fat percentage and body shape/size. My lung functions (lung volumes) will be measured with a spirometer, the strength and endurance of my respiratory muscles will be tested.
 - 2.3 No invasive procedures (e.g. injections, draw of blood) or administration of any substances will be administered.
 - 2.4 I understand that I can stop the exercise tests at any time when I experience one of these symptoms.
 - 2.5 The researcher/test observers and/or the University of Stellenbosch cannot be held responsible for any injuries that might occur during any of the tests included in the project. I was also informed that a medical doctor (Dr. P.L. Viviers or Dr. A. Williams) will be present at all times at Coetzenburg to take care of medical emergencies.
3. I was informed that the information which will be obtained through this project will be handled confidentially, but that the results will be published in research journals. However, the results within the publications will be done anonymously.

4. The above information was explained to me in English by Jacolene Kroff. I was given the opportunity to ask questions and all the questions were satisfactorily answered. If I have any questions, I can contact: Me. J. Kroff (Tel. 021 808 2818 of 082 448 3778; e-mail: jkroff@sun.ac.za) or Prof. E. Terblanche (Tel. 021 808 2742 of 082 707 6501; e-mail: et2@sun.ac.za).
5. It was explained to me that my participation in this project is voluntary and that I may withdraw from the study at any time. I also understand that the researcher or medical doctor may withdraw me from the study if deemed necessary for medical purposes.
6. I was informed that there are no costs involved for my participation in this project. I was also informed that I will receive no payment for participation in the research.
7. I was informed that I can contact Me. Hunter-Husselman (tel. 021 808 4623; e-mail: mh3@sun.ac.za) if I have any questions regarding my rights as a subject in a research project.

I voluntarily agree to participate in the abovementioned project.

I take the responsibility to endeavour to complete all tests.

I realise that the outcome of each test depends on how well motivated I am to do my best.

I take the responsibility to be and stay **highly motivated** throughout the duration of the project and to complete each test to the best of my ability.

Signed at _____ on _____ 20 _____.

Subject

Witness

STATEMENT BY RESEARCHER

I, _____, declare that I:

1. Explained the information in this document to _____;
2. requested him/her to ask questions if anything was unclear;
3. that this conversation took place in English.

Signed at _____ on _____ 20 _____

Researcher

Witness

APPENDIX G

Table B: The mean \pm SD values of respiratory muscle function variables at the different time points during the respiratory muscle training and detraining period for the experimental (EXP) and control (CON) groups.

<i>Variable:</i>	<i>12 week RMTP</i>			<i>20 week DT</i>	
	<i>Baseline</i>	<i>6 weeks</i>	<i>12 weeks</i>	<i>21 weeks</i>	<i>32 weeks</i>
<i>FVC</i>					
EXP	4.07 \pm 0.6	4.23 \pm 0.7	4.28 \pm 0.7	3.97 \pm 0.7	4.10 \pm 0.7
CON	4.26 \pm 0.5	4.44 \pm 0.5	4.49 \pm 0.5	4.23 \pm 0.5	4.29 \pm 0.5
<i>FEV₁</i>					
EXP	3.40 \pm 0.4	3.54 \pm 0.5	3.58 \pm 0.5	3.40 \pm 0.4	3.44 \pm 0.5
CON	3.62 \pm 0.3	3.81 \pm 0.3	3.81 \pm 0.4	3.56 \pm 0.4	3.62 \pm 0.4
<i>PEFR</i>					
EXP	7.66 \pm 1.1	7.98 \pm 1.1	8.01 \pm 1.1	7.76 \pm 0.8	7.66 \pm 0.8
CON	7.96 \pm 1.1	8.21 \pm 1.0	8.32 \pm 1.1	8.27 \pm 1.4	8.04 \pm 1.3
<i>MEF_{50%}</i>					
EXP	3.95 \pm 0.7	4.11 \pm 0.8	4.20 \pm 0.8	4.14 \pm 0.8	4.17 \pm 0.9
CON	4.38 \pm 0.9	4.75 \pm 0.9	4.80 \pm 1.2	4.31 \pm 1.1	4.41 \pm 1.2
<i>MIP</i>					
EXP	115 \pm 24	123 \pm 25	129 \pm 27	120 \pm 23	120 \pm 23
CON	144 \pm 22	145 \pm 20	147 \pm 22	142 \pm 10	145 \pm 23
<i>MEP</i>					
EXP	130 \pm 27	141 \pm 34	141 \pm 35	135 \pm 31	132 \pm 31
CON	138 \pm 31	138 \pm 32	148 \pm 31	135 \pm 20	135 \pm 19
<i>MVV</i>					
EXP	141.0 \pm 19.1	144.7 \pm 17.8	151.8 \pm 18.0	143.6 \pm 18.6	142.4 \pm 19.3
CON	149.7 \pm 19.8	150.1 \pm 19.0	158.6 \pm 26.5	155.3 \pm 25.5	154.5 \pm 27.0

RMTP: respiratory muscle training programme; DT: respiratory muscle detraining period. Variables: FVC – forced vital capacity; FEV₁ – forced expiratory volume in one second; PEFR – peak expiratory flow rate; MVV – maximum voluntary ventilation; MEF_{50%} - maximum instantaneous flow at 50% FVC; MIP – maximum inspiratory mouth pressure; MEP – maximum expiratory mouth pressure.

Table C: Mean \pm SD values of the number of shuttles achieved during the 20 m multistage shuttle run tests at different time points for the experimental (EXP) and control (CON) groups.

<i>Variable:</i>	<i>12 week RMTP</i>		<i>20 week detraining</i>	
	<i>Baseline</i> <i>(Pre-with RMT)</i>	<i>12 weeks</i> <i>(post-with RMT)</i>	<i>32 weeks</i> <i>(pre-without RMT)</i>	<i>44 weeks</i> <i>(post-without RMT)</i>
<i>BLEEP SHUTTLES</i>				
EXP	90 \pm 15	97 \pm 25	93 \pm 21	89 \pm 19
CON	102 \pm 6	114 \pm 8	108 \pm 4	102 \pm 10

RMTP: respiratory muscle training programme; RMT: respiratory muscle training