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THE EFFECT OF UPPER RESPIRATORY TRACT ILLNESS ON EXERCISE PERFORMANCE

D A VILJOEN

DECLARATION

I, Deon Andre Viljoen, hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise), and that neither the whole work, nor any part of it has been, is being, or is to be submitted for another degree in this or any other University.

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Portions of this work have already been presented at the SASMA (South African Sports Medicine Association) Congress in 1997 and the ACSM (American College of Sport Medicine) in 1998.

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ABSTRACT

Two studies were undertaken to investigate the relationship between upper respiratory tract infection (URTI) and exercise performance. The first study documented the incidence of URTI in an athletic population and the second study determined the effect of URTI on exercise performance during the recovery period. Endurance runners (n=29) were used for these studies and the athletes were monitored for 45 months. During this time the subjects ran an average of 40 kilometers/week.

In the first study, 22 reports of symptoms (n=22) of URTI occurred during the 45 month period. Of these, 10 subjects (n=10) were ill for less than 3 days. All 10 subjects reported their symptoms directly following an endurance event. The other 12 subjects (n=12) were ill for 5 days and longer and fulfilled the inclusion criteria for illness due to infection. The incidence of symptoms of URTI/1000 hours of training for the group of 29 runners was 1,26. The incidence of symptoms for the 10 athletes not fulfilling the inclusion criteria for illness was 0.58 and the incidence for the 12 athletes fulfilling the inclusion criteria was 0.69. The odds ratio for the athletes (n=22) for developing symptoms of URTI during a year is 1.03 compared to the odds ratio of 2.5 for the general population during the similar period. These results seem to indicate that 45% of athletes (n=10) who reported URTI symptoms directly following strenuous exercise do not have clinical infection. Furthermore, the study indicates that regular, moderate, endurance exercise may afford protection against URTI, when compared to the general population.

In the second study, 5 athletes (n=5) of the original 12 subjects with URTI complied with all the test protocols. On recruitment, baseline tests were done for muscle strength, muscle endurance, aerobic endurance and maximal effort to exhaustion. Following the development of the URTI, the above parameters were tested over six days on days 0, 2, 4 and 6. After regaining their pre-

illness fitness levels over a three month period, the subjects were detrained for comparative periods and the above tests repeated on similar days for comparative purposes.

Parameters for muscle strength and muscle endurance [Work (Joule), Power (Watt), Torque to mass (Nm/kg), and Total power (Watt)] appeared to be unaffected following periods of illness and following comparative periods of detraining.

Following detraining, respiratory oxygen absorption (VO_2) (Fully fit 4.28 ±1.98 ml/kg/min ; Detrained 6.14 ±1.03 ml/kg/min [p=0.03]), respiratory carbon dioxide expiry (VCO_2) (Fully fit 277.8 ±102.4 ml/kg/min; Detrained 402.8 ±100.1 ml/kg/min [p=0.04]) and metabolic units (METS) (Fully fit 1.22 ±0.55 METS; Detrained 1.74 ±0.27 METS [p=0.04]) were increased significantly at rest on day 2 and respiratory frequency (RF) (Fully fit 70.22 ±10.44 breaths/min; Detrained 51.04 ±7.83 breaths/min [p=0.03]) were significantly decreased and volume of air inspired and expired (V'e) (Fully fit 94.86 ±32.16 l/min; Detrained 130.4 ±23.9 l/min [p=0.05]) were increased significantly on day 0 at maximal performance. Following illness, VO_2 and METS were significantly increased on day 0 (VO_2 – Illness 43.06 ±5.52 ml/kg/min; Detrained 40.74 ±4.23 ml/kg/min [p=0.03]) (METS – Illness 12.30 ±1.57 METS; Detrained 11.64 ±1.22 METS [p=0.02]) and day 2 (VO_2 – Illness 41.48 ±4.61 ml/kg/min; Detrained 39.72 ±3.15 ml/kg/min [p=0.04]) (METS – Illness 11.82 ±1.24 METS; Detrained 11.36 ±0.88 METS [p=0.04]) after running for 40 minutes at 70% of maximal effort. Furthermore, at maximal effort following illness, VO_2 and METS were significantly decreased on day 0 (VO_2 – Illness 53.4 ±8.06 ml/kg/min; Detrained 58.7 ±7.45 ml/kg/min [p=0.01]) (METS – Illness 15.26 ±2.32 METS; Detrained 16.78 ±2.12 METS [p=0.01]) and VCO_2 and V'e were significantly decreased on day 0 (VCO_2 – Illness 3920 ±663 ml/kg/min; Detrained 4753 ±835 ml/kg/min [p=0.002]) (V'e – Illness 103.7 ±18.7 l/min; Detrained130.4 ±23.9 l/min [p=0.02]), day 2 (VCO_2 – Illness 4253 ±913 ml/kg/min; Detrained 4553 ±694 ml/kg/min [p=0.02]) (V'e – Illness 114.4 ±25.1 l/min; Detrained 125.6 ±21.8 l/min

[p=0.02]) and day 6 (VCO_2 – Illness 4566 ±734 ml/kg/min; Detrained 4689 ±1077 ml/kg/min [p=0.01]) (V'e – Illness 132.4 ±19.3 l/min; Detrained 135.9 ±22.4 l/min [p=0.003]).

The study indicates that URTI has a significant influence on parameters of respiratory function (increased VO_2 and METS) during submaximal endurance on the first two days only following the illness. However, URTI has a significant influence on parameters of respiratory function (decreased VO_2, METS, VCO_2 and V'e) at maximal exercise during the first six days following the illness. Furthermore, the significant effects on respiratory function are not due to detraining alone.

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GLOSSARY OF TERMS

Aerobic endurance	Physiological endurance parameters of respiration measured during prolonged submaximal exercise.
Borg RPE	A graded scale of perceived exertion measured from 6 to 22, where 6 represents no effort and 22 represents total exhaustion. The Borg Scale starts at six and ends at 22.
Cardiac output	Outflow of blood volume from the heart determined by the rate of pumping (heart rate) multiplied by the quantity of blood ejected from the heart with each stroke (stroke volume), measured in litres.
Diffusion capacity (DC)	The passive process when a gas flows from an area of high concentration to an area of low concentration. Measured by Breven 1962 as the diffusion capacity of carbon monoxide across the alveolar membrane, indicated in ml/min/mmHg/m2.
Functional residual capacity (FRC)	The volume of air retained in the lungs at the end of a normal, unforced expiration (litres).
Heart volume (HV)	Determined in prone position by 2-plane roentgenogram and measured in milliliters

METS	A measurement of work rate. One MET represents the resting oxygen consumption in $mlO_2/kg/min$. Work rate is indicated in multiples of resting oxygen consumption.
Skeletal muscle endurance	Physiological measurements of exhaustion measured on a muscle group during maximal contractions over a set time period against light resistance.
Residual volume	The volume of air retained in the lungs following a maximal expiration (litres).
Respiratory exchange ratio (RER)	The ratio of metabolic gas exchange, measured as the quantity of carbon monoxide produced in relation to the quantity of oxygen consumed during exercise. During exhaustive exercise the RER may rise above 1.00 due to an increase in the CO_2 production.
Respiratory quotient (RQ)	The ratio of metabolic gas exchange, measured as the quantity of carbon dioxide produced in relation to the quantity of the oxygen consumed (as in RER). This is a reflection of the oxydation of nutrients during a steady state of exercise.
Stroke volume	The quantity of blood ejected from the heart with each ventricular contraction (millilitres).
Total lung capacity (TC)	The total volume of air in the lungs measured by the sum of the vital capacity and the residual capacity (litres).

URTI	Upper respiratory tract illness.
Vital capacity (VC)	The total volume of air expelled with full, forced expiration following a maximal inspiration (litres).
VO_2 max and VO_2 peak	The maximal use of oxygen at the point of exhaustion measured in $mlO_2/kg/min$.

THE EFFECT OF UPPER RESPIRATORY TRACT ILLNESS ON EXERCISE PERFORMANCE

CHAPTER 1

INTRODUCTION AND SCOPE OF THE THESIS

One of the concerns all sports people have is the possibility of developing an illness. Achieving success in sport requires dedication and consistent training. This is especially true of endurance sports where the preparation for an event can involve many months of carefully calculated and scheduled training. The aim of training is to allow the athlete to reach his or her peak of ability on the day of the event and an untimely, forced break from training program may negatively affect optimum performance.

Upper respiratory tract illness (URTI) occurs more commonly than all other illnesses combined in the general population, and therefore has a frequent and often negative influence on an athlete's training or performance (Nieman 1994). However, there is conflicting anecdotal evidence concerning the incidence and relative risk of acquiring upper respiratory tract illness among sportspersons. Some athletes and coaches believe that consistent training affords protection against acquiring upper respiratory tract illness, while others claim that exercise training increases the risk of acquiring such illness.

When contracting an upper respiratory tract illness the athlete needs to stop training for the duration of the illness. In addition to this period of detraining the illness per se might have a debilitating effect on fitness levels. It is thus quite understandable that the individual will limit this period of inactivity, contrary to sound medical advice as well as any physiological impediment caused by the disease. Many athletes therefore return to training before regaining full health. The effects of upper respiratory tract illness on exercise performance in the recovery period have not as yet been well investigated. Most studies done on this subject investigated the effects illness have on athletes whilst they are symptomatic. The effects of the upper respiratory tract illness on exercise performance immediately following the illness are not known, nor is it clear how much of this effect is due to detraining.

This study documents the incidence of upper respiratory tract illness in a cohort of active runners over a period of 45 months. In those who developed upper respiratory tract illness further measurements were made in order to determine the relative contribution of the illness itself and a possible detraining effect to any post-illness performance deficit. Physiological parameters, exercise performance and muscle strength were measured in those athletes who developed upper respiratory tract illness once they were asymptomatic, and then again after a period of detraining of equal duration to their illness.

The results of these investigations are then examined in relation to the published literature on the incidence of upper respiratory tract illness and the effect these have on exercise performance. These findings serve to clarify an understanding of exercise as a risk factor for upper respiratory tract illness and also the effect of such an illness on exercise performance during the recovery period.

This information has important applications for the athlete who develops upper respiratory tract illness during training or prior to competition. The athlete requires guidance on the effects that the illness might have on his/her training schedule and performance. The clinician must be able to give advice, that is based on sound clinical evidence, on when an athlete could resume training and return to competition. Furthermore the medical practitioner or coach should be able to advise the athlete on how the period of detraining, in addition to being ill, could affect the exercise performance immediately following upper respiratory tract illness.

CHAPTER 2

LITERATURE REVIEW

2.1 UPPER RESPIRATORY TRACT ILLNESS

2.1.1 INTRODUCTION

The common cold is the most frequently occurring upper respiratory tract illness (URTI) in humans, accounting for 50% of absenteeism from work or school (Aquilina et al. 1980). Two thirds of respiratory tract illnesses are caused by viruses and most involve the upper respiratory tract alone. Spread of the illness readily occurs in the course of sporting activity, where transmission can take place in change rooms, hotels, living areas, eating and travelling together (Sharp 1989), and as such, this illness is a persistent threat to the training and performance of the athlete. Nevertheless, it is believed by many athletes and the general public that good physical conditioning helps prevent illness, and being deconditioned increases susceptibility to common ailments (Nash 1986; Simon 1987).

Upper respiratory tract illness (URTI) may impair physical performance due to a variety of reasons. It has been shown that acute febrile illness may adversely affect neuromuscular transmission (Friman et al. 1977a). Myocarditis may develop as a complication of acute infectious illness (Friman 1976) and in response to URTI, skeletal muscle can develop enzymatic and ultrastructural alterations which correspond to those found in other forms of muscular diseases (Astrom et al. 1976; Friman et al. 1991). A factor that can also influence physical performance during URTI is the effect of medication taken during febrile illnesses. It is documented that decreased physical performance may be prevented during URTI by symptomatic treatment. It is suggested that this apparent beneficial effect may be influenced by the degree of averse subjective impressions of a patient with respect to the severity of the illness. A feeling of well-being seem to prevail in athletes whose performance did not deteriorate appreciably. This result might have been due to the effect of aspirin and/or propoxyfene which was administered to

the athletes during the study. (Beisel et al. 1974).

2.1.2 DEFINITION, TERMINOLOGY AND CLASSIFICATION OF UPPER RESPIRATORY TRACT ILLNESS.

Upper respiratory tract illness consists of a number of acute infections that occur in the upper portion of the respiratory tract. The upper respiratory tract is defined as the nasal passages, naso pharynx, pharynx, larynx, trachea, and main bronchi. URTI is caused by viral, bacterial or fungal organisms that cause inflammation of the upper respiratory tract organs. The term "cold" is usually defined as "an acute illness that involves nasopharyngitis characterised by catarrh, little or no fever and insignificant systemic symptoms" (Heath et al. 1992). Viral infections can also result in widespread systemic inflammation including cardiac muscle. This could result in injury to the miocardium when stressed during exercise. If unchecked, some of these infections can easily progress to the bronchioli and lung tissue proper. The symptoms and signs of the illness are important clinical indicators to differentiate among the many causative agents (Heath et al. 1992).

2.1.3 MICROBIOLOGY OF UPPER RESPIRATORY TRACT ILLNESS.

Many organisms cause upper respiratory tract illness (Table 2.1). These infective agents will be discussed briefly.

2.1.3.1 Rhino Viruses

These viruses have more than 100 serotypes and are the major cause of URTI in adults. These infections are characterised by the common symptoms of nasal congestion, rhinorrhoea, pharyngeal irritation, sore throat, malaise and sometimes headache. Symptoms begin after an incubation time of 24 to 48 hours, are self limited and usually subside in less than a week. Viral shedding by nasal discharge begins before the onset of symptoms and may continue for 2 to 3 weeks thereafter.

Spread of the rhino virus is enhanced by crowded living conditions and extended periods of exposure.

The peak incidence of rhino viral infections occur during the autumn and spring and continues during the winter months (Casey et al. 1990).

Table 2.1 Microbial agents responsible for upper respiratory tract illness.

RNA Viruses	
	Rhino viruses
	Corona viruses
	Influenza viruses
	Para-influenza viruses
	Respiratory Syncytial virus
	Enteric RNA Viruses
DNA Viruses	
	Herpes Simplex Virus
	Adenoviruses
Mycoplasma pneumoniae	
Chlamydia Infections	
	C trachomatis
	C psittaci
Bacterial Infections	
	Streptococcus pyogenes
	Streptococcus pneumoniae
	Staphylococcus aureus
	Haemophilus influenza
Legionnairs Disease	
	Legionella pneumophila

(Casey et al. 1990)

2.1.3.2 Corona Viruses

The second most frequent cause of URTI amongst adults are Corona Viruses. Only two strains have been isolated in the laboratory. Although the incubation period is slightly longer and the period of illness is usually a bit shorter, the incidence and symptoms are similar to those experienced with Rhino Viruses (Casey et al. 1990).

2.1.3.3 Influenza Viruses

The next most frequent cause of URTI in adults are Influenza Viruses of which three types (A B and C) are recognised. Although these viruses have the same incubation period, symptoms and incidence as Rhino- and Corona viruses, they usually cause epidemics that result in severe complications like viral pneumonia or secondary bacterial infections resulting in pneumonia (Casey et al. 1990).

2.1.3.4 Para-influenza and Respiratory Syncytial Viruses

In infants and young children these viruses are important causes of severe respiratory illnesses. Since most adults have been infected with these viral serotypes during childhood, re-infection usually causes mild illness. The incubation, symptoms and incidence mimics those of Rhino- and Corona Viruses (Casey et al. 1990).

2.1.3.5 Enteric Viruses

Several of the enteric viruses, including the Echo and Coxsackie Viruses, may cause upper respiratory tract illness in infants, children and adolescents during the summer and autumn months. These do probably not cause acute adult respiratory illness (Casey et al. 1990).

2.1.3.6 Herpes Simplex Type I and II

Herpes simplex type I and II cause upper respiratory tract illness which is characterised by symptoms of mild cold followed by oropharyngeal ulcers, tonsillitis, pharyngeal inflammation and exudate, tender cervical and submandibular adenopathy and fever. The illness is self limited lasting 2 to 3 days and have no distinct seasonal peak (Casey et al. 1990).

2.1.3.7 Adenoviruses

Adenoviruses are not common agents for illness in the adult population and infections occur only sporadically during winter and early spring and are characterised by fever, pharyngitis, laryngitis, rhinorrhoea and hoarseness. The incubation period is 6 to 9 days and the disease has a clinical course of 1 to 2 weeks. Viral pneumonia is a common complication (Casey et al. 1990).

2.1.3.8 Mycoplasma pneumoniae

Respiratory infections caused by Mycoplasma organisms may be seen throughout the year. Incubation takes from 1 to 3 weeks and the illness is almost invariably self limited to 3 weeks or less. Symptoms of infection usually begin in the upper airway with pharyngitis and tracheitis being the most prominent. Malaise, mild fever and myalgia may be present and cough is the most common symptom. The disease can be complicated by pneumonia when the infection spreads to the bronchioles and lungs (Casey et al. 1990).

2.1.3.9 Chlamydiae

The Chlamydiae genus contains two species, C trachomatis and C psittaci. C trachomatis is known to cause pneumonia in infants and immunocompromised adults. C psittaci causes pneumonia in healthy adults but is almost universally contracted through exposure to infected birds.

Recently a new Chlamydia has been isolated and named strain TWAR and this may be an important cause of respiratory disease in healthy young persons. The new strain emerged from epidemics of mild pneumonia caused by C psittaci where the illness could not be linked to birds (Casey et al. 1990)

2.1.3.10 Bacterial Infections

Among these infections Streptococcus pyogenes remains an important cause of acute pharyngitis of which β-haemolytic Group A is the most important one. Streptococcal pharyngitis occurs most commonly during the winter months and has an incubation period of 2 to 4 days. This is followed by rapid onset of sore throat, fever, headache, myalgia and malaise.

Clinically the uvula, pharynx and tonsils are diffusely erythematous and oedematous and there is associated cervical adenopathy which may progress to suppurative tonsillitis. Complications known to be associated especially with Streptococcus Group A infections are Acute Rheumatic Fever and Glomerulonephritis.

Of the several bacterial pneumoniae only Streptococcus pneumonia will rarely produce a primary

illness in young adults. When bacterial pneumonia complicates influenza, Streptococcus pneumonia is the most frequent secondary invader followed by Staphylococcus aureus and Haemophilus influenza (Casey et al. 1990).

2.1.3.11 Legionnaires disease

Legionella pneumophila is a unique species in microbiology and not related to any other environmental or pathological organism. The organism is part of flora, naturally occurring in water and damp environment, and capable of surviving in extreme ranges of environmental conditions. Human infections have been associated with contaminated cooling towers, air conditioning systems, damp excavation sites and respiratory equipment. The incubation period is 2 to 14 days and the illness presents as acute bacterial pneumonia. Symptoms are malaise, fever, muscle aches, dyspnoea and headaches. Other symptoms may include confusion, ataxia, abdominal pain and diarrhoea. On examination pulmonary rhonchii are usually heard and a chest X-ray shows patchy consolidation (Strebel et al. 1988; Marik 1989).

2.1.3.12 Summary of microbiology of URTI

It is clear that many organisms can be the cause of URTI. This presents a challenge to the clinician in diagnosis and treatment. All of the organisms discussed have similar symptoms and signs of URTI. Some bacterial infections have unique additional clinical signs that assist in diagnosis, but the other organisms can only be identified serologically or by culture. Irrespective of the causative organism the illness results in a period of debilitation. At present it is not known what effect the illness and it's duration has on exercise performance.

2.1.4 PATHOGENESIS OF VIRAL UPPER RESPIRATORY TRACT ILLNESS.

Influenza can spread rapidly and should never be underestimated. It may have serious consequences for the athlete who continues training or competing. Attempting to "sweat it out" can be an extremely dangerous practise if the individual has systemic symptoms such as fever and tachicardia and may lead to permanent damage to the myocardium (Sharp 1989). The many causative agents, as discussed,

have different pathological effects on the tissues they invade.

Viruses cause an acute interstitial infiltration of lymphocytes, histiocytes and plasma cells resulting in widespread inflammatory response of the upper respiratory tract tissues. Bacteria tend to localise the inflammation to the infected area or tissue but can also cause widespread granuloma formation in cases of tuberculosis and other mycobacterium (Knight 1977; Breven 1962).

Viral upper respiratory tract illness is primarily an infection of the respiratory epithelium that is transmitted from one person to another by either inhalation of infective droplet nuclei or by ocular inoculation most commonly when the eye is rubbed by a hand carrying the virus. After nasal infection the virus multiplies rapidly and spreads to involve the entire nasopharyngeal and tracheobronchial tree. At first the mucosa becomes swollen and hyperaemic and loses its normal ciliary activity and is then followed by necrosis of the respiratory epithelium. Although the infection is largely confined to the respiratory tract, viral products can enter the circulation and cause viraemia affecting the heart, kidney and other extra pulmonary tissues. Recovery from uncomplicated disease is often complete after 5 to 7 days (Knight 1977).

Contrary to popular belief, exposure to a cold environment does not increase the risk of acquiring or aggravating an URTI (Simon 1987; Heath et al. 1992). Smoking and psychological stress have been shown to be risk factors for developing URTI (Nash 1986; Schouten et al. 1988; Heath et al. 1992).

2.1.5 EPIDEMIOLOGY OF URTI IN THE GENERAL POPULATION.

A number of different viruses and bacteria can cause upper respiratory tract illness, all of which have the ability to change their antigenic characteristics every few years. As a result they continue to challenge personal immunity, despite the availability of vaccines. In general, the number of infections acquired per year decrease with age. Infants and children have the highest incidence with 4 to 8 infections per year. This rate may even double when children are in day care or in nursery school. In school-aged children the incidence is 2 to 6 upper respiratory tract infections per year. Adults acquire 2

to 5 upper respiratory tract infections per year, with women and adults who live in households with children tending to suffer even more upper respiratory tract infections per year (Heath et al. 1992).

Table 2.2 Summary of the frequency of causative microbials in upper respiratory tract infections (Casey et al. 1990)

Causative agent	Frequency (%)
Unknown cause	40%
Rhino viruses	25%
Corona viruses	3%
Influenza viruses	4%
Para-influenza viruses	4%
Respiratory Syncytial virus	4%
Enteric viruses	1%
Herpes Simplex virus	2%
Adeno viruses	1%
Mycoplasma	3%
Chlamydiae	3%
Streptococcus pyogenes	6%
Bacterial pneumoniae	4%
	100%

Although the incidence of URTI is highest during the winter months, evidence suggests that it is not low temperatures per se that are responsible (Simon 1987; Schouten et al. 1988; Heath et al. 1992). The environmental conditions during winter lead to overcrowding, which increases the exposure of persons to etiologic agents (Brenner et al. 1994).

Transmission of both rhino- and corona viruses appear to result mainly from inhaling infected droplets. Another method is self-inoculation of nasal mucosa and conjunctivae by fingers that have been in contact with the virus (Brenner et al. 1994; Heath et al. 1992). In addition, strenuous exercise may decrease local nasal mucosa antibodies and thereby reduce resistance to respiratory infections (Aquilina et al. 1980).

2.1.6 CLINICAL PRESENTATIONS AND COMPLICATIONS OF UPPER RESPIRATORY TRACT ILLNESS.

Acute upper respiratory tract illness is an acute febrile illness lasting about one week and characterised by fever, cough, hoarseness and sore throat. Pharyngitis is the most prominent localised manifestation of the disease and reaches maximum severity after about three days. The illness is accompanied by general malaise and headache. Nasal congestion and obstruction occurs commonly and cough and hoarseness of the voice is frequent. Diffuse myalgia occurs in almost half the cases and this is particularly marked in the legs and over the lumbosacral area. A rise in temperature occurs which is accompanied by tachycardia and can last for up to seven days (Knight 1977).

The main complication of upper respiratory tract illness is pneumonia. This occurs as either primary pneumonia due to the virus infection per se, or secondary pneumonia due to bacteria which cause infections on the lesions produced by the virus. Other complications are bacterial infections of the paranasal sinuses or middle ear. Rarely viral upper respiratory tract illness can be fatal in individuals with pre-existing pulmonary or cardiac disease, regardless of age (Knight 1977).

Sudden death of athletes during participation in sport has been linked to viral myocarditis caused by febrile illness such as upper respiratory tract illness. In these cases, cardiac abnormalities at autopsy are focal microscopic myocyte necrosis and lymphocyte infiltrate. It is speculated that acute exercise in the face of myocarditis triggers an arrhythmia which can be fatal (Eichner 1993).

2.1.7 MANAGEMENT OF UPPER RESPIRATORY TRACT ILLNESS.

The severity of upper respiratory tract illnesses in practise can be a mild, uncomplicated, symptomatic, common cold or be more debilitating influenza. Influenza can be complicated by primary or secondary infections of related organs leading to otitis media, sinusitis, bronchitis or pneumonia (Knight 1977). In general, the initial treatment of URTI is focussed on symptomatic relief. If URTI's are prolonged, severe or complicated they are best treated with bed rest combined with the appropriate antibiotic.

Physiotherapeutic measures can be employed to assist in clearing chest and sinus infections (Beisel et al. 1974).

The sports physician is often faced with the decision whether athletes should be allowed to exercise when they have an upper respiratory tract illness. The accepted recommendation is that if the athlete has symptoms of a common cold with no constitutional involvement, regular training may be safely resumed a few days after resolution of symptoms (Simon 1987). However, if there are symptoms or signs of systemic involvement such as fever, fatigue, muscle aches or lymphadenopathy, then 2 to 4 weeks should be allowed before resumption of more intense training. These precautions are advised because of the potential relationship between intense exercise and the risk of developing viral cardiomyopathy and other severe forms of viral infection (Heath et al. 1992).

Eichner (1993) describes a simple protocol (the 'neck check') for determining whether exercise is appropriate or not under conditions of URTI. If all symptoms were ' above the neck' (runny or blocked nose, sore throat), the athlete could commence an exercise routine at below normal intensity for about 10 minutes. If such activity makes them feel worse, they should stop exercising but if they feel better they may continue the training session. If symptoms were ' below the neck' however (muscle aches, cough, vomiting, diarrhoea or fever), training should be discontinued.

2.1.8. PREVENTION OF UPPER RESPIRATORY TRACT ILLNESS.

Although clinical experience and epidemiological data seem to indicate that there is increased risk of upper respiratory tract illness in some athletes (Eichner 1993), the balance of evidence favours the view that psychosocial variables play an important role in effecting immunological competence (Heath et al. 1992). This together with the physiological stress from heavy endurance exercise may lead to suppression of the immune system (Heath et al. 1992; Eichner 1993).

Precautions that can help decrease the risk of illness include the spacing of high intensity acute exercise bouts and race events as far apart as possible, eating a well balanced diet, keeping other life

stresses to a minimum, avoiding overtraining and chronic fatigue and obtaining adequate sleep.

Table 2.3 Precautions to decrease the risk of acquiring URTI in athletes (Heath et al. 1992).

Space strenuous exercise bouts far apart
Eat well balanced diet
Stress management
Avoid overtraining
Avoid chronic fatigue
Adequate sleep pattern
Avoid contact with people that have URTI
Isolate team members that have URTI
Wash hands regularly
Avoid rubbing of eyes and nose
Inoculate with flu vaccine

To minimise exposure to infection athletes should avoid contact with individuals who have symptoms or signs of an infectious illness. A team member who develops symptoms should be isolated from the other athletes as soon as is practicable. The hands should be washed regularly and rubbing of the eyes and nose should be avoided since viruses are often transmitted by hand contact. Inoculation of viruses endemic to the area where the athlete will compete may also be helpful (Brenner et al. 1994).

2.1.9 SUMMARY AND CONCLUSIONS.

It is well known that upper respiratory tract illnesses are widespread and common in the normal population. There is evidence that athletes following vigorous training schedules may be more susceptible to URTI. This observation is probably due to an increase in psychological and physiological stress that may result in a suppression of the immune system. The many causative agents are mainly viral and this can occasionally be complicated by secondary bacterial infections. The dangerous complications that can manifest when athletes exercise whilst suffering from upper respiratory tract illness are well documented. However, the effect that upper respiratory tract illness has on exercise

performance during the recovery period is not known.

There are general guidelines for the athlete to avoid acquiring upper respiratory tract illness. Clinical symptoms and signs can be used to assess the severity of the URTI and these parameters can be used to advise athletes on whether or not to continue or discontinue exercise training. These parameters are however based on common sense rather than sound scientific observations.

It is clear that there is a definite need for more information on the effect that upper respiratory tract illness has on exercise performance. The sports physician should then be able to advise the athlete on possible decreases in exercise performance during the recovery from URTI.

2.2 THE IMMUNE SYSTEM

The immune system comprises naturally occurring as well as acquired components that function in a co-ordinated manner. The main functions are to protect against, recognise, attack and destroy foreign infectious agents. The naturally occurring factors serve as a first line of defence when the body encounters an infectious agent. Failing this, the acquired factors are activated to combat the ensuing infection. This system aids the body to recover from the infection and also develops a memory for the infectious agent (Brenner et al. 1994; Nieman et al. 1991; Nash 1986).

This review will outline the components and functions of the immune system and indicate the process of the immune and inflammatory response. This information will then be applied to illustrate how an acute exercise bout affects the immune parameters and functions.

2.2.1. THE FUNCTION OF THE IMMUNE SYSTEM

The immune response has both cellular and humoral (antibody mediated) factors. The cellular components are leukocytes that comprise granulocytes, lymphocytes and monocytes. Granulocytes account for 60 to 70% of the circulating leukocytes and consist of neutrophils, eosinophils and

basophils. Lymphocytes account for 20 to 25% of the white cells and consist of T-cells, B-cells and natural killer cells. In this group the T-cells are classified as either T-helper cells or T-suppressor cells. Macrophages are monocytes that enlarge in response to ingestion of foreign material with resultant increase in synthesis of lysosomal enzymes. These macrophages have the function of phagocytosis of foreign components and they contribute to the rest of the cellular response (Figure 2.1) (Nieman et al. 1991).

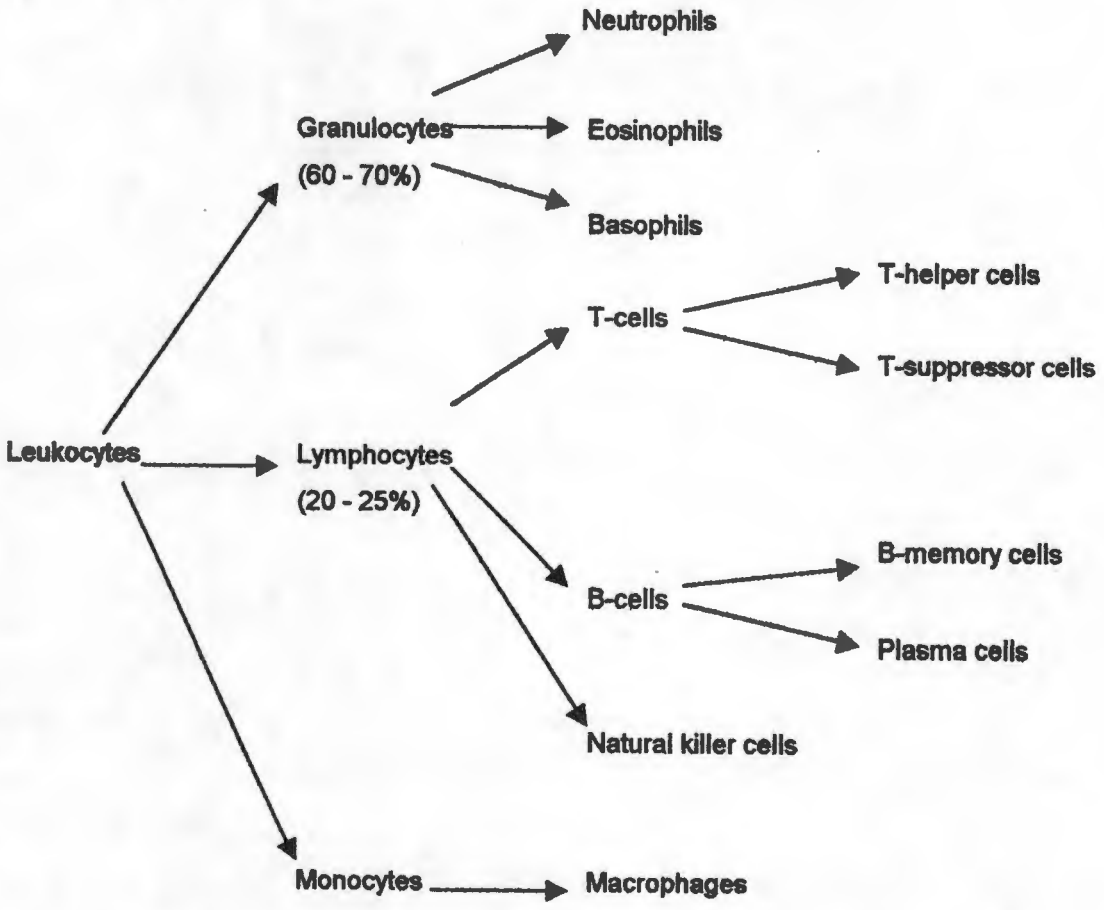

Figure 2.1 **Cellular components of the immune response (Nieman et al. 1991)**

T-helper cells recognise antigen presenting or virally infected cells and release cytokines. These are proteins involved in the communication between and activation of different immune cells and include interleukins, interferons, tumour necrosis factors and colony stimulating factors, each with diverse functions. (Nieman et al. 1991; Brenner et al. 1994). One of the main functions of these proteins is to attract and activate the host macrophages, which together with monocytes, neutrophils and natural killer cells are responsible for the phagocytic function of the immune system. T cells also play a crucial

role in modulating and controlling the differentiation of immunoglobulin-bearing B cells to immunoglobulin-secreting plasma cells in response to antigen invasion, with T helper cells aiding and T suppressor cells inhibiting the process.

Immunoglobulins are components of the humoral response and are produced by the B-cells and plasma cells. Five immunoglobulins are recognised: IgG, IgA, IgM,IgD and IgE. The immunoglobulins are known as antibodies and they bind with specific antigens which characterise foreign micro-organisms. The B-lymphocytes carry the IgG, IgA, IgM and IgE on their surface membrane. When the immunoglobulins on a B-cell interacts with antigen, the B-cell differentiates into a plasma cell. The plasma cells are short lived cells which actively secrete immunoglobulin antibodies. These antibodies are specific to the class of antigen they bind with. The antibodies protect the host by binding with the surface antigens and agglutinising the micro-organisms. This in turn facilitates phagocytosis, activates the complement system, produces opsonins and neutralises bacterial toxins (Brenner et al. 1994). The T-cells have receptors for IgD which are expressed when exposed to antigen and which is associated with augmented antibody production (Swenson et al. 1996; Swenson et al. 1998).

The complement system consists of more than 20 proteins, some of which are proteolytic enzymes. These proteins bind with antibodies and are responsible for stimulating phagocytosis, stimulating antigen presentation and stimulating the killing of infected cells. IgM is especially effective in activating complement to produce immune lysis of foreign cells (Irvine 1974).

It is clear that the body has an effective mechanism to defend against foreign organisms. Infection initiates an immediate immune response which destroys the organisms with enzymes released from host cells and engulfment by host cells.

2.2.2 IMMUNITY AND THE INFLAMMATORY RESPONSE

The immediate response of the body to infection is acute inflammation. In the first instance blood flow is increased to the infected area and vascular permeability is enhanced to allow the entry of leukocytes

and plasma proteins to the infected area (Brenner et al. 1994). This inflammatory reaction also serves to mobilize nutrients for the increased needs of the immune system (Friman et al. 1998).

The immune response is initiated when a macrophage engulfs a foreign organism and then processes and incorporates the foreign proteins into its own cell surface. The foreign proteins are antigens and the macrophage is known as an antigen presenting cell. This antigen presenting cell comes into contact with B-cells and T-cells (Brenner et al. 1994; Austen 1977).

The B-cells produce immunoglobulin antibodies that bind with the antigen to form free immune complexes that are rapidly removed from the circulation by phagocytosis. The antibodies also bind with tissue and activate complement which is responsible for triggering phagocytosis. The B-cells differentiate into either plasma cells that secrete large amounts of the specific immunoglobulin, or into resting B-memory cells. The B-cells rapidly transform into plasma cells following a second exposure to the antigen. Resistance to the infection is thus in part due to the presence of adequate levels of antigen specific antibodies (Nieman et al. 1991; Austen 1977).

T-cells are also activated by the presenting antigen and they serve to modulate the development of immunoglobulin secreting plasma cells from B-cells, with T-helper cells aiding and T-suppressor cells inhibiting the process. The activated T-helper cells produce soluble factors (cytokines) that bind to the specific receptors in the activated B-cells to give signals for growth and differentiation. These soluble factors are interleukin, interferon, B-cell growth factors and B-cell differentiation factors (Nieman et al 1991). Different components of the immune response react to different types of infectious agents. Bacteria and fungi are removed through phagocytosis by macrophages and neutrophils, eosinophils are responsible for the elimination of parasites and T-cells are prominent in the defence against viruses (Brenner et al 1994).

2.2.3 THE EFFECTS OF EXERCISE ON THE IMMUNE RESPONSE

Numerous studies (listed in Table 2.4) have documented the effects that exercise has on the immune

system. There is evidence that prolonged submaximal exercise might stimulate the immunity and thus have a protective effect, whereas acute and high intensity bouts might suppress the immunity and have a negative effect. In studies describing the relationship between an acute bout of exercise and the immune system, there has been considerable variations of exercise intensities. For the purpose of this dissertation the exercise intensities have been graded as mild, moderate or strenuous. Table 2.4 summarises the type of physical activity used by various researchers to achieve mild, moderate or strenuous effort.

Table 2.4 Summary of physical activities used to achieve different exercise intensities by researchers examining the effect of exercise on immune system function.

Authors	Levels of Exertion		
	Mild	Moderate	Strenuous
Poortmans 1970			Graded maximal treadmill test
Eberhardt 1971		20min. cycle @ 130kgm/min	
Eskola et al. 1979		Trained runners running marathon @ submaximal pace	Elite runners running marathon @ maximal pace
Hanson et al. 1981		13km running @ 72% VO2 max	
Stephenson et al. 1985			Graded maximal treadmill test
Mac Kinnon et al. 1987		2 hours cycling @ 70 to 75% VO2 max	
Lewicki et al. 1987			Graded maximal cycle test
Schoutes 1988			Graded maximal treadmill test
Nieman et al. 1989			Graded maximal treadmill test
Tvede et al 1989		1 hour cycle @ 80% VO2 max	
Pedersen et al. 1990		60 minutes Cycling @ 75% VO2 Max	
Smith et al. 1990	1 hour cycle @ 60% VO2 Max		
Nieman et al. 1990	45min. walking @ 60% VO2 Max 5x per week		More than 97km running per week
Rickan et al. 1990		60min. Running @ 63% VO2 max	
Hansen et al. 1991			Running 1,7 to 10,5 km at close to maximum speed
Nieman et al. 1991	45min. walking @ 60% VO2 Max 5x per week	Running less than 40km @ 70% VO2 Max	Running 45 to 75km
Nieman et al. 1991		3 hours running @ marathon pace	
Lee et al 1992	Basic army cadet orienteering	Basic army cadet training -32% increase in rate of perceived exertion	
Eichner 1993		Running less than 20 miles per week	Running more than 60 miles per week
Gabriel et al. 1997		<2 hours @ 85% of Individual Anaerobic Threshold (IAT)	Exhaustive exercise @ 100% IAT or above
Mackinnon et al. 1998	Running a single bout of 40 to 90 minutes @ 55% to 75% of VO2 max.	Running for 90 min @ 75% VO2 max on three consecutive days.	

Based on the information presented in Table 2.4 mild exercise can be defined as training at 60% of VO_2 max, moderate exercise as running less than 40km in one bout at 75% VO_2 max and strenuous exercise when running more than 45km in one bout or completing a graded maximal exercise test (Table 2.5).

Table 2.5 Stratification of exercise intensity based on information detailed in Table 2.4 above

	Level of exertion		
	Mild	**Moderate**	**Strenuous**
Exercise type	Training at 60% of VO_2 max	Running less than 40km in one bout at 75% VO_2 max	Running more than 40km in one bout Or a graded maximal exercise test

Studies have been conducted to determine the effect of various intensities of exercise on the immune parameters. Results of some of these studies during mild, moderate and strenuous exercise are summarised in Tables 2.6, 2.7, 2.8, 2.9 and 2.10. Most studies conclude that all intensities of exercise cause an increase in the leukocyte count due to specific increases in natural killer cells, neutrophils and monocytes. There is a concomitant decrease though in the T and B lymphocytes with all intensities of exercise.

Studies on the effect of exercise on humoral parameters have produced conflicting results (Nieman et al. 1991; Nieman 1997a). It has been suggested that the changes in plasma volume due to exercise is responsible for the apparent increases in soluble factors. Following maximal exercise, plasma volume reductions of 12% - 16% has been reported (Nieman et al. 1989; Stephenson et al. 1985). These plasma volume changes could explain the 8% - 18.5% increases in immunoglobulin and complement reported by Nieman et al. (1989) immediately following graded maximal exercise. Conversely, it has been argued that exercise has no effect on the humoral factors (Brenner et al. 1994; Lewicki et al. 1987; Fitzgerald 1988).

Tables 2.6, 2.7 and 2.8 list the findings of various authors on the effects of mild, moderate and strenuous exercise on cellular and humoral immune factors.

Table 2.6 The effect of mild exercise on cellular and humoral immune factors.

Mild Exercise										
Author	**Exercise type and Intensity**	**Immune Parameters**								
		Cellular					**Humoral**			
		T-lymph Func	B-lymph Func	NKC Act	Neut Func	Mono Count	IgG	IgA	IgM	Comp
Lee et al. 1992	Basic army cadet orienteering	↓								
Smith et al. 1990	1 hour cycle @ 60%VO2 Max	N			↑↓					
Nieman et al. 1990	45min. walking @ 60% VO2 Max 5x per week			↓			↑	↑	↑	
Mackinnon et al. 1994	Jogging for 40 min @ 55% to 75% of VO_2 max							N		

Abbreviations: T-lymph func =T-lymphocyte function, B-lymph func =B-lymphocyte function, NKC act=Natural Killer Cell activity, Neut func =Neutrophil function, Mono count =Monocyte count, IgG=Immunoglobulin G, IgA=Immunoglobulin A, IgM=Immunoglobulin M, Comp=Complement, ↓=decrease, ↑=increase, N = no significant increase or decrease.

Table 2.7 The effect of moderate exercise on cellular and humoral immune factors.

Moderate Exercise										
Author	**Exercise Type and Intensity**	**Immune Parameters**								
		Cellular					**Humoral**			
		T-lymph Func	**B-lymph Func**	**NKC Act**	**Neut Func**	**Mono Count**	**IgG**	**IgA**	**IgM**	**Comp**
Gabriel et al. 1997	Exercise <2 hours at 85% of individual anaerobic threshold					↑				
Nieman 1997a	Long term exercise training			↑						
Lee et al. 1992	Basic army cadet trainng- 32% increase in rate of perceived exertion	↓								
Ricken et al. 1990	60min running @ 63% VO_2 Max	↓	N							
Pederson et al. 1990	60min cycling @ 70% VO_2 Max			↑	↓	↑				
Tvede et al. 1989	60min cycling @ 80% VO_2 Max	↓	↓							
MacKinnen et al. 1987	120min cycling @ 70-75% VO_2 Max						N	N	N	
Hanson et al. 1981	13km running @ 72% VO_2 Max						N	N	N	
Eskola et al. 1978	Trained runners running marathon @ submaximal pace	N	N							
Eberhardt 1971	20min cycling @ 130kgm/min						N	N	N	

Abbreviations: T-lymph func =T-lymphocyte function, B-lymph func =B-lymphocyte function, NKC act=Natural Killer Cell activity, Neut func =Neutrophil function, Mono count=Monocyte count, IgG=Immunoglobulin G, IgA=Immunoglobulin A, IgM=Immunoglobulin M, Comp=Complement, ↓=decrease, ↑=increase, N = no significant increase or decrease.

Table 2.8 **The effect of strenuous exercise on cellular and humoral immune factors.**

Strenuous Exercise										
Author	Exercise type and intensity	Immune Parameters								
		Cellular					Humoral			
		T-lymph Func	B-lymph Func	NKC Act	Neu Func	Mono Count	IgG	IgA	IgM	Comp
Shephard et al. 1990	Athletes during periods of heavy training							↓		
Pyne et al. 1995	Elite swimmers during Training						↓		↓	
Nieman 1997a	Periods of heavy training				↓					
Nieman 1997b	Heavy exertion	↓	↓	↓	↓					
Gabriel et al. 1997	Exhaustive exercise at 100% of individual anaerobic threshold				↓					
Hansen et al. 1991	Running 1,7 to 10,5 km at close to maximum speed	↓	↓							
Nieman et al. 1990	More than 97km running per week			↑						
Nieman et al. 1989	Graded maximal treadmill test						↓	↓	N	↓
Schouten et al. 1988	Graded maximal treadmill test							↑↓		
Lowicky et al. 1987	Graded maximal cycle test				↓	↑				
Stephenson et al. 1985	Graded maximal treadmill test						↑	N	N	
Eskola et al. 1978	Elite runners running marathon @ maximal pace	↓	↓							
Poortmans 1970	Graded maximal treadmill test						↑	↑		

Abbreviations: T-lymph func =T-lymphocyte function, B-lymph func =B-lymphocyte function, NKC act=Natural Killer Cell activity, Neut function =Neutrophil function, Mono count=Monocyte count, IgG=Immunoglobulin G, IgA=Immunoglobulin A, IgM=Immunoglobulin M, Comp=Complement, ↓=decrease, ↑=increase, N = no significant increase or decrease.

Table 2.9 Summary of immune responses during mild, moderate and strenuous exercise.

Immune Response	Exercise intensity		
	Mild	Moderate	Strenuous
Cellular			
Leukocyte count	↑	↑	↑
Lymphocyte function	↓	↓	↓
Natural killer cell activity	↑	↑	↑
Neutrophil function	↓	↓	↓
Monocyte count		↑	↑
Humoral			
Immunoglobulin	↑↓	↑↓	↑N
Complement		↓	

Abbreviations: ↓=decrease, ↑=increase, N = no significant increase or decrease.

Strenuous exercise comprises long endurance exercise and acute bouts of high intensity exercise. As indicated in Table 2.5, most authors accept long endurance exercise as running more than 40km (Nieman et al. 1991; Nieman et al 1990) and acute exercise as a graded maximal exercise test. The graded maximal exercise tests are commonly performed on a treadmill where the subjects are exercised to exhaustion (Nieman et al. 1989b; Schouten et al. 1988; Stephenson et al. 1985; Poortmans 1970).

Changes in the cellular components during exercise have been associated with increases in serum cortisol concentrations and serum epinephrine concentrations that take place in response to an acute

exercise bout (Eichner 1993). The cortisol and epinephrine blood concentrations increase when exercise intensity rises above 60% of maximal performance. Epinephrine has an effect within the first hour of exercise and cortisol has an effect after 2 to 3 hours of exercise. During exercise, leukocytes are mobilised from the lungs, liver, spleen and muscle due to the increase in cardiac output and the rise in the cortisol and epinephrine levels. Cortisol and epinephrine inhibit lymphocyte entry into the bloodstream and increases lymphocyte migration into the lymphoid tissue which results in lymphopaenia (Eichner 1993). The mobilisation of neutrophils and monocytes result in the leukocytosis and the total white cell count does not reflect the associated lymphopaenia. Natural killer cells have a high density of beta-adrenergic receptors and the natural killer cell count thus increases during exercise in response to the increase in serum epinephrine concentration (Eichner 1993).

Where the above represents the cellular immune response during exercise, most studies show that following exercise activities, cellular functions are suppressed (Table 2.10).

Table 2.10 Summary of immune responses following prolonged endurance and acute strenuous exercise.

Immune Response	Exercise intensity	
	Long endurance	Acute strenuous
NKC count	↓	↓
NKC function	↓	↓
Neutrophil function	↓	↓↑
T-lymphocyte count	↓	↓
T-helper cells	↓	↓
T-suppressor cells	↑	↑
Macrophage function	↓	↑

Abbreviations: ↓=decrease, ↑=increase, NKC=natural killer cell.

Natural killer cell count and activity is suppressed within 1 to 2 hours following exercise and returns to baseline within 6 to 24 hours (Fitzgerald 1988; Pederson et al. 1990; Eichner 1993; Nieman 1997b). Monocytes release prostaglandins during exercise and it is thought that these prostaglandins suppress natural killer cell activity after exercise. It has been demonstrated that natural killer cell activity increases after exercise if the prostaglandin production is inhibited by ingestion of indomethacin (Pederson et al. 1990).

It has been shown that neutrophil phagocytic activity is inhibited following long term endurance exercise and acute strenuous exercise (Lewicki et al. 1987; Gabriel et al. 1997; Bury et al. 1998). Contrasting evidence demonstrated increased neutrophil microcidal activity after acute strenuous exercise but inhibited activity after long endurance exercise. Conversely macrophagic phagocytic activities were inhibited both following acute strenuous and long endurance exercise (Smith et al. 1990).

Studies on T-lymphocytes also indicated a decrease after acute strenuous and long endurance exercise with a return to baseline levels after 24 hours (Nieman 1997b). Observations on the T-lymphocyte fractions though indicated that the T-helper fractions decrease but the T-suppressor fractions show an increase following exercise (Fitzgerald 1988; Ricken et al. 1990). It has been suggested that a reduction in the helper to suppressor cell ratio below 1.0 to 1.5 might indicate immunosuppression and could be used to identify athletes with an increased susceptibility to infection (Nash 1986; Brenner et al. 1994).

All of the above have the effect of suppressing the immune response by impairing phagocytosis, antibody formation, migration of cells to infection site and the ability to kill microbial particles. The transient suppression of cell - mediated immunity following exercise might be sufficient to allow micro-organisms time to evade early immunological recognition and thus establish infection. These results might explain the anecdotal observation by athletes and coaches that acute strenuous and prolonged endurance exercise, appear to cause an increased risk of upper respiratory tract illness.

2.3 EXERCISE AND THE RISK OF DEVELOPING UPPER RESPIRATORY TRACT ILLNESS.

Physical exercise has an influence on the immune system and changes due to exercise can be induced in both the naturally occurring as well as the acquired immune processes (Brenner et al.1994). Immune system function can also be an important determinant of the outcome of exercise performance following upper respiratory tract illness.

There is anecdotal evidence that upper respiratory tract illness occurs less commonly in well trained athletes than in the general population (Eichner 1993; Nieman et al. 1998). Many athletes believe that exercise improves their resistance to infectious disease (Nieman et al. 1989a; Heath et al. 1992; Nash 1986). In contrast there are reports of an increase in upper respiratory tract symptoms following ultra marathon participation and following intensive training (Brenner et al. 1994; Heath et al. 1992). Many coaches and athletes believe that strenuous exercise during training or competition may cause the athletes to be unusually prone to illness, especially upper respiratory tract illness (Nash 1986).

2.3.1 THE RISK OF UPPER RESPIRATORY TRACT ILLNESS FOLLOWING AN ACUTE EXERCISE BOUT.

Mechanical effects of exercise that could result in exposure to infectious micro-organisms include mouth-breathing and drying of the mucosa, slowing of ciliary action due to thickening of the bronchial mucosa by inspiration of cold dry air thus slowing clearance of micro-organisms. During contact sports damage of the skin could occur through trauma which could lead to invasion by micro-organisms (Brenner et al. 1994; Heath et al. 1992; Aquilina et al. 1980; Schouten et al. 1988). In interviewing marathon runners after a 56km race in the two weeks immediately following the race it was reported that 33.3% of the runners developed symptoms such as runny nose, cough or sore throat, compared with 15,3% of non-runners. The faster runners also seemed the most likely to report such complaints (Brenner et al. 1994).

In comparison athletes training and participating in shorter distance events did not however seem to

alter the likelihood of respiratory complaints. The incidence of respiratory symptoms in 273 runners was similar before and after groups participating in short road races of 5, 10 and 21km (Nieman et al. 1989a). This would further support the finding that running less than 40km rates as moderate exercise and does not necessarily contribute to increased upper respiratory illness.

Pederson et al. (1994) suggests an "open window" for increased risk of URTI, in the first few hours following strenuous exertion. During this period there is a transient immunosuppression due to decreases in the lymphocyte and natural killer cell counts (Table 2.10). It has been reported that these cells remain suppressed for 6 to 24 hours following strenuous exercise (Fitzgerald 1988; Pederson et al. 1990; Eichner 1993). During this period the athlete is most vulnerable to URT infection.

Nieman 1994 proposes a "J" shaped curve when comparing moderate, regular exercise and strenuous exercise to sedentary controls with the risk of acquiring URTI (Figure 2.2). The curve indicates that regular moderate exercise decreases the risk for contracting URTI and strenuous exercise increases this risk.

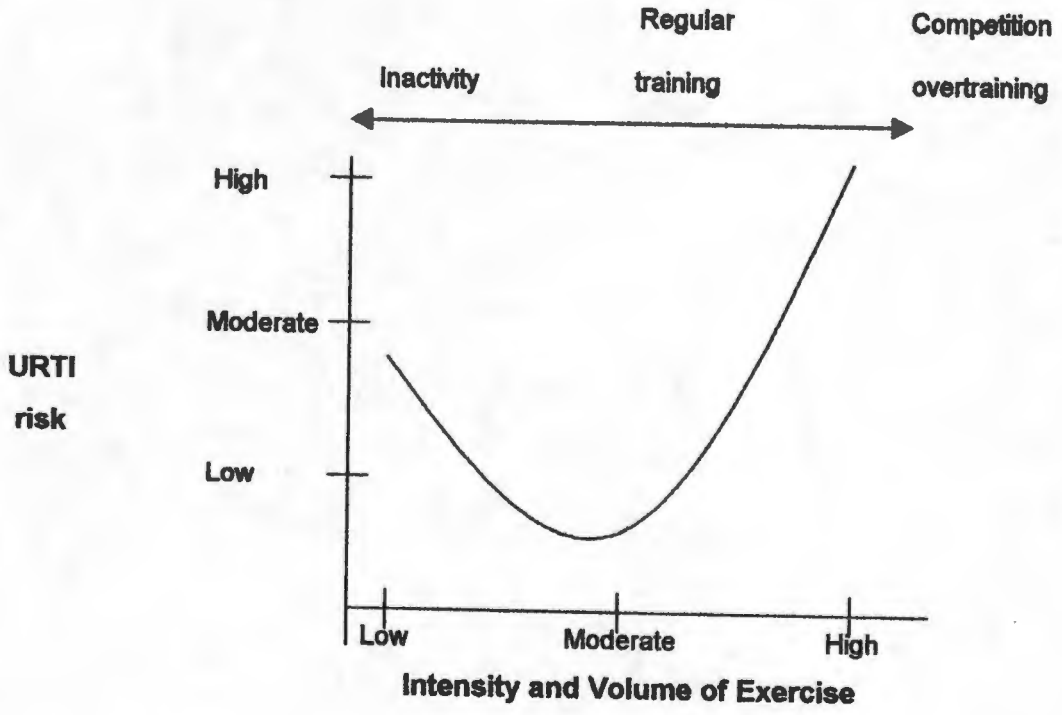

Figure 2.2 "J" shaped model of relationship between ranging amounts of exercise and risk of URTI (Nieman 1994).

In a study by Nieman et al. (1989a) runners were analysed according to average miles per week in training (those training more than 15 miles per week and those training less than 15 miles per week). The study compared the incidence of upper respiratory infectious episodes in 273 runners during a 2 month training period prior to a 5km, 10km and half marathon race. Furthermore the incidence on infectious illness following the races were studied. 25% of runners training more than 15 miles per week reported one infectious episode compared to 34.3% of runners training less than 15 miles per week. Only 6.8% of runners preparing for the half marathon reported sick with flu compared to 17.9% of the 5km and 10km runners. During the week following the race runners did not report an increase in infectious episodes as compared to the week prior to the race. These findings suggest that runners exercising at high intensity are less at risk for acquiring infectious illness compared to recreational runners. Furthermore, that the act of participating in a race does not appear to be associated with an increase in risk of acquiring upper respiratory tract illness.

Table 2.11 Summary of studies on the risk of illness and upper respiratory tract illness

Exercise and the risk of upper respiratory illness				
Author	**Exercise**	**Subjects**	**Study period**	**Findings**
Increased risk				
Douglas et al. 1978	Conditioned athletes with high VO_2 Max	61 100%M	9 weeks	↑ incidence *vs* controls
Nieman et al. 1989a	Running < 15km/week	137 66%M 33%F	8 weeks	↑ incidence *vs* controls
Heath et al. 1991	Running > 486miles/week	530 82%M 18%F	48 weeks	↑ incidence vs general population
Risk unchanged				
Osterback et al. 1987	Various 3 to 4 x / week	137 55%M 45%F	48 weeks	No difference to controls
Schouten et al. 1988	Various @ >700mets/week	199 46%M 54%F	192 weeks (4 years)	No difference to controls
Decreased risk				
Nieman et al. 1989a	Running > 15km/week	136 66%M 33%F	8 weeks	↓ incidence *vs* controls
Nieman et al. 1990	Walking 45min 5x/ week @ 60%max HR	36 100%F	15 weeks	↓ URTI symptomatology
Heath et al. 1991	Running ave. 4,7km/day	530 82%M 18%F	48 weeks	1,2 events/month *vs* 2,3 events/month in general population

Abbreviations: M=male, F=female, <=less than, >=more than, ↑=increase, ↓=decrease, HR=heart rate, min=minutes, km=kilometres, URTI=upper respiratory tract illness.

In contrast Douglas et al. (1978) compared 61 conditioned athletes (VO_2 max 55-76ml/kg/min) to 126 unconditioned cadets (VO_2 max 30-54ml/kg/min) as to the frequency and severity of upper respiratory tract illness over a 9 week period. This study found that the high exercise group experienced upper respiratory tract illness more frequently than the control group.

Osterback et al. (1987) studied the incidence of upper respiratory illness over a 12 month period in children (mean age 12,7 years), who actively participated in gymnastics, swimming and ice hockey. No differences in the incidence of URTI were found between the sports and control groups. They concluded that sports participation does not seem to have a preventative effect on the occurrence of upper respiratory tract illness in children. The Amsterdam Growth and Health Study in 1988 confirmed these results as reported by Schouten et al. (1988). The exercising group was classed as expending more than 700 mets/week. The study found that an increased level of habitual physical activity in a young and normal population does not show lower incidence and shorter duration of upper respiratory tract symptoms.

Two further studies indicated a positive effect of exercise on upper respiratory illness. Nieman et al. (1990) trained 36 mildly obese and sedentary women over a 15 week period by walking for 45 minutes at 60% of maximum heart rate five times a week. Compared to a control group the exercised group reported significantly fewer days with upper respiratory tract illness symptoms during the 15 week period.

In the Heath et al. (1991) study on a group of 530 runners over a period of one year the incidence of upper respiratory tract illness was investigated and the total annual mileage was related to the incidence of upper respiratory tract illness. The subjects ran an average of 4.7 miles per day, but a summary of the total mileage done in the year showed an increased odds ratio (the predicted number of bouts of upper respiratory tract illness for the year) for contracting upper respiratory tract illness with an increase in the total mileage (Table 2.12). The average incidence of upper respiratory tract sickness events in this study was 2.1 events per year. (Heath et al. 1991). This incidence is lower when compared to the incidence of upper respiratory tract illness in the general population of 2.3 events per year (Badger et al. 1953).

Table 2.12 **Summary of total miles run per year and the odds ratio for contracting upper respiratory tract illness in a year (Heath et al. 1991).**

Total miles for year	Odds ratio
< 486	1.0
486 – 865	1.7
866 – 1388	2.6
> 1388	2.0

Contrary to anecdotal evidence, some studies listed in Table 2.11 indicate no difference in the risk of acquiring upper respiratory tract illness when comparing athletes to controls and one study shows an increase in the risk when associated with mild to moderate levels of exercise. With more strenuous levels of exercise one study shows a decrease, while two studies show an increase in the incidence of upper respiratory tract illness. In summary it appears that mild forms of exercise do not result in an increase in upper respiratory tract illness and may even be beneficial in protecting against the illness, but as exercise intensity increases, so does the risk of acquiring upper respiratory tract illness.

From the summary in Table 2.11 it is clear that there are conflicting reports on the risk of acquiring upper respiratory illness whilst exercising. However, the J-shaped curve proposed by Nieman (1994) seems to reflect the findings of most studies, that regular moderate exercise is beneficial in preventing URTI and strenuous exercise increases illness risk.

2.3.2 THE EFFECT OF REGULAR EXERCISE TRAINING ON THE RISK OF UPPER RESPIRATORY TRACT ILLNESS.

In general, athletes believe that regular physical activity protects them against upper respiratory illness or that regular exercise training might shorten the period of illness (Nash 1986). A study by Nieman et al. 1998 supports the belief that regular exercise might shorten the period of illness. In this study obese women were exercised with five 45 minute walking sessions per week at 60% to 75% of their maximum heart rate. The results indicated that the number of days with symptoms of upper respiratory tract infection was reduced to 5.6 in the exercise groups compared to 9.4 in the non-exercise control groups. However there is little scientific evidence to support the belief that regular exercise might decrease in the risk of contracting upper respiratory tract illness. Results of studies to this effect are summarised in Table 2.11 where the studies of Schouten et al. (1988), Nieman et al. (1990) and Osterback et al. (1987) are significant.

The Amsterdam growth and health study of Schouten (1988) studied the relationship between regular physical activity and upper respiratory infections in a normal population of young men and women (n=199). Subjects were 92 men and 107 women (age 20 to 23 years) and they were followed up for 4 years. The subjects participated in various sports and activities and the intensity of activity was reported in metabolic equivalent (MET) which is the ratio between metabolic rate during steady-state work and the basal metabolic rate of the subjects. Three levels of activity were identified, 4-7 METS for low intensity physical activities, 7-10 METS for medium intensity activities and 10 METS or more for high intensity activities. Furthermore, a group was identified who spent more than 700 METS per week and this group was divided into an outdoor and an indoor group to examine the possible influence of exercise in a cold environment. The incidence rates for upper respiratory illness in all the study groups were similar to the incidence rates among the general population reported by general practitioners in the Netherlands. The duration of upper respiratory tract symptoms was not related to the levels of physical activity in the study groups. The study also found that sports outdoor in the winter season at an intensity level of more than 700 METS did not increase the incidence of upper respiratory tract illness symptoms compared with indoor sports (Schouten et al. 1988).

The incidence of respiratory tract infections in children (mean age 12,7 years), who participated in regular, supervised training in gymnastics (n=16), swimming (n=14) and ice hockey (n=32) was also reported for a 12 month period (Osterback et al. 1987). The children had been training regularly three to four times a week for four years. They were compared to a control group of children (n=75) who did not exercise regularly. All the children contracted on average 2,8 upper respiratory tract infections during the 12 months. In the incidence of respiratory infections there was no significant difference between the sports groups and control groups of children. There was also no significant difference between the different types of sports in the incidence of respiratory infections. (Osterback et al. 1987). In this study, there was no indication of the intensity or duration of exercise and these results can thus be viewed as a general observation.

Table 2.13 Summary of studies on the incidence of upper respiratory tract illness with regular exercise.

Regular exercise and the incidence of upper respiratory illness			
Reference	**Physical activity**	**Duration**	**Incidence**
Badger et al. 1953	General population	——	2,3 events/year
Osterbach et al. 1987	Gymnastics, swimming, ice-hockey	12 months	2,8 events/year Equal to control group
Schouten et al. 1988	Various sports with intensity of > 700 METS/week	4 years	Equal to general population
Heath et al. 1991	Running ave. 4,7 miles/day	12 months	2,1 events/year

In summary, there is evidence that regular participation in exercise decreases the risk of upper respiratory illness in athletes. Where Schouten (1988) and Osterbach (1987) found their athletes to be no different to the general population, Heath et al. (1992) reported a slight decrease in the upper respiratory tract illness events over one year (Table 2.13). However, from studies by Nieman et al. (1989a), Douglas et al. (1978) and Heath et al. (1992) there is evidence that increase in intensity and frequency of exercise may be responsible for an increase in the risk for acquiring upper respiratory tract

illness. There is also evidence that regular exercise decreases the period of illness when compared to the sedentary population (Nieman et al. 1998).

2.3.3 SUMMARY AND CONCLUSIONS.

Present evidence seems to indicate that an increase in training results in an increase in psychological as well as physical stress. This leads to changes in the natural as well as acquired immune processes that have the effect of suppressing the immune response. This in turn increases the risk for acquiring upper respiratory tract illness. If this is true, exercise must then be a risk factor for acquiring upper respiratory tract illness and athletes must then have a higher incidence of upper respiratory tract illness events over a period when compared to the general population. Studies to this effect have some conflicting results. Where most studies indicate that exercise leads to an increased risk of contracting upper respiratory tract illness, some show a decrease or no difference in risk and incidence. In analysing these studies, the results seem to indicate that the risk of acquiring upper respiratory tract illness is related to the intensity and duration of the exercise.

A summary of the evidence thus seems to indicate that regular participation in mild to moderate exercise offers protection against contracting upper respiratory tract illness. Athletes who train at more strenuous levels and with longer duration seem to be more at risk for acquiring URTI, as is explained in the J-shaped curve for the risk of contracting URTI (Nieman 1994). A possible explanation for some studies which found that strenuous exercisers have a benefit in that they have slightly fewer illnesses over a given period when compared to the general population, might be due to the athletes actively selecting to avoid situations where they could become infected. The slight decrease in incidence of upper respiratory illness might thus not be due to the exercise *per se.*

2.4 THE EFFECT OF UPPER RESPIRATORY TRACT ILLNESS ON EXERCISE PERFORMANCE.

2.4.1 INTRODUCTION.

The effect of an upper respiratory tract illness on exercise performance in the recovery period has not been well studied and is hence poorly understood. Athletes report that there is a decline in exercise performance post-illness . This effect, if present, may be the result of detraining during the illness as upper respiratory tract illness can force an athlete out of training for periods ranging from a few days to a few weeks. In addition, the decrease in performance may be due to additional pathological effects of the illness itself (Nash 1986).

Despite the fact that an URTI occurs commonly, particularly in highly trained athletes (Nieman 1994), it is not known how long it might take the athlete to regain the level of performance after such an illness. This review will examine evidence relating to 1) the degree to which an athlete's fitness declines during the URTI, 2) the time period necessary for recovery and return to pre-illness performance levels. Both animal and human studies pertinent to these issues will be reviewed.

2.4.2 ANIMAL STUDIES.

Friman et al. (1991) observed changes in metabolic parameters and exercise performance rats infected with bacteria. In order to determine whether shortages in fuel reserves or alterations in energy substrate utilization might contribute to a decrease in performance during bacterial infection, oxygen consumption, lipid and carbohydrate metabolism were measured during swimming exercise to exhaustion in these infected rats.

All the infected rats were febrile and experienced a further increase in body temperature during exercise. Compared to pre-infection recordings there was an increase in oxygen consumption and 60% decrease in swimming performance (time to exhaustion) post-infection. Glycogen stores and plasma

free fatty acid levels were decreased, while plasma insulin, glucagon and ketone levels were increased (Friman et al. 1991).

The changes in lipid and carbohydrate metabolism suggested that the carbohydrate stores become reduced during the febrile illness and that fat is increasingly utilised during exercise. The authors concluded that there was a probable deranged oxidative metabolic capacity of the exercising muscles during the infective period and they speculated that this was evident to be the cause for the impaired exercise performance. Raised serum lactate concentrations during exercise indicated increased reliance on oxygen independent metabolism (Friman et al. 1991).

In another study, Ilback et al. (1989) investigated similar metabolic and exercise changes during illness. Similar methods were used with *Streptococcus pneumoniae* infected rats and the same metabolic testing was done. The results were similar to the study of Friman et al. (1991) and also indicated that there was a shift from carbohydrate metabolism toward fat metabolism with exercise during the febrile illness.

These studies illustrate that there is an apparent switch from glycogen to fat metabolism during illness and that this metabolic change is concurrent with a decrease in physical performance. This however is not proof that the poor physical performance is due to this metabolic change. To date no animal studies have been done during the recovery period following the illness.

2.4.3 HUMAN STUDIES.

A number of studies have been conducted in humans to investigate the effect of febrile illness on physical exercise performance, cardio-pulmonary function, metabolism and skeletal muscle parameters.

In a case study an elite cyclist underwent repeated physiological assessments over a 15 month period whilst training for a 100km national event. During this period the cyclist developed fever and reported a

decrease in performance. Subsequent testing indicated raised antibodies to Coxsackie virus (titres >300) (Jakeman 1993). Exercise testing was resumed one week after body temperature returned to normal. During prolonged submaximal exercise the cyclist had an increase in heart rate and perceived exertion with a decrease in total exercise time (pre-illness = 180 minutes, post-illness = 150 minutes). It was found that the respiratory exchange ratio (RER) decreased during exercise indicating a higher percentage of fat oxidation. Blood lactate concentrations were higher at all levels of submaximal exercise and this was interpreted as an increase in oxygen independent metabolism. The increase in oxygen independent glycolysis could be due to a greater use of carbohydrate during the infectious illness, which would then rapidly deplete the carbohydrate stores leading to increased reliance on lipid metabolism. The subject completed six stages of a graded exercise protocol pre-illness. Following the first week post infection the subject could only complete four stages of the protocol and five of the six stages thereafter. There was a pronounced increase in heart rate at all levels of exercise. The author speculates that the increase in heart rate could be due to myocarditis or pericarditis resulting from the infection (Table 2.14). Clinical electro - cardiographic studies were done during exercise but did not however reveal any abnormalities in the subject.

Table 2.14 Summary of results from studies on the effects of upper respiratory tract illness on exercise performance variables.

Physiological effects of upper respiratory tract illness			
Reference	**Parameters tested**	**Results**	
		During illness	**Post illness**
Jakeman 1993	VO2 max (litres/min) HR max (beats/min) RER (units) RPE (units) Glucose (mmol/litre) Lactate (mmol/litre)	 ↑ ↑ 	↑ ↑ ↑ ↑ ↓ ↑
Daniels et al. 1985	HR max (beats/min) Muscle strength (isometric and dynamic contractions) Submax. walking (graded treadmill test)	↑ ↓ ↓	N N N
Friman et al. 1985	Muscle enzymes Muscle ultrastructure Stroke volume Cardiac output	N N ↓ N Due to increased HR	N N ↓ ↓
Friman 1977b	Muscle strength (isometric contractions)	↓	N
Friman et al. 1977a	NMT (single fibre EMG)	↓	N
Astrom et al. 1979	Muscle enzymes Muscle ultrastructure	↓ Abn.	
Breven 1962	HV (ml) HR max (beats/min) VC (litres) FRC (litres) TC (litres) FEV1 (litres) DC (ml/min/mmHg/m2)		↓ ↑ ↓ ↓ ↓ ↓ ↓

Abbreviations to Table 2.14: VO_2 Max = maximal oxygen uptake in ml/kg/min, HR max = maximal heart rate achieved during testing, RER = respiratory exchange ratio, RPE = rate of perceived exertion on the Borg scale, NMT = neuromuscular transmission, HV = heart volume, VC = vital capacity, FRC = functional residual capacity, TC = total lung capacity, FEV1 = forced expiratory volume in one second, DC = diffusion capacity, ↓ = decrease, ↑ = increase, N = Normal.

These observations concur with the results of the studies in infected rats where carbohydrate stores seem to rapidly become reduced with an increased utilisation of fat metabolism. The increase in blood

lactate during and after illness might be due to impaired oxidative capacity of the muscle which then requires a greater contribution from oxygen independent metabolism and could result in muscle acidosis which might be one of the reasons for fatigue. It might be significant that the subject was unable to complete a graded exercise test one week after fever had subsided. This may be due to metabolic inhibition and/or physiological abnormalities due to the illness and/or may be ascribed to a central protective mechanism to spare vital functions from being damaged due to excessive exercise in a state of illness.

At the United States Army Research Institute exercise parameters were tested before, during and after an infective process with the sandfly fever virus. The disease is characterised by the rapid onset of flu-like symptoms. Nine subjects volunteered, seven were inoculated with the virus and two were used as controls and inoculated with saline. Submaximal walking on a treadmill and muscle strength tests were performed before inoculation, 4 days after inoculation and 7 days after fever had subsided. The study found that during pyrexia measurements of isometric muscle strength were decreased and submaximal work performance was limited in some individuals. During a submaximal walking test in the febrile state, three subjects were unable (or unwilling) to complete the test. In the early convalescent stage after the fever had subsided, the submaximal exercise performance and muscle strength returned to near normal (Daniels et al. 1985) (Table 2.14).

It was noted that there seemed a marked unwillingness of subjects to perform some types of exercise during pyrexia and they seemed to voluntarily terminate the activity. This might indicate a central defence mechanism to protect against injury of vital organs due to exertion during febrile illness. It is significant that the muscle strength and submaximal exercise performance returned to normal rapidly, once the fever subsided. This might indicate that the fever itself contributed to the decrease in performance

In the above mentioned study by Daniels et al. (1985), Friman et al. (1985) participated to investigate the effect of the sandfly fever viral infection on muscle enzymes, muscle ultrastructure, cardiac output and stroke volume. The muscle and serum enzymes recorded were creatinine kinase (CK), lactate

dehydrogenase (LDH), triose phosphate dehydrogenase, citrate synthase and lysosomal enzymes. None of the enzymes changed notably during the illness nor during the post febrile phase. No abnormalities were found on muscle biopsies in the ultrastructure of skeletal muscle. It was documented that cardiac output was maintained at pre-illness levels during the febrile illness although the stroke volume was decreased. After the fever had subsided the cardiac output decreased. The maintenance of cardiac output during the illness was as a result of the increase in the heart rate. The lower cardiac output after the fever corresponded to a slower pulse rate and the smaller stroke volume (Friman et al. 1985) (Table 2.14).

In this study there were no significant alterations in the activities of oxidative or glycolytic enzymes. This could support the hypothesis that oxygen independent metabolism during febrile illness increases. Although the cardiac output and stroke volumes were decreased, a tachycardia seemed to adequately compensate for the cardiac output. Decreases in exercise performance following upper respiratory tract illness might occur if the increase in heart rate reaches a stage where it is ineffective in compensating for a reduced stroke volume.

Isometric muscle strength was measured by Friman (1977b) who tested a group of 39 patients suffering from various infectious diseases of viral and bacterial origin. A control group consisted of healthy subjects that were confined to bed for the same period of time as the patients. Recordings were made immediately after the fever subsided, at one month post fever and at 4 months post fever. Muscle strength was found to be decreased in the patients once the fever had subsided compared to the control subjects, but were similar compared to the controls, one and four months afterwards (Table 2.14).

In another study by the same author (Friman et al. 1977a) neuromuscular transmission during febrile illness due to influenza or echovirus infection was measured in 14 patients with influenza and 9 patients with mumps. Single fibre electromyography (EMG) was performed during the febrile period, immediately after the fever and 2 weeks later. Mildly abnormal transmission characteristics during the acute phase of the disease were documented. The authors concluded that abnormal transmission is

part of the disease process in the muscle and this might explain muscle weakness experienced during the febrile phase of illness (Table 2.14).

Enzyme activities and muscle ultrastructure changes were measured by Astrom et al. (1976) during viral and mycoplasma infection. Thirteen patients were studied and they were compared to eight healthy controls who were confined to bed for time periods similar to the confinement to bed of the patients. It was documented that the activities of glyceraldehyde-3-phosphate dehydrogenase (GPD), lactate dehydrogenase (LDH), citrate synthetase (CS) and cytochrome oxydase (cytox) were lower in the skeletal muscle of patients than in the muscle of healthy control subjects. These changes were comparable to patterns documented in progressive muscular dystrophy. The ultrastructure of skeletal muscle was also investigated using electron microscopy and some focal deviations abnormalities demonstrated. The myofibrils were reduced in diameter, the Z-lines irregular and the mitochondria enlarged with inclusion particles. The sarcoplasmic reticulum was distended. These changes were consistent with degeneration in the muscle ultrastructure. The enzymatic and ultrastructural changes documented in the muscle of infected individuals corresponded to those documented in other degenerative muscle diseases. This finding could explain the reduction in performance that athletes experience during upper respiratory tract illness (Astrom et al. 1976) (Table 2.14)

Changes in cardio-pulmonary function were investigated in the post infectious stage of atypical pneumonia to determine if recovery *per se* could have any effect on cardio-pulmonary function (Breven 1962). Sixteen cases were investigated during the convalescent stage of atypical pneumonia and were compared to 12 controls who were recovering from a non-pulmonary disease. The same parameters of heart rate (HR), heart volume (HV), vital lung capacity (VC), functional residual capacity (FRC), total lung capacity (TC), forced expiry volume in one second (FEV1) and diffusion capacity (DC) were investigated in both the atypical pneumonia and non-pulmonary groups. The results of investigations in the non-pulmonary group were similar to parameters previously reported in normal subjects. In the atypical pneumonia group there was an increase in HR, but decrease in HV and respiratory parameters of VC, FRC, TC, FEV1 and DC. It is speculated that the decrease in the diffusion capacity could be caused by a loss of alveoli due to the process of the disease or due to a thickening of the alveolar

membranes leading to a change in the permeability of the membranes (Breven 1962) (Table 2.14).

These findings confirm the decreases in cardiac function reported by Friman et al. (1985) as well as showing attendant decreases in lung function. This could explain the reluctance of subjects to complete exercise protocols following illness. The compromised cardiac and lung functions might lead to premature exhaustion with resultant termination of exercise even though the subject might seem afebrile with the absence of clinical symptoms.

A study was conducted by Lloyd et al. (1988) on patients diagnosed as post viral fatigue syndrome (myalgic encephalomyelitis) to determine whether their complaints of debilitating fatigue were could be measured by a decrease in exercise parameters. These patients fulfilled the diagnostic criteria for the condition including subjective fatigueability of muscles, lymphadenopathy and T-cell lymphopenia. However, measurements of muscle strength, endurance and recovery were all entirely normal among the 20 individuals who reported severe muscle fatigue, suggesting that the fatigue is associated with disordered perception of muscle force and effort rather than actual force production (Lloyd et al. 1988). This indicates that psychological perception of exhaustion can play a role and thus have a negative influence on exercise performance.

Table 2.15 **Summary of data on the effects of upper respiratory illness on exercise performance variables.**

Summary of physiologic effects of upper respiratory illness			
System tested	**Parameter tested**	**Results**	
		During illness	**Post-illness**
Respiratory	Vital capacity (litres)		↓
	Total capacity (litres)		↑
	FRC (litres)		↓
	FEV1 (litres)		↓
	VO_2 max (ml/kg)		↓
	DC (ml/min/mmHg/m2)		↓
	RER (units)		↓
Cardiac	HR max (beats/min)	↑↑	↑↑N
	Stroke volume	↓	↓↓
	Cardiac output	N	↓
Muscle	Muscle strength (isometric and dynamic)	↓↓	NN
	NMT (single fibre EMG)	↓	N
	Muscle ultrastructure	N-Abn	N
Metabolic	Glucose (mmol/litre)		↓
	Lactate (mmol/litre)		↑
	Muscle enzymes	↓N	N
Endurance performance	Submax. Walking (graded treadmill test)	↓	N
	RPE (units)	↑	↑

Abbreviations: VO_2 Max = maximal oxygen uptake in ml/kg/min, HR max = maximal heart rate achieved during testing, RER = respiratory exchange ratio, RPE = rate of perceived exertion on the Borg scale, NMT = neuromuscular transmission, HV = heart volume, VC = vital capacity, FRC = functional residual capacity, TC = total lung capacity, FEV1 = forced expiratory volume in one second, DC = diffusion capacity, ↓ = decrease, ↑ = increase. N = normal.

From the results summarised in Table 2.15 it seems clear that cardio-respiratory function is compromised during and following upper respiratory tract illness. The decrease in lung function as well

as the decrease in diffusion capacity in the lungs may result in inadequate gas transmission during exercise and could contribute to decrease in exercise performance following an illness. Furthermore, the decrease in cardiac output and stroke volume does not seem to be adequately compensated for by the increase in heart rate. This inadequate circulation could lead to premature exhaustion due to diminished oxygen supply to the muscles.

The muscle tests on the other hand indicate abnormalities during the illness which seem to return to normal almost immediately after the febrile period of the disease. Decreased serum glucose and increased serum lactate concentrations following respiratory tract illness support the reports of a rapid depletion of glycogen and subsequent reliance on fat metabolism with exercise during and after the illness. It is not clear if this finding might contribute to a decrease in exercise performance as both fat and glycogen are adequate sources for energy. The report that exercise parameters tested during submaximal walking seems to be normal following upper respiratory illness might indicate that decrease in exercise performance could be limited to strenuous and maximal effort only. Finally the increased rate of perceived exertion during and after illness might be as a result of the compromised cardio-respiratory functions and changes in metabolic energy utilization.

It is not known how detraining during the period of illness could influence the decrease in exercise performance. Studies measuring the decrease in exercise performance following URT infection have never been repeated in the same subjects after a similar detraining period whilst unaffected by URTI.

2.5 OVERALL SUMMARY AND CONCLUSIONS.

Upper respiratory tract illness occurs commonly among the general population including the sporting community. Although URTI are caused by many microbials, the rhinovirus is the most common. The measures for prevention and management of upper respiratory illness are well documented, but little is known of what effect upper respiratory tract illness has on exercise performance. There is evidence that the immune response is suppressed for 6 to 24 hours following an acute exercise bout and that this

could be a window period during which the athlete is susceptible for contracting URTI. Studies also indicate that the intensity and duration of exercise seems to be important in either providing protection against URTI or increasing the risk for contracting the illness. Overall evidence seem to indicate protection against URTI to the athlete undergoing regular, moderate training and an increased risk of contracting URTI during strenuous and prolonged exercise.

Most authors agree that definite pathological changes take place during upper respiratory tract illness and these changes lead to decrease in exercise performance. Decreases in isometric muscle strength, dynamic muscle strength and muscle endurance during upper respiratory tract illness have been documented in both animal and human studies. During upper respiratory tract illness studies also indicate decreases in cardiac output and physical respiratory values as well as some metabolic changes. Evidence suggests that the pathology rapidly returns to normal once the fever has disappeared. Few studies though have examined the levels of exercise performance during this recovery phase, nor whether the decrease in performance is due to the illness itself or the result of detraining.

CHAPTER 3

THE INCIDENCE OF UPPER RESPIRATORY TRACT ILLNESS IN DISTANCE RUNNERS - A 45 MONTH FOLLOW UP.

3.1 INTRODUCTION

Adults in the general population contract 2 – 5 upper respiratory tract illness (URTI) events per year with an average of 2.3 events per year (Heath et al. 1992). Anecdotal evidence suggests that regular exercise training can protect against contracting URTI (Nash 1996; Nieman et al. 1989a; Heath et al. 1992; Eichner 1993). This observation has been scientifically confirmed for regular, moderate exercise but the effect appears to be reversed under conditions of strenuous or acute exercise (Nieman 1994). Thus the "J" shaped curve proposed by Nieman (1994) which depicts the risk of URTI in individuals participating in regular, moderate exercise to be less than that of the sedentary population, while those engaging in strenuous and acute exercise were at highest risk of acquiring an URTI.

3.2 AIM OF THE STUDY

The aim of the study was to closely follow a cohort of athletes over a period of 45 months (May 1992 – Feb 1996) and to record the incidence and duration of URTI contracted during this time.

3.3 METHODOLOGY

3.3.1 SUBJECTS

For the study, 42 distance runners were recruited from the Empangeni and Richards Bay running clubs (KwaZulu-Natal, South Africa). Each club has an active running population of about 100 individuals, from a total population of around 60 000. These athletes were followed over a period of 45 months for the development of URTI. Of the original 42 athletes recruited, 29 were retained in the study and 13

dropped out (4 subjects relocated and 9 subjects became untraceable).

The athletes were all active endurance runners aged between 25 and 50 years. During the 45 month study period they were asked to maintain their levels of fitness for the duration of the study. All the subjects were instructed to run between 30 and 50 kilometres per week and to record their weekly training schedule on a log sheet (Appendix 4). This criterium of running 30 to 50 kilometres per week was calculated from the average distances the subjects reported running at the time of their recruitment. The subjects were instructed to run an 8 kilometre time trial weekly and to record these times as an indicator of their endurance performance (Appendix 4). The total training hours of each athlete over the two year period was estimated.

3.3.2 TESTING PROCEDURES

Subjects were asked to report any symptoms of an URTI immediately to the principle investigator (Deon Viljoen). When athletes reported symptoms of URTI they were medically examined to confirm the symptoms and signs of URTI using specific criteria (Appendix 5). Symptoms of sore throat, headache, blocked nose, painful chest, coughing, sore eyes and muscle pains were confirmed. The athlete was examined for signs of pyrexia, conjunctivitis, pharyngitis, rhinitis, otitis media and bronchitis. Once the diagnosis of URTI was confirmed, the athletes took home a standardised form for the daily listing of their symptoms and signs (Appendix 6). Once the URTI symptoms and signs had disappeared, the athletes reported back to the principle investigator for a medical examination to confirm that they have recovered from the illness. The number of URTI events per 1000 training hours was calculated as the incidence of URTI in this group.

When this study commenced, the prolonged time it took to collect the data was not anticipated. It was initially expected that enough subjects should report with clinical illness and thus be tested within six to eight months. The limited number of subjects and the inclusion/exclusion criteria eventually dictated a period of 45 months. Due to this extended time subject compliance for diligent data recordings of distances run and time trial times were poor. When subjects reported an URTI, the data for weekly

distances run and time trial times for four weeks prior to illness were recorded. This data was correlated with their entries on the study forms (Appendix 4), their own logbooks and the logs on time trial times kept by their clubs. Following illness the subjects were allowed a period of more than 3 months to regain their fitness levels. During the four weeks prior to detraining the subjects were instructed to run similar weekly distances as recorded prior to falling ill. Their time trial times were compared to the times they ran for the four weeks prior to illness to ensure that they had achieved the same level of fitness.

The inclusion criterium for URTI was a period where symptoms lasted for five days or more and the exclusion criterium was an illness with symptoms lasting less than 5 days. These criteria were decided upon to maximise the chance of upper respiratory tract illness resulting from infection. It was also thought that URTI symptoms lasting less than 5 days might be due to mechanical effects of strenuous exercise or long endurance events and not due to infection.

3.3.3 RESULTS

As depicted in Figure 3.1, in the group of 29 athletes there were 22 reports of upper respiratory tract illness (76%) of which 12 (55%) lasted longer than 5 days which complied with the URTI inclusion criterium for clinical illness (CI). The other 10 subjects (45%) had symptoms for only 2 to 3 days and all these episodes immediately followed their participation in a long distance endurance event. These subjects did not comply with the URTI inclusion criterium and had mild illness (MI). Seven subjects (24%) did not report URTI symptoms during the 45 month period and thus had no illness (NI).

The twelve subjects out of the 29 with clinically confirmed illness (CI), reported URTI lasting 5 days or longer over a period of forty five months. When projected into four years, 3.2 subjects (12%) out of the study group experienced clinical upper respiratory tract infections per annum, which is an odds ratio (incidence of upper respiratory tract illness in one year) of 1.03 upper respiratory tract infections per year.

Ten of 29 subjects reported URTI of less than three days duration and occurring directly following an endurance event. The inclusion criteria for an URTI was a minimum of five days illness. These events

were thus excluded from the study.

The incidence of URTI in 1000 hours of training was estimated and calculated as follows. Athletes in the study trained for 30 to 50 km/week (average of 40 km/week) at an average speed of 5min/km. During the study period of 45 months each athlete ran on average 600 hours. The 29 athletes participating in the study ran an estimated total of 17400 hours in the 45 months. The total incidence for URTI (n = 22) per 1000 training hours was 1.26. The incidence for the athletes with mild URTI with the symptomatic period being less than 5 days (n = 10) is 0.58. The incidence for athletes with moderate URTI with the symptomatic period more than 5 days (n = 12) is 0.69.

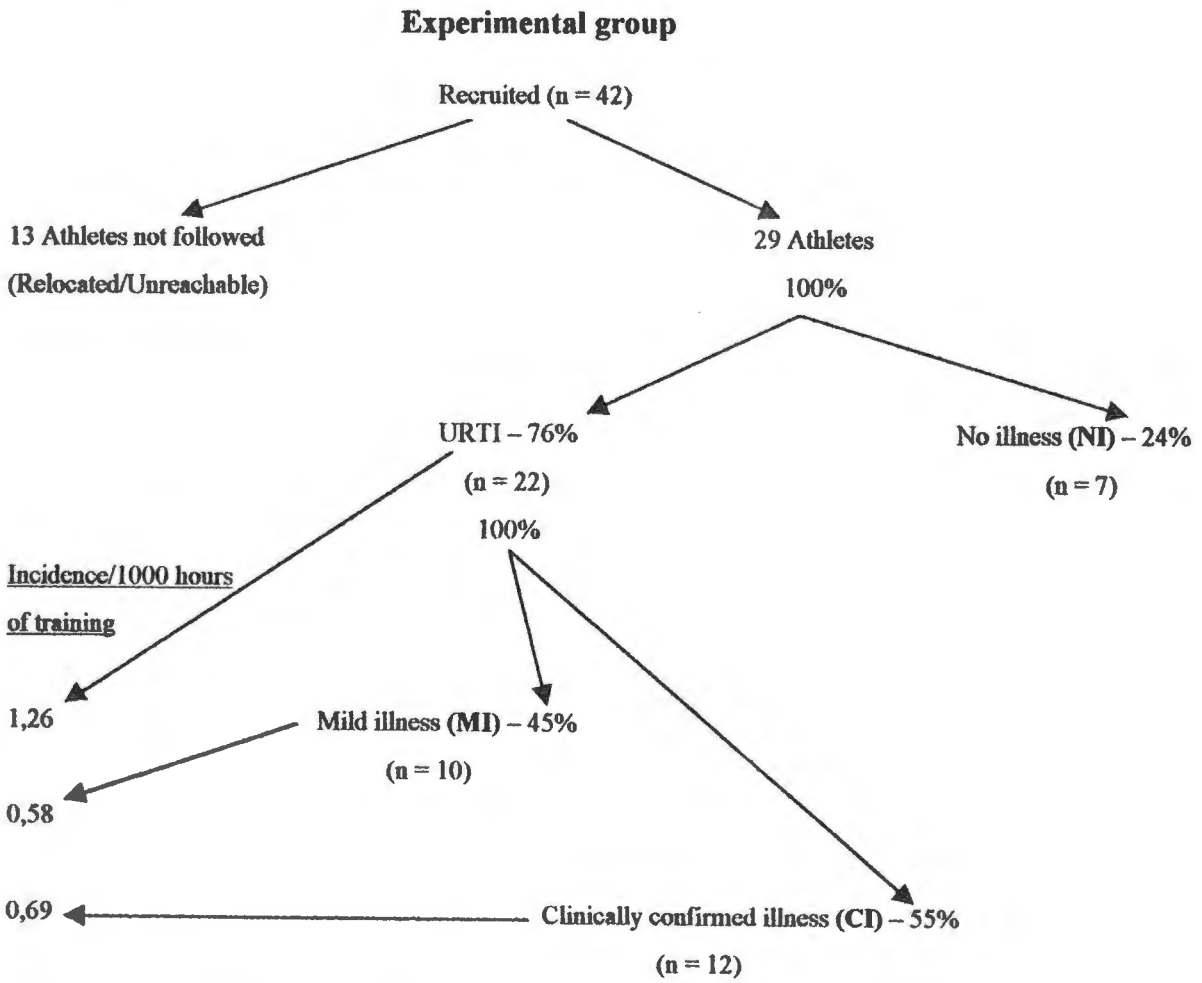

Figure 3.1 **Flow diagram of experimental group.**
(NI = no illness. MI = mild illness. CI = clinically confirmed illness.)

3.4 DISCUSSION

In this study, the odds ratio for a runner running an average of 1920km per year to acquire an upper respiratory tract illness is 1.03 per year. This correlates well with Heath et al. (1991), who found that athletes running less than 782km per year had an odds ratio of 1.0 and when they ran 782 to 1392km per year their odds ratio was 1.7. These ratio's are much lower when compared to the general sedentary population where an odds ratio of 2.3 for contracting upper respiratory tract illness per year has been documented (Badger et al. 1953).

It therefore appears that running 1920km per year affords protection against acquiring upper respiratory tract illness. However, the improved odds ratio might also be due to the athletes being a selected group who are highly motivated and practice preventative measures against contracting upper respiratory tract illness. For example, they may avoid crowded places and wash their hands and not touch their faces for fear of contracting the illness. Athletes are a self-selected group and cannot be compared to the general population. Other studies have also documented regular exercise training does not necessarily afford protection against upper respiratory tract illness (Osterback et al. 1987; Schouten et al 1988; Nieman et al. 1990).

It has been shown that exercise promotes an increase in free radicals. Free radicals can alter and inactivate enzyme complexes and damage cells, damage DNA and RNA and promote mutations and cancer (Kanter 1998). Thus free radicals can be important in the initiation of exercise-induced muscle damage and in the initiation and propagation of the subsequent acute muscle inflammatory response. Antioxidants have the potential to limit muscle oxidative stress during the postexercise period, but direct evidence for their role is limited (Tiidus 1998). There is also evidence that physical training results in increased activity of several major antioxidant enzymes and overall overall antioxidant status of the trained individual (Dekkers et al. 1996). This study did not record the intake of supplements by the athletes and the effect of free radicals due to disease and/or training is not known.

The symptoms of URTI which developed in 10 athletes (45%) directly followed their participation in a strenuous endurance event. These symptoms could possibly be attributed to the event itself rather than

to infection. This finding would correlate with a study by Brenner et al. (1994) who showed that faster runners complained more frequently of upper respiratory tract illness symptoms. The 10 athletes in this study who reported symptoms of URTI following participation in strenuous endurance events, were ill for a maximum of three days. This period of illness is typically too short to indicate an infection because it is well accepted that URTI illness due to infection will last for longer than 5 days (Knight 1977). Studies investigating the immune system during and after strenuous events, indicate that cellular elements responsible for the immune response are depressed immediately following the exercise (Eichner 1993). This suppression of immunological parameters following strenuous exercise might manifest with symptoms of URTI for a limited period and thus might explain the short term symptoms of URTI for a few days following these events. Furthermore, there is evidence that regular exercise stimulates the immune response (Nieman et al. 1990) and thus protects against URTI (Heath et al. 1992). This might also protect against acquiring URTI for longer than 3 days following strenuous exercise.

A limitation of this study is the absence of a control group of sedentary subjects. Such a group would have enabled the study to compare the incidence of URTI in a sedentary population to the incidence of URTI in a population of trained athletes over a period of 45 months. Furthermore valuable data would have been available to indicate the periods of symptomatic illness in the sedentary group compared to the exercise group. It is also possible that the intensity and detail of the exercise protocols might have been responsible for some athletes not making themselves available for this study, thus the small number of subjects.

3.5 SUMMARY

This study indicates that runners who perform regular, moderate, endurance exercise have a low odds ratio for contracting upper respiratory tract illness. Endurance exercise training does not increase the incidence of upper respiratory tract illness, and may even decrease the risk for acquiring the illness. This might be due to a self-selection process with regard to the testing programme as well behaviours which minimise the risk of infection. Participants in this study were generally well informed of the

methods of transmission of disease and protect themselves from being contaminated and infected. This finding means that these athletes are a selected group and cannot be compared to the general population. Furthermore, simpler protocols with less variables and less frequent testing might have resulted in more athletes becoming available for testing.

CHAPTER 4

THE EFFECTS OF UPPER RESPIRATORY TRACT ILLNESS ON EXERCISE PERFORMANCE IN THE RECOVERY PERIOD

4.1 INTRODUCTION

Anecdotal evidence suggests that there is a decrease in exercise performance in the days or weeks following an upper respiratory tract illness. Evidence from a limited number of scientific studies indicate that the pathological changes that occur during upper respiratory tract illness may be responsible for the decrease in exercise performance (Breven 1962; Astrom et al. 1976; Friman 1976; Friman et al. 1977a; Friman et al. 1985; Nash 1986; Friman et al. 1991). However, the extent and duration of any decreases in performance is unknown, as is the degree to which detraining contributes to this post-illness decline. This study was designed to answer the question of what the extent of decline in exercise performance is following URTI and how much of the decline is due to the illness and how much is due to the detraining alone.

4.2 MATERIALS AND METHODS

This is a cohort prospective clinical trial of 45 months duration to investigate the effect of URTI and detraining alone on exercise performance.

4.2.1 SUBJECTS

The athletes were selected and tested following an URTI that complied with the inclusion/exclusion criteria for this study.

4.2.2 TESTING PROCEDURES

All laboratory tests were conducted at the laboratory of the Department of Human Movement Sciences at the University of Zululand.

Over a period of two years, baseline tests were performed on 42 athletes after which they were monitored for the development of a URTI. Initially three athletes were recruited for pilot studies and the protocol was tested, designed to avoid errors and to test the practical feasibility of the protocol. The protocol allowed for baseline tests to be performed on the subjects and these tests were used to calculate the workload for follow-up testing.

4.2.2.1 PRE TRIAL SCREENING

The subjects chosen for this study completed a medical questionnaire (Appendix 2) and also signed a consent and indemnity form (Appendix 1). A full medical examination was performed on all the athletes at the time of recruitment following prescribed parameters (Appendix 3).

Base-line tests were performed on the subjects and their maximum aerobic capacities were measured (Appendix 7). On Appendix 7 the time on the treadmill was recorded in the left column and the corresponding treadmill speed in the right column. When this data was compared to the respiratory parameters recorded with the Oxycon apparatus, it was possible to calculate at what treadmill speed the subject achieved his VO_2 max. From this data the treadmill speed at a VO_2 of 70% of max could be calculated and this 70%VO_2 max was used in the actual testing protocol as outlined in section 4.2.3.2. Isokinetic muscle strength and muscle endurance was also measured. The exact testing procedures are outlined in paragraphs 4.2.3.1. and 4.2.3.2. The maximum aerobic capacities were used to calculate 70% of their workload. The maximum oxygen uptake (VO_2 peak in ml/kg/min) was measured at the point of maximal effort and 70% of this parameter was calculated. This was the intensity that testing would be performed at when athletes contracted an upper respiratory tract illness.

4.2.2.2 DIAGNOSIS AND MANAGEMENT OF URTI

Once an athlete developed an upper respiratory tract illness the athlete reported to the investigating doctor to confirm the clinical diagnosis. Medical examinations were conducted to confirm that the signs were consistent with upper respiratory tract illness and not due to localised illness such as a suppurative tonsillitis. Only subjects with periods of illness lasting 5 days and longer were admitted to the study (Table 4.1). Some subjects reported upper respiratory tract illness symptoms immediately after completing endurance races where the symptoms only lasted for 2 to 3 days.

Table 4.1 Criteria for admission to the study

Inclusion	Exclusion
General clinical symptoms and signs of URTI: **Symptoms:** Sore throat, headache, blocked nose, painful chest, cough, sore eyes, muscle pains. **Signs:** Pyrexia, conjunctivitis, pharyngitis, rhinitis, otitis media, rhonchii, wheeze.	Localised infection
Illness lasting > 5 days	Illness lasting < 5 days

Subjects were instructed not to take any medication on commencement of the illness prior to the medical examination. This was to prevent treatment from masking any symptoms. Once the diagnosis was established, symptomatic treatment consisting of antihistamines, cough syrup and analgesics were allowed during the period of illness. Medication was discontinued 24 hours before the final medical examination after all the symptoms subsided and during the testing period after the illness.

During the period of illness, the subject recorded the symptoms on a daily symptom chart (Appendix 6). During the time of illness no training was allowed. Strict instructions were given to athletes so that they accurately recorded data on the symptom chart. Symptoms were graded for severity on a scale from 0 to +++ with 0 - no symptoms, + - slight symptoms, ++ - mild symptoms and +++ - severe symptoms.

Patients were requested to record their oral temperature and resting pulse rate each morning on waking up. The temperature was taken with an oral thermometer that was held under the tongue for two minutes. The pulse rate was measured by palpating the carotid artery and timed on a wrist watch for 60 seconds. Once all clinical symptoms had disappeared, the subjects reported to the doctor to confirm the end point of the illness. The end point of the illness was therefore defined as the point where all clinical symptoms and signs were absent and performance testing could commence.

4.2.3 PERFORMANCE TESTING FOLLOWING URTI

The athletes were tested within 24 hours from the designated end point of illness (Day 0). Isokinetic strength and muscular endurance were first tested. In addition, subjects performed an exercise test at 70% of their maximum aerobic workload for 40 minutes after which the effort was incrementally increased to exhaustion. These tests were repeated every two days over a period of six days following the illness. No exercise other than the prescribed tests was allowed during the six days of testing. These repeat tests will be referred to as Day 2, Day 4 and Day 6 after illness.

4.2.3.1 MUSCLE STRENGTH AND ENDURANCE TESTING

Muscle strength and endurance of the quadriceps and hamstring muscle groups of the right leg were tested using an Akron isokinetic unit (Akron, Great Britain). The apparatus was set to accommodate the leg length of each individual according to the published manual. The right leg of the subject was positioned so the knee was free to move in flexion and extension. The right thigh was firmly strapped to the seat of the machine to prevent movement during the test. The right ankle was strapped to the lever arm of the Akron and the lever was set to ensure that the arc of movement was smooth for the leg, and that there was no friction during movement between the leg and the lever. The athlete was allowed to hold onto the sides of the seat during the tests.

Once the athlete was secured in the seated position the angle was set to zero with the knee in the fully extended position. The athlete was then assisted in familiarising himself with the operation of the

apparatus. Following this the subject was allowed to perform warming up exercises by extending and flexing his leg for 20 seconds with the speed set at 180 degrees/second. A compulsory rest period of 5 minutes was then allowed.

The subject was first tested for muscle endurance. The right knee was extended and flexed with maximal effort for 35 seconds at 180 degrees/second. The subject was then rested for ten minutes before the test for muscle strength was commenced. In this test the right knee was extended and flexed with maximal effort for 10 seconds at 60 degrees/second and the data for these tests were recorded by using the Akron computer program. During the tests the subjects were continuously motivated verbally to deliver their maximal performance.

The parameters of muscle strength were recorded during the 10 seconds of maximal repetitive flexion and extension of the right knee at a speed of 60 degrees/second. Muscle strength was measured in Joules, power was measured in Watts and flexion and extension torque to body weight in Newton meters/kg.

The muscle endurance was recorded as the total work done (in Joules) during the full period of 35 seconds of repetitive flexion and extension of the knee at a speed of 180 degrees/second.

The protocols for muscle strength and endurance was used for the baseline tests and the format was kept exactly the same for the tests following the illness period as well as the tests following the detraining period. All the subjects on the study had performed baseline tests. During these tests they were familiarized with all the apparatus and study protocols.

4.2.3.2 ENDURANCE PERFORMANCE TESTING

The test for aerobic endurance performance was performed 10 minutes after the muscle endurance and strength tests. Endurance performance was tested with the subject running on a treadmill (Powerjog E10 - Powerjog, Great Britain) and coupled to a respiratory analysis system (Oxycon Sygma

- Mijnhardt, Holland). The treadmill was set at zero elevation. Core temperature was continuously monitored by a recto thermo coupler (YSI - part of Oxycon Sygma apparatus with a sensor that is inserted in the rectum and connected to the apparatus by a wire lead) and recorded by the resident computer program. The rectal temperature probe was kept in place by taping the lead to the gluteal area. The respiratory analysis system consisted of a mouthpiece, exhaust controller and wide diameter tube connected to the Oxygon Sygma apparatus which is a free standing unit housing the respiratory analysis apparatus and the computer hardware. The respiratory variables were captured by the resident computer program. Prior to testing the Oxycon system was calibrated in a standard fashion using 0% oxygen, 4,9% carbon dioxide and air volume. During the closed circuit breathing the subjects nose was clamped to prevent nasal breathing. A twelve lead ECG (Cardiovit At6 - Schiller, Switzerland) was used to monitor the electrocardiogram during the first test on day 0 following illness. Before commencing the test each subject was familiarised with the apparatus and the testing procedure.

During baseline testing (Appendix 7), the subject first stood on the treadmill at rest for one minute. The treadmill was then started and accelerated to 3 kilometres per hour (kph) over one minute. The subject then walked at this speed for the next two minutes. The treadmill speed was once more increased to 7 kph over one minute at which speed the subject jogged for three minutes. The treadmill speed was increased at one kph every minute until the athlete reached exhaustion. During the test the athlete was verbally encouraged to complete the test to exhaustion.

A submaximal treadmill exercise test was performed and the data from the baseline test was used to calculate the workload required to achieve 70% of maximal effort. For this test the athlete stood on the treadmill for one minute during which the resting parameters were recorded. The treadmill was started and incrementally accelerated over five minutes to reach the calculated speed to achieve 70% of maximal effort. The subject then ran at this speed for 40 minutes after which the treadmill speed was increased by 1 kph every minute until the subject reached ultimate exhaustion. The treadmill speed was then rapidly decreased to a walking speed of 3 kph and this was maintained for two minutes during which the recovery parameters were recorded. Respiratory parameters were recorded in the first 5 minutes and the last 5 minutes of submaximal exercise, and continuously during the exercise to

exhaustion. During the test the athlete was verbally encouraged to perform at maximal capacity.

For the endurance test, the Oxycon system measured the (VO_2) oxygen in ml/kg/min, the (VCO_2) carbon dioxide in ml/kg/min and respiratory frequency as breaths per minute (RF). The system calculated the respiratory quotient (RQ), metabolic units (METS) and the volume breathed in and out (V'e) in litres per minute. The measured and calculated parameters were recorded every minute during the test.

During the endurance testing the rate of perceived exertion (RPE) was included in the protocol. This data was incompletely recorded and thus excluded in this report.

4.2.4 RETRAINING PERIOD

Following the period of illness and the subsequent performance tests the athlete was encouraged to commence normal training in order to regain the same state of conditioning prior to developing the illness. This was conducted by adhering to a prescribed exercise schedule until the athlete was able to run 30 to 50 km per week, corresponding to the distance run prior to illness. Weekly 8 km time trial times were again recorded. The time period allowed for the athletes to regain their fitness in all cases exceeded three months.

4.2.5 DETRAINING AND REPEAT PERFORMANCE TESTING

After the illness and subsequent testing, the athlete was allowed to regain his/her fitness level comparative to before the illness (Table 4.2) after which the final phase of the study was commenced.

Table 4.2 **The weekly distance (km/week) and 8km time trial running time (min) of the runners 4 weeks pre – illness and 4 weeks pre – detraining. Values are mean ±SD.**

	Pre – illness	Pre – detraining	P value
Weekly distance (km/week)	50.5 ± 5.8	49.9 ± 5.9	0.92
8 km Time trial	33.2 ± 0.5	33.0 ± 0.49	0.24

During the final phase the athlete stopped training for the same period he/she detrained during the illness. After the simulated detraining period the subject repeated all the testing for muscle strength, muscle endurance and aerobic endurance parameters duplicating the tests that were done following the period of illness. The tests following the detraining period were compared to the tests following the illness period. This was done to determine whether there was a difference in performance when comparing detraining associated with illness to detraining alone. As in the case of the tests following the illness period the tests following the detraining period are also referred to as Day 0, Day 2, Day 4 and Day 6.

4.2.6 STATISTICAL ANALYSIS

Statistical analysis was done at the Department of Statistics, Medical School of Natal, South Africa, using SAS Statistical Software, SAS Institute, USA. Means and standard deviations were used to summarise the data. Paired t-tests were used to compare mean of performance parameters between tests done on days following illness versus days following detraining. A probability of $p < 0,05$ was considered statistically significant. Performance measurements were not done on one of the subjects who developed a bacterial infection of the lower leg. Thus, statistics for all the days were accumulated and calculated on five subjects except for the last of each test day following detraining where the calculations were made on four subjects.

4.3 RESULTS

4.3.1 INCIDENCE OF URTI IN AN ATHLETIC POPULATION

A total of 42 volunteers were recruited and performed the baseline tests. During the time the study was performed four athletes relocated and nine students from the University of Zululand became uncontactable. These subjects were effectively lost to the study, thereby reducing the total to 29 athletes. Among these 29, there were 22 incidences subjects developing upper respiratory tract illness symptoms during the study period. Of these, 3 athletes (10%) reported illness that complied to the criteria for URTI, but could not participate further in the study due to political unrest on the University of Zululand campus. Another 3 athletes (10%) reported their illness that was clinically confirmed by the investigating doctor. Two of these athletes declined to participate further in the study and one completed the protocol for post-infection testing but subsequently refused to detrain for the comparative study. This athlete had been ill for 14 days and was disinclined to break a rigorously maintained training schedule to detrain for an additional 14 days.

Six patients completed all the test protocols. Of these, one did not maintain his level of fitness after his URTI, which rendered his detraining tests invalid. One athlete developed a septic lesion on the leg before the final detraining test and this test was not performed. The five subjects who completed both test protocols were ill for periods ranging from 5 to 13 days and the mean period of illness was 7.8 days.

4.3.2 PERFORMANCE PARAMETERS POST-ILLNESS

4.3.2.1 MUSCLE STRENGTH AND ENDURANCE

Muscle strength parameters following the upper respiratory tract illness and the detraining periods in the athletes were tested and is depicted in Table 4.3.

Table 4.3 **Muscle strength (maximal contraction of Quadriceps muscle for 10 seconds at 60 degrees/second) following URTI and a detraining period of equal duration.**

Muscle Strength								
	Work/ flexion (Joule)	Flexion power (Watt)	Flexion torque to mass (Nm/kg)	Work/ extension (Joule)	Extension power (Watt)	Extension torque to mass (Nm/kg)	Total work (Joule)	Total power (Watt)
Baseline	598 ±112	119 ±25	2.32 ±0.42	439 ±129	95 ±24	1.65 ±0.39	1037 ±224	834 ±313
Day 0 Illness	586 ±53	117 ±16	2.10 ±0.40	444 ±86	96 ±19	1.58 ±0.36	1029 ±132	841 ±204
Day 2	511 ±140	102 ±30	1.97 ±0.58	410 ±108	88 ±23	1.47 ±0.36	921 ±232	730 ±297
Day 4	571 ±40	113 ±12	2.20 ±0.43	455 ±129	97 ±23	1.59 ±0.43	1026 ±155	731 ±187
Day 6	559 ±70	109 ±17	2.09 ±0.48	417 ±116	90 ±23	1.52 ±0.36	976 ±177	735 ±175
Day 0 Detrain	594 ±83	112 ±15	2.13 ±0.34	403 ±96	91 ±24	1.54 ±0.38	997 ±177	737 ±152
Day 2	605 ±91	115 ±20	2.19 ±0.41	399 ±103	89 ±21	1.52 ±0.41	1004 ±190	723 ±139
Day 4	558 ±143	106 ±23	2.02 ±0.49	392 ±66	88 ±18	1.51 ±0.34	950 ±177	690 ±123
Day 6	484 ±84	98 ±12	1.86 ±0.26	396 ±88	83 ±23	1.48 ±0.40	880 ±172	646 ±150

Actual values reported in Mean ±Standard Deviation. Nm/kg = Newton metre/kilogram.

There were no significant differences in Total work in flexion (Joule), Power in flexion (Watt), Flexion torque to mass (Nm/kg), Total work in extension (Joule), Extension power (Watt), Extension torque to mass (Nm/kg), Total work (Joule) and Total power (Watt) between the illness and detraining days on days 0, 2, 4 and 6. There were also no significant differences when the post illness and post detraining parameters were compared to the baseline tests.

Muscle endurance parameters were measured following the illness and the detraining periods and is depicted in Table 4.4.

Table 4.4 **Comparison of muscle endurance (total work done in 35 sec of maximal flexion and extension of Quadriceps muscle at 180 degrees) following URTI and a detraining period of equal duration.**

Muscle endurance (Joule)			
Baseline tests			
3789 ±717			
Days	Illness	Detraining	p-values
0	4169 ±765	3996 ±620	0.45
2	3841 ±814	4130 ±781	0.32
4	4130 ±873	3939 ±759	0.39
6	4044 ±962	3949 ±1013	0.15

Actual values reported in Mean ±Standard Deviation.

There were no significant differences in muscle endurance work (Joule) when comparing the illness period to the detraining period on days 0, 2, 4 and 6. Furthermore there were no significant differences when comparing baseline data to the post illness and post detraining data.

These data show that muscle strength and endurance does not seem to be affected by upper respiratory tract illness immediately and for at least six days after the resolution of symptoms. The muscle strength and endurance also does not seem to be affected by a comparable period of detraining.

4.3.2.2 AEROBIC PHYSIOLOGICAL PARAMETERS

The parameters measured during the baseline tests at rest and at maximal effort were compared to the parameters measured following the illness period and following the detraining period at rest and during maximal exercise. Furthermore the tests done following the period of illness were compared to the tests done following the detraining period at rest, after 40 minutes endurance at 70% of maximal effort and at exhaustion.

4.3.2.2.1 RESTING VALUES

The baseline performance variables recorded at rest and the performance variables recorded following the illness period and following the detraining period for each of the four testing days (days 0, 2, 4 and 6) are depicted and compared in Table 4.5.

Table 4.5 Comparison of baseline values to the test values, following illness and detraining periods for physiological parameters at rest, and comparison of the illness period to the detraining period for physiological parameters at rest on testing days 0, 2, 4 and 6.

	Physiological parameters at rest						
	VO_2 (ml/kg/min)	VCO_2 (ml/kg/min)	RQ	METS	RF (breaths/min)	V'e (l/min)	Temp (degrees Celsius)
Baseline	4.28 ±1.98	277.8 ±102.4	0.92 ±0.07	1.22 ±0.55	15.34 ±6.80	10.44 ±3.22	37.84 ±0.48
Illness- Day 0	5.94 ±0.99	394.5 ±86.7	0.95 ±0.06	1.70 ±0.31	14.56 ±7.67	13.44 ±4.50	37.56 ±0.33
Day 2	6.36 ±4.23	481.0 ±309.3	1.02 ±0.23	1.80 ±1.20	15.12 ±3.77	14.54 ±7.96	37.56 ±0.47
Day 4	4.68 ±1.34	313.5 ±97.7	0.92 ±0.03	1.32 ±0.37	13.72 ±5.70 *	11.28 ±1.37	37.40 ±0.29
Day 6	5.97 ±3.02	423.0 ±273.7	1.03 ±0.12	1.70 ±0.84	17.87 ±8.65	15.50 ±6.83	37.67 ±0.48
Detrain-Day 0	6.22 ±3.02	406.6 ±208.0	0.92 ±0.05	1.78 ±0.85	15.22 ±6.31	13.30 ±5.77	37.58 ±0.32
Day 2	6.14 ±1.03 *	402.8 ±100.1 *	0.92 ±0.09	1.74 ±0.27 *	15.02 ±6.65	13.14 ±3.37	35.12 ±5.50
Day 4	6.32 ±2.37	409.2 ±139.3	1.11 ±0.40	1.80 ±O.65	14.34 ±5.29	14.62 ±4.08	37.42 ±0.49
Day 6	7.60 ±4.09	480.0 ±271.8	0.87 ±0.04	2.17 ±1.12	13.27 ±4.12	15.37 ±7.68	37.60 ±0.36

Actual values are reported as Mean ±Standard Deviation. (VO_2 = oxygen uptake recorded. VCO_2 = carbon dioxide expired. RQ = respiratory quotient. METS =metabolic units. RF = respiratory frequency in breaths/minute. V'e = volume air inspired andexpired. Temp = rectal temperature. * denotes p-value < 0.05.)

There was considerable individual variation in actual parameters as is indicated by the large standard deviations. In comparison, the resting VO_2, VCO_2 and METS were significantly greater ($p < 0.05$) than baseline values on Day 2 following the detraining period. The resting RF was significantly decreased ($p < 0.05$) on Day 4 following the period of illness when compared to baseline values.

There were no significant differences between the illness and detraining periods in any of the resting measurements.

4.3.2.2.2 SUBMAXIMAL EXERCISE PARAMETERS

The performance variables following the period of illness and the performance variables following the detraining period taken after 40 minutes endurance running at 70% of maximal effort on testing days 0, 2, 4 and 6 are depicted and compared in Table 4.6. METS was significantly higher ($p < 0.05$) in the period following illness compared to the same period following detraining on day 0 and day 2. Likewise, VO_2 was increased ($p < 0.05$) on these two days post-illness compared to post-detraining.

Table 4.6 Comparison of physiological parameters after a 40 min run at 70% of VO_2 peak post-illness and post-detraining.

Physiological parameters after 40 minutes exercise at 70% of maximum effort								
		VO_2 (ml/kg/min)	VCO_2 (ml/kg/min)	RQ	METS	RF (breaths/min)	V'e (l/min)	Temp (degrees Celsius)
Illness	Day 0	43.06 ±5.52	3128 ±614	0.97 ±0.03	12.30 ±1.57	30.92 ±7.73	73.63 ±14.07	38.88 ±0.71
	Day 2	41.48 ±4.61	3046 ±583	0.98 ±0.05	11.82 ±1.24	34.78 ±8.99	75.64 ±15.61	39.03 ±0.58
	Day 4	32.58 ±13.78	2203 ±628	0.75 ±0.49	9.32 ±3.93	32.08 ±6.15	69.26 ±10.71	35.64 ±7.39
	Day 6	41.00 ±3.83	2889 ±615	0.99 ±0.05	11.53 ±1.08	36.48 ±7.25	75.73 ±15.30	38.75 ±.58
Detrain	Day 0	40.74 ±4.23 *	2773 ±497	0.95 ±0.04	11.64 ±1.22 *	33.02 ±6.31	72.86 ±12.88	34.44 ±8.98
	Day 2	39.72 ±3.15 *	2689 ±424	0.95 ±0.04	11.36 ±0.88 *	34.40 ±6.26	72.36 ±12.59	34.52 ±6.44
	Day 4	36.74 ±6.65	2453 ±492	0.94 ±0.04	10.50 ±1.93	30.40 ±6.96	62.14 ±11.49	36.90 ±3.08
	Day 6	40.93 ±5.05	2811 ±553	0.95 ±0.02	11.70 ±1.47	27.97 ±2.80	74.13 ±11.83	38.67 ±0.71

Actual values are reported as Mean ±Standard Deviations. (VO_2 = oxygen uptake. VCO_2 = carbon dioxide expired. RQ = respiratory quotient. METS = metabolic units. RF = respiratory frequency in breaths/minute. V'e = volume air inspired and expired. Temp = rectal temperature. * denotes p-values < 0.05)

4.3.2.2.3 MAXIMAL EXERCISE TEST PARAMETERS

The baseline performance variables at maximal exercise and the performance variables following the period of illness and following the detraining period on testing days 0, 2, 4 and 6 are depicted and compared in Table 4.7. There were considerable individual variations as depicted by the large standard deviations.

When comparing the baseline tests with the tests following the illness and the detraining periods, there were a lower respiratory frequency (RF) and a higher ventilation volume (V'e) on day 0 following the detraining period. There was no significant difference when baseline tests were compared to tests following the period of illness.

At maximal effort, there were significant differences on comparative days of the post-illness and post detraining periods as depicted in Table 4.7. VO_2 and METS were significantly lower on day 0 post-illness, while VCO_2 and V'e were significantly lower on days 0, 2 and 4 post-illness when compared to the corresponding detraining periods.

Table 4.7 **Comparison of baseline values to the test values following the illness and detraining periods for physiological parameters at maximal effort, and comparison of the illness period to the detraining period for physiological parameters at maximal effort.**

	Physiological parameters at maximal effort						
	VO_2 (ml/kg/min)	VCO_2 (ml/kg/min)	RQ	METS	RF (breaths/min)	V'e (litres/min)	Temp (degrees Celsius)
Baseline	49.7 ±13.0	3839 ±1306	1.07 ±0.13	14.16 ±3.63	70.22 ±10.44	94.86 ±32.16	38.12 ±0.61
Illness Day 0	53.4 ±8.06	3920 ±663	1.08 ±0.07	15.26 ±2.32	50.60 ±27.71	103.7 ±18.7	38.92 ±0.65
Day 2	54.1 ±6.86	4253 ±913	1.12 ±0.09	15.46 ±1.94	44.12 ±7.57	114.4 ±25.1	35.64 ±8.27
Day 4	56.3 ±4.02	4594 ±639	1.17 ±0.09	16.08 ±1.15	47.34 ±8.12	120.4 ±13.4	35.36 ±7.60
Day 6	58.3 ±6.43	4566 ±734	1.17 ±0.84	17.37 ±2.96	49.22 ±10.40	132.4 ±19.3	39.02 ±0.80
Detrain Day 0	58.7 ±7.45 *	4753 ±835 *	1.13 ±0.06	16.78 ±2.12 *	51.04 ±7.83 *	130.4 ±23.9 *	35.16 ±7.72
Day 2	57.8 ±6.92	4553 ±694 *	1.11 ±0.01	16.50 ±1.94	48.02 ±7.23	125.6 ±21.8 *	34.14 ±7.76
Day 4	54.9 ±5.23	4345 ±388	1.13 ±0.06	15.50 ±1.73	48.08 ±4.88	116.7 ±12.8	33.84 ±7.82
Day 6	56.6 ±10.26	4689 ±1077 *	1.15 ±0.03	16.13 ±2.94	46.73 ±3.38	135.9 ±22.4 *	38.83 ±0.86

Actual values are reported as Mean ±Standard Deviations. (VO_2 = oxygen uptake. VCO_2 = maximal carbon dioxide expired. RQ = respiratory quotient. METS = metabolic units. RF = respiratory frequency in breaths/minute. V'e = volume air inspired and expired. Temp = rectal temperature. * denotes p-values < 0.05.)

Figures 4.1, 4.2, 4.3 and 4.4 depict the VO_2, VCO_2, V'e and METS at maximal exercise intensity during the post illness and post detraining periods respectively.

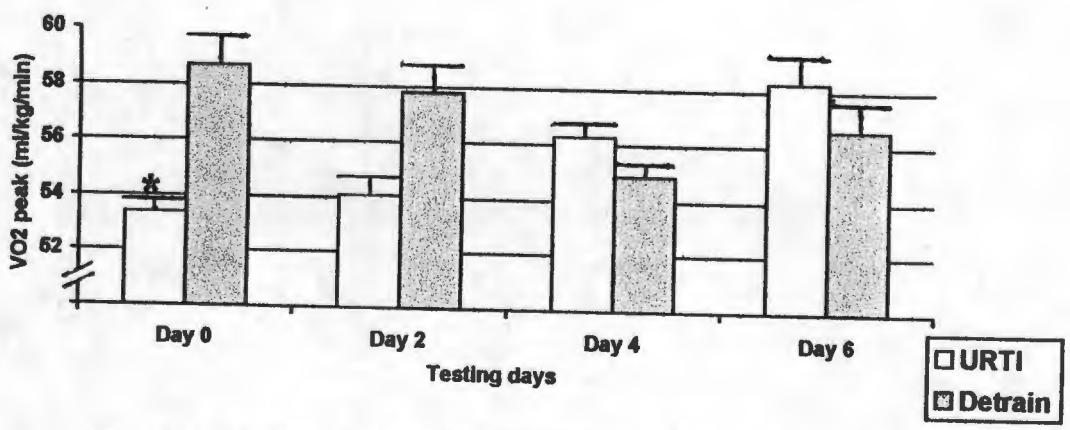

Figure 4.1 **Effect of URTI and detraining on VO_2 peak in runners**

(* : $p < 0.05$)

The mean values of VO_2 peak following the period of noticeable illness and the period of detraining at maximal effort is depicted in Figure 4.1. There is a progressive increase in VO_2 peak from day 0 to day 6 following illness. The difference in VO_2 peak following illness and following detraining is significant only on day 0.

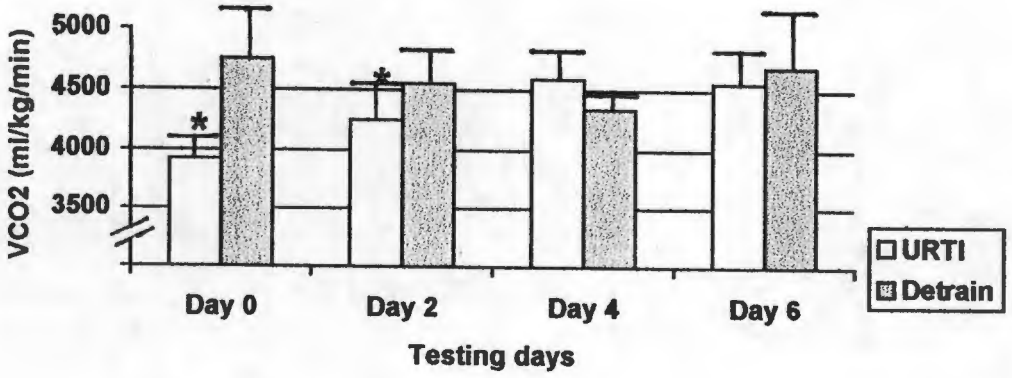

Figure 4.2 Effect of URTI and detraining on VCO_2 expiry in runners
(* : $p < 0.05$)

Comparisons of VCO_2 expiry at maximal effort following the illness and detraining periods are depicted in Figure 4.2. There is an incremental increase in the actual mean values of VCO_2 expiry following illness when compared to detraining and the difference is significant on days 0 and 2.

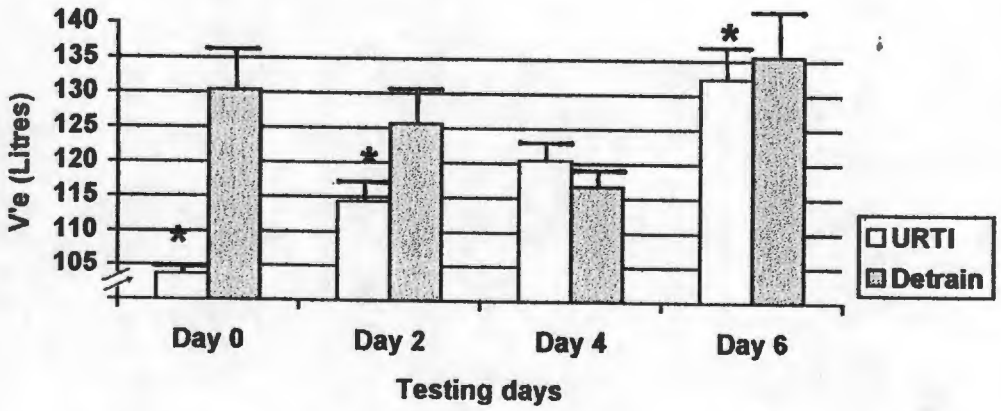

Figure 4.3 The effect of URTI and detraining of V'e in runners
(* : $p < 0.05$)

The V'e (volume of air inspired and expired in litres) at maximal effort following the illness and the

detraining periods are depicted in Figure 4.3. Following illness there is a progressive increase in the volume of air expired from day 0 to day 6 and the differences following the illness and detraining periods are significant on days 0, 2 and 6.

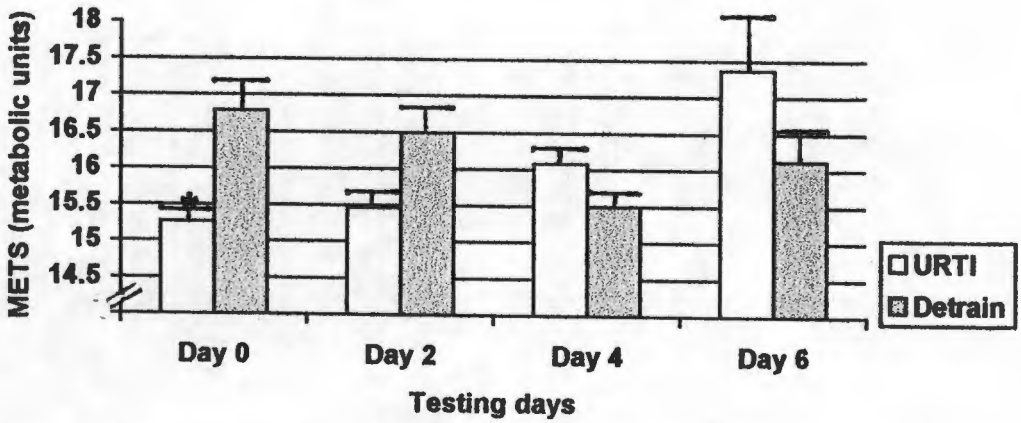

Figure 4.4 Effect of URTI and detraining on METS in runners
(* : $p < 0.05$)

The mean values for METS (metabolic units) at maximal performance following the illness and the detraining periods are depicted in Figure 4.4. The tests following URTI improve incrementally from day 0 to day 6 and the values are significantly decreased for the illness test on day 0 only.

4.3.2.2.4 TIME TO EXHAUSTION

A comparison of the time it took the athletes to reach their levels of exhaustion following the illness and the detraining periods is indicated in Table 4.8. There were no differences between days 0, 2, 4 and 6 following the detraining period. However, there was a significant decrease in the time to exhaustion following illness on day 0.

Table 4.8 **Comparison of the time to exhaustion (mean ± SD) following illness period and following detraining period during a graded treadmill test to exhaustion after running for 40 minutes at 70% of maximal performance on testing days 0, 2, 4 and 6.**

Time to exhaustion			
Days	**After illness**	**After detraining**	**p-values**
0	5.20 ±2.49	6.80 ±2.17	0.03 *
2	6.40 ±2.30	7.00 ±1.14	0.30
4	6.60 ±1.95	7.20 ±1.64	0.43
6	7.27 ±2.63	7.25 ±2.22	1.00

Actual values reported as means ± standard deviation (SD). * indicates $p < 0.05$.

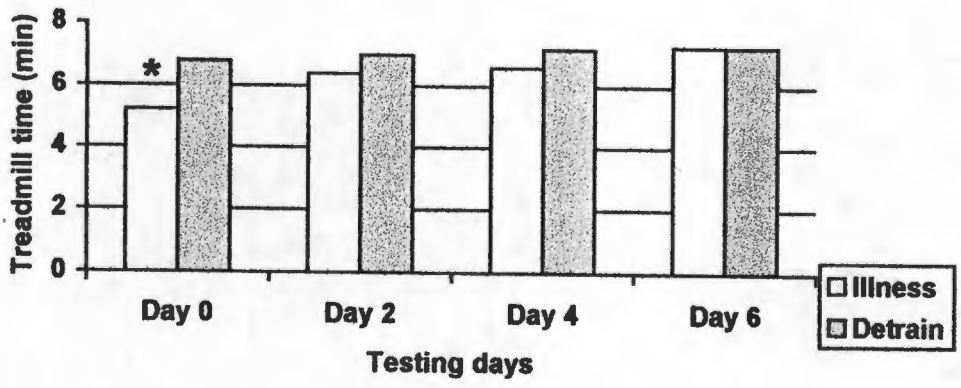

Figure 4.5 Effect of URTI and detraining on treadmill time (min) in runners
(* : $p < 0.05$)

There was a progressive increase in the time to exhaustion from day 0 to day 6 following the URTI (Table 4.7 and Figure 4.5). Comparison of the period following illness to the period following detraining indicates a significant difference between illness and detraining on day 0 only.

4.4 DISCUSSION

The objectives of this study were to determine the effect of URTI on exercise performance during the recovery period from the illness.

The baseline tests which determined the athletes' physiological parameters both at rest and at exhaustion were compared to the tests performed following upper respiratory tract illness and then following a comparable period of detraining. This was done to demonstrate possible decreases in performance due to the illness and detraining periods.

Of the original group of 42 subjects who underwent baseline tests, 29 remained in the study. Twelve athletes out of the 29 developed upper respiratory illness over a 4 year period and only five became available for testing in the study. This is a small group with large variations as is evident from the maximum and mean differences of the values. Differences in the aerobic parameters measured seem to follow a trend that can be related to the illness itself and not the concomitant detraining effect. Similar differences in muscle strength and muscle endurance tests were not documented. Thus, even with the small sample size, the results seem to indicate that URTI has a negative effect on aerobic endurance due to the illness.

When compared to baseline tests at rest, a significant decrease ($p < 0.05$) in RF was recorded on day 4 following illness with no significant difference following detraining. The significant difference in RF at rest on day 4 following illness is not clear, but might indicate some subclinical impairment of respiratory function, or might be due to the small cohort and thus large individual variation as is illustrated by the standard deviations.

Furthermore, when compared to baseline tests, significant increases for VO_2, VCO_2 and METS were recorded at rest on day 2 following detraining with no significant differences following illness. The reason for these increases in VO_2, VCO_2 and METS following detraining are not clear but might indicate that the period of detraining has a detrimental effect on respiratory function at rest that

manifests on day 2 following the initial exercise bout. These increases in VO_2, VCO_2 and METS following detraining may also indicate an increased metabolic rate following the testing on day 0, which had not fully recovered prior to the testing on day 2. The increases in VO_2, VCO_2 and METS following detraining had disappeared on test day 4 and might indicate that the subject had fully recovered the respiratory function and/or metabolic rate as a result of detraining.

When comparing baseline tests with tests following illness and detraining at maximal performance, there is a significant decrease in RF and an increase in V'e on day 0 following detraining. This might once more be evidence of some loss of respiratory function during the detraining period which is apparent at maximal performance on day 0, but rapidly recovers once exercise is recommenced and has disappeared on day 2.

The physiological parameters measured at rest, after 40 minutes at 70% of maximal exertion and at exhaustion, in both the post-illness and post-detraining periods were compared to determine whether possible decreases in performance are due to the illness or due to the detraining period. At rest no significant differences were found following the illness or detraining periods. After 40 minutes running at 70% of maximal exertion, it was found that the VO_2 and METS were increased significantly on days 0 and 2 following illness. At maximal effort, all the recorded values were lower following URTI than following detraining and decreases were significant in VO_2 and METS on day 0, and VCO_2 and V'e on days 0, 2 and 6 following the URTI period but not after detraining.

The reason for the significant increase in the VO_2 peak and the METS following illness on days 0 and 2 after 40 minutes exercise at 70% of maximal exertion is not clear. This might have been due to an increase in effort resulting from a subclinical myopathy with resultant increase in oxygen demand and increased muscle metabolism. These effects disappeared on days 4 and 6 and this might be due to the athlete having recovered by this time after the illness.

The significant decreases of VO_2 and METS following illness on days 0 at maximal exercise and the significant decreases of VCO_2 and V'e following illness on days 0, 2 and 6 at maximal effort are also not

clear but might be due to subclinically impaired respiratory function and metabolism as result of the upper respiratory tract illness.

In summary, comparison of the baseline tests with the tests following illness and the tests following detraining seem to indicate that an upper respiratory tract illness lasting from 5 to 13 days might have a subclinical effect on the respiratory function and metabolism at rest once the subject has recovered from the illness. Furthermore, the period of detraining resulted in a significant loss of aerobic endurance ability at maximal effort on day 2 only following this period. Significant differences between the two experimental periods of illness and detraining at rest were also found on day 0. This seems to indicate that there might be a decrease in athletic performance following detraining due to a loss of endurance capacity which recovers after the first two days on commencement of training.

In comparing the period following detraining to the period following illness at maximal exercise to exhaustion, there were decreases in physiological parameters indicating possible subclinical pathological sequelae of the illness that may be interfering with respiratory or skeletal muscle function. This was demonstrated by decreases in VO_2 max, VCO_2, METS and respiratory volume (V'e) during the first day following the illness and decreases in VCO_2 and V'e for the full six days duration of the testing. The testing did not extend beyond the first six days following illness and it is not known when the VCO_2 and V'e returned to normal.

This study is the first to show, that upper respiratory tract illness lasting on average 7,5 days has a detrimental effect on exercise performance immediately following the illness. Physiological parameters were unaffected at rest. However, there was evidence of increased oxygen consumption and increased metabolism during the first two days following illness, when subjects performed submaximal exercise by running for 40 minutes at 70% of maximal performance. This would support the findings of a study (Daniels et al. 1985) which showed that submaximal exercise performance was near normal following febrile illness.

Our study also showed that athletes terminated the maximal test earlier following illness than after detraining alone. This finding was also reported by Jakeman (1993) who found that an elite cyclist who exercised to exhaustion by completing six stages of a graded exercise protocol, was able to complete only four stages of this protocol during the first week following a coxsackie viral illness (sp. not identified). The reasons for the decreased performance are not clear but might be the result of compromised respiratory functions due to the illness. As demonstrated in this study, the muscle strength and muscle endurance do not seem to be affected by the illness, therefore the premature termination of exercise to exhaustion might not be due to an abnormality in skeletal muscle functions.

These findings seem to indicate that there is a significant negative effect of upper respiratory tract illness on submaximal exercise performance during the first two days only following the illness, and that the negative effect on maximal exercise lasts longer.

In this study no significant differences between URTI and detraining states were documented in the total work performed by isokinetic testing on skeletal muscle (Joule). Likewise for indicators of skeletal muscle strength [total power (Watt), work done (Joule) and torque produced (Nm) during flexion and extension] there were no significant differences post illness and post detraining. These results seem to indicate that an upper respiratory tract illness lasting from 5 to 13 days has no negative effect on either skeletal muscle strength nor muscle endurance. This finding is in agreement with previous studies which found that changes in muscle parameters due to URTI return to normal almost immediately following febrile illness (Friman 1977; Astrom et al. 1979; Friman et al. 1985; Daniels et al. 1985).

A limitation of this study is the absence of a control group which could have been useful in detecting a drift in calibration of apparatus over time. However, the air mixture used during recording of the endurance parameters were from one cylinder only and this cylinder was exclusively kept for this study. The aerobic apparatus was calibrated prior to each test according to specified parameters and by using the same cylinder of air. A further limitation to the study could be a possible learning effect due to repeated physiologic testing. The only familiarising with testing procedure was done during the baseline testing and that did not include an endurance run for 40 minutes at 70% of maximal endurance.

Intensity and duration of training during this study should not be limiting factors as the distances run per week were dictated and the time trial times monitored. The intake of anti-oxidants and dietary factors could be limitations of this study as they were not controlled nor recorded.

4.5 SUMMARY

This study has shown that following an URTI, aerobic endurance capacity but not muscle strength nor muscle endurance, is negatively affected. Upper respiratory tract illness has a significant effect on performance parameters during submaximal endurance exercise in the first two days only following an URTI. However, during maximal exercise, upper respiratory tract illness has a significant detrimental effect on endurance parameters during the first six days immediately following the illness and possibly for longer.

The illness appears to be responsible for some loss of respiratory function which recovers by the fourth day following the active period of illness. Detraining alone significantly decreases endurance capacity, but this recovers rapidly within 2 days of recommencing endurance training.

In future studies, it may be necessary to extend the testing period beyond six days following upper respiratory tract illness to determine how long it takes for respiratory parameters to recover such that exercise to exhaustion is not impaired. Such a study could also attempt to determine whether the premature termination of effort to exhaustion following illness is due to respiratory factors, skeletal muscle factors or central factors. Another question that remains unanswered is whether the apparent protective effect that regular endurance exercise has on the risk of developing an upper respiratory tract illness is due to the exercise *per se* or due to the athletes being a selected group who conscientiously practice methods to avoid contracting illness.

In summary this thesis shows that there is a low risk of URTI in moderately trained endurance athletes. However, in athletes an URTI can also decrease submaximal endurance performance for the first two days following the illness, and decrease maximal performance for longer than six days following the

illness. This decrease in performance is not only due to detraining. As muscle strength and endurance does not appear to be negatively affected by URTI, athletes participating in events that depend on strength and power as opposed to aerobic endurance would probably not be handicapped.

APPENDICES

Appendix 1

UNIVERSITY OF ZULULAND

DEPARTMENT OF HUMAN MOVEMENT SCIENCE

SUBJECT CONSENT FORM

I,_____ of (address)

_____

having been fully informed of the nature of the research entitled:

THE EFFECT OF UPPER RESPIRATORY TRACT ILLNESS ON EXERCISE PERFORMANCE

do hereby give my consent to act as a subject in the above-named research.

I am fully aware of the procedures involved as well as the potential risks and benefits attendant to my participation as explained to me. In agreeing to participate in this research, I waive any legal recourse against the researchers of the University of Zululand, any and all claims resulting from personal injuries sustained. This waiver shall be binding upon my heirs and personal representatives. I realise that it is necessary for me to promptly report to the researcher any signs or symptoms indicating any abnormality or distress.

I am aware that I may withdraw my consent and withdraw from participation in the research at any time. I am aware that my anonymity will be protected at all times and agree that the information collected may be used and published for statistical or scientific purposes.

I have read the foregoing and understand it. Any questions which may have occurred to me have been answered to my satisfaction.

Subject:

_____ _____ _____

(Print name) (Signature) (Date)

Person administering informed consent:

_____ _____ _____

(Print name) (Signature) (Date)

Witness:

_____ _____ _____

(Print name) (Signature) (Date)

Project supervisor:

_____ _____ _____

(Print name) (Signature) (Date)

Appendix 2.

Personal and Medical History

Name:		
Sex:	Race:	Date of birth:
Occupation:		

In which sports do you participate?

Have you ever been prohibited from participating in sport for medical reasons?
If Yes, describe:

Have you had an operation or ever been advised to have an operation?
If Yes, describe:

Do you have or have you ever suffered from the following:

Indicate with an X	No	Yes	Indicate with an X	No	Yes
Ear Nose Throat			Sinusitis		
Frequent colds			Frequent headaches		
Hay fever			Migraine		
Asthma			Allergies		
Rheumatic fever			Painful joints		
Tuberculosis			Epilepsy		
Serious accident			Head injury		
Thyroid problems			Persistent coughing		
High blood pressure			Palpitations		
Low blood pressure			Diabetes		

If Yes, describe:

Signed: _____ Date: _____

Appendix 3

Medical Examination Date: _____

Name:	Age:

General medical examination	Normal	Abnor	Describe abnormality
Head, face, neck			
Nose			
Sinuses			
Mouth and throat			
Ears			
Chest and lungs			
Heart			
Vascular system			
Abdomen			
Endocrine system			
Upper limbs			
Spine			
Lower limbs			
Neurological			

Weight:			Length:	
Body Build:	Slender	Average	Muscular	Fat
Blood pressure:	Supine:		Sitting:	
Resting pulse rate:				
Urine:	Protein	Blood	Leukocytes	Glucose

ECG comment:

Summary of disabilities and diagnosis:

Appendix 4

Weekly training schedule																	Starting date:							
Kilometres run per week																								
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Mon																								
Tue																								
Wed																								
Thu																								
Fri																								
Sat																								
Sun																								
8km time trial times weekly																								

Appendix 5.

Medical examination on commencement of Upper Respiratory Tract Illness

Name:	Date:

Symptoms and signs were noted as being either present (+) or absent (-)

Symptoms	
Sore throat	
Headache	
Blocked nose	
Painful chest	
Cough	
Sore eyes	
Muscle pains	
Other	

Signs	
Temperature	
Conjunctivitis	
Pharyngitis	
Rhinitis	
Otitis media	
Rhonchii	
Wheeze	

Appendix 6.

Listing of Symptoms and Signs during Upper Respiratory Tract Illness

Date when last trained:	Date when illness started:

Scale for grading of symptoms and signs How do you perceive your symptoms? If the symptom is absent you mark 0 for none. If there is slight discomfort you mark + for light. If there is definite discomfort you mark ++ for moderate. I there is extreme discomfort you mark +++ for severe.

None	0
Light	+
Moderate	++
Severe	+++

Symptoms	Days																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Sore throat																				
Headache																				
Blocked nose																				
Painful chest																				
Cough																				
Sore eyes																				
Muscle pains																				
Other																				

To be taken each morning on waking up

Pulse rate																				
Temperature																				

Other comments or symptoms not listed above:

Appendix 7.

Respiratory analysis

Name:		
Date of birth:	Height:	Mass:

Time in minutes	Activity
Comments:	

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