Infection in Athletes

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Summary

Coaches and athletic team physicians have provided anecdotal information and case studies to support their beliefs that athletes may be unusually prone to illness during strenuous training or competition. Many athletes, in contrast, believe that physical activity improves their resistance to infectious disease. However, it is generally agreed that the stress of competition may make athletes temporarily more susceptible to infectious illness. A review of the literature shows that upper respiratory tract infections and skin infections are more prevalent in top level athletes than in the general population, particularly during periods of intensive training. Exercise induced changes occur in both the innate and adaptive components of the immune system; however, the relative importance of each component is unknown. Strenuous exertion and contact sports may compromise host defence both by reducing physical protection and by impairing immunosurveillance. Skin lacerations, vigorous sweating and maceration of the dermis impair the defence normally provided by the skin surface. In addition, adverse changes in soluble and cellular components of the immune system can increase susceptibility to infection. Persistence with strenuous training during an infectious illness can have deleterious effects; not only is athletic performance impaired, but the severity of the disease process can be augmented.
Elite athletes train mainly to improve sport specific components of skill and physical fitness (Niinemaa 1982) and others participate in regular physical activity to reap physiological, psychological and health benefits (Morgan 1985; Paffenbarger et al. 1984, 1993). Regular physical activity generally has positive effects on the cardiorespiratory and musculoskeletal systems and it often induces positive changes in mood state. However, controversy exists regarding the acute and chronic effects of physical activity on the immune system and thus, resistance to disease (Fry et al. 1991a,b; Keast & Morton 1992; Mackinnon 1992; Nash 1986; Verde 1992).

Recent research suggests that although regular participation in moderate exercise has an immunoenhancing effect, very strenuous exercise and high levels of athletic competition may suppress immune function, increasing susceptibility to infections (Cannon 1993; Eichner 1993; Fitzgerald 1991; Heath et al. 1992; Liesen & Uhlenbruck 1992; Linde 1987; Linenger et al. 1993; Nash 1986; Nieman & Nehlsen-Cannarella 1992; Nieman et al. 1989b, 1990a,b; Simon 1987). Infections are often associated with a reduction in athletic performance and can have permanent or lethal consequences (Daniels et al. 1985; Jokl 1974; Midtvedt & Midtvedt 1982; Munschek 1976; Roberts 1986).

As a result of the training methods of athletes, the physical and psychological environments encountered and the consequences of musculoskeletal injuries, certain types of infection are more common in athletes than in the general population (Diop Mar 1988; Midtvedt & Midtvedt 1982). This review provides an overview of the immune system and its role in infection and summarises the types of infections where athletes are at increased risk and the postulated mechanisms that enhance their susceptibility.

1. The Immune System

The immune system protects against, recognises, attacks and destroys elements which are foreign to the body. It comprises innate (naturally occurring) and adaptive (acquired) components which work together in a coordinated manner (Nieman & Nehlsen-Cannarella 1991; Roitt et al. 1985) [table I]. The first line of defence, the innate immune system, is encountered immediately when an infectious agent attempts to enter the body (Roitt et al. 1985). If this system fails, an acute infection may result and the adaptive immune system is activated. The adaptive immune system helps the body to recover from the infection and develops a 'memory' for the infectious agent, decreasing the likelihood of reinfection (Roitt et al. 1985).

1.1 Cells and Soluble Factors

Both cells and soluble factors contribute to the immune response. The white cell population (leucocytes) consists of granulocytes, lymphocytes and monocytes. Granulocytes (neutrophils, eosinophils and basophils) normally account for 60 to 70% of circulating leucocytes and 20 to 25% of circulating leucocytes are lymphocytes. Various subsets within the lymphocyte cell population have been identified through the use of monoclonal antibodies, which bind to unique cell surface proteins (cluster designators, CD) [Mackinnon 1992]. Important subsets of the T cell population (CD3) include T helper cells (CD4) and T cytotoxic/suppressor cells (CD8). Other lymphocytes include the B cells (CD19) and natural killer (NK) cells (CD16).

The B cells and plasma cells produce and secrete immunoglobulins which can be assayed in serum and other body fluids (for example, the saliva). There are 5 major groups of immunoglobulins: IgA, IgG, IgM, IgD and IgE. IgG and IgM are found predominantly in the serum, whereas secre-

| Soluble factors | Complement | Antibodies |
|-----------------|------------|------------|
|                 | Acute phase proteins | T lymphocytes |
|                 | Lysozyme | B lymphocytes |
| Cells           | Phagocytes (macrophages, monocytes, neutrophils) | T lymphocytes |
|                 | Natural killer cells |

Table I. Major elements of innate and adaptive immunity (Nieman & Nehlsen-Cannarella 1991; Roitt et al. 1985)
IgA is found mainly in mucosal fluids. When immunoglobulin molecules have a capacity to react with specific antigens they are described as antibodies. Antibodies protect the host by combining with surface antigens and agglutinating microorganisms, facilitating phagocytosis, activating the complement system, producing opsonins and neutralising bacterial toxins (Mackinnon 1992).

Other soluble factors include cytokines, complement and acute phase proteins. Cytokines are proteins involved in the communication between different immune cells; they include interleukins, interferons, tumour necrosis and colony-stimulating factors, each with diverse functions. Complement is a system of at least 20 proteins that stimulate phagocytosis, antigen presentation and the killing of infected cells. Acute phase proteins are involved in immune and inflammatory responses (Kumar et al. 1992), encouraging cell migration to sites of infection and injury, activating complement, stimulating phagocytosis and minimising tissue injury.

1.2 Innate and Adaptive Immunity

The innate immune system restricts entry of micro-organisms into the body through physical and chemical barriers plus phagocytosis by selected cells (Mackinnon 1992). The physical barriers to micro-organisms include the skin, epithelial linings and mucus secretions. The pH of body fluids and soluble factors such as complement, acute phase proteins and lysozyme create an adverse chemical environment for the invading micro-organism. Finally, neutrophils and macrophages can phagocytose and destroy bacteria either in the surface secretions or after penetration of the physical and chemical barriers.

The adaptive immune system depends on the body's ability to recognise antigens on an invading micro-organism. It, also, involves cells and soluble factors: T cells, B cells and immunoglobulins. Activation of lymphocytes (T cells) induces an alteration in their design to destroy the specific invading micro-organism. B cells secrete immunoglobulins specific for the antigens presented by the invading micro-organism, and in the process acquire a 'memory' of the exposure that facilitates the response to a subsequent challenge by the same micro-organism.

1.3 General Scheme of the Immune Response

Inflammation works in concert with the immune response to eliminate infectious micro-organisms (Kumar et al. 1992) [fig. 1]. The body's immediate response to infection is acute inflammation. Blood flow is increased to the infected area and vascular permeability is augmented to facilitate the entry of leucocytes and plasma proteins into the infected tissue.

Details of the immune response vary, depending upon the type of infection (parasitic, bacterial, fungal or viral), but the general pattern of response is similar (fig. 2). The process is initiated when a macrophage engulfs an invading micro-organism. The micro-organism is killed by lysozymes and/or oxidising agents released from within the macrophage. Foreign proteins, normally found on the surface of the micro-organism, are processed by the macrophage and are incorporated into its own cell surface, where they can be presented to other cellular components of the immune system.

T-helper (CD4) cells play a major role in coordinating the immune response. Once they recognise antigen-presenting or virally infected cells, they release cytokines to activate other immune
cells. They also stimulate B lymphocytes to secrete antibodies and to become memory B cells.

The type of infectious agent will determine which component of the immune response will predominate (Thong 1986). Phagocytic cells (macrophages and neutrophils) play a major role in the defence against bacteria and fungi. Eosinophils are important in the elimination of parasites, whereas cell-mediated immune mechanisms involving T cells predominate in the defense against viruses.

The cytokines (interleukin-1, interferon-α) stimulate T-cytotoxic cells and NK cells to kill virally infected cells. B cells, antibodies and complement are also effective against certain viruses and toxin-producing micro-organisms.

1.4 Infection

Resistance to infection depends upon the general state of health of an individual as well as the
individual's innate and adaptive immunological competence (Clarke 1984). Other factors which influence susceptibility include the dose and virulence of the infecting micro-organism (Clarke 1984).

Exercise affects several of the above variables. It can improve the individual's general state of health, but it may also modify exposure (for example, by causing mouth breathing, bypassing the nasal filter) [Niinimaa et al. 1980] and affect innate defences (for example, drying bronchial secretions and slowing ciliary action) [Rylander 1968]. Moderate training improves acquired immune function, but athletes who exercise strenuously may have a reduced immunological competence, particularly in situations where heavy training is combined with the stress of international competition (Fry et al. 1991a, b; 1992).

2. Influence of Exercise on Immune Function

Rigorous athletic training and sports participation modify various components of the innate and adaptive immune systems [Fry et al. 1991a, b, 1992; Keast & Morton 1992], potentially increasing an athlete's immediate susceptibility to infectious illness (fig. 3). The psychological stress of athletic competition may further compromise host defences. However, there has been minimal research directly linking infection in athletes with altered immune parameters (Keast & Morton 1992; Nieman et al. 1990b). Nieman and associates (1990b) demonstrated that 15 weeks of regular moderate exercise (five 45-minute sessions/week at 60% of the heart rate reserve) significantly increased NK cell activity in 18 females relative to 18 matched sedentary females. Moreover, the increased NK cell activity was linked to a reduction in the duration of symptoms associated with upper respiratory tract infections. More recently, Rhind et al. (1993) have found a close correlation between maximal oxygen intake and NK cell activity, with well trained individuals showing a much greater development of the 70 to 75 kDal interleukin-2 β receptor.

2.2 Exercise and Adaptive Immunity

Components of the adaptive immune response such as total lymphocyte counts, the ratio of helper to suppressor T cells, lymphocyte transformation and serum or secretory immunoglobulins (IgG and IgA) are all modified by acute bouts of strenuous exercise (Esperson et al. 1990; Green et al. 1981; Mackinnon et al. 1992; Shinkai et al. 1992). However, a given bout of exercise may have a smaller acute impact on a trained than on an untrained individual (Verde et al. 1991, 1992a, b).

2.1 Exercise and Innate Immunity

The physical barrier of the skin is often damaged through trauma or maceration during sports participation, thus setting the stage for invasion by micro-organisms, particularly if there is an associated immunosuppression. Likewise, the normal filtration mechanism of the nose is bypassed and the inspiration of cold dry air thickens bronchial mucus and slows ciliary clearance of micro-organisms. Other alterations in innate immune parameters associated with very vigorous bouts of exercise include an acute increase of C-reactive protein levels (Fitzgerald 1988, 1991; Liesen & Uhlenbruck 1992), a decrease in resting levels of serum complement (Berk et al. 1988; Nieman et al. 1989c; Smith et al. 1990a) and C-reactive protein (Dufaux et al. 1984), a reduction in neutrophil adherence and phagocytic activity (Gabriel et al. 1993; Lewicki et al. 1987; Smith et al. 1990b) and a post-exercise suppression in NK cell activity (Berk et al. 1990; Mackinnon et al. 1986; Shinkai et al. 1992).

On a more long term basis, regular moderate exercise may enhance innate immunity (Crist et al. 1989; Keast & Morton 1992; Nieman et al. 1990b). Nieman and associates (1990b) demonstrated that 15 weeks of regular moderate exercise (five 45-minute sessions/week at 60% of the heart rate reserve) significantly increased NK cell activity in 18 females relative to 18 matched sedentary females. Moreover, the increased NK cell activity was linked to a reduction in the duration of symptoms associated with upper respiratory tract infections. More recently, Rhind et al. (1993) have found a close correlation between maximal oxygen intake and NK cell activity, with well trained individuals showing a much greater development of the 70 to 75 kDal interleukin-2 β receptor.
the helper to suppressor T cell ratio below 1.0 to 1.5 is thought to indicate immunosuppression (Kest et al. 1988; Shephard et al. 1991). A reduced ratio has been observed following acute bouts of both strenuous submaximal exercise (Espersen et al. 1990; Verde et al. 1992a) and maximal exercise (Berk et al. 1986; Hack et al. 1993; Lewicki et al. 1988); however, reduced rates are not invariably observed in athletes who are showing other signs of overtraining (Fry et al. 1992).

Prolonged moderate intensity aerobic exercise (greater than 60% maximum oxygen uptake) and participation in a marathon run have both reduced in vitro lymphocyte proliferation response to external mitogens (Eskola et al. 1978; Hedfors et al. 1976; Shinkai et al. 1992; Verde et al. 1991, 1992a). Verde and colleagues (1991, 1992c) reported that a deliberate 38% increase in the training volume of endurance runners, who already had a heavy conditioning schedule, reduced the cumulative lymphocyte proliferation rates immediately after an acute exercise bout. They noted that 2 of the 10 runners developed rhinoviral infections during the 3-week period when immune function was suppressed by the deliberate increase of training load.

Reductions in secretory immunoglobulins (IgA) have also been observed following prolonged exercise in treadmill runners (Hack et al. 1993; McDowell et al. 1992), in cyclists (MacKinnon & Jenkins 1993; MacKinnon et al. 1986; Pedersen &
Tvede 1993), in cross-country skiers (Tomasi et al. 1982), in distance runners (Müns et al. 1989), in kayakers (Mackinnon et al. 1993) and in swimmers (Mackinnon et al. 1992), although repeated participation in basketball games increased resting salivary IgA (Thorp 1991). When athletes had low salivary immunoglobulin levels (IgA), susceptibility to upper respiratory infections was increased (Mackinnon et al. 1991; Order et al. 1989). Ninety-two percent of athletes who developed upper respiratory infections had low secretory IgA concentrations 2 days before developing their illness (Mackinnon et al. 1992).

The primary mechanism responsible for many of these changes is still unknown. Changes in blood levels of both hormones (particularly catecholamines and cortisol) and cytokines may play a role. The salivary and mucosal content of immunoglobulins is also affected by the rate of secretion of these fluids and any impairment in the function of mucosal B cells because of changes in the mucosal membranes (Tomasi et al. 1982).

2.3 Hormonal Effects

Changes in plasma concentrations of such hormones as adrenaline (epinephrine) [Field et al. 1991; Kappel et al. 1991] and corticosterone (Pedersen et al. 1991; Tonnesen et al. 1987), induced by either exercise or psychologically stressful situations can have important immunomodulatory effects, altering the rate of transport of cells between lymph tissue and the blood stream, modulating the attachment of immunocompetent cells to the vascular endothelium, and reducing lymphocyte proliferation rates (Keast et al. 1988). Hydrocortisone can also interfere with the availability of immunoglobulin (Fc) receptors on neutrophils, impairing the phagocytosis of IgG sensitised particles (Klempner & Gallin 1978).

3. Physical Activity and Infection
3.1 Upper Respiratory Tract Infections

Coaches and athletic team physicians have used anecdotes and case studies to support their uncritical contention that a combination of excessive training and the psychological stress of competition increase an athlete's risk of developing an upper respiratory tract infection (Eichner 1993; Fitzgerald 1988; Heath et al. 1992; Jokl 1974; Keast & Morton 1992; Keast et al. 1988; Legwold 1982; Linde 1987). In contrast, most athletes believe that normal physical training enhances their resistance (Green et al. 1981; Nieman et al. 1989a, 1990b), although they agree that intensive competition can increase susceptibility (Nieman et al. 1989a, 1990a). Problems in understanding the extent to which exercise increases susceptibility include: mechanical effects of exercise that modulate exposure to infectious micro-organisms [for instance, mouth-breathing (Niinimaa et al. 1980), drying of the mucosa and slowing of ciliary action (Rylander 1968)]; and a confounding of symptoms from exercise and from any super-imposed infection or bronchospasm.

The most common upper respiratory tract symptom is an acute coryza, usually caused by viral infection (the 'common cold'). Almost 200 different viruses have been implicated, but the rhinovirus or coronavirus is the most common pathogen (Heath et al. 1992; Roberts 1986; Thornton 1990). Typical symptoms include nasopharyngitis, nasal catarrh, cough and sore throat, with minimal fever and little or no systemic involvement. Other less common manifestations of upper respiratory tract infections include pharyngitis, croup, bacterial tracheitis or epiglottitis (Heath et al. 1992). In general, the risk of respiratory infection decreases to early adulthood (Heath et al. 1992), but increases again in older adults. Infants and young children have from 4 to 8 infectious episodes per year. In young adults the incidence ranges from 1 to 6 episodes per year (Badger et al. 1953; Fox et al. 1972; Gwaltney et al. 1966; Roberts 1986), common population averages being 1.5 to 2 episodes per year (Berglund & Hemmingson 1990).

Studies which have examined the relationship between exercise and upper respiratory tract infections yield conflicting results (table II). Some authors have reported an increase in the incidence, duration and/or perceived severity of upper respi-
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Infection in physically active individuals (Douglas & Hanssen 1978; Heath et al. 1991; Linde 1987; Linenger et al. 1993; Nieman et al. 1990a; Peters & Bateman 1983; Seyfried et al. 1985). Other studies have reported either no difference (Lee et al. 1992; Österback & Qvarnberg 1987) or even a reduction in the number and severity of such infections (Karper & Boschen 1993; Nieman et al. 1989b; Nieman et al. 1990b; Schousten et al. 1988). Some research has suggested that top level athletes have a greater risk of upper respiratory tract infections compared with moderately active or sedentary control groups (Douglas & Hanssen 1978; Linde 1987). Recreational athletes were also shown to have a predisposition to upper respiratory tract infection, particularly when participating in certain high risk activities such as swimming (Seyfried et al. 1985). Other studies have examined the impact of major competitions on the subsequent development of infections (Nieman et al. 1989b, 1990a; Peters & Bateman 1983).

Douglas and Hanssen (1978) compared upper respiratory infections in university rowers with a sedentary group. The athletes reported symptoms more frequently and perceived their symptoms (stuffy nose, cough, malaise, laryngitis, aching joints and muscles, watery or burning eyes, nasal discharge and sore throat) as being more severe than those reported by the nonathletes.

Linde (1987) examined the incidence of respiratory tract infections in 42 elite orienteers (14 female, 28 male) and 41 nonathletes (14 female, 27 male), matched for age, sex and occupation. 2.5 episodes of upper respiratory tract infections were reported in orienteers compared with 1.7 episodes in nonorienteers (p < 0.05). The symptoms also lasted longer in the orienteers than in the nonathletes (7.9 vs 6.4 days). However, no objective measures of immune function were made. Thus, it seems possible that the athletes (through their exercise) were more conscious of mild symptoms and as a result were more likely to seek medical care.

A prospective study of recreational swimmers and nonswimmers (Seyfried et al. 1985) found morbidity rates of 7.0 and 3.0% respectively. Respiratory ailments were most common in both groups, followed by gastrointestinal, eye, ear, skin and allergic symptoms. Overall, the swimmers reported more symptoms for each route of infection. Swimmers who swam with the head under water reported a substantially higher incidence of eye, ear and skin infections compared with those who swam with the head above water, but people who swam with the head above the water still reported more frequent respiratory, gastrointestinal and skin infections than nonswimmers.

Peters and Bateman (1983) interviewed 150 marathon runners and 150 age-matched sedentary individuals before and after the runners had participated in a 56km ultramarathon. In the 2 weeks immediately following the race 33.3% of the runners reported symptoms such as runny nose, cough or sore throat, compared with 15.3% of nonrunners. The faster runners also seemed the most likely to report such complaints.

These results are supported by the findings of Nieman et al. (1990a). A questionnaire was used to examine the effects of exercise and perceived stress on the incidence of respiratory complaints (sore throat, cold or influenza) among almost 5000 participants in the 1987 Los Angeles marathon. The percentage of questionnaires returned was low (46.9%), and was probably biased by those who had developed infections. However, the results showed that the most active runners had an increased likelihood of reporting such symptoms, both during training and following the marathon race. During training, the odds ratio was doubled in individuals who ran more than 97 km/week relative to those running less than 32 km/week. In the week following the race, respiratory symptoms were reported 5 times more frequently by participants than by runners who trained but did not compete in the event.

In contrast, participation in less strenuous and less competitive events did not alter the likelihood of respiratory complaints (Nieman et al. 1989b); the incidence of respiratory symptoms in 273 runners was similar before and after short road races (5, 10 and 21km).
### Table II. Studies on exercise and upper respiratory tract infections (URI)

| Reference | Design | Participants | Age (years) | Evaluation methods | Findings |
|-----------|--------|--------------|-------------|--------------------|----------|
| **Incidence studies** |        |              |             |                    |          |
| Strauss et al. (1988) | Prospective | 87 athletic team members | 18-33 | Weekly interviews | 86% report 1 URI over 8 weeks |
| Budgett & Fuller (1989) | Retrospective | 69 oarsmen | 18-33 | Questionnaire | URI in 59% over 52 weeks |
| Berglund & Hemmingson (1990) | Prospective | 121M, 53F cross-country skiers | 16-21 | Diary technique | URI accounts for 92.3% of all illness |
| **Risk of URI increased** |        |              |             |                    |          |
| Douglas & Hanson (1978) | Prospective | 61 rowers, 126 cadets | 18-26 | Validated symptom checklist | ↑ frequency & severity of URI in athletes |
| Peters & Bateman (1983) | Prospective | 150 runners vs nonrunners | 18-65 | Questionnaire, interview | ↑ URI in athletes, seen in faster runners |
| Seyfried et al. (1985) | Prospective | 8000 swimmers vs nonswimmers | 0-70 | Questionnaire, telephone | ↑ URI in swimmers |
| Linde (1987) | Prospective | 42 orienteers, 41 controls | 19-34 | Diary | ↑ URI incidence and duration in orienteers |
| Nieman et al. (1989a) | Retrospective | 9M, 1F marathon runners | 27-44 | Questionnaire | 7 perceive ↑ risk of URI following competition |
| Nieman et al. (1990a) | Retrospective | 2311 runners vs nonrunners | 34-39 | Questionnaire | 2 x ↑ URI with training, 6 x ↑ post-race |
| Heath et al. (1991) | Prospective | 447M, 83F runners | 13-75 | Questionnaire | ↑ URI with ↑ running mileage |
| Linenger et al. (1993) | Prospective | 482 trainee-months (special warfare trainees) | 18-31 | Physician diagnosed | High incidence of URI |
| **Risk unchanged** |        |              |             |                    |          |
| Österback & Qvarnberg (1987) | Prospective | 62 sports participants vs 75 controls | 11-14 | Interview | ↔ in URI sports vs controls |
| Schouten et al. (1988) | Retrospective | 92M, 107F active people | 20-23 | Questionnaire | URI not related to fitness or activity level |
| Nieman et al. (1989b) | Retrospective | 183M, 90F runners | 31-38 | Questionnaire | ↔ pre- vs post-race |
| Lee et al. (1992) | Prospective | 96 Air Force cadets | ? | Medical charts, symptom checklist | Immune function depressed but no increase in infections |
| **Risk reduced** |        |              |             |                    |          |
| Green et al. (1981) | Retrospective | 20M marathon runners | 23-46 | Questionnaire | 9 perceive risk of URI ↓ |
| Schouten et al. (1988) | Retrospective | 92M, 107F active people | 20-23 | Questionnaire | ↓ URI symptoms in active females |
| Nieman et al. (1989a) | Retrospective | 9M, 1F marathon runners | 27-44 | Questionnaire | 9 perceive risk of URI ↓ |
| Nieman et al. (1990b) | Prospective | 36F | 25-45 | Log books | Physical conditioning ↓ URI symptom duration |
| Karper & Boschen (1993) | Prospective | 16 (10F, 6M) | 60-72 | Log books | Physical conditioning reduced infections relative to initial values (retrospective) |

*a Epidemiological study.

**Abbreviations and symbols:** F = female, M = male, ↑ = increase, ↓ = decrease, ↔ = no change; ? = unknown.
A large proportion of athletes (e.g., cross-country skiers, gymnasts, oarsmen, swimmers, and wrestlers) suffer from upper respiratory tract infections, but no more frequently than would be anticipated in sedentary people (Berglund & Hemmingson 1990; Budgett & Fuller 1989; Strauss et al. 1988).

Strauss and colleagues (1988) interviewed men's intercollegiate athletic teams (wrestling, swimming, and gymnastics) over an 8-week competitive season. Of 87 athletes, 75 (86%) developed at least 1 upper respiratory infection. Moreover, the incidence of illness was highest during the first 5 weeks of training. Such figures certainly exceed the usual rates for the general population of 1.5 to 2.0 episodes per year (Berglund & Hemmingson 1990), although observations were collected during the winter months of January and February (when respiratory infections are somewhat more frequent).

Berglund and Hemmingson (1990) had 174 individuals maintain a 1-year diary in which they noted their reasons for absence from training. The main reason cited was an upper respiratory tract infection; however, the frequency reported in their study (1.5 incidents/year) was no greater than in the general population. Other reasons for halting training included skin infections and gastroenteritis. Budgett and Fuller (1989) used a retrospective study to investigate illnesses in 30 international oarsmen; 83% of them reported upper respiratory tract infections, with an incidence of 1.4 episodes per year.

The relationship between the weekly walking/running distance and the risk of upper respiratory infections is best explained by a ‘J’ shaped model (Heath et al. 1992; Nieman & Nehlsen-Cannarella 1992). The model is descriptive rather than quantitative, implying that excessive training or a single very severe bout of exercise increases the risk of upper respiratory tract infections, whereas habitual moderate exercise lowers the risk relative to a sedentary lifestyle. Nieman et al. (1990a) found that the distance runner’s risk of illness increased when training exceeded 97 km/week. Heath et al. (1991) analysed upper respiratory tract infections in 530 runners. Risk factors included running more than 776 km/year (15 km/week), living alone and a low body mass index. It was suggested (although without strong evidence) that social support protected against infection and that the stress of living alone predisposed to the development of illness. In support of the adverse effect of a low body mass index, recent biochemical studies suggest that the reserves of amino acid in muscle tissue may be quite important to immune function (Parry-Billings et al. 1990, 1992).

One study of heavy military training (Linenger et al. 1993) noted a high incidence of both musculoskeletal injuries and respiratory infections. A second report (Lee et al. 1992) demonstrated a reduction of in vitro immune responsiveness during basic cadet training, but in this study there was no associated increase in symptomatology or illness.

Schouten et al. (1988) reported a negative correlation between the incidence of upper respiratory tract symptoms and a moderate level of sports participation in female study participants. Nieman et al. (1990b) also demonstrated that 15 weeks of physical conditioning (45 minutes, 5 days/week) at a moderate intensity (60% of the heart rate reserve) helped to reduce the duration of symptoms of upper respiratory tract infections in a group of women 25 to 45 years of age. Karper and Boschen (1993), likewise, trained 16 seniors 3 times a week for 9 to 12 months; 12 of the 14 who completed the programme showed a reduced incidence of respiratory symptom relative to their pre-programme experience.

Evidence of the relationship between other types of sports activities (Österback & Qvarnberg 1987; Schouten et al. 1988) and the incidence of upper respiratory tract infections is inconsistent, and in some instances the ‘J’-shaped model does not appear to apply. For example, Schouten et al. (1988) found no relationship between either the incidence or the duration of upper respiratory tract infections and the volume of habitual physical activity or the resulting maximal oxygen consumption. Schouten et al. (1988) quantified the weekly dose of physical activity as the product of duration (min/week) and energy expenditure (METs/week),...
making the study difficult to compare with other investigations that have specified running distances; however, the exercise levels considered (> 700 METs-min/week) seem less than in some studies where adverse effects were observed.

Österback and Qvarnberg (1987) conducted a prospective study of respiratory infections in children who participated in extracurricular physical activity (gymnasts, swimmers and ice hockey) 4 times per week. The incidence of infection was the same as in other elementary school children and music students who participated only 2.7 times per week (intensity and duration not specified). However, no record was made of spontaneous leisure activity, and it seems likely that the total dose of exercise even in the more active group was less than in some studies reporting adverse effects.

Overall, these studies suggest that elite and endurance athletes may be more susceptible to upper respiratory tract infections than the general population during periods of excessive training and/or intense competitive stress. In contrast, recreational athletes do not appear more susceptible to upper respiratory infections than the general population and some groups have even shown an enhanced resistance to infectious illness. Further research is required to determine the type, intensity and duration of exercise most likely to be immunoenhancing. It would also be helpful to obtain data on such potential modifiers of the immune response as lean tissue mass, nitrogen balance and tissue injuries. Finally, it seems desirable to examine possible protection of immune function from the use of prostaglandin inhibitors such as indomethacin (Pedersen et al. 1990; Shephard & Shek 1993).

3.2 Cutaneous Infections

Athletic activity, particularly contact sports, is associated with an increased risk of skin infections (table III). Bacterial, viral and fungal infections may develop through direct contact with others and may be facilitated by excessive exposure to ultraviolet radiation, sweating and skin friction (Conklin 1990; Sharp et al. 1988). Sweating macerates the skin, softening the stratum corneum and impairing the body’s natural defence against infection (Basler 1983). Trauma also breaches the physical barrier of the skin. A moist and softened skin facilitates both adherence and skin penetration by micro-organisms (Cole & Silverberg 1986). Athletic clothing and footwear offer a warm, moist environment that is conducive to the growth of both bacteria and fungi and problems are particularly likely if athletic clothing is not washed frequently (Allen & King 1978; Basler 1983; Duncan et al. 1969).

3.2.1 Bacterial Infections

Sports activities contribute to the development of such bacterial infections as impetigo, furunculosis and folliculitis (Basler 1983; Conklin 1990). Impetigo is common in wrestlers and rugby players (Becker et al. 1988; Dorman 1981; Glezen et al. 1972; Ludlam & Cookson 1984). It is usually caused by *Streptococcus pyogenes*, although there may be secondary *Staphylococcus aureus* infection (Bergfeld 1984; Conklin 1990). Otitis externa is seen frequently in swimmers (Hicks 1977; Hoadly & Knight 1975).

Furuncles and abscesses usually occur because of localised *S. aureus* infections (Bergfeld 1984). Such lesions are common in basketball players (Sosin et al. 1989), football players (Bartlett et al. 1982; Sosin et al. 1989) and in river rafters (Decker et al. 1986). A persistent superficial infection of the hair follicles, termed ‘acne mechanica’ (Basler 1983; Farber et al. 1977), commonly occur in athletes, such as football and hockey players who need to wear heavy protective clothing.

3.2.2 Viral Infections

Viral infections of the skin in sports participants include herpes simplex, molluscum contagiosum, and verrucae vulgaris (Basler 1983). The most frequently reported skin infection is herpes simplex type I virus (HSV-1). The incubation period for HSV-1 ranges from a few days to 2 weeks (Nelson 1992). Prodromal symptoms such as a burning, stinging or itching of the skin or an erythema may precede infection by a few hours. The condition is characterised by an extensive vesiculopustular eruption, with an underlying erythema (Nelson
Table III. Sports activities and skin infections

| Sport         | Disease | Micro-organism          | Type of infection               | Reference                          |
|---------------|---------|-------------------------|---------------------------------|------------------------------------|
| Basketball    | Bacterial | *Staphylococcus aureus* | Furunculosis                     | Sosin et al. (1989)               |
| Boxing        | Viral   | Smallpox                | Vaccinia                        | Hanssen (1948)                    |
| Figure skating| Not identified |                        | ‘Skin infection’                | Smith & Michel (1982)             |
| Football      | Bacterial | *Streptococcus pyoderm* | Scrum strep/pox (impetigo)      | Gliezen et al. (1972)             |
|               |         | *Staphylococcus albus,* | Acne mechanica                  | Farber et al. (1977)              |
|               |         | *Corynebacterium*       |                                 |                                    |
|               |         | *Staphylococcus aureus* | Furunculosis                     | Bartlett et al. (1982); Sosin et al. (1989) |
| River rafting | Bacterial | *Staphylococcus aureus* | Furunculosis, abscesses, cellulitis | Decker et al. (1986) |
| Rugby/soccer  | Viral   | Herpes simplex virus I  | Scrum pox, herpes rugbeiorum, herpes venaturum | Mare et al. (1978); Shute (1979); White et al. (1984); Verbov & Lowe (1974) |
|               |         | *Streptococcus pyogenes*| Scrum kid (impetigo)            | Dorman (1981)                     |
|               | Fungus/bacterial |                      | Tinea pedis                     | Ludlam & Cookson (1984)           |
| Skiing        | Viral   | Herpes simplex virus I  | Herpes labialis                 | Mills et al. (1987); Spruance et al. (1988) |
| Swimming      | Viral   | Molluscum contagiosum   |                                 | Postlewaite & Watt (1967); Nilizeki et al. (1984) |
|               | Bacterial | Herpes simplex virus I  | Herpes                          | Hicks (1977)                      |
|               |         | *Pseudomonas aeruginosa*| Otitis externa                  | Hicks (1977); Hoadly & Knight (1975) |
|               | Fungus/bacterial | Trichophyton, Candida albicans | Tinea pedis                  | Gentles et al. (1974); Hicks (1977) |
| Wrestling     | Viral   | Herpes simplex I virus  | Herpes gladiatorum              | Becker et al. (1988); Belongia et al. (1991); Duda (1989); Dyke et al. (1965); Nelson (1992); Porter & Baughmann (1965); Selling & Kibrick (1964); Wheeler & Cabaniss (1965); White (1992) |
|               | Bacterial | Molluscum contagiosum   | Impetigo                        | Becker et al. (1988)              |
|               |         | *Streptococcus pyogenes*|                                 |                                    |

1992; Porter & Baughmann 1965). 25% of athletes may experience systemic effects, including fever, myalgia, lethargy, headache, sore throat, and regional lymphadenopathy (Nelson 1992).

HSV infection has been observed among athletes involved in contact sports and recreational athletes who have had prolonged exposure to ultraviolet radiation. Wrestlers affected often refer to it as ‘herpes gladiatorum’ (Becker et al. 1988; Belongia et al. 1991; Duda 1989; Dyke et al. 1965; Nelson 1992; Porter & Baughmann 1965; Selling & Kibrick 1964; Wheeler & Cabaniss 1965; White 1992). In rugby or soccer, it is has been termed ‘herpes rugbeiorum’, ‘herpes venaturum’ or ‘scrum pox’ (Maré et al. 1978; Shute et al. 1979; Verbov & Lowe 1974). Athletes in contact sports are at particular risk of infection because of skin abrasions during direct skin contact with infected indi-
individuals. The most common sites of infection are the head, neck, upper extremities and trunk.

Infection of the lips (‘herpes labialis’) is common in skiers (Mills et al. 1987; Spruance et al. 1988). Excessive exposure to ultraviolet radiation and cold are triggering factors for development or reactivation of herpes labialis (Mills et al. 1987). The stress of high altitude exposure or increased physical activity may also provoke a reaction (Mills et al. 1987). The average time between first exposure to the sun and reactivation of the skin condition is 3.5 days (Mills et al. 1987). It is believed that sun-induced skin irritation stimulates nerve fibres originating in the trigeminal ganglion, and that this reactivates the virus (Mills et al. 1987). Frequent exposure to the virus may increase epidermal susceptibility (Mills et al. 1987).

Molluscum contagiosum infection is common in swimmers. It is acquired through water contact in pools, pool decks and changing areas (Niizeki et al. 1984; Postlewaite & Watt 1967), but may also be transmitted through skin contact and trauma (Becker et al. 1988; Roberts 1986). Verrucae vulgaris is not particularly frequent in athletes (Basler 1983; Conklin 1990; Freeman & Bergfeld 1977; Houston & Knox 1977; Resnik et al. 1977). Plantar warts can be transmitted on swimming pool decks and floors of shower areas, whereas hand warts can be transmitted by the handling of gymnastic equipment (Basler 1983; Conklin 1990). Athletes may have an increased risk of developing warts because of the macerating effect of water or sweat on the skin (Basler 1983). The resulting discomfort and pain can impair performance (Houston & Knox 1977).

### 3.2.3 Fungal Infections

Tinea pedis is transmitted through swimming pools and the floors of gymnasium and locker rooms (Gentles et al. 1974; Hicks 1977). Organisms potentially involved include *Trichophyton rubrum*, *Trichophyton mentagrophytes* and *Candida albicans* (Resnik et al. 1977). There may also be a mixed fungal and bacterial infection (Conklin 1990). Small vesicles between the toes, sides of the feet and the soles may progress to an advanced scaling dermatitis with a reddened border (Bergfeld 1984). Analogous infections include tinea cruris (‘jock itch’) and monilial intertrigo (Houston and Knox 1977).

### 3.3 Other Infections

Pools, rivers, lakes and sea water may contain a mixture of avirulent, virulent, and opportunistic pathogenic micro-organisms (Joseph et al. 1991) which can lead to outbreaks of gastrointestinal illness (nausea, vomiting, stomach cramps, and diarrhoea) amongst swimmers. Infection can occur in individuals following ingestion of contaminated water while swimming (Rosenberg et al. 1976). Contamination of swimming pool water and subsequent disease transmission may be the result of either a malfunction in the pool chlorination system or a faecal accident (D’Alessio et al. 1981; D’Angelo et al. 1979; Harter et al. 1984; Kappus et al. 1982; Lenaway et al. 1989; Porter et al. 1988; Turner et al. 1987). Inefficient disposal of sewage and waste waters has also resulted in elevated counts of infectious virus particles (Norwalk agent), bacteria (Enterococci) as well as parasites (*Shigelloides sonnei*) in fresh and salt water (Balrajan et al. 1991; Baron et al. 1982b; Joseph et al. 1991; Makintubee et al. 1987; Rosenberg et al. 1976; Von Schirnding 1989).

The severity of pneumonia, hepatitis and poliomyelitis (table IV) are all augmented if exercise is performed during the infectious stage of the disease (Berg et al. 1971; Cowles 1918; Hargreaves 1948; Krinkler & Zilberg 1966; Russell 1947, 1949). One outbreak of hepatitis A in members of a football team (Morse et al. 1972) was traced to use of a contaminated water source near a playing field; however, exercise-related immunosuppression may have also made the team more susceptible to the infection. Baron et al. (1982a) reported that athletes have an increased vulnerability to an enterovirus, with some athletes developing aseptic meningitis.

There have been suggestions that human immunodeficiency virus (HIV) infection could be spread by physical contact between 2 wounded athletes,
## Table IV. Physical activity and other infections

| Infection                                | Observation                                                                 | Reference                                                                 |
|------------------------------------------|-----------------------------------------------------------------------------|---------------------------------------------------------------------------|
| Viral hepatitis                          | Vigorous physical activity associated with fulminating hepatitis             | Krinkler & Zilberg (1966)                                                 |
|                                          | Unusual outbreak of hepatitis A observed in football team members            | Morse et al. (1972); Friedman et al. (1985)                               |
| Case study of a football player           |                                                                             | Bowman (1976)                                                            |
| Epidemic of serum hepatitis in cross-country track runners |                                                                             | Berg et al. (1971)                                                       |
| Physical activity in early convalescence did not cause relapse or prolong recovery |                                                                             | Edlund (1971)                                                            |
| Light exercise (e.g. walking) was not harmful in the acute phase of hepatitis |                                                                             | Chalmers et al. (1955); Nefzger & Chalmers (1963)                         |
| Aseptic meningitis (Entero-viral illness) | 70% of football team members affected vs 10% of non-team members            | Baron et al. (1982a)                                                     |
| Human immunodeficiency virus (HIV)       | Acquired immune deficiency syndrome in a bodybuilder may have been the result of sharing of infected needles while injecting anabolic steroids | Torre et al. (1990); LaPerriere et al. (1990, 1991); Schlenzig et al. (1990) |
|                                          | Report of transmission of HIV during a football match                        |                                                                           |
|                                          | Aerobic exercise training in previously sedentary seropositive males         |                                                                           |
|                                          | attenuate acute stress related decrements in immune function. Training      |                                                                           |
|                                          | increased helper-T cell concentration and improve psychological status      |                                                                           |
| Mononucleosis (Epstein Barr virus)       | Noncontact physical activity can safely be initiated after the febrile stage| Welch & Wheeler (1986)                                                   |
|                                          | Contact sport can be resumed 1 month after illness provided                  | Eichner (1987)                                                           |
|                                          | splenomegaly is not present                                                  |                                                                           |
| Pneumonia                                | Respiratory infection progressed to pneumonia after intense exercise         | Cowies (1918)                                                            |
|                                          | and physical sport                                                          |                                                                           |
| Poliomyelitis                            | Increase in incidence and severity of paralysis associated with physical     | Russell (1947); Hargreaves (1948); Russell (1949); Horstmann (1950)      |
|                                          | activity. Paralysis located in active body regions                           |                                                                           |
|                                          | Poliomyelitis infected members of football and soccer teams                 | Weinstein (1973)                                                         |

but the World Health Organisation has noted that this is an extremely remote risk (Sharp et al. 1988). Only 2 instances of transmission between athletes have been described to date, although it is possible that such incidents may be underreported. Torre et al. (1990) reported transmission of HIV between 2 players as a result of a collision during a football match. Sklarek et al. (1984) suggested that the sharing of needles for anabolic steroid injections may have led to HIV infection and acquired immune deficiency syndrome (AIDS) in at least one bodybuilder.

In a recent review, Calabrese and LaPerriere (1993) summarised the current state of knowledge regarding the effects of exercise on the immune system of individuals infected with HIV. Regular moderate physical conditioning can improve the quality of life of HIV infected individuals (Macarthur et al. 1993) as well as decrease HIV disease progression, regardless of their stage of infection [asymptomatic HIV infected, AIDS related complex (ARC), AIDS] (Calabrese & LaPerriere 1993). Also, in individuals who have become seropositive, regular moderate physical activity can not only improve physical fitness (Macarthur et al. 1993; Rigsby et al. 1992; Spence et al, 1990), but also attenuate decrements in immune function (LaPerriere et al. 1990, 1991) with increases in helper T cell counts (LaPerriere et al. 1990, 1991; Schlenzig et al. 1990).
4. Exercising Before and During an Acute Infectious Illness

4.1 Morbidity and Mortality

Interactions between exercise and infection are complex and multidimensional (Ilbäck et al. 1989). The effect of exercise on the progression of a disease depends upon the type of infection, the quantity and quality of the exercise that is undertaken and the timing of exercise relative to the course of the infection. In general, moderate exercise before an infection may enhance resistance, whereas exhaustive exercise during the infectious stage of an illness may be detrimental (Cannon & Kluger 1984). During convalescence, moderate exercise does not seem to have any adverse effects (Chalmers et al. 1955; Nefzger & Chalmers 1963; Welch & Wheeler 1986).

4.1.1 Bacterial Infections

Early researchers initially hypothesised that fatigue might increase susceptibility to infectious disease (Bailey 1925; Nicholls & Spaeth 1922; Oppenheimer & Spaeth 1922). Several animal species (guinea pigs, rabbits, rats) were exercised to fatigue before experimental infection with type I pneumococcus. Such studies suggested that exhausting exercise before inoculation increased the animal’s resistance to infection. More recently, studies on mice infected with Salmonella typhimurium or Francisella tularensis have also demonstrated that physical conditioning prior to infection augments the production of antibodies (Liu & Wang 1987). Moreover, survival rates have remained the same (Ilbäck et al. 1984) or have even been enhanced by previous exercise (Cannon & Kluger 1984).

4.1.2 Parasitic and Viral Infections

In contrast to the findings for bacterial infections, exercise before and during a parasitic or viral infection increases morbidity and mortality (Elson & Abelmann 1965; Ilbäck et al. 1984). Individuals who exercise during a viral illness can develop myocarditis, with some increase in the risk for sudden death (Drory et al. 1991; Phillips et al. 1986), although a fatal viral myocarditis is a relatively rare occurrence. Animal models have examined the effects of forced swimming on the virulence of Coxsackie B3 virus (Cabinian et al. 1990; Gatmaitan et al. 1970; Kiel et al. 1989; Lerner & Wilson 1973; Reyes & Lerner 1976; Reyes et al. 1981; Tilles et al. 1964). Mice who were exercised demonstrated extensive myocardial inflammation and necrosis, with an increased mortality. However, forced treadmill exercise in mice increased morbidity but had no effect on mortality (Ilbäck et al. 1989).

Studies involving humans, monkeys and mice have demonstrated that acute exercise during infection with anterior poliomyelitis or hepatitis can augment the severity of the disease (Hargreaves 1948; Horstmann 1950; Krinkler & Zilberg 1966; Levinson et al. 1945; Russell 1947, 1949; Rosenbaum & Harford 1953; Weinstein 1973). In a study of 411 patients from 3 epidemics of anterior poliomyelitis, Horstmann (1950) noted that when physical activity was undertaken at the onset of the illness, it resulted in a significant increase in the severity of disease process. More of those who escaped eventual paralysis had bed rest during the first 3 days of the infection. Likewise, Krinkler and Zilberg (1966) reported that 5 patients with fulminant hepatitis had all undertaken vigorous physical activity during the early stages of the disease.

The finding that the course of bacterial infections is less influenced by exercise than the course of viral or parasitic infections may be explained by the differing immune responses that they elicit. Bacterial infections involve predominately phagocytic cells, whereas the immune response to viral and parasitic infections is predominately T cell mediated (Thong 1986). This suggests that heavy exercise adversely affects immune function primarily through T cell mediated mechanisms; a deterioration in cell-mediated immunity may well explain the greater virulence of infections in animals that are exercised during infection (Cabinian et al. 1990; Kiel et al. 1989).
4.2 Decrements in Physical Performance

Animal studies have demonstrated impaired physical performance, as reflected by a reduced time to exhaustion, during various types of acute infection (Friman et al. 1982, 1991; Ilbäck et al. 1983, 1991). One of the first studies reporting a decrement of physical performance during an infection was on patients with pulmonary tuberculosis (Dorman & Friedlander 1942); a decrement in physical ability was suggested as an early indicator of tuberculosis infection, although it is possible that tuberculous disease sufficient to limit performance would be regarded as relatively advanced by modern standards. In humans, aerobic power, isometric and isokinetic strength are all adversely affected during acute infectious illnesses (Daniels 1985; Friman 1977, 1985). Athletic performance is also commonly impaired during an infectious illness, but the effects are transitory, and the customary level of performance is quickly restored after convalescence (Friman 1977).

Various factors contribute to these decrements in performance. Infection is a physiological stressor. It disturbs resting homeostasis and induces an inflammatory response (Cularke 1984). Metabolic shifts occur during the early phase of infection (for example, the core temperature, heart rate and oxygen consumption of the host are all increased) [Ilbäck et al. 1991]. Later, during the post-infective phase, there may be changes in the activities of performance-related enzymes in the skeletal muscle and myocardium (Friman et al. 1985; Ilbäck et al. 1991). In addition, an athlete may perceive the infection as imposing a handicap, increasing their psychological stress and thus impairing performance (Clarke 1984; Friman et al. 1985). Performance decrements have also been observed to occur in association with sleep deprivation and shifts in time zones (Shephard 1984). Circadian rhythms may contribute to variations in performance dependent upon reaction speed and muscle force (Shephard 1984).

5. Clinical Implications

This review has illustrated that athletes are not always resistant to infectious illness and that in some circumstances they may even have an increased susceptibility to upper respiratory tract and skin infections. Preventive measures should be instituted to reduce the risk of infection including: minimising exposures to pathogens, monitoring of the training plan, maintaining an adequate diet and limiting exposures to stressors (Shephard & Shek 1993).

To minimise exposure to infection, athletes should avoid contact with individuals (especially small children) who have symptoms or signs of an infectious illness. Any team members who develop symptoms should be isolated from their peers as soon as is practicable. Since viruses are often transmitted by hand contact, the hands should be washed regularly (especially before eating) and any rubbing of the eyes or of the nose should be avoided (Shephard & Shek 1993).

As overtraining and the stress of competition increase susceptibility to infection, athletes should avoid excessive conditioning and ensure that other life stresses are minimised (Fry et al. 1992; Heath et al. 1992). Sufficient recovery time should be allotted between strenuous practices and competitive events, the diet should be well balanced and sufficient to maintain a positive nitrogen balance, and adequate sleep should be encouraged (Heath et al. 1992), particularly if circadian rhythms have been disturbed by travel. Treatment modalities such as sauna, massage, hydrotherapy and physiotherapy may help to reduce the level of stress (Shephard & Shek 1993). Other possible measures include counselling, progressive muscle relaxation, distraction or visual imagery. Muscular immunoglobulin injections may reduce the incidence, duration and severity of upper respiratory tract illness in athletes (Billigmann 1991; Nieman & Nehlsen-Cannarella 1991; Weiss 1993). The inoculation of viruses endemic to the area where an athlete will compete may also be useful (Shephard & Shek 1993).

Athletes who have developed a minor upper respiratory tract infection without systemic involve-
ment may continue to exercise during the infection (Roberts 1986; Simon 1987). Decongestants should be taken during the day, with addition of an antihistamine at night (Thornton 1990), although care must be shown to avoid conflict with antidoping rules as competition is approached. Use of most aerosol β₂-agonists [salbutamol (albuterol), terbutaline, orciprenaline, rimiterol and bitolterol], with the exception of fenoterol, are permitted during international competition (Morton & Fitch 1990). Other acceptable medications include: sodium cromoglycate, nedocromil sodium, theophylline, aerosol ipratropium bromide and beclomethasone (Morton & Fitch 1990).

In view of the occasional findings of cardiomyopathy, the training intensity should be reduced if there are symptoms or signs of systemic involvement (such as fever, myalgia, fatigue or lymphadenopathy). In such individuals, 2 to 4 weeks should be allowed for recovery before strenuous exercise is resumed. Eichner (1993) recently provided a ‘neck check’ for anyone wanting to exercise while they were sick. If all symptoms were ‘above the neck’ (runny, stuffy nose or scratchy throat), the athlete could begin their exercise routine at a below normal intensity for 10 minutes. If such activity made them feel worse, they should stop exercising, but if they felt better, they should continue the training session. However, if any symptoms were ‘below the neck’ (muscle aches, cough, vomiting, diarrhoea or fever) training should be discontinued.

Since most athletic skin infections are spread through direct contact, competitors should be screened for skin lesions before participation in contact sports. Individuals infected with HSV should be excluded from competition until the lesions are no longer contagious (Duda 1989; Simon 1987). Oral aciclovir can be helpful in the relief of symptoms (Duda 1989; White 1992). A combination of sunscreen and aciclovir is recommended in individuals who are susceptible to herpes labialis (Nelson 1992). Other skin infections such as folliculitis and tinea pedis may be treated by a combination of general hygienic measures, antibiotic and antifungal treatments.

Coaches and athletes should be aware of the hazards of HIV infection. Special precautions should be used when dealing with body fluids. Open wounds should be covered immediately and any spills of blood on the floor or clothing should be cleaned as quickly as possible. According to the American Pediatric Society Policy Statement (1992), HIV infected individuals should be allowed full participation in sports programmes. Their serum positive status should not be disclosed, and the testing of athletes for HIV infection should be performed only on a voluntary basis.

6. Conclusions

Epidemiological data indicate some increase in the risk of upper respiratory tract and skin infections in athletes who are engaged in heavy training. The risk is further enhanced immediately following athletic competition, because of the psychological stress of the competitive event itself. Alterations in immune surveillance associated with intensive training and competition include a reduction in lymphocyte proliferation rates, a reduction in NK cell activity and low concentrations of immunoglobulins in serum and body secretions.

Data from both animal and human studies suggest that the intensity and duration of exercise, its timing relative to exposure to an infectious agent, and the type of infection all influence the effect of exercise on the immune response and vice versa. Overall, regular moderate physical conditioning apparently improves immunosurveillance, enhances resistance and decreases the duration of disease symptoms, but intensive training impairs the immune response, particularly when it is combined with the stress of major competition. Intensive or exhausting physical activity immediately before or during the early stages of an infection may increase the severity and lethality of a disease. Such an effect is greatest for viral infections, perhaps as a consequence of differences in the immune responses to viral and other types of infection.
More research is needed on the interactions between athletic endeavour, immunity and infection, to improve guidelines for athletic training and competition. Researchers should seek the critical change in immune function that increases an athlete's susceptibility to infection. Based upon this finding, specific prophylactic measures may then be designed to minimise the adverse effects of heavy training and to maximise immunosurveillance during competition.

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