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CONTAGIOUS INFECTIONS IN COMPETITIVE SPORTS

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KEY POINTS

1. Exercise can change blood levels, proportions, and functions of white blood cells, especially natural killer cells. These changes are generally modest and brief, but they may have clinical importance.
2. Whether exercise helps or harms immunity or increases the chance of acquiring upper respiratory tract infection (URTI) or other infections is still debated, but may depend on the stress level involved.
3. The risk today of contracting HIV through competitive sports is next to zero, although a theoretical risk exists for wrestling and certain other "blood sports."
4. Prevention of infections among athletes hinges on common sense, good hygiene, prudent immunization, wise training, and "universal precautions."
5. A practical gauge for the athlete with URTI is the "neck check," wherein symptoms below the neck should preclude strenuous exercise, whereas those above the neck may be less serious.

INTRODUCTION

Many athletes believe that physical training enhances immunity and helps prevent upper respiratory tract infection (URTI) like the common cold or "flu" (influenza). They also believe that physical activity and fitness help them overcome any minor infections they do get. On the other hand, based largely on anecdotes and on popularization of a link between physical or psychological stress and "impaired immunity," the athletic community in general seems persuaded that intensive training, exhaustive exercise, or competition predisposes athletes to infections.

Concerned athletes, trainers, and coaches often ask: 1) Does exercise really enhance immunity? 2) How can athletes avoid URTI when "peaking" or competing? 3) Are their special risks of infection from team sports? 4) Should athletes get the "flu shot," the hepatitis B vaccine, or other immunizations? 5) If an athlete does get an URTI, are there guidelines for training or competing? 6) What is the risk of transmitting human immunodeficiency virus (HIV) through competitive sport?

Applied and epidemiologic research on exercise, stress, immunity, and UTRI has burgeoned, as has been previously reported in the Gatorade *Sports Science Exchange* (Nieman, 1992). Some trends are clear, but their clinical importance is unclear. This review: 1) updates the established immunologic changes in exercisers; 2) offers a perspective on the epidemiologic studies of URTI in athletes and nonathletes; 3) highlights the practical, clinical aspects of infections in athletes; and 4) discusses current concerns about HIV and sports.

IMMUNOLOGIC CHANGES IN EXERCISERS

Exercise can change the blood concentrations, proportions, and functions of white blood cells, especially polymorphonuclear leukocytes (PMNs), natural killer (NK) cells, and lymphocytes, and can affect immunoglobulins and other immune factors.

Rises and Falls in PMNs and Lymphocytes. Vigorous exercise lasting minutes to hours evokes leukocytosis (Eichner, 1993; Gabriel et al., 1992; Nieman, 1992), mainly of PMNs and lymphocytes. The less fit the athlete and the more intense the exercise, the greater the degree of leukocytosis.

The early leukocytosis of exercise is a result of the mechanical action of an increase in cardiac output and the physiologic effect of adrenaline. These two forces move PMNs from along the walls of blood vessels into the circulating blood and release them from reservoirs in lungs, spleen, and liver. The same forces also shape the lymphocytosis of exercise; infusing adrenaline into humans causes both granulocytosis and lymphocytosis (Eichner, 1993; Kappel et al., 1991). Acute exercise may also activate PMNs (Smith et al., 1990).

If the exercise is strenuous and "eccentric" (e.g., downhill running for 30 minutes or longer), there may be a "second rise" in PMNs over the next two to four hours. This "delayed leukocytosis" is due mainly to cortisol, which spurs release of PMNs from the marrow and slows their egress from blood to tissues. The "acute phase response" may also play a role, as blood monocytes infiltrating damaged muscles release interleukins (Weight et al., 1991).

After brief, easy exercise, the PMN count soon returns to baseline, but after long, hard exercise, this return may take 24 hours. The lymphocytosis, however, always fades quickly when exercise ends. After only five minutes of recovery, the lymphocyte count is falling; over the next two hours, it often falls below the resting baseline. Usually within six hours of recovery, and nearly always by 24 hours, the lymphocyte count is back up to normal (Gabriel et al., 1992; Neiman, 1992).

During early recovery, the lymphocyte count likely falls for the same reason the PMN count rises - the unopposed action of cortisol. The action of adrenaline, which brings lymphocytes into the blood, stops when the exercise ends, allowing the longer-lasting action of cortisol to "redirect the traffic" of lymphocytes from blood into lymph organs (Eichner, 1993).

Function of Lymphocytes and NK cells. Exercise lymphocytosis comprises mainly NK cells (to a lesser extent, T-suppressor lymphocytes), which provide a first-line defense against certain viruses (Nieman, 1992). Exercise can also acutely activate NK cells, perhaps due partly to adrenaline (Kappel et al., 1991; Shinkai et al., 1992). These changes vary in degree with the intensity of exercise (Nieman et al, 1993a; Nieman et al, 1994).

If immunity is "enhanced" during exercise, it is briefly "suppressed" afterwards. During early recovery, the aforementioned lymphocytopenia is accompanied by a blunting of lymphocyte function (mitogenesis). It is unclear whether the blunted mitogenesis is real (mediated perhaps by cortisol and/or prostaglandins released from monocytes) or is an artifact of relative enrichment of blood samples by NK cells, which do not undergo mitogenesis (Nieman, 1992; Nieman et al., 1994).

Whether physical training enhances basal NK function is also unclear. In a randomized, controlled study of training (brisk walking) in young women, basal NK activity was enhanced at six weeks but not at 15 weeks (Nieman et al., 1990). In a follow-up study of elderly women, training did not enhance basal NK function (Nieman et al., 1993b). Some cross-sectional studies suggest that athletes, such as highly conditioned elderly female athletes or male marathon runners, have higher basal NK function than do sedentary controls (Nieman et al., 1993b, 1995).

Immunoglobulins and Other Immune Factors. Exercise-related changes in serum immunoglobulins are minor and seem trivial clinically (Nieman, 1992). Salivary immunoglobulin A (IgA) levels are low in skiers at rest and fall further after a ski race. Similar falls can occur after strenuous cycling, swimming, or rowing, and in squash or hockey players; levels generally return to normal within 24 hours. Whether these brief, mild changes impair mucosal defense against URTI is postulated but not proved (Mackinnon et al., 1993).

Strenuous or prolonged exercise can activate complement and spur the release of tumor necrosis factor, interferons, and interleukins, but whether these events alter immunity is unclear.

EPIDEMIOLOGIC AND OBSERVATIONAL STUDIES

Some epidemiologic studies suggest that hard-training athletes are prone to URTI; others do not. Most studies, however, are limited by self-reporting. URTI is generally not proven, but assumed, on the basis of questionnaires.

For example, "high-mileage" runners training for a marathon were twice as apt to report URTI as "low-mileage" runners, and in the week after the marathon, marathon finishers were six times more apt to report URTI as were last-minute marathon nonparticipants. Yet the same researchers, polling half-marathoners, found opposite trends (Nieman, 1992).

Were the half-marathoners exercising just enough to boost immunity and resist URTI? Were the marathoners overdoing it, impairing immunity, and inviting URTI? Or are both studies inconclusive because some symptoms were not from URTI but from allergy, overtraining, or fatigue? Can one validly equate self-reported "colds, flu, or sore throat" with URTI?

Other epidemiologic and observational studies also seem inconclusive. Four examples: 1) In epidemiologic studies, elite Danish orienteers, but not elite Swedish skiers, have more URTIs than do control subjects. 2) In the two weeks after a 56-km marathon, fast runners, but not slow runners, were more apt to report URTI than were controls. 3) A one-year cohort study of 530 runners links a higher incidence of URTI to higher running mileages, but cannot adjust for all confounders and lacks nonrunning controls (Eichner, 1993). 4) In a randomized, controlled training study, the exercisers (walkers) reported only fewer URTI "symptom-days," not fewer URTIs, than did the nonexercisers (Nieman, 1992).

Psychological stress must also be considered. In college students, NK function is inversely related to mental stress. In medical and dental students, NK function and salivary immunoglobulin levels decline as final exams near. Declines in NK function are also seen after divorce, during depression, and even after losing a few hours of sleep (Eichner, 1993; Nieman, 1992).

If physical stress can promote URTI, so can psychological stress. In a prospective epidemiological study in Australia, people under the most mental stress acquired the most URTIs. When 394 healthy volunteers were given nasal drops containing respiratory viruses, the risk of acquiring documented URTI increased in proportion to the degree of mental stress (Cohen et al., 1991).

Conversely, relieving stress may improve immunity. Research suggests that relaxation methods can increase salivary immunoglobulin levels, and that writing essays to "divulge personal traumas" can boost lymphocyte mitogenesis and improve health (Eichner, 1993).

In summary, it is uncertain whether most athletes or active people are more - or less - apt to acquire URTI than nonathletes or sedentary people. Yet, recent authoritative reviews agree that top athletes get more URTIs than the general population, and that - in light of the physical and psychological stress involved - athletes seem to be most vulnerable to URTI around the time of competition (Brenner et al, 1994; Nieman, 1992).

CLINICAL ASPECTS, PRACTICAL IMPLICATIONS

A recent review of the medical literature (going back to 1966) finds 38 reports of infections from competitive sports: 24 outbreaks or instances of person-to-person transmission; nine common-source transmissions; and five airborne transmissions. It also finds 28 newspaper reports of infectious outbreaks or exposures or issues concerning vaccination. The infectious agents were mainly viruses but also fungi and gram-positive and gram-negative bacteria (Goodman et al., 1994).

Of the 24 medical reports of person-to-person spread, most were herpes simplex infections in wrestlers ("herpes gladiatorum") or rugby players, or other skin infections (fungal or bacterial) in wrestlers, football players, or rugby players. Of the nine common-source outbreaks, eight were enteroviral illnesses, either aseptic meningitis or pleurodynia (abrupt onset of chest or abdominal pain, usually with fever), in football players. All five of the airborne outbreaks were measles. The 28 newspaper reports were mainly of flulike illnesses or viral outbreaks (measles, mumps, chickenpox) in football and basketball team-

mates. Measures to prevent such infections are discussed below.

An outbreak of herpes gladiatorum in 60 (35%) of 175 young wrestlers at a Minneapolis training camp suggests means for future prevention. Besides a vesicular rash, some wrestlers also had fever, chills, sore throat, headache, and/or enlarged lymph nodes. Some wrestlers were allowed to compete despite a rash. Transmission was mainly by skin-to-skin spread, aided by skin abrasions from mat burns. No transmission was thought likely via soap, saliva, or shared water bottles (Belongia et al., 1991).

Shared water bottles, however, can be a problem among athletes. In Ohio in 1991, three young football teammates, the coach, and a student manager all developed viral meningitis the same week. In response, the health department began an educational campaign to promote hand washing and to discourage players from sharing ice buckets and drinking vessels (Eichner, 1993).

A similar enteroviral outbreak, this one pleurodynia occurred the same year in high school football players in upstate New York. Behaviors linked to becoming ill were: 1) eating ice cubes from the team ice chest and 2) drinking water from the team cooler. Measures to avoid future outbreaks included: 1) educating students and coaches on how enteroviruses are spread; 2) discouraging direct oral contact with common drinking containers; 3) use of ice packs rather than ice cubes from the team chest for injuries; and 4) use of disposable cups or individual drinking containers (Ikeda et al., 1993).

Measles is a hazard of indoor sports. Airborne outbreaks have occurred in gymnasts, wrestlers, and basketball players (and spectators) competing in packed, humid gyms or "skydomes," ideal settings for respiratory transmission of viruses. Also, sports teams may unwittingly transmit measles when they go on the road. In the spring of 1991, a high school wrestling tournament spread measles all over the state of Maryland, resulting in 126 cases. Perhaps the measles vaccine should be used more widely among young athletes. Some experts also advise giving the influenza vaccine ("flu shot") to team athletes and the hepatitis B vaccine to "contact-sport" athletes or even to all adolescents and young adults (Mast et al., 1995; Mellman, 1994).

RISK OF HIV TRANSMISSION

Concern about HIV in sport is where the ancient mystique of blood meets the primal fear of contagion. The risk today is next to zero, yet concern is great. Two common questions are: What is the risk of transmitting HIV from sports? and 2) Should athletes be screened for HIV?

Research is reassuring. HIV does not grow in sweat; the risk from sweat is zero. HIV can be grown from tears and saliva, but there is no known case of spread this way (Calabrese et al., 1993).

It boils down to potential blood transmission during sports. Only one "case" has been reported, and it is widely debunked. Italian soccer players bumped heads, cut eyebrows, and bled freely. One was HIV-positive; the other, who had been HIV-negative one year earlier, turned positive two months after the game. The two viruses were not typed to prove identity. Experts reject this anecdotal report as unproven (Mast et al., 1995).

In professional football, there are almost four bleeding injuries per game, yet researchers estimate the risk of HIV transmission in an NFL game to be less than one in one million (Brown et al, 1995). In "analogies" to other "bloody" sports (boxing, wrestling, hockey), there are two reports each of HIV transmission from bloody fistfights and from household blood contact between siblings (Mast et al., 1995). There are also reports of HIV transmission when bodybuilders shared needles to inject anabolic steroids (Scott & Scott, 1989).

Other useful analogies relate to hepatitis B tranmission and to the health care field. After needlestick exposure, hepatitis B is transmitted from six percent to 30 percent of the time, whereas HIV is transmitted only 0.3 percent of the time. In other words, hepatitis B is up to 100 times easier to acquire through blood than is HIV. Yet only one clearcut case of hepatitis B transmission has occurred through sport, when five of 10 young Japanese sumo wrestlers were infected from a teammate who bled on them during matches (Kashiwagi et al., 1982). No case of hepatitis B transmission from sports has been reported in the United States.

Reassuring from the health care field is that no HIV transmission has occurred among 2,700 cases of blood splashed from a known carrier onto normal skin, and only four cases of tranmission have occurred when large amounts of blood splashed into eyes or mouth or onto denuded skin. But these were freak accidents, unlikely to apply to sports (Calabrese et al., 1993).

Arguing by analogy, then, the risk of getting HIV from most sports is next to zero. Yet controversy rages on whether athletes should be screened for HIV. Most experts argue against screening (Mast et al., 1995); some call for screening in wrestling; and some screening occurs in professional boxing (Calabrese et al., 1993; Mitten, 1995).

Screening policies among NCAA institutions range from the sublime to the ridiculous. Of 860 institutions polled, 548 responded: 78 percent had no HIV screening; 18 percent, voluntary or "on-request" screening; and four percent, mandatory screening of athletes. Twelve institutions had found HIV-positive athletes, most of whom were no longer competing. Fifteen institutions barred HIV-positive athletes from hockey and wrestling; 14 barred them from football; 13 from basketball; 11 from baseball; and six barred them even from golf and tennis (McGrew et al., 1993).

Short of screening, "universal precautions" against potential spread of HIV during sports are widely published (Mast et al., 1995). The key features are: 1) Wear gloves when touching or wiping up body fluids ("If it's wet and not yours, don't touch it without gloves"); 2) For athletes in the "contact" sports, securely cover or wrap skin wounds (scratches, abrasions, lacerations) and potentially infectious skin lesions (vesicular or weeping lesions) to prevent leakage of blood or serum during the game; 3) Cleanse bloody skin immediately with soap and water or a premoistened towelette; 4) An injured athlete can return to the game only after the wound has been securely covered or wrapped; 5) Any bloody uniform or equipment (tape, padding) must be changed at the earliest convenience (such as when play is stopped for other reasons); 6) Disinfect (and let dry) all bloody surfaces or equipment, using disposable toweling and a 1/100 dilution of household bleach (sodium hypochlorite, l cup to 4 gallons water) made fresh daily; 7) After each practice or game, launder bloody uniforms or gloves, using standard laundry cycles and detergents.

CONCLUSION

The risk today of getting HIV through sports play is next to zero, although a theoretical risk exists for "blood sports" like wrestling and boxing. As for URTI, whether exercise "prevents or promotes" may depend on whether the exercise routine is "a joy or a stress."

Common sense and "universal precautions" can help prevent infections in athletes. Vaccines should be used more widely. Hygiene should be taught and enforced. Team physicians should clear any athletes with signs or symptoms of illness, or with rashes. Coaches can help prevent "overtraining", which may predispose athletes to URTI and other infections, by: 1) balancing training and rest; 2) monitoring mood, fatigue, symptoms, and performance; 3) reducing distress; and 4) ensuring optimal nutrition (Eichner, 1995). Athletes should be educated on how HIV spreads and how to avoid it off the field: abstinence, monogamy, and condoms. Athletes should never share needles, razors, earrings, or even toothbrushes.

What to tell athletes about exercising with an URTI? First, an acute, febrile viral infection saps both strength and endurance, so any workout by a sick athlete will be subpar. Why do it?

Second, it is hard to know early on that an "URTI" is only a rhinovirus, and not an incipient mycoplasmal pneumonia, for example, or a Coxsackie virus that can cause myocarditis and sudden death during exercise. Why not rest when sick?

Third, alas, some athletes are so driven that they must work out despite the above advice. For them, use the "neck check," in their language:

If symptoms are "above the neck" (stuffy or runny nose, sneezing, watery eyes, scratchy throat), try a "test drive" at "half speed;" if you feel better after 10 minutes, you can "rev up" and finish the workout.

But do not work out if you have fever or symptoms "below the neck" or bodywide (aching muscles, hacking cough, nausea, vomiting, diarrhea). Your body will recover faster with rest (Eichner, 1993).

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