Editorial

There is a popular belief in the medical world today that a fatigued individual is more susceptible to disease than a non-fatigued individual.

Oppenheimer and Spaeth, 1922[1]

From the clinical perspective, the effect of exercise on the immune system remains an enigma. That exercise has both immunostimulatory and immunosuppressive effects has been acknowledged for at least the last century. Over the last decade in particular, substantial evidence from both laboratory-based investigations into specific aspects of immune functioning and epidemiological studies of athletic populations supports this dualistic relationship.

Athletes, coaches and sports doctors believe, with some justification, that sportspeople are more susceptible to certain illnesses during intense training and major competition. This is obviously a problem both in terms of their ability to compete, but there are also long term effects on training and health to consider. Somewhat paradoxically, there is the common perception that those who exercise regularly are less susceptible to certain illnesses, such as the common cold.

While neither of these observations has been rigorously assessed at an epidemiological level, the evidence would point to a dual effect of exercise: intense exercise increases illness susceptibility while moderate exercise does the opposite. Nieman[2] has depicted this relationship as a "J" shaped curve. Moderate regular exercise can reduce URTI risk to below that of sedentary population. However, beyond a point specific to each individual, the risk of infection rises exponentially with an increase in exercise frequency, duration and intensity.

The exact mechanisms of this immunosuppression are far from understood. The human immune system is highly complex and precisely ordered. It comprises cells, hormones and soluble immunomodulators that inhabit the bone marrow, lymphoid tissues and ducts and peripheral circulation. Because of the number and complexity of its interactions with other body systems, it is almost impossible to delineate, isolate or ascribe a specific function to any single pathway or mechanism. Moreover, although significant exercise-induced immune alterations can be demonstrated in *in vivo* assay systems, there are presently no *in vitro* studies to confirm that these changes are biologically meaningful.

Nevertheless, highly trained and competitive athletes in a variety of sport disciplines, do appear to be at risk of developing an infection, particularly those of the upper respiratory tract (URTIs). Edith Peters, author of two papers in this edition, was the first South African researcher together with (Eric Bateman) to attempt to quantify rates of post-race illness in ultramarathon runners.[3] They confirmed that those athletes who ran the faster times, and trained the highest mileages were more likely than the slower, less well trained runners and the sedentary control subjects to develop URTIs. Subsequent studies on Comrades runners have not entirely supported the ides that it is the more competitive athlete who is more likely to develop post-race URTI. Ms Peters reviews both her own work and that of other exercise immunologists in this publication.

Ever since Linus Pauling published his treatise on vitamin C and the common cold, this anti-oxidant has enjoyed good press. However, for once there is some fact behind the hype. Peters and her colleagues at Wits have shown that vitamin C supplementation, even more so than the other anti-oxidants (beta-carotene, vitamin E), reduced the incidence of URTIs in ultradistance runners in the two week period after the Comrades marathon.

Interest in the immune response to exercise has arisen for reasons other than those related to sports. One is the possible clinical implications. For example, could exercise play a role in the prevention and treatment of certain illnesses such as cancer and acquired immunodeficiency syndrome (AIDS) ? Epidemiological evidence certainly suggests an association between regular physical activity and a lower incidence of certain cancers. Animal studies have shown exercise training to enhance resistance to experimentally induced tumour growth.

Exercise is currently used as an adjunctive therapy to counteract the physically debilitating effects of the illness and treatment, and to improve the patients' physical and psychological state. However, as the immune system plays a fundamental role in the progression of cancer and AIDS, the interactive effect of exercise on the immune system could itself prove to be therapeutically useful.

Although there is only one documented case of HIV transmission as a result of participation in contact sport, athletes engaging in high risk behaviour are at the same risk of infection as non-sports persons. This means that the HIV epidemic will eventually affect physically active people. Dr Martin Schwellnus discusses the effect HIV disease has on the ability to perform exercise, and what role exercise has on disease progression. Despite a lack of epidemiological data from which to calculate the risk of HIV transmission during sports participation, Dr Schwellnus addresses the concerns of sports administrators and participants with regard to this emotive subject.

In reading these reviews it may occur to you that in trying to quantify and explain the effect that exercise has on immune functioning, scientists have overlooked a fundamental factor: the mind. Although the concept of psychoneuroimmunology, (the interaction between the neuroendocrine and immune systems) is alluded to in the more recent studies, there is as yet no data to back up the theory. Exercise may be immunostimulatory because it reduces negative affective states, increases the release of endogenous opiates and reduces HPAC activation. On the contrary, hard training and competition is immunosuppressive, because it serves as an additional stressor to an already overloaded system.

Thus the sportsperson who is most likely to develop a URTI is not the elite athlete whose entire lifestyle and infrastructure is geared around his or her training. It is the individual who, on top of professional, domestic and social responsibilities, attempts to withstand the same training load as the top class athlete. Dealing with their URTI's is going to require more than just judicious doses of Vitamin C.

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CONTENTS

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Exercise and the immune system: A review

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Exactly how and why the immune system is challenged by exercise has been the subject of a concerted research effort over the last decade. What has emerged is a highly inconsistent puzzle, because of the extreme complexity of the immune system and the barely quantifiable factors that influence it. Further disparity is caused by the large variation in the age, gender and fitness levels of the subjects, the extent or lack of training, the duration and type of exercise protocol and the timing and frequency of sampling. Host defence parameters measured in these studies range from simple determination of leukocyte counts to sophisticated evaluations of lymphocyte subsets and functions. Large variations in the reliability of both in vivo and in vitro assay techniques further compromise the reliability of studies in this field.[1]

Although there are few data on the effects of exercise on the immune system as a whole, there is a great deal of information on the influence it has on individual components thereof. These studies form the basis of this review.

Exercise and leukocytes

Perhaps the best documented effect of exercise on the host defence system is an acute and immediate leukocytosis, the magnitude of which is directly related to the intensity and duration of work, and inversely proportional to fitness level (for review, see McCarthy and Dale[2]). The increase in leukocyte number, which can be up to four times the resting level, is predominantly due to increases in neutrophil and, to a lesser extent, lymphocyte counts, although monocyte numbers also increase.

At the end of exercise lasting up to 30 minutes, the leukocyte count usually falls to normal levels within 10-30 minutes. Lymphocyte counts drop 30-50% below pre-exercise levels, remaining low for 3-6 hours. Eosinophils also vacate the blood in large numbers while basophils are largely unaffected.[3] After sustained exercise lasting 2-3 hours, leukocyte counts may still be elevated for up to 24 hours. During very prolonged exercise (16-24 hours), baseline levels may be reached even before the end of exercise.[4] Moderate intensity exercise induces a much less pronounced leukocytosis, lymphocytosis, neutrophilia and lymphocytopenia.[5]

There appears to be no effect of training on resting leukocyte numbers, with virtually all papers reporting clinically normal values.[2] Some investigators have reported that training blunts the leukocytosis of exercise [6], while others have not.[7,8]

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Leukocyte function and exercise

Exercise produces a decrease in neutrophil adherence and bactericidal capacity in conditioned athletes but not untrained subjects. Conversely, phagocytic activity is improved in untrained men but not athletes.[9] It is tempting to speculate that the leukocytosis of exercise could protect athletes from bacterial infection. However this is unlikely as the response is so acute and transient and there is no evidence that regular exercise produces long-term adaptation in leukocyte numbers, mobilisation or function.

The mechanisms of exercise leukocytosis

At rest, more than half of the body's mature leukocytes are sequestered in the lungs, liver and spleen. An increase in cardiac output and perfusion of the microvasculature, as well as adrenergic-receptor mediated changes in the interaction between leukocytes and endothelial cells of the capillaries would mobilise this pool of marginal cells into the blood stream.[10,11] Epinephrine is a potent beta-adrenergic agonist, and high intensity exercise is known to induce a rapid increase in the density of $beta_2$ adrenoreceptors.[12] Cortisol, while inducing a strong and sustained neutrophila, also inhibits the entry of lymphocytes into the circulation and facilitates their egress from the blood into other lymphoid compartments.[13] Accordingly, this post-exercise leukocytosis is predominantly a neutrophilia with a concurrent lymphopenia. Immature leukocytes may also be released from the bone marrow. Hemoconcentration effects are minimal.

The mechanisms of exercise leukocytosis therefore involve a redistribution of existing cells, rather than synthesis of new cells. McCarthy and Dale's[2] model of exercise leukocytosis is based on the differential activity of catecholamines and cortisol. Blood concentrations of epinephrine and cortisol rise when exercise intensity exceed 60% of VO_2 max. Epinephrine effects circulating leukocyte numbers during brief (<1 hr) exercise, while cortisol only comes into play after two to three hours exercise and during recovery. There is a high correlation between serum cortisol levels and total white cell count post-exercise.[14,15] In fact, Belgian researchers have proposed that the 3h post-exercise neutrophil-to-lymphocyte ratio is a reliable index of exercise-induced stress in race horses because of its high correlation with post-exercise plasma cortisol concentrations.[16] Between 3-4 hours, the total number of leukocytes in the vascular compartment seems to reach a plateau, representing a temporary homeostatic readjustment to the prevailing higher plasma cortisol concentration.[2]

Exercise and lymphocytes

Resting lymphocyte numbers are usually normal in athletes.[2] A lymphocytosis, proportional to exercise intensity and fitness level occurs during and immediately after exercise of as little as 10 minutes to several hours

duration. Return to baseline levels follows a similar time-sequence to that of the leukocytes, but lymphocytes numbers may drop to below pre-exercise levels during recovery.

The various lymphocyte subsets appear to respond differently to exercise. This is partly explained by their complexity and the techniques used to measure them. What has been consistently demonstrated is that there is a proportionally greater increase in B cells, natural killer (NK) cells and monocytes than in T cells. The T:B ratio decreases, although there does not appear to be a significant change in the T helper:T suppressor ratio. Most of these changes are transient and exercise training has no long-term effect on lymphocyte distribution.[17] Cannon[18] argues that these acute and transient effects are often attributable to circadian rhythms or hemoconcentration effects. Studies claiming post-exercise 'immunosuppression' rarely include measurements from non-exercising time controls, which may well exhibit a similar pattern of leukocyte distribution due simply to circadian variation.

Effects of exercise on lymphocyte function

Determination of the proliferative response (blastogenesis) of human lymphocytes upon stimulation with mitogens in vitro is a well-established test to evaluate the functional capacity of T lymphocytes. Exhaustive exercise suppresses mitogen-stimulated proliferation of separated mononuclear cells by 50%, while short to moderate exercise has little or no effect.[19] The decrease in mitogenic response is probably due to the relative decrease in T cells compared to NK cells in the post-exercise in vitro assay sample. That is, there is a smaller percentage of cells that respond to mitogens, due to the differential changes in circulating lymphocyte numbers.[3] Both cortisol and epinephrine inhibit mitogen-induced lymphocyte proliferation directly and indirectly via their effects on interleukin-2 production.[13] This may explain the reduction in lymphocyte proliferation after high but not moderate intensity exercise. Training may attenuate this suppression.[20]

Mechanisms of lymphocyte activation

Evidence from canine but not human studies is that exercise stimulates lymph flow from skeletal muscle and that this 'tissue pump' delivers large numbers of lymphocytes to the circulation, as does an increased perfusion of the lung.[11] Epinephrine promotes exercise-lymphocytosis, while cortisol exerts a limiting effect.

Exercise and innate immunity

The principle components of innate immunity are structural and chemical barriers that limit entry into the host. This first line of defence is augmented by phagocytic cells that kill foreign micro-organisms and release soluble factors that initiate the immune response.

Phagocytic functions increase, decrease or remain unchanged, depending on the type of exercise and the source of cells.[21] Macrophage activity appears to be enhanced by exercise, and there is a suggestion that exercise may enhance macrophage secretion of tumour necrosis factor (TNF) that inhibits tumour growth.

Neutrophil activation and microbicidal activity is either enhanced or unchanged by exercise,[6,20] and appears to be lower in trained athletes than non athletes.[20] Moderate exercise is associated with a prolonged improvement in the killing capacity of neutrophils, while exhaustive exercise may have the opposite effect. Both complement and C-reactive protein (CRP) levels tend to be lower in athletes than non-athletes, but increase after prolonged exercise.[14,23] While the lower resting acute phase protein concentrations may represent adjustments to chronic training-induced inflammation the implications of these changes for resistance to infection is unknown.[21]

Exercise and humoral (anti-body mediated) immunity

The B lymphocytes, when exposed to a specific antigen and/or in co-operation with macrophages, T lymphocytes and their soluble products, replicate and mature into plasma cells. These synthesise and secrete five classes of antibodies, the immunoglobulins IgG, IgM, IgA, IgG and IgD, which react with, and lead to the destruction of the antigen. Resistance to infection is due in part to the presence of sufficient levels of serum and secretory immunoglobulins, especially the antigen-specific IgG and IgA. A pool of antigen-specific lymphocytes forms the basis of long-term immunity for an individual.

Exercise and the Immunoglobulins

Most researchers have reported trained athletes to have resting serum immunoglobulin (Ig) levels within the normal reference range and similar to those of sedentary controls.[14,24] Intense exercise during regular training does not appear to alter serum Ig levels, although some elite athletes may experience low levels during the competitive season.[25,26] Serum Ig changes following less than 40km of running are generally minor, but can be depressed for up to two days after completion of a run longer than 40km. Prolonged endurance exercise has been associated with low salivary IgA levels.[27] In contrast, moderate exercise training may even improve Ig levels.[27]

Secretary IgA is a glue-like substance of the mucosal barrier that has anti-body activity against certain viruses, bacteria and common allergens. Exercise-induced decreases (within the clinically normal range) in salivary and nasal wash IgA levels have been reported in a variety of competitive athletes inducing Nordic skiers [28], cyclists[29], swimmers[30], runners[31], hockey and squash players and kayakers.[32] The intensity of the exercise session and the psychological stress of competition profoundly affect salivary IgA concentrations.[21]

These depressed Ig levels are implicated in increased susceptibility to bacterial and viral infections following prolonged exercise. Simon[1] argues however that exercise-induced alterations in secretory antibodies levels are not likely to be functionally significant, as they are so transient. He points out that the majority of the 1 in 650 adults who are entirely deficient in IgA are healthy and do not experience an increased incidence of infection.

Exercise and Immunoglobulin function

In general, following acute prolonged exercise or several weeks of heavy exercise training, in vivo and in vitro antibody production, at least in animal models remains unaltered.[20,33] Whether in vitro Ig synthesis is an accurate reflection of that occurring in vivo is however debatable. Helper T lymphocytes are essential for B cell differentiation and antibody synthesis. As there is a temporary reduction in the T helper ratio after exercise, the in vitro reduction in Ig synthesis could simply reflect the change in lymphocyte subsets at the time of sampling.

Exercise training may also enhance specific antibody formation in response to immunologic challenge. For example, higher titres of serum Ig specific to injected tetanus toxoid was observed in males completing a 42km marathon compared to controls. This occurred despite a significant decrease in mitogen-stimulated T-cell transformation.[7] However the sample size in this experiment was very small (4 runners, 59 controls). Trained mice immunised with injected *Salmonella typhi* produced markedly higher specific antibody than did the non-exercising controls.[34]

Mechanisms of exercise-induced immunoglobulin redistribution

Plasma volume changes explain most of the acute exercise-related increases in immunoglobulins, together with an increase in Ig-containing lymph flow into the vascular compartment.[35] However, over the long term, baseline Ig production does not appear to be affected by exercise, as evidenced by generally normal resting Ig levels in athletes (Green et al. 1981). There is as yet no explanation for the higher specific antibody response observed after acute exercise and training, although this may represent an enhanced capacity of the immune system to respond to antigenic challenge.[21]

Exercise and the cytokines

Monocytes and macrophages exert a pervasive influence on host defences by secreting cytokines, which play a central role in initiating and regulating the immune response. Principally, these proteins stimulate the cells mediating specific immunity and also induce a wide array of non-specific host defence adaptations.[18] There are few published studies on the effects of exercise on these intracellular messengers.

Interleukin-1 (IL-1) is a protein with pleitropic effects, produced by mononuclear cells in response to endotoxin, immune complexes, phagocytosis and other stimuli such as exercise. Interleukin-1 and interleukin-6 (IL-6) levels and activity has been reported to increase during or in the recovery period after exercise,[15,36,37] and to be higher in endurance athletes at rest compared to non-athletes.[38]

These observations of exercise-induced increases in IL-1 provide a potentially important link between exercise and host defence mechanisms. Firstly, IL-1 mediates the acute phase response to infection and inflammation by, among other things, producing a leukocytosis. It is reasonable to speculate that IL-1 exerts a similar effect during exercise. Secondly, IL-1 increases the activity of both B and T lymphocytes. However, whether this is related to changes in immune functioning has not been established. It is possible that the immunodepressive effects of IL-1 are countered by a decrease in the Th:Ts ratio or other factors that oppose IL-1.[36]

Interleukin-2 (IL-2) levels in plasma and in vitro IL-2 production are reduced following brief maximal exercise[39], possibly because of a shift in the Th:Ts ratio and the ability of the lymphocytes to respond to a immunogenic challenge. IL-2 production following prolonged exercise has not been measured.[21] IL-6 release is dependent on IL-1, and it is suggested that there is a co-ordinated release of IL-1, IL-6 and CRP during or after exercise, which may be related to muscle damage and proteolysis, or a general inflammatory response.[15,38] Both interferon (IFN) levels and activity increase transiently after one hour moderate exercise.[40] Although IFN exhibits anti-viral activity, stimulating cytotoxic cells and inhibiting viral replication the transient and minor fluctuations measured post exercise are not likely to have any biological significance.[1] Likewise, tumour necrosis factor (TNF) levels are moderately elevated post-exercise in trained and untrained individuals[41], but the functional significance is unknown.[21]

Levels of all cytokines studied to date appear to be within clinically normal ranges, even when elevated after exercise.[41] It is impossible to determine the effects on immune functioning of an exercise-induced change in a single cytokine. Biological activity of any cytokine depends on a complex network of interactions, and cytokines may behave differently in vivo compared to in vitro.

Exercise and cytotoxic cells

Cytotoxic (killing) activity is exhibited by several types of immune cells, in particular cytotoxic T lymphocytes, natural killer (NK) cells and monocytes/macrophages. The effects of exercise on cytotoxic cells, particularly NK cells, is currently the focus of much research interest. Exercise appears to influence host resistance to both cancer and viral infections, due to its effect on the cytokines involved in resistance to tumour growth and viral infection.

Exercise and NK cells

NK cells, which comprise about 15% of lymphocytes recognise and kill virally infected cells, certain tumour cells and some micro-organisms without prior exposure. They also exhibit spontaneous cytolytic activity. Total NK activity is generally increased during and immediately after brief, prolonged, moderate and intense exercise, and contributes substantially to the overall lymphocytosis (for review see MacKinnon).[42] NK cells are selectively recruited into the circulation early in exercise. NK cells have a greater density of beta2 adrenergic receptors than do other lymphocytes, which explains why circulating levels increase dramatically after high intensity exercise when epinephrine concentrations are high.[12] Possibly epinephrine, IL-1, IFN, TNF and B-endorphin act synergistically to augment NK activity during exercise.

Immediately following exercise the cytotoxic capacity of the blood is markedly improved (by 40-100%)[43], but the effect is transient. One to two hours after maximal or prolonged exercise, NK activity is low, due to cortisol-mediated decrease in total circulating numbers and the inhibitory influence of prostaglandins from activated monocytes and neutrophils.[44] Nieman[3] argues that per cell, NK cytotoxic activity is actually increased after high but not moderate intensity exercise. There are simply less NK cells around, due to post-exercise egress to peripheral tissues. However, even this is a transitory effect, as baseline NK activity is restored around 6 hours post-exercise.[43] Resting NK activity in athletes may be slightly elevated compared to non-athletes, but within the normal range.[45]

Exercise and non-specific host defence mechanisms

Anatomical and physiological barriers form the first line of defence against infection, by preventing penetration of micro-organisms from the environment and the cutaneous and mucosal surfaces of the body into vulnerable body tissues. Examples of these non-immunological mechanisms are the skin and mucous membranes, the cough and gag reflexes, gastric acidity and intestinal motility.

The stress of exercise has a negligible effect on non-

specific defence mechanisms. Strenuous exercise and sports can result in trauma that disrupts the integrity of the cutaneous barrier to infection, but this is not usually a clinically important problem. Maceration through perspiration can predispose to annoying superficial fungal infections such as athletes foot.[1]

Exercise and the acute phase response

The acute phase response involves the complement system, neutrophils, macrophages, various cytokines and acute phase proteins acting together to clear damaged tissue and initiate repair and regrowth.[46] Lymphocytes, neutrophils and macrophages are attracted to the injured muscle cells where they variously phagocytize tissue debris and release soluble products such as cytokines which mediate the inflammatory response .[3] During this process, macrophages produce prostaglandin E_2 which suppresses NK cell activity. There is speculation that when muscle damage is severe, the immune system, partly disabled by prostaglandin is unable to perform its other host-defence duties. That this renders the individual susceptible to opportunistic infection has yet to be substantiated by objective measurement.[3]

The immune response to chronic exercise

Several studies have made cross-sectional comparisons of the immune systems of athletes and non-athletes,[24,45] or followed immune parameters in sedentary individuals undergoing exercise training.[17] Most of these studies have failed to demonstrate any important effects of regular exercise training on circulating concentrations of total leukocytes or lymphocytes or their various subpopulations. Generally, trained athletes have resting serum immunoglobulin levels within the normal reference range and similar to those of sedentary controls .[27] Longitudinal studies of collegiate teams over a competitive season show decrements in specific immune parameters, which could however be simply due to established seasonal variations in immune function.[50] However, the question of whether the exercise-induced transitory insults to leukocyte functioning are related to illness susceptibility is still open to speculation.[21]

Long term leukocyte function is relatively unaffected and mitogen-stimulated lymphocyte proliferation is not altered by exercise training in young adults.[47] Suppression of lymphocyte function has been reported in exhaustively trained athletes[48] and neutrophil-killing activity was found to be lower in elite athletes compared with untrained controls.[22] In contrast, NK cell activity is improved with exercise training.[45,49] Nevertheless, results from animal studies consistently show that chronic exertion is related to negative changes in immune functioning.[8,22]

Towards an integrated model of the immune response to exercise

There are no data on the effects of exercise on the immune system as a whole, and no models that can fully explain the complex interaction of the physiological responses to exercise and how these influence immune function. While individual models may vary in complexity, all have neuroendocrine factors playing a pivotal role.

Psychoneuroimmunology (PNI) or behavioural immunology is an emerging field that is concerned with the study of the relationships among psychological variables (such as stressors or affective states such as anxiety and depression), the nervous system and the immune system. It is generally accepted that there is a two-way communication between the neuroendocrine and immune systems.

Exercise, as a form of physical stress can be considered one of the many lifestyle factors such as diet, sleep or work stress that influence the immune system. In addition, exercise training may reduce negative affective states, increase the release of endogenous opiates, reduce HPAC activation and enhance immunity.[53]

Many hormones capable of immunomodulation are released during exercise, and affected by training. Smith and Weidemann[51] propose that moderate exercise increases the release of immunostimulatory hormones, such as growth hormone, prolactin and B endorphin, as well as cytokines such as IL-2 and TNF. Immonosuppressive hormones such as cortisol and catecholamines are generated during intense exercise. The latter would provide some explanation for the anecdotal evidence of increased rates of infection in elite athletes at times of intense physical and emotional stress such as occurs during major competitions.

Pedersen and Ullum[52] propose an 'open window' hypothesis. During moderate and severe exercise, immune system functioning is enhanced, but followed by a period of immuno-depression post-exercise. During this 'open window' period, microbacterial agents may invade the host, and infection be established. They suggest that regular moderate exercise may enhance immune functioning, thereby protecting the individual even after a severe exercise bout.

Exercise, stress and illness can be viewed as three points on a triangle. Each has independent effects on the immune system, while being mutually interactive.[54]

For every individual, there is an optimal level of regular physical activity conducive to illness resistance.[54]

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Exercise and upper respiratory tract infections: A review

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ABSTRACT

Despite equivocal findings in early laboratory and longitudinal studies investigating the relationship between exercise and infection risk, recent carefully controlled work appears to point towards a paradoxical response of the host defence and immune systems to endurance exercise; whereas long-term regular training is thought to stimulate these systems, acute exposure to exhaustive endurance exercise has been associated wiith transient suppression thereof. Several studies have confirmed that athletes are more susceptible to upper respiratory tract infection (URTI) symptoms following participation in competitive marathon and ultramarathon running events, while others have produced evidence of a protective effect and lower incidences of URTI symptoms following prolonged endurance training programmes. Nieman (1993) proposes a "J" shaped relationship between URTI risk and exercise intensity, whereas Pederson and Ullman (1994) describe an " open window" period during which the athlete is most vulnerable to infection. How is the athlete best managed during periods of heavy training and competition? What influences the size of the "open window" and URTI risk? These are some of the questions to be addressed in this paper.....

INTRODUCTION

Upper Respiratory Tract Infection (URTI) frequently results in inopportune disruption of the training programmes of serious athletes. In 1975 Ryan et al[1] reached the conclusion that "upper respiratory illness causes more disability among athletes than all other diseases combined." This was confirmed by Berglund and Hemmingsson[2] who reported that infectious diseases, of which URTI was the most common, was the main medical reason for absence from training in elite skiers monitored over a 12 month period [Figure 1].

Regular prolonged exercise, is however, generally recognised as a therapeutic modality providing numerous health benefits; cardiovascular, respiratory and metabolic adaptations have indeed been linked to longevity.[3] Many also believe that regular aerobic exercise training improves their resistance to infection and anecdotal reports of fewer colds and URTI infections in well-conditioned athletes abound.[4]

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Studies investigating URTI incidence in sportspersons reveal a paradoxical response. On the one hand, a high level of physical conditioning has, in few carefully controlled recent studies, provided evidence of a lower incidence of infection symptoms which is possibly associated with chronic immunomodulation and an increased resistance to infection.[5,6,7,8] On the other hand, overtraining and the combined psycho-physical stress of competitive endurance events and acute bouts of exhaustive endurance exercise have been linked to an increased incidence of URTI symptoms possibly associated with transient suppression of host and immune defences.[9,10,11,12]

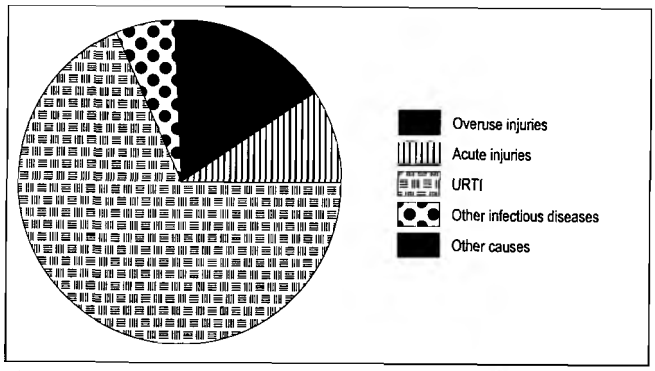

Figure 1: URTI, the main medical reason for absence from training in elite Swedish cross country skiers. Data adapted from Berglund and Hemmingsson.[2]

Nieman[13] has graphically depicted the relationship between exercise and URTI incidence in a "J" shaped model proposing that a moderate and regular exercise load can reduce URTI risk to below that found in sedentary population, whereas a high volume and intensity of exercise can increase URTI risk to almost double that found in the sedentary population [Figure 2].

It is thus the purpose of this review to provide a brief overview of the epidemiological and laboratory evidence which is currently available in support of this dual response and to provide practical guidelines to the athlete, coach and medical practitioner regarding the prophylactic management of the athlete during periods of increased susceptibility to infection and treatment of the athlete once URTI has set in.

CHRONIC TRAINING AND URTI INFECTION RISK

A number of longitudinal studies have been designed specifically to consider the chronic effects of repeated bouts of exercise (ie training and racing) on the incidence of URTI. While Linde[14] found that on average elite Danish orienteers experienced 2,5 infectious episodes (lasting for more than three days) during a one-year period as opposed to 1,7 in non-athletic controls and a higher average duration of symptoms, Health et al[15], examining the illness patterns of a cohort of 530 male and female runners over a period of 12

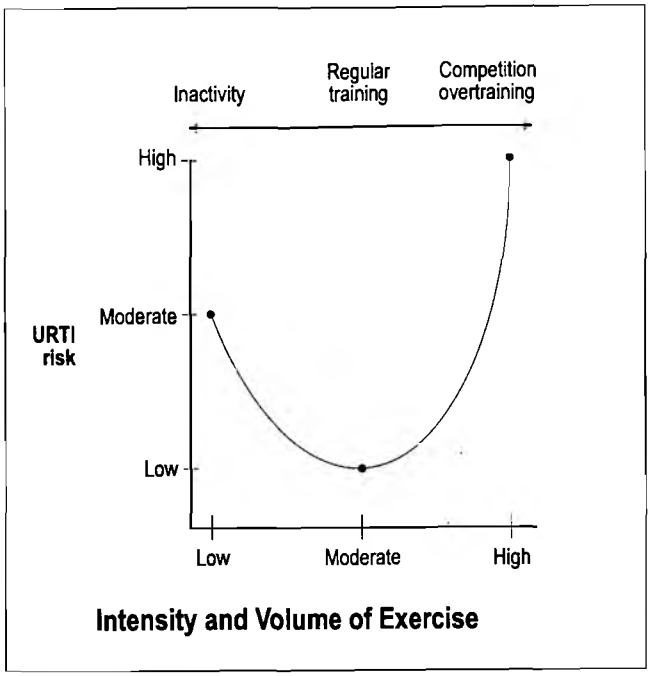

Figure 2: "J" Shaped model of relationship between ranging amounts of exercise and risk of URTI. This model suggests that moderate exercise may lower risk of respiratory infection below that evidenced in sedentary individuals while excessive amounts may increase the risk. Adapted with permission from Nieman.[13]

months, found that the average number of self-reported illness patterns per runner per year was only 1.2 and suggested that chronic high mileage training may pose as great a risk of an infectious episode as racing.[18] A relatively low incidence of URTI symptoms was confirmed by Berglund and Hemmingsson[2] who reported an average of 1,5 and 1,88 URTI infections per year in groups of Swedish national team [n=36] and elite college [n=136] cross country skiers. This study further revealed the incidence of URTI symptoms to be greatest in the winter months. Osterback and Qvarnberg[16], surveying the incidence of respiratory infections in 12yr old children participating in swimming, ice-hockey and gymnastics on a regular basis, however, found no differences in infection rates when these children were compared to an age-matched control group.

Schouten et al[17] studied the relationship between the incidence and duration of URTI symptoms (over a 6 month period) and level of sport activity in 92 men and 107 women participating in the Amsterdam Growth and Health study and found statistically negative correlation between the incidence of URTI symptoms and the level of sport activity in the women only.

Variance in the findings of these longitudinal studies could well be attributed to disparity in the quality, quantity and frequency of the training completed by the subjects studied. More recent randomized, well-controlled studies on circulating immune system variables and incidence of URTI symptoms, however, may support the common belief that a moderate level of physical training may lower infection risk.

Nieman et al[6,7] performed two tightly controlled consecutive studies which investigated the effect of endurance training on the incidence of URTI symptomatology. Firstly, in a randomised controlled study of 36 women (mean age 36 years) five 45 min sessions.wk $^{-1}$ of brisk walking at 60% heart-rate reserve resulted in a significant reduction in URTI symptomatology with less than 50% the number of days with symptoms in the walkers than in the sedentary control group over the 15 week period[7] [Figure 3]. In a later study URTI symptomatology in a group of highly conditioned elderly female subjects who exercised moderately each day for about 1,5 hr [n=12] was compared to a group exercising for 40 min 5 times per week [n=14] and control group who only participated in calisthenic exercises [n=16] over a 12 wk period.[7] The lowest incidence (8%) of URTI was reported in the highly-conditioned group who exercised daily, followed by the next lowest incidence in the group which trained at a moderate intensity 5d.wk^{-1} over the 12 wk period [Figure 4]. As the highly-conditioned distance runners trained more frequently than the lower-distance runners (4.6 vs 2.7 sessions per week), frequency of exercise was suggested as a factor which may improve immunosurveillance.[7]

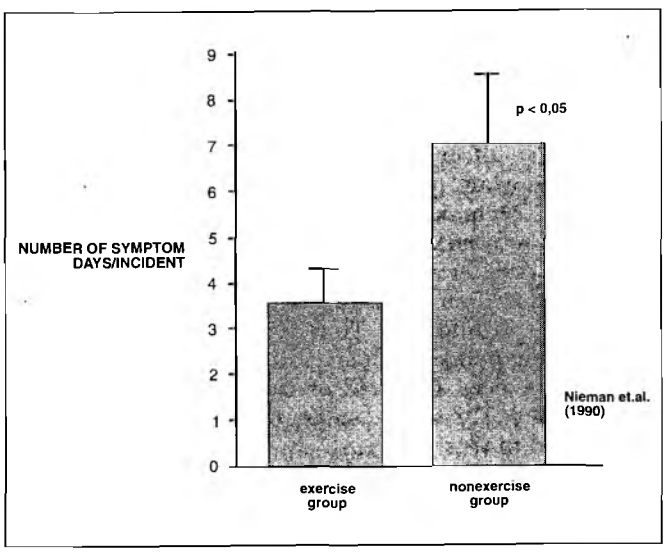

Figure 3: Mean number of symptom days per URTI incident in exercise and non-exercise groups during a 15 wk study period. Adapted with permission from Nieman et al.[6]

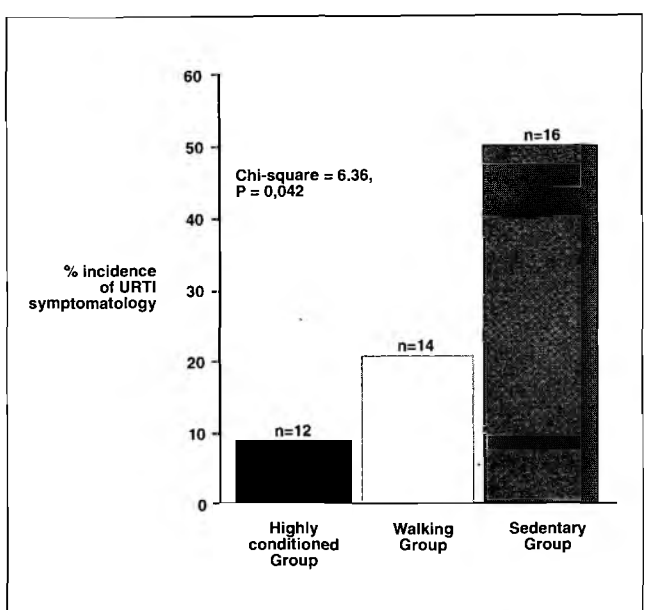

Figure 4: Incidence of URTI (expressed as a percentage of the group) during a 12 wk study period in highly conditioned, walking and sedentary control groups women (n=42). Adapted with permission from Nieman et al.[7]

Mackinnon and Hooper[5] examining secretory IgA response to various exercise conditions, support these findings showing evidence of a cumulative effect of intense daily exercise on local mucosal resistance against pathogenic organisms causing URTI. A group of well trained Australian swimmers presented with significantly higher secretory IgA levels than "stale", overtrained swimmers over a 6 month session. In contrast, previous studies have shown that salivary IgA levels are depressed in elite well trained athletes[18,19] and linked these to an increased risk of infection in elite athletes.[19] These data may, however, also point to an endurance training threshold above which URTI risk is increased supporting the "J" shaped model proposed by Nieman.[13]

ACUTE EFFECTS OF COMPETITIVE PROLONGED EXERCISE ON URTI RISK

A number of epidemiological surveys performed locally and abroad in the last 12 years suggest that athletes engaging in marathon and ultramarathon type events are at increased risk for URTI during the two-week post race period.[20,21,22,23,24,25,26]

The first preliminary investigation was conducted by Peters and Bateman[20] at the 1982 Two-Oceans Ultramarathon (56 km) in Cape Town. A simple epidemiological survey on 150 successful finishers in this race, and their age-matched non-running controls who resided in the same households, revealed that whereas a mere 15.3% of the non-running controls reported URTI symptoms during the two-week post-race period, thirty-three percent of the runners completing the race reported URTI symptoms during this same period [Figure 5]. The incidence of URTI symptoms was highest in the fastest runners ($p > 0.01$) and 47% of those completing the 56 km race in less than 4 hrs reported post-race URTI symptoms. Sore throats and nasal symptoms were the most prevalent with more than 80% of the symptoms reported lasting more than 4 days.

The finding of a significantly higher incidence of URTI symptoms among runners during the post-race period was confirmed by Peters in 1989[21] when the study was repeated at the Milo Korkie Marathon , a 56 km race taking place between Johannesburg and Pretoria [Figure 5]. Nasal symptoms and sore throats were once again the most prevalent of self-reported symptoms during the two-week post-race period and 39% of symptoms lasted more than 7 days. In this study, it was the runners in the low pre-race training status group who had the highest incidence of post-race infection.

To date, an increased incidence of post-race URTI symptoms has, however, only been reported following events falling into the marathon and ultramarathon categories. A two-month investigation conducted into the pre- and post-event incidence of URTI in a group of 273 participants in 5,10 and 20 km events in California[22] revealed no increase in URTI incidence in the runners during the 7-d post race period when compared to the incidence in the week prior to the race. Although the URTI incidence was highest (33.3%; $p = 0.092$) in runners completing less than 25 km.wk^{-1} in training, a correlation between pre-race training distance and finishing times in the race was not found. As intensity of training is also an important factor in the preparation for these shorter events, weekly training distance may not provide an accurate reflection of pre-race training status in participants in shorter races.

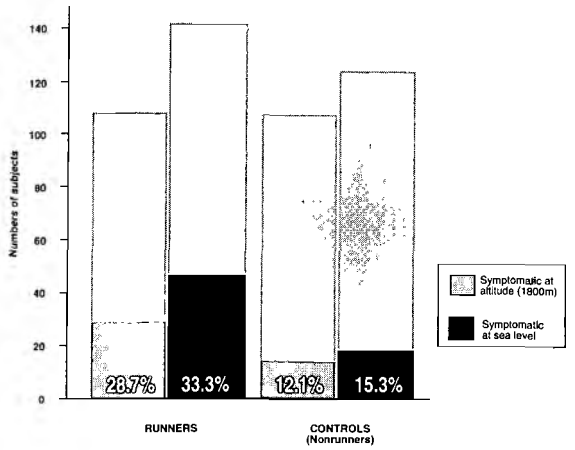

Figure 5: The incidence of URTI symptoms during the post-race fortnight in runners completing 56 km Ultramarathon at sea level and moderate altitude and matched sedentary controls. Data from Peters and

An investigation into the URTI symptom incidence in 2311 participants before and after the Los Angeles Marathon(LAM)[23] revealed an increase in odds ratio of infectious episodes (IE) with an increase in pre-race training distance (km.wk^{-1}) ($p = 0.04$). Reported incidence of illness was highest in those runners who completed >97km.wk^{-1} while training in preparation for the event. Of the 1828 LAM competitors without infectious episodes (IE) before the race, 12.9% reported IE during the week following LAM vs 2.2% in controls (well trained non-participating runners). These researchers concluded that runners may experience increased odds for IE during heavy training or following a marathon race.

More recent nutritional intervention studies performed by Peters et al[24,25,26] on participants in the 90 km Comrades Marathon, also confirm these findings of greater incidence of infection in the runners during the post-race fortnight. Of interest are the observations, that in the case of the 90 km ultramarathon, both conditions of "under-" and "overtraining" appeared to predispose to higher post race URTI risk. [24,25,26]

RECENT HYPOTHESES SUPPORTED BY LABORATORY FINDINGS

The paradoxical response to exercise

A perspective which explains the dual, paradoxical response to exercise is provided by Smith and Weiderman[27] who propose that while moderate exercise activates the immunostimulatory release of growth hormone, prolactin and cytokines into the circulation resulting in both neutrophilia and increased neutrophilic activity[27,28], during very exhaustive prolonged exercise, the immunosuppressive arm of the pituitary-adrenal axis is activated with subsequent release of ACTH and cortisol and suppression of the activity of the polymorphonuclear neutrophils(PMNs.)[27]

A further explanation of the exercise paradox is contained in the theory that initial pro-oxidative effect on PMN function becomes immunosuppressive as exercise is prolonged and microbicidal reactive oxygen species produced by the PMNs have an auto-oxidative effect reducing the "killing capacity" of the PMNs.[29] This was recently confirmed in a study conducted on the nasal

lavage of runners completing a 20 km event.[30] Although the total number of PMNs rose, percentage active phagocytes and number of ingested microorganisms were reduced.[30]

Further Smith et al[29], comparing neutrophil oxygenative activity in elite trained Australian cyclists to that in untrained subjects, showed that endurance training decreases the auto-oxidative phagocytic activity in the PMNs. Further evidence of reduced oxidative activity in trained individuals has most recently been reported by Pyne et al[31] who in a study conducted on elite swimmers over a 6 month period showed that (i) elite swimmers undertaking intensive training have a significantly lower neutrophil oxidative activity at rest than do age- and sex-matched sedentary individuals and (ii) aspects of oxidative activity in swimmers are further suppressed during periods of strenuous training. In this study the extent of the suppression did, however, not appear to be of clinical significance as no significant difference was reported in URTI incidence between swimmers and sedentary individuals.[31]

Increased post-event susceptibility to URTI

Consistent evidence exists of increased post-race URTI symptomolology in runners competing intensively in events of the marathon-ultramarathon category. Many existing laboratory findings also appear to lend support to these epidemiological findings and, in particular, the recent hypothesis of Pederson and Ullman[32] that the during first few hours following strenuous exertion, an "open window" exists. This Danish group contend that previously reported reversal of the exercise-induced increment in leucocyte number,[33,34,35,36,37] commonly referred to as a "biphasic" response[9], contributes, together with the post-exercise decrease in NK cell number[32,38], complement[39] and mucosal IgA levels[5], to transient immunosuppression during this open window period [Figure 6]. Pederson and Ullman suggest that, at this time, the athlete is most vulnerable to infection; microbacterial agents can invade the host and infections are easily established [Figure 6]. According to the findings of Nieman[9,13,35] the phase of transitory immunosuppression lasts between 6 and 20 hours post-exercise.[35]

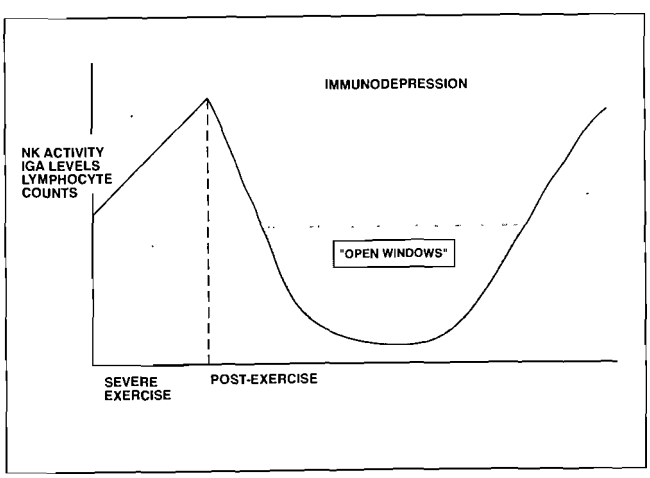

Figure 6: The open-window hypothesis of Pederson and Ullman.[42] Severe exercise is followed by a period of transitory immunodepression during which the athlete possesses an increased susceptibility to infection. Adapted from Pederson and Ullman.[42]

MANAGEMENT OF THE ATHLETE ... PRACTICAL GUIDELINES FOR ATHLETE, COACH AND CLINICIAN

Advice for the athlete in possession of URTI symptoms

The effects of systemic infections on impairing muscle and circulatory function and reducing physical work capacity are well established.[40] Nieman[9] thus proposes that if an athlete experiences sudden and unexpected deterioration in performance during training or competition, viral infection should be suspected. While Fitzgerald[41] warns that exercising during the incubation period may worsen the illness, participation during the presence of systemic infection is generally regarded as counterproductive resulting in both poor performance and a prolongation of the recovery period.[12] In view of the additional risk of myocarditis, a recognized, although rare cause of death in exercising individuals possessing influenza virus[42], athletes with constitutional symptoms of infection should not be expected to participate in competition; even strenuous training should be discouraged in the presence of fever, myalgia, swollen lymph glands, extreme tiredness or other symptoms which are suggestive of systemic infection.[12] Two to four weeks should probably be allowed before resuming hard training.[9]

However, in the case of mild common cold with no constitutional symptoms, complete interruption of exercise schedules is not generally required and mild exercise does not appear to be contraindicated. In fact, some individuals report relief of symptoms, probably, according to Simon[12], because of the increased mucous flow associated with exercise. Simon[12] recommends gentle stretching sessions, permitting ordinary daily activities to the limit of tolerance with a programme of graded aerobic exercise and reassurance on resumption of training. Most clinical authorities in this area[12,43] recommend that regular training may be safely resumed a few days after the resolution of symptoms and warn that admonitions for prolonged bed rest are responsible for deconditioning which can give rise to protracted fatigue even after infection has subsided.

Eichner[43] recommends a "neck-check". He suggests that if the athlete has "below-the-neck" symptoms such as aching muscles, a hacking cough, vomiting or diarrhoea, all training should be halted; in the case of "above-the-neck" symptoms including stuffy or runny nose, sneezing or a scratchy throat, he recommends that athletes "go ahead and plough cautiously" through their scheduled work-out. "If, after 10 minutes, your head is clear and you feel better, you can speed up and finish your workout. If, instead, your head pounds and you feel like you are running through water, stop, go home and rest!", advises Eichner.

In terms of the use of cold remedies, caution is advised. Most South Africans recall the unfortunate controversy surrounding the stripping of the title from Comrades gold-medallist, Charl Matheus, in 1992. Most cold remedies do contain sympathominetric agents which are on the list of banned substances. Disqualification of athletes from high-level competitive events is thus often a possibility when cold remedies are being used.

The advisability athletes using anti-biotics when contracting an apparently severe local bacterial infection, is an area which does not yet appear to have been investigated. Whereas this would no doubt assist in early combating of the infectious microorganisms and prevent extension of nasal infections into the lower respiratory tract and ears, hence resulting in a shortening of the lay-off period, anecdotal reports of transitory periods of general weakness and lethargy resulting from anti-biotic ingestion, do abound. At this stage it would appear that athlete and clinician would need to weigh up the advantages of a greater possibility of overcoming the infection sooner against the transitory period of reduced performance which may result from the intake of anti-biotics. As large individual differences in response to administration of anti-biotics may also exist, well controlled clinical studies are needed in this area.

Prophylactic measures

(1) *Avoidance of exposure to infectious agents*

Since endurance exercise is associated with muscle cell damage and an increased intake of potential pathogens through increased ventilatory flow rates, Weidner[44] suggests that particularly during the winter months, exposure to the cold virus in crowded dormitories, classrooms, and gymnasiums perhaps accounts for the high incidence of colds during the cooler months. He further recommends "careful handwashing, avoidance of skin-to-skin contact or contact with contaminated tissues, sporting equipment, and appliances."

(2) *Immunization of athletes*

At present, the evidence regarding effectiveness of the immunization of athletes against influenza appears to be anecdotal and variant; successful in some individuals, but bringing on flulike symptoms in others. Melman[43] reports the use of the anti-viral agents amantadine hydrochloride and rimantadine hydrochloride which are both effective against influenza A as a prophylactic measure among the group when the first athletes develop flulike symptoms. This may be particularly helpful for athletes who play winter sports as close contact among teammates can result in rapid spread of influenza and thus disrupt the whole season.[13]

(3) *Enhanced intake of Vitamin C*

Recent placebo-controlled studies performed on Comrades athletes[25,26] have provided evidence of decreased incidences of URTI symptoms during the post-race "open window" period following three weeks of supplementation with Vitamin C, and Beta Carotene-Vitamin C and E combinations. In both studies a total daily intake of approx 1g Vit C a day resulted in a significantly lower incidence of URTI symptoms during the two-week post-race period when compared to the incidence in runners receiving placebo's. We hypothesised that phagocytic production of reactive oxygen species (ROS) during prolonged exercise suppressed neutophilic function and that the anti-oxidant properties of Vitamin C contributed to a neutralization of the ROS, lifting the transient post-race immunosuppression. Although these hypotheses are supported by laboratory work on patients possessing auto-immune respiratory disorders and a link between plasma Vitamin C levels and neutrophilic activity has been established, it does require further verification on athletes participating in ultradistance events. At this stage, anecdotal reports of many coaches who find it of benefit to increase Vitamin C intake of athletes in heavy training, particularly during the winter months, do abound. As Vitamin C is water-soluble and no negative side-effects have been documented at these levels, an enhanced Vitamin C intake is not contra-indicated as a prophylactic measure.

(4) *Training progression*

Careful attention should be paid by coaches to slow progression in training intensity and duration and the avoidance of overtrained conditions and the resultant immunocompromising effects thereof.[39] Coaches report that more frequent, less intense sessions, have been shown to be more effective in building resistance to infection than very heavy training sessions at less frequent intervals. As evidence in this field is largely anecdotal, well controlled studies are, however, required.

CONCLUSION

Although many findings in this field remain inconclusive, current evidence would appear to suggest that while exercise provides favourable physical and psychological stress, a therapeutic and immunostimulatory effect is obtained. It would seem that once exercise is too intense, frequent and/or long in duration, particularly when coupled with exposure to pathogenic microorganisms, the favourable stress situation develops into one of distress and a concomitant increment in susceptibility to infections, results. Greater awareness of the predisposing factors can assist coach, athlete and clinician in lessening the onslaught and reducing the length of the lay-off period when the athlete is exposed to a high bacteriological/virological count.

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HIV infection in sport: A review of current issues

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ABSTRACT
Regular participation in physical activity is well recognized as an important preventative health measure. In contrast, in the last decade, human immunodeficiency virus (HIV) infection has become a major global public health threat. The impact of HIV infection is likely to have a major effect on the South African population as a whole, including the sporting population. In this article issues relating to HIV infection and participation in physical activity are reviewed. These issues include the risk of HIV transmission during sports participation, the effects of HIV infection on exercise performance and the effects of regular exercise on the clinical outcome of HIV infection. In addition practical guidelines are offered to i) decrease the risk of HIV transmission during sports participation, and ii) to ensure safe participation in physical activity for patients with documented HIV disease.

INTRODUCTION
The health benefits of regular participation in physical activity are well established (Astrand 1992). Regular physical exercise is an essential component of a healthy lifestyle. Public awareness about the health benefits of regular exercise has resulted in an increase in the number of participants in all types of sport (contact and non-contact sport). It is estimated that more than 4.3% of the South African population regularly participate in sport (HSRC 1982). Recent surveys indicate that the most popular sport in South Africa is soccer followed in popularity by racquet ball sports, athletics and tennis (COSAS, 1990). In contrast to positive lifestyle changes such as regular exercise, the HIV pandemic has in recent years become a major global as well as South African public health threat (Ijsselmuiden et al, 1988a, Schall 1990, Schoub et al 1990).

The acquired immune deficiency syndrome (AIDS) was first recognized as a clinical entity by the Centers for Disease Control (CDC) in 1981 with the description of 5 cases of Pneumocystis carinii infection and 26 cases of Kaposi's sarcoma in homosexual males (MMWR 1981a, MMWR 1981b). The first cases of AIDS in South Africa were described one year later (Ras et al 1982). The transmission (Friedland et al 1987, Lifson 1988), clinical features (Sher 1988), epidemiology (Moodie 1988a), serology (Moodie 1988b), and mortality associated with human immunodeficiency virus (HIV) infection have been well described. Although there is considerable debate on the precise impact of the HIV epidemic on the South African society (Schall 1990), it seems certain this disease will have a considerable effect on the health of the South African population, including the sporting population. The association between physical activity and HIV infection can therefore not be ignored. Specific issues that need to be considered are the risk of HIV transmission during sports participation, the effect of HIV infection on sports performance and the effects of regular physical activity on the clinical outcome of HIV infection.

The aim of this article is i) to review the current knowledge on the risk of HIV transmission during sports participation, ii) to establish guidelines to reduce the possible risk of HIV transmission during sports participation, iii) to review the effects of HIV infection on exercise performance and iv) to review the effects of regular physical activity on the outcome of HIV infection.

The risk of HIV transmission during sports participation
Since its discovery in 1983, the human immunodeficiency virus (HIV) has been isolated in blood, semen, cervical secretions, lymphocytes, serum, plasma, cerebrospinal fluid, tears, saliva, urine, breast-milk and alveolar fluid of infected subjects (Friedland et al 1987, Ziegler et al 1986). However, the transmission of the virus from one individual to another has only been linked directly to blood, semen and cervical secretions with likely transmission occurring through breast-feeding (Lifson 1988). The primary routes of transmission are therefore by sexual contact with an infected person, parenteral exposure to infected blood or blood products and perinatally from an infected mother to her child.

Prior to 1989, there has been no documentation of HIV infection occurring as a result of participation in sports. However, the theoretical possibility of HIV transmission through open bleeding wounds in contact sports has been recognized by sports physicians (Alcena 1988, Loveday 1989). The first case of possible HIV transmission as a result of sports participation was published in 1990 (Torre et al 1990). This involved an Italian soccer player in whom HIV seroconversion was documented weeks after a traumatic incident during a soccer match. During the soccer match the player collided with another player who was later documented as being HIV seropositive. Both players sustained open bleeding wounds resulting in possible mixing of blood. There was no indication that the player may have been infected through any other route of HIV transmission. The authors concluded that this was the first case of

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HIV transmission which occurred directly as a result of sports participation (Torre et al 1990). Although this case report has been criticized on epidemiological grounds (Goldsmith 1992), it nevertheless has clear implications for the risk of HIV transmission during sports participation. In particular, those sportspeople that participate in contact sports such as boxing, wrestling, rugby and soccer are potentially at risk.

It must be emphasized that to date no cases of HIV transmission have been recorded in either the sporting or the non-sporting population through contact with saliva, social contact or sharing facilities such as living space, toilets, bathrooms, eating and cooking facilities (Loveday 1989). It must also be borne in mind that the risk of HIV infection in sportspeople is the same as that in the general population if there is a history of engaging in well established high risk behaviors for HIV transmission (Table 1).

Table 1 : High risk behaviour for HIV transmission

- Anal sexual intercourse with or without the use of a condom
- Sexual intercourse with multiple partners or with one person who has sexual intercourse with multiple partners (eg. prostitutes)
- Vaginal or anal sexual intercourse with someone who uses intravenous drugs or engages in anal sexual intercourse
- The sharing of needles, razors, toothbrushes or any instrument that may cause bleeding (including mouthguards)
- Sexual intercourse with an HIV infected person - especially if a condom is not used
- The use of drugs or excessive alcohol may result in participation in the above mentioned behavior patterns

Although there is documentation of only a single possible case of HIV disease as a result of sports participation in contact sports, there is a need to establish clear guidelines for the prevention of HIV transmission during sports participation. As early as 1989 the International Sports Medicine Federation (FIMS), together with the World Health Organization, published guidelines for the prevention of HIV transmission during contact sport (WHO position statement 1989). Subsequently, other organizations such as the Australian Sports Medicine Federation (Sheridan, 1992), the American Academy of Pediatrics (American Academy of Pediatrics, 1991), and most recently, the National Football League in the USA (Brown et al 1994) have published similar guidelines. In South Africa, to date only the South African Football Medical Association (SAFMA) have published an official position statement on HIV disease in sport.

The question that is foremost in the minds of sports administrators and participants, is what is the risk of HIV transmission in sport. In answer to this, there are no epidemiological data available to date to calculate the risk of HIV transmission during sports participation. At best, a theoretical risk of transmission in a sport can be calculated by considering the following variables (Sheridan, 1992):

- the estimated carrier rate of HIV in the sports participants (% participants that are HIV positive)
- the estimated chance of an open bleeding wound in a sports participant (Incidence of open bleeding wounds)
- the estimated chance of two players with open bleeding wounds making contact that could result in blood to abrasion or blood to mucous membrane exposure (incidence of physical contact between two participants)
- the estimated chance of transmission of the virus when infected blood makes contact with an open bleeding wound (estimated to be 0.3-0.5% which is similar to that calculated for a needlestick injury)

If all the above data are available for a particular sport, the estimated risk of HIV transmission can be calculated (Table 2). At present, accurate data are only available for one sport, American football (Calabrese 1993). In American football the risk of HIV transmission in a game has been calculated as 0.0000000104. This can be translated to approximately one player becoming infected per 100 million games. Clearly, this is a very low risk and is probably the reason why there is no widespread documentation of HIV infection in American football players.

Table 2: Calculation of the theoretical risk of HIV transmission in sport

Risk = Seroprevalence of HIV (%) X Risk of open bleeding wound (%) X Risk of contact with a bleeding player (%) X 0.03 *

*: Estimated to be similar to the risk of seroconversion after a needlestick injury

However, it must be pointed out that the seroprevalence in American football players was estimated to be 0.5%, and that both the risk of an open bleeding wound (0.9%) and the risk of contact between players (7.7%) were low. These estimates will differ between different populations (higher seroprevalence) and sports (higher risks of bleeding and player contact). For instance, in a boxing fight of 12 rounds, the risk of an open bleeding wound is approximately 33%, and the risk of contact is probably 100%. The risk of seroconversion after contact between two boxers may also be higher than that of a needlestick injury for at least two reasons. Blood may be forced into the wound by the nature of the blow, and contact may be repetitive. Finally, if the seroprevalence of HIV in boxers is high, the risk of transmission in boxing may be much higher than that reported for American Football. There is thus a need to obtain accurate research data on the incidence and nature of open bleeding wound injuries as well as the risk of contact between players if more accurate assessments on the risk of HIV transmission during sports are to be made.

Despite the lack of accurate scientific data, the prevention of HIV disease in sportspeople has to be addressed by establishing clear guidelines for sports participants, administrators and medical personnel involved in sport. A number of practical recommendations can be made to decrease the risk of HIV transmission in sport. These are summarized in Table 3.

Table 3: Guidelines for the prevention of HIV transmission in sports

a. General guidelines:

The following general guidelines are suggested to reduce the risk of transmission of HIV in sport:

- In general the risk of HIV transmission as a result of sports participation is very low
- The risk is higher in contact sports where there is a risk of transmission through contamination of open lesions, wounds or mucous membranes of a non-infected individual with infected blood or blood products
- At present there is no risk of transmission from saliva, sweat, tears, urine, respiratory droplets, hand-shaking, swimming pool water, communal bath water, toilets, food or drinking water

b. Specific guidelines for sportspeople:

The following are specific guidelines for the individual sportsperson to reduce the risk of HIV transmission during sports:

- A sportsperson who engages in high risk behavior (Table 1) is advised to seek medical attention regarding possible HIV infection
- Sportspersons with known HIV infection should seek medical and legal counselling before considering further participation in sport in order to assess risks to their own health as well as the theoretical risk of HIV transmission to other sportspeople
- Sportspeople with known HIV infection should inform medical personnel of their condition if they sustain an open wound or skin lesions during sports participation so that these can be managed appropriately

c. Specific guidelines for sports administrators:

Sports administrators, including coaches and managers have special opportunities for meaningful education of sportspeople with respect to HIV disease. They should encourage sportspeople to seek medical counselling where appropriate. Finally, they also have an important role in ensuring that adequate medical care is available for their sportspeople.

d. Specific guidelines for medical personnel attending to sportspeople:

Guidelines for medical personnel in preventing HIV disease in sportspeople are:

- In general the guidelines for management of HIV-positive patients that have been published in a policy statement by the College of Medicine of South Africa can be applied by medical personnel that attend to sportspeople with suspected HIV disease (Policy statement, 1991)
- All open skin lesions sustained during sports participation should be treated appropriately before allowing the sportsperson to return to the playing field
- The following treatment of open skin lesions is recommended:
  - immediate cleaning of the wound with a suitable antiseptic such as hypochloride (bleach, Milton), 2% gluteraldehyde (Cidex), organic iodines or 70% alcohol (ethyl alcohol, isopropyl alcohol)
  - the open wound should be covered securely so that there is no risk of exposure to blood or blood products prior to returning to the playing field
- It is recommended that all first aiders and medical personnel attending to sportspeople with open wound lesions wear protective gloves to decrease the risk of HIV transmission to themselves and other sportspersons

The effects of HIV infection on exercise performance

The HIV epidemic will inevitably affect physically active people. This would include people that are physically active in sports (recreational or competitive) as well as manual laborers. Two of the questions that will arise as a result of HIV infection in physically active individuals are i) whether HIV disease affects the individuals ability to perform physical work and ii) whether regular physical work has a beneficial or perhaps a detrimental effect on the outcome of the disease? These two questions have important implications for the sportsperson as well as advice regarding the occupation of an infected individual.

There is very little information available in the medical literature on the effect of HIV infection on the ability to perform physical work. Indeed to date only a few studies have addressed this issue. In one study exercise performance was investigated in 32 patients with a clinical diagnosis of AIDS (Johnson et al 1989). Patients with documented AIDS and complaining of dyspnea on exertion were enrolled in the study and their exercise performance was compared to a group of age, height and weight matched normals. HIV infected patients with evidence of pulmonary infection, atherosclerotic heart disease, asthma, pre-existing pulmonary disease or other significant complicating medical disorders were excluded from the study.

The exercise test to which patients and control subjects were subjected consisted of an incremental exercise test to exhaustion during which cardiorespiratory variables were measured. Arterial oxygen saturation was measured by finger oximetry. Spirometry was done before and immediately after the exercise test. The results of this study were that the patients with documented AIDS exercised to a significantly lower workload than controls, had a lower maximum oxygen uptake (VO_2 max) than controls (not statistically significant), had similar maximal heart rates as controls, had a lower "ventilatory anaerobic threshold" than controls, and did not have bronchospasm post-exercise.

Nine subjects achieved an oxygen consumption (VO_2) less than 81% of the predicted whereas none of the controls exhibited this. The authors concluded that some AIDS patients have impaired exercise performance and this was attributed to a central (cardiac) limitation. The possibility of a direct effect of the HIV infection or its complications on muscle function was not considered.

In another study exercise testing was included as part of a clinical trial evaluating the effectiveness of corticosteroids on Pneumocystis carinii pneumonia in AIDS patients (Montaner et al 1990). In this study AIDS patients with Pneumocystis pneumonia were randomly allocated to either an experimental (receiving corticosteroids) or a control (receiving placebo) group and then monitored for 4 weeks. A maximal incremental exercise test was performed on days 0, 3, 7, 14 and 30. Effort tolerance was very poor in both groups on day 0. After treatment the exercise tolerance improved sevenfold in the treatment group whereas it remained the same in the control group. This positive effect was attributed to the prevention of early clinical deterioration by the administration of corticosteroids in the treatment group. It is obvious that this study was not primarily aimed at investigating effort tolerance in these patients. However, the results do indicate that

Table 4: Summary of clinical trials: HIV infection and exercise

PATIENTS	EXERCISE PROGRAMME CHARACTERISTICS					OUTCOME (EXERCISE GROUP)	REFERENCE
	FREQUENCY	INTENSITY	DURATION	PERIOD	TYPE		
HIV +ve (n=21)	2/week	?	1 hr	8 wks	Various sports games	- Reduced anxiety/ depression - CD4 count - CD4-Cd8 ratio	Schlenzig et al, 1989
Asymptomatic HIV +ve (n=16)	3/week	70-80% of HR max	45 min	10 wks	- Cycle ergometer - Internal type training	- Reduced anxiety/ depression - Non significant decline in NK - Trend for increase in CD4 count	LaPerriere et al, 1991
HIV +ve (n=37)	3/week	60-80% of HR reserve (20 mins)	1 hr	12 wks	- Cycle ergometer - Strength/ flexibility training	- Improved muscle strength and endurance - No change in lymphocyte	Rigsby et al,1992

physical work is severely impaired in AIDS patients with associated pneumocystis infection and that this can be improved by appropriate therapy.

The effects of regular exercise training on patients who were seropositive for the HIV but who were otherwise asymptomatic, have also been documented. In one study 45 male volunteers who were allocated randomly to an exercise and a counselling group (Rigsby et al, 1992). The groups were well matched according to modified Walter Reed criteria. The patients in the training group underwent exercise training consisting of cycling, weight-training and flexibility exercises for 12 weeks. Exercise testing and immunological parameters were assessed before and after the training period. One of the significant findings of the study was that the trained group showed evidence of adaptation to exercise. This study therefore indicated that 12 weeks exercise training in HIV seropositive patients can result in improved physical work capacity.

In summary, patients with AIDS appear to have impaired physical work capacity, the precise mechanism of which is not clear. Asymptomatic patients with HIV disease appear to respond well to exercise training.

The effects of regular physical activity on the outcome of HIV infection

It is well recognized that an acute bout of physical exercise, as well as exercise training, has significant but variable effects on a number of immunological parameters (Keast et al, 1988, McCarthy et al, 1988, Nehlsen-Cannarella et al, 1991, Nieman et al 1989, Nieman et al 1991, Oshida et al, 1988, Ricken et al, 1990). An acute bout of exercise will result in a leucocytosis, the magnitude of which is related to the intensity of exercise, the duration of exercise and the state of exercise training of the athlete (McCarthy et al, 1988). The nature of the leucocytosis is variable and can either be a predominant neutrophilia or a predominant lymphocytosis. Furthermore, changes in lymphocyte subsets can also occur in response to an acute bout of exercise (Oshida et al, 1988). It has been documented that there is an increase in the absolute cell numbers of all lymphocyte subsets, but that the increase is greater in B cells compared to T cell subsets; thereby decreasing the T cell to B cell ratio (Keast et al, 1988, McCarthy et al, 1988). It has also been demonstrated that a greater increase occurs in T8 compared to T4 cells during exercise; thereby decreasing the T4/T8 ratio (McCarthy et al, 1988). In addition, total lymphocyte proliferation to antigens and mitogens is typically reduced in response to exercise (Keast et al, 1988).

The response of T lymphocytes to an acute bout of exercise has clear implications for individuals infected by the HIV. Of particular importance is the effect of exercise and exercise training on T cell counts and the CD4/CD8 ratio. To date there are no published studies on the possible association between a programme of regular physical activity and the long term clinical outcome of HIV infection. However, it has been suggested by long term AIDS sufferers that regular exercise contributed to their longevity (Solomon et al 1987).

In recent years, several groups of investigators have been concerned with and have published data on the effects of exercise on the clinical course of HIV infected individuals. The results of three well controlled randomized clinical trials on the influence of regular exercise training on immunological and psychological parameters in HIV positive patients are presented in Table 4. In one of the earliest reports from the University of Miami, the beneficial effects of regular exercise training on CD4 cells of HIV-infected individuals has been documented. In this study, 10 weeks of exercise training at moderate intensity (< 80% of maximal heart rate), for 45 minutes three times a week resulted in an increase in CD4 cells (LaPerriere, 1991). In another aspect of the study, high risk individuals who were regular exercisers, showed less anxiety and depression after receiving the news that they were HIV seropositive. Exercise appeared to provide a "buffer" to the psychological sequelae of a powerful acute stressor in these patients. The findings from the group at the University of Miami have the following possible practical applications: i) that exercise can play a beneficial role in the pre-HIV test counselling of potentially infected patients, and ii) that regular, moderate intensity exercise can play a role in the management of early, asymptomatic individuals with HIV infection (Calabrese et al 1993). Recently, specific recommendations on the role of regular exercise in the management of patients with HIV disease have been made. These are summarized in Table 5.

Summary

Regular participation in physical activity is advocated as an important preventative health measure. However, the global pandemic of HIV infection is likely to influence physically active individuals. The association between HIV infection and physical activity therefore requires attention. In this review the risk of HIV transmission during sport and physical activity, the effects of

Table 5: Recommendations for exercise in patients with HIV infection

a. General

Before initiating any type of exercise-training, all HIV-infected individuals regardless of age or stage of disease should:

- have a complete physical examination
- discuss exercise plans with a physician or exercise specialist
- comply with ACSM testing and prescription guidelines.

b. Healthy asymptomatic HIV seropositive

- Unrestricted exercise activity
- Continue competition
- Avoid overtraining

c. AIDS-related complex

- Continue exercise training on symptom listed basis
- Avoid strenuous exercise
- Reduce or curtail exercise during acute illness.

d. Diagnosed AIDS

- Remain physically active
- Continue exercise training on symptom limited basis
- Avoid strenuous exercise
- Reduce or curtail exercise during acute illness

HIV infection on exercise performance, and the effects of regular physical activity on the outcome of HIV infection was discussed.

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Vitamin C as effective as combinations of anti-oxidant nutrients in reducing symptoms of upper respiratory tract infection in ultramarathon runners.

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Abstract

The effect of anti-oxidant supplementation on the incidence of symptoms of upper-respiratory-tract infection (URTI) was determined during the fortnight following the 1993 Comrades Marathon (90km). Runners (n=178) and sedentary matched controls (n=162) were randomly divided into groups receiving 500mg Vit C (C; n=86), 500mg Vit C and 400IU Vit E (CE; n=90) or 300IU Vit E, 300mg Vit C and 18mg Beta Carotene (CEB; n=73) or placebo (P; n=93) daily for 21 days prior to participation in the ultramarathon. Total pre-race dietary vitamin and mineral intakes and post-race self-reported URTI symptoms were recorded in all subjects (n=340). The incidence of the URTI symptoms in P runners (40.4%) was significantly higher ($p<0.05$) than that in C (15,9%) and CEB (20,0%) runners, and also greater than that in matched, non-running controls receiving placebo (24,4%). The group of runners reporting the lowest incidence of URTI symptoms during the post-race period, had the lowest mean age and the highest (i) total mean daily Vit C intake (1004 mg); (ii) pre-race training status and (iii) percentage of black runners.

This study suggests that Vitamin C alone is as effective as combinations of Beta Carotene, Vitamin E and Vitamin C in reducing the incidence of post-race URTI symptoms and that age, training status and genetic make-up also may influence the susceptibility to the development of URTI symptoms in ultramarathon runners.

Key Words: Anti-oxidant Vitamins, Upper Respiratory Tract Infections, Ultramarathon Runners.

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Introduction

Two separate epidemiological surveys performed on ultradistance runners[1,2] have reported an increased incidence of symptoms of upper respiratory tract (URTI) following participation in ultramarathon events. A subsequent study[3] found that daily administration of 600mg Vitamin C for three weeks prior to the 90km Comrades Marathon, resulted in a significantly lower ($p<0.05$) incidence of symptoms of infection in runners during the fortnight after the race, compared to runners ingesting placebo. This was attributed to the anti-oxidant properties of Vitamin C, which suggests that athletes participating in prolonged exercise have an increased daily Vitamin C requirement.[3]

Evidence from more recent studies[4,5] is that the anti-oxidant nutrients may be more effective when used in combination. The aim of this study was therefore to compare the efficacy of supplementation with combinations of Vitamin E, Vitamin C and Beta Carotene and Vitamin C alone in reducing the incidence of post race URTI in ultra marathon runners.

Method

The protocol was approved by the Committee for Research on Human Subjects of the University of the Witwatersrand. Two hundred and twenty entrants for the 1993 Comrades Marathon volunteered to participate in the study. Each runner was matched (n=220) with a control of similar age who resided with the runner, but did not participate in running regularly. A double-blind, placebo-controlled study design was used in which the runners, in addition to their matched, non-running controls, were randomly divided into four groups. Each group received either anti-oxidant supplements or placebos for three weeks prior to the race. The pre-race training status, state of health and dietary vitamin and mineral intake of athletes and their age-matched controls was recorded by means of a questionnaire which each runner and control subject completed prior to the race. Demographic data including running distance per week, average running speed, number of weeks spent training and the number of other ultramarathons in which the athlete had recently participated were also recorded. In addition, the pre-race incidence of self-reported symptoms of URTI was documented. Runners with a history of sinusitis, hay-fever or both were excluded.

Each runner and matched control (n=55) was required to take three tablets of similar appearance daily. These contained either 500mg Vit C (C), 500mg Vit C & 400IU Vit E (CE), 300mg Vit C & 300IU Vit E, 18 mg Beta

Carotene (CEB) or lactose as placebo (P).

The total daily vitamin A, C and E intakes including that derived from any additional vitamin and mineral used by the athlete of all subjects was determined by using the Dietary Manager computer programme (Program Management, Randburg, South Africa). For the purpose of this study, the total vitamin A, C & E intake of each subject was thus calculated from the sum of the (i) daily dietary intake, (ii) additional supplements used and (iii) the anti-oxidant supplements given to the subjects.

Two weeks after the race all runners and controls who had originally volunteered to participate in the study were telephonically interviewed and questioned regarding: (i) whether the prescription of supplements had been adhered to or not; (ii) the race distance covered and the time taken by each athlete to complete this distance; (iii) the incidence and duration of symptoms of URTI. The number and duration of self-reported symptoms including sneezing, running nose, sore-throat, cough and fever were documented. All reports of trivial symptoms were excluded by including in the final analysis only reports of single URTI symptoms which lasted >1 day or a combination of at least 2 URTI symptoms each of which lasted < 1 day.

The training status ratio of each runner was calculated from the following formula:

$$\frac{\textit{Weekly training distance (km)} \bullet \textit{no of weeks spent in training}}{\textit{average speed at which those kilometres were covered}}$$

As in our previous study[3], runners who reported a ratio of > 450 fell into the high-training status category, whereas those with a ratio < 300 were classified in the low training status category.

Statistics

A chi-square statistic was used to establish whether the incidence of symptoms of URTI was significantly different between the four groups of runners and controls. Multivariate analysis of variance and several one-way analyses of variance were used to analyze the significance of the difference between the four groups in terms of age, Vitamin C, E and Beta Carotene intake and training status ratio. For all statistical analyses the Statgraphics and Excel computer software programs were used and the level of significance were set at 0.05.

Results

Of the initial 220 runners and matched controls, 178 runners (23 female; 155 male) and 162 (116 female ; 46 male) controls compiled fully with the requirements of the study. Reasons for exclusion from the study included a previous history of allergic rhinitis, failure to take the prescribed medications, failure to complete at least 60 km of the race and inability to establish contact with the subjects after the race.

The size, gender distribution and mean age of the 4 groups of runners and their matched controls is given in Table 1. Among the runners who ranged in age from 19 to 65 years, 46 were < 30 y old, 119 were aged between 31 and 50 y and 13 were > 50 y in age. Although the two groups with the highest incidence of reports of URTI possessed the highest mean age, this was not significantly different from the mean age of the groups with lower incidence of infection (p>0.05).

Table 1: *The size, gender distribution and mean age (+/- SD) of the 4 study groups (n=340).*

EXPERIMENTAL GROUP	RUNNERS (n = 178)			CONTROLS (n = 162)		
	N		Age	N		Age
	M	F	(Years)	M	F	(Years)
Group C	44		34,3	41		33,1
	36	8	(5,2)	11	30	(9,5)
Group CE	47		37,6	43		33,7
	41	6	(8,4)	11	32	(9,2)
Group CEB	40		35,1	33		29,7
	36	4	(10,0)	8	25	(11,9)
Group P	47		39,2	45		32,8
	42	5	(9,5)	16	29	(11,8)

The total mean Vitamin A, E and C intakes of the runners in each of the four groups is shown in Table 2. The highest total mean intake of Vitamin C was reported in group C, whereas the P group reported a mean intake of 585mg of Vitamin C. Groups CE & CEB also reported high mean intakes of this Vitamin. Group CEB was the only group with a Vitamin A intake which exceeded the RDA for sedentary individuals[6], whereas group CEB was the only group which exceeded the RDA[6] of Vitamin E for sedentary individuals. The group with the lowest incidence of infection (C) reported a total mean Vitamin C intake of 1004mg.

Table 2: *The mean Vitamin A, C and E intakes of the runners (n=178) in the 4 groups.*

		GROUP C	GROUP CE	GROUP CEB	GROUP P
Food	Vit A (IU)	3170	4405	3915	3110
Sources	Vit C (mg)	72	93	95	80
	Vit E (IU)	25	22	26	18
Additional	Vit A (IU)	1446	1737	2348	2992
Supplements	Vit C (mg)	432	305	271	505
	Vit E (IU)	12	22	8	43
Anti-oxidant	Vit A (IU)	-	-	30 000	-
Supplements	Vit C (mg)	500	500	300	-
provided	Vit E (IU)	-	400	300	-
Total	Vit A (IU)	4616	6142	36 263	6102
	Vit C (mg)	1004	893	665	585
	Vit E (IU)	37	444	334	61

The incidence of symptoms of URTI in the runners and controls are given in Figure 1. The difference between the incidence of symptoms of infection in the runners ingesting placebo (40.4%) and their sedentary, matched controls (24.4%) was not significantly different (X^2=1.99; p = 0.16). The lowest incidence of infection amongst the runners (15.9%) was reported in group C. This was significantly different (X^2=5.54; p < 0.05) from the incidence of URTI symptoms in group P. A 20% incidence of infection was reported in group CEB. The incidence of symptoms of URTI in group CE was also significantly lower than in group P (X^2 = 6.24; p > 0.05). The incidence of symptoms of infection in group CE (25.6%) was not significantly different (p > 0.05) than the incidence in group P (40.4%).

The incidence, nature and mean duration of the symptoms of URTI among runners and control subjects is presented in Table 3. The most common URTI symptoms reported by runners in the 4 groups were nasal (n = 35). Included in this category were reports of running noses and sneezing. Symptoms lasting more than 7 days occurred in 20 cases (nasal), 13 cases (sore throat) and 12 cases (coughing). Twelve of the runners reported fever in conjunction with their URTI symptoms. Only 24,2% of the reported runners symptoms lasting 1-3 days. The mean duration of symptoms in the runners was not significantly different from the mean duration of symptoms in the control subjects (p > 0.05). Although the mean duration of symptoms was higher in runners on placebo than in runners who received the different combinations of anti-oxidants, this difference was not statistically significant (p > 0.05).

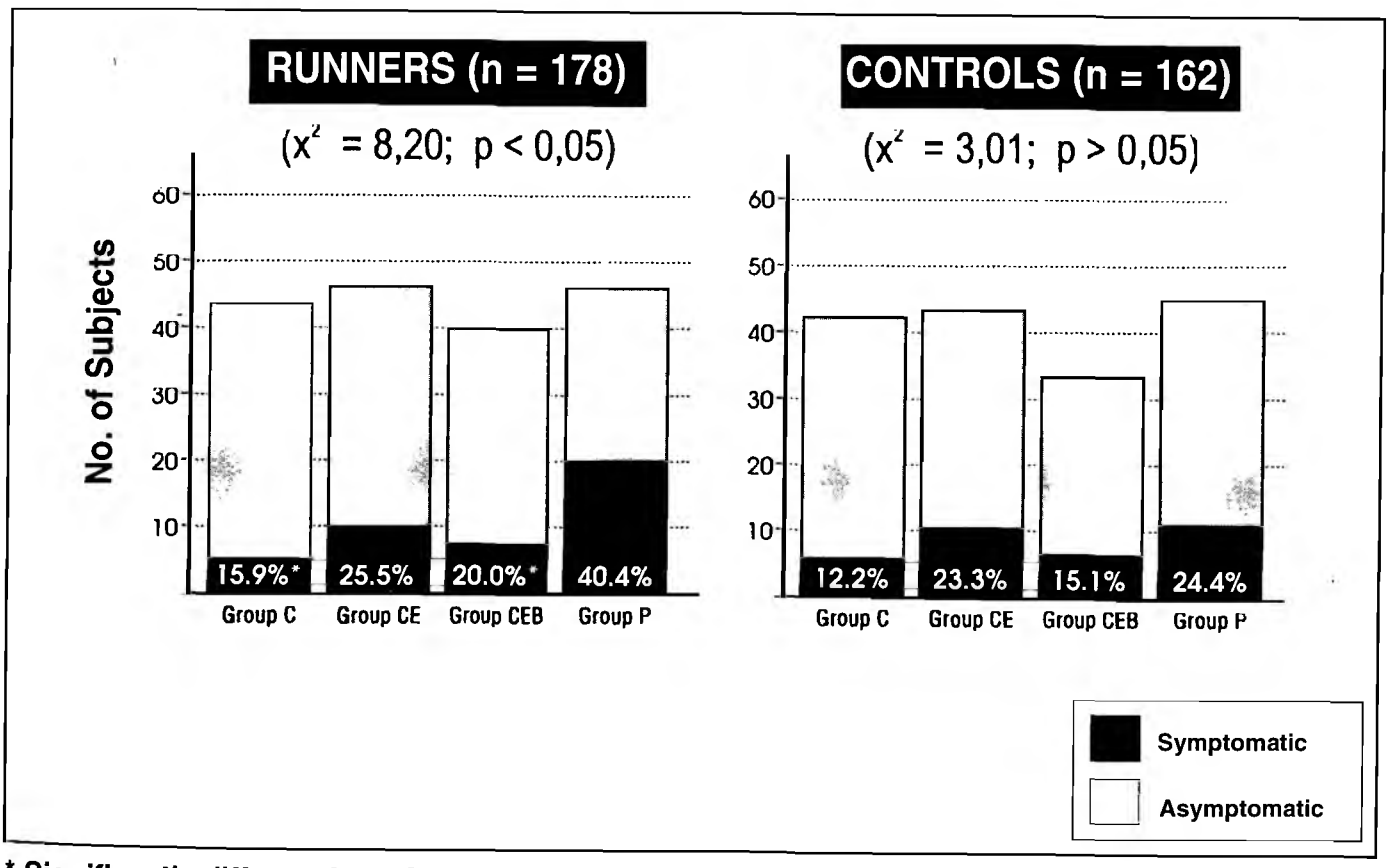
Table 3: *Incidence, nature and duration of post-race symptoms of URTI among runners and control subjects.*

EXPERIMENTAL GROUP	N	% SYMPTOMATIC	MEAN (± SD) TRAINING STATUS RATIO
Group C	42	20,2*	311 (±150)**
Group CE	47	25.8	274 (±127)
Group CEB	40	16,7*	328 (±166)**
Group P	47	40,4	236 (±111)

* P < 0.05 vs Group P
** P. <0.01 vs Group P

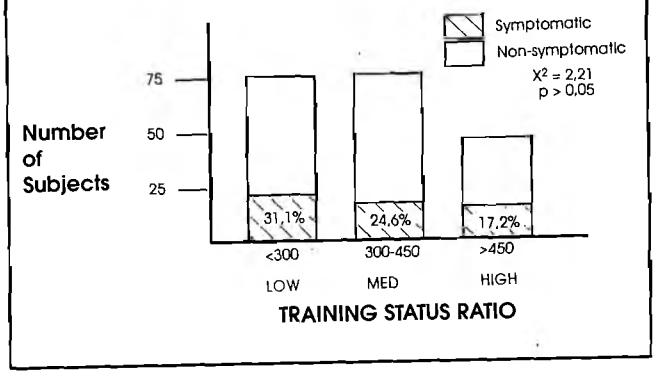
The incidence of infection in the low, medium and high training status groups is shown in Figure 2. Those runners who reported a low pre-race training status (<250) group reported the highest incidence of infection symptoms. This finding was confirmed when the incidence of post-race URTI symptoms was related to the mean training status of the 4 groups of runners (Table 4). The two groups with an incidence of URTI symptoms of more than 25%, reported the lowest mean training status. The difference between the training status of groups C and CEB (possessing the lowest incidence of infection) and groups CE and P (possessing the highest

Figure 1: *Incidence of Symptoms of URTI in runners (n = 178) and controls (n = 162) during the 14 day post race period.*



*** Significantly different from Group P (p<0,05)**

Figure 2: *The incidence of post-race URTI symptoms in low, medium and high training status groups (n=176)*



incidence of infection) was highly significant ($p > 0.01$). When the incidence of URTI symptoms were related to weekly training distance completed in preparation for the race, this trend was confirmed. Once again, the runners who had done the least pre-race training, reported the highest incidence of URTI symptoms.

A total of 16 black runners participated in the study. None of these athletes reported symptoms of URTI. When excluding the black runners from their respective groups, the percentage incidence of infection symptoms in the respective groups was 25,7(C); 41,3(P); 26,7(CE) and 23 (CEB). The difference between the three groups receiving additional anti-oxidant supplementation was not significant ($p > 0.05$). Despite a substantial difference in percentage incidence in the placebo group and the groups receiving anti-oxidant supplementation, this difference was no longer significant ($p > 0.05$).

No relationship between running time and incidence of URTI symptoms in the four groups was found in this study. Although the incidence was highest (35,5%) in those runners who took >10hrs to complete the race, this incidence was not significantly greater than the incidence in groups taking 7-8 hrs (23.5%) and 8-9 hrs (27,8%) to finish the race.

Discussion

The data obtained in this study confirms our previous findings[1,2,3,7] of a greater incidence of post-race symptoms of infection in ultramarathon runners compared to sedentary controls (Figure 2). The comparatively lower general incidence of symptoms in this study than in our previous study[3] must be seen in the light of the fact that (i) in this study, reports of symptoms lasting one day or only part thereof were not included in the final calculation and (ii) prevailing virological or bacteriological counts may have varied greatly at the time that these two separate studies were undertaken.

This study confirms previous findings[3] that daily intake of an excess of 1g of Vit C is effective in lowering the incidence of symptoms of URTI during the post-race period. Although the incidence of symptoms of URTI was substantially lower in all three groups of runners receiving anti-oxidant supplementation than in the group of runners receiving placebos, no correlation was obtained between the total amount of anti-oxidant nutrients ingested and the incidence of symptoms of URTI. Rather, it was the group with the highest total daily intake of Vit C which had the lowest incidence of URTI symptoms. Several factors could account for this.

Table 4: *The mean training status ratio and incidence of post race URTI symptoms in the 4 groups of runners (n=176)*

EXPERIMENTAL GROUP	A : RUNNERS								B : CONTROLS							
	Group C (n=44)		Group CE (n=47)		Group CEB (n=40)		Group P (n=47)		Group C (n=41)		Group CE (n=43)		Group CEB (n=33)		Group P (n=45)	
Post Race Symptoms	n	duration	n	duration	n	duration	n	duration	n	duration	n	duration	n	duration	n	duration
Nasal Symptoms	4	5,3	10	5,8	7	7,6	14	8,6	5	7,0	8	22,8	5	3,2	11	7,8
Sore Throat	5	5,8	9	4,7	5	5,8	12	4,5	5	7,8	8	9,3	3	1,3	8	8,1
Cough	2	9,0	2	7,0	4	4,8	8	7,8	4	9,6	8	11,1	1	7,0	7	10,3
Fever and URT Symptoms	1	3,0	2	1,8	4	4,3	5	6,4	1	14,6	2	17,5	6	0,0	4	2,0
Total Symptomatic**	7		12		8		19		5		11		5		11	
Mean Duration of Symptoms		5,8		4,8		5,6		6,8		9,4		15,2		2,3		6,9

Nasal symptoms include runny nose and sneezing.
** No. of persons in the group who presented with 1 or more symptoms lasting≥ 1 day
or 2 more symptoms lasting ≤ 1 day
Duration = mean no. of days

Vitamin C is regarded as a first line anti-oxidant in the defence against phagocyte- derived reactive oxidants[8,9,10]. These immunosuppressive free radicals are known to be autotoxic, causing inhibition of chemotaxis, phagocytosis, the proliferation of T-lymphocytes and B-lymphocytes as well as the cytotoxic activity of natural killer cells [11,12,13]. Evidence is mounting in favour of Vitamin C- mediated neutralization of these reactive oxidants[8,9,10].

The relatively smaller protective effect of vitamin E and Beta Carotene supplementation may, be partially attributable to the slow elevation in plasma Vitamin E and Beta Carotene levels[15] and the fact that a 21 day supplementation period is too short to elevate plasma levels to reach protective levels. Secondly, variance in age[14], training status[15], environmental training conditions[16], and genetic make-up between the groups studied, may have obscured the effect of the other anti-oxidant nutrients.

As in our previous study[3], age does not appear to have played a significant role in the risk of infection. Training status, however, appeared to be an important factor. The highest incidence of symptoms of URTI was found in those runners who fell into the low training status category (Figure 2). Further confirmation of a possible beneficial effect of training is found when examining the mean training status of the four groups. It was found that the two groups possessing the highest training status had the lowest incidences of infection symptoms (Table 4). These findings were further supported when the incidence of URTI symptoms was analysed as a function of weekly training distance.

Two factors might support this finding. First, endurance training results in a lower catecholamine levels at a given exercise workload. This is potentially important as catecholamines affect free radical production[16]. Second, endurance training increases concentrations of endogenous antioxidant enzymes in skeletal muscle[15]. That endurance training exerts a protective effect on oxidative stress thus warrants further investigation in humans.

The finding that Black runners who participated in this study did not develop infection symptoms, also requires further investigation. This difference might be explained by chance; socio-economic or hereditary factors may also have played a role.

To conclude, the findings of this study appear to indicate that large intakes of Vitamin C alone (>1000mg) are more effective than combinations of Vitamin E, Vitamin C and Beta Carotene in lowering the incidence of URTI in ultradistance runners. This study, however, also indicates that besides Vitamin C ingestion, training load and ethnic background, are important variables which may influence the susceptibility to infection in ultramarathon runners.

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A social cognitive perspective on promotive and preventive health behaviour in post-apartheid South Africa

I Miller

Abstract

Non-communicable diseases or chronic diseases of lifestyle are on the increase amongst all South African population groups. There are, however, disparities in the health profile of black and white communities which can be attributed to the discriminatory health care services of the apartheid era. Sport and Exercise Scientists who strive to enhance health and prevent disease and injury by facilitating participation in physical activity and sport, should therefore endeavour to address these unbalances. However, their efforts to promote health and fitness amongst all communities should be guided by an understanding of the social, psychological, political and cultural factors which could foster hypokinetic lifestyles or deter participation in health-related physical activity. Albert Bandura's social-cognitive theory is therefore used to explain and predict health behaviour in the South African context.

It is further advocated that research in the sport and exercise domain should be re-orientated to develop a knowledge base which could guide the design and implementation of effective community-based, health-related physical activity programmes in post-apartheid South Africa.

Introduction

Professionals and academics concerned with the promotion of health and fitness through sport, physical exercise and recreation, could be regarded as promotive and preventive health care workers. As such, they should be concerned about indications that non-communicable diseases or chronic diseases of lifestyle are on the increase amongst all population groups in South Africa. As far back as 1988, life-style related diseases already accounted for 24,5% of all deaths reported in South Africa. Research conducted in 1992 revealed that 55% of the population within the 15-64 age group were in need of life-style modification and that 16,5% received medical treatment for preventable hyperlipidaemia and hypertension (in that particular year).[1]

In addition to the physical diseases referred to above, it can also be expected that the mental health of South African citizens has been affected by the political turmoil which has prevailed in South Africa over the past few decades. (Van Rensburg and Benatar 1993)[2] ascertained that high profiles of stress, trauma, powerlessness and instability exist in non-white communities. They further cautioned that South Africans have internalised apartheid to the extent that it influences their "psychic make-up and self-image,... minds and emotions,... attitudes and behaviour patterns".[2] It can therefore be presumed that South Africans' perceptions of themselves, as well as their views of themselves in relation to society, have been influenced in a positive or negative manner by their socio-political experiences, and that this has ultimately impacted on the psychological (and general) health and social well-being of our nation.

The health problems cited above can certainly be addressed by Human Movement Specialists (sport and exercise scientists, biokineticians, physical educators, recreation officers) by means of health-related physical activity programmes or rehabilitative movement therapy. Sufficient evidence exists to verify that regular physical activity can bring about:

* modifications in blood fat profiles and associated reductions in obesity;
* reductions in blood pressure for moderately hypertensive individuals;
* reductions in coronary heart disease (CHD);
* organic changes in the lungs and resultant increases in respiratory efficiency;
* reductions in low back pain and postural defects;
* preservation of joint and muscle function;
* reductions in constipation through improved efficiency of the digestive system;
* promotion of bone health and consequent reductions in fractures and osteoporosis in later life;
* reductions in anxiety and depression;
* reductions of the adaptations to psychological stress;
* improvement of sleeping habits;
* and enhancement of self-image and self-confidence.[3-8]

Admittedly, improvement of health cannot be attributed to regular exercise alone and the positive impact of exercise alluded to above may be limited to specific populations under specific conditions. However, while the rehabilitative and curative value of exercise is acknowledged and advocated, it is regrettable that the preventive potential of regular physical activity is not promoted amongst all sectors of the South African community. X[9] observed that good health has been deemed more important for whites than for blacks and that a lack of interest in the health status of black patients has been deemed more important for whites than for blacks and that a lack of interest in the health status of black patients has been discernible to date. Although this observation refers to the discriminatory provision of curative health care, the lack of sport, gymnasium and recreation facilities in black communities suggests that similar inconsistencies exist with regard to health

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The health plan proposed by the African National Congress advocates a "redress of the negative effects of apartheid health care services".[10] However, the topics under discussion at the 1994 congress of the South African Federation for Movement and Leisure Sciences indicates that this discipline has not re-orientated itself to the needs of the "new" South Africa. Instead, it affirms the emphasis which Human Movement Scientists place on understanding the capacity and performance of a relatively small percentage of the community who maintain active lifestyles and who participate in elite, competitive sport. The valuable contribution of Sport Science, Exercise Science and Biokinetics in this regard, especially in view of South Africa's re-entry into the international sport arena, is not contested. However, it can be questioned whether the debilitating social, political, economic and nutritional variables which impact on the motor development and physical performance of the majority of South Africans, does not warrant more attention. Moreover, this discipline should be concerned with the factors which foster hypokinetic lifestyles and deter health-seeking behaviour.

A Social-Cognitive analysis of Health Behaviour

The World Health Organization (WHO) defined health as an integrated, multifaceted phenomenon which encompasses physical, psychological and social well-being. Schlebusch[11] concurred that age, gender, psychological status, socio-cultural and political experiences, education, religion, the media, networks which exist in society, as well as personal beliefs, values and attitudes could influence health behaviour. Health workers should therefore acknowledge that individuals' perceptions of health and health care will be influenced by the reciprocal interaction between their behaviour, internal make-up and the external environment within which they function.[12]

The social-cognitive theory proposed by Bandura could be used to account for the variability and consistency which exist with regard to promotive health behaviour in general, and participation in regular physical activity in particular. The theory postulates that "... behaviour is influenced by three self-regulatory mechanisms operating in concert: perceived self-efficacy for outcome attainment, outcome expectations, and personal goal-setting".[13] It is suggested that perceptions about the outcome of the behaviour (eg. monetary reward, social approval, self-satisfaction or health benefits) will influence decisions about participation in physical activity. Similarly, individuals' beliefs of self-efficacy (skills, information and ability) serve as an internal or personal form of behaviour control which determine involvement with physical activity.[14]

The notion that health behaviour is influenced by personal perceptions and beliefs, is further demonstrated by the "locus-of-control" theory delineated by Lau .[14] He contended that individuals who believe that they have no or little control over the negative situation in which they find themselves, may demonstrate "learned helplessness". Similarly, some individuals, the so-called internals, may perceive their circumstances and experiences to be a consequence of their own actions and will therefore believe that they can control it, whereas others, the so-called externals, will regard events in their lives to be unrelated to their actions and therefore beyond control.[14] The fact that apartheid legislation has influenced the environment in which

people lived, the jobs and level of education they obtained and health care which they received, may indeed have caused disadvantaged individuals to believe that their quality of life cannot be improved through their own efforts. Individuals who assume that they have little or no control over their living environment or that they lack the necessary skills and knowledge (self-efficacy) to improve their health may thus be disinclined to try. It is not surprising that these individuals (externals) experience ill-health as more debilitating and stressful than internals who believe that they can control their health.[14]

However, proponents of emancipatory health education[15,16] cautioned that movement specialists need to move beyond the individualistic perception of health behaviour which currently characterises health-related physical activity programmes. It is indeed simplistic to view health behaviour as a matter of individual responsibility and control only, without acknowledging the impact of the social and environmental variables cited above. This reductionist ideology should be rejected as it could proliferate stereotyping of individuals who are unable to modify their health risk behaviour as weak, lazy, lacking will-power, self-discipline or motivation.[15]

According to Bandura's social-cognitive theory, social or environmental factors such as socio-economic circumstances and social relationships interact in a reciprocal manner with psychological traits such as attitude and motivation. Similarly, actions and cognitions (beliefs) are interactive behavioural variables. It can therefore be deduced that the social networks which exist within a community can also influence the health-seeking behaviour of an individual. Geertsen[17] and Ritter[18] reminded us that the socio-cultural perceptions about health which are demonstrated by significant others in the social network, may persuade or dissuade an individual to adopt health-promoting behaviour. The dynamics within the group as well as the individuals' position with the group (superior or inferior) will of course also play a role. Ritter[18] reported that "... a lack of ties may itself be a source of stress, created by not having sufficient social ties to provide for intimacy, or a sense of belonging, and the opportunity to provide as well as receive nurturance". These psychological factors are likely to impact negatively on the health-seeking behaviour of most sectors of the black community who do not have access to adequate gymnasium, recreation and sports facilities within their residential areas and subsequently participate in less supportive social networks outside their communities.

Bandura hypothesized that behaviour could be acquired by observing role models. However, white South Africans generally dominate the fitness, recreation and sport domains and blacks who have been conditioned to perceive themselves as an inferior race, may find it difficult to identify with or emulate white role models. Negative self-efficacy thoughts may in fact discourage blacks from engaging in activities which have traditionally been practised by whites, regardless of its health promotive potential. Allen[12] intimated that low self-efficacy can be boosted by personal performance accomplishment or positive vicarious experiences, particularly when the inhibited behaviours are performed by individuals who experienced similar fears and self-doubt. Wankel[19] also observed that feelings of competence, choice and perceived control translates into enjoyment and intrinsic motivation and that this in turn could foster exercise adherence, positive adjustment and overall well-being. The critical contribution of sport development programmes in this regard is obvious, but the need to promote a broader range of physical activities amongst communities who are not involved with mainstream sport (eg. older adults) should not be disregarded.

Socio-economic circumstances largely determine whether people have access to health care facilities and services and whether they will be motivated to adopt health-seeking behaviour. Maslow's hierarchy of needs[8] postulates that the behaviour of individuals is determined by their needs which in turn are defined by beliefs. It can be assumed that the behaviour of individuals who have a low socio-economic status will, to a large extent, be influenced by physiological and safety needs. More affluent individuals who are able to fulfill basic needs such as nutrition and safety, will probably be driven by esteem and self-actualization needs. A survey conducted in 1989[10] revealed that 52,7% of Africans, 28,1% of Coloureds, 10,7% of Indians and 1,6% of Whites live below the so-called breadline. It can therefore be assumed that a smaller percentage of the black community will be encouraged to participate in physical activity in order to look or feel good, while it appears to be a major incentive amongst whites or more affluent individuals.

Duda and Allison[20] expressed concern about the lack of cross-cultural analysis in Exercise and Sport Science. They cautioned that differences which may exist between whites and blacks with regard to the performance and preference of physical activity cannot necessarily be attributed to biological and physical characteristics as these explanations do not account for inter-and-intragroup differences. Instead, they proposed that exercise scientists should analyse the impact of sociological processes such as socialization differences and stereotypical reinforcement of certain skills and behaviour, as well as psychological processes such as motivation, expectations or perceived ability, from a cross-cultural perspective. Their suggestion that the exercise fraternity only acknowledges the culture of white mainstream participants is supported by Colquhoun[15] who observed that the media promotes Western values which associate mesomorphic images with achievement, dynamism, discipline, conformity, efficiency, "manliness" and femininity; ectomorphy with weakness, maladjustment, neuroticism, anti-social tendencies, unattractiveness and coldness; and endomorphy with laziness, inefficiency, self-indulgence, unhealthiness and unattractiveness. It should be acknowledged that these perceptions are not necessarily supported by all cultural groups, and that some communities may be less inspired to change their life-styles in order to attain the presumed ideal images of health and attractiveness.

Women are apparently very susceptible to pressure from society and the media to emulate images of the ideal woman who is depicted as thin, active and fit. They are therefore coerced to reduce weight, often leading to bulimia and anorexia nervosa. The impact of gender stereotyping is also demonstrated in other aspects of health behaviour. The fact that most societies frown upon women who abuse alcohol or smoke, and generally associate these behaviours with "manliness", may account for the higher incidence of these risk-taking behaviours amongst men. However, men are more likely to engage in physical and recreational activ-

ity due to the fact that they generally have less child-care or domestic responsibilities.[21] Women nevertheless demonstrate more concern about health as they are generally responsible for the health care of the family. They are also more dependent on medical care due to their complex and demanding reproductive functions. Duncan, Duncan and McAuley[22] identified that social integration or network support appears to enhance exercise compliance amongst men, while attachment and emotional support as well as reassurance of worth motivate women to persist with exercise.

It could be expected that individuals' level of education will impact on their health behaviour. However, Bailey and Macphee[23] observed that people who have the necessary knowledge about the repercussions of health-risk behaviour will not necessarily be dissuaded from engaging in it. According to the so-called Health Belief Model, people will only be prepared to modify their lifestyles if they perceive themselves to be at risk. The Self-Regulation Model contends that individuals' responses are determined by their understanding and personal experience of health problems. These reasons were nevertheless found to be poor motivators to exercise, and it is believed that knowledge of the positive outcomes of regular physical activity, rather than information about future health risks, may be greater incentives to exercise. This observation concurs with Bandura's social cognitive theory which hypothesizes that "... the perception that physical activity will lead to a valued outcome will motivate individuals to participate".[13]

In conclusion, Exercise Scientists are urged to adopt a multi-factorial and trans-cultural orientation to promotive health care. Schlebusch's[11] admonition that health-risk behaviour and unhealthy lifestyles are almost intractable in developing communities and that concentrated efforts are required to facilitate change, should serve as a challenge for Exercise Scientists who wish to contribute to the reconstruction and development of their communities.

At issue is the accommodation of health professional roles and the structure of the health care system to a changing knowledge base that incorporates culture and social process as important influences upon health behaviour.[24]

The social-cognitive theory outlined in this paper is not the only framework which can be used to analyze human thoughts and actions, but it can nevertheless serve as a useful guide to predict variations and consistencies regarding participation in health-promotive physical activity in post-apartheid South Africa. However, additional research on the impact of the aforementioned variables is essential to ensure that physical activity programmes which are aimed at community-based health promotion are relevant and sustainable.

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