HIGHLIGHTED TOPIC | Exercise and Inflammation

Immune function in sport and exercise

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Gleeson M. Immune function in sport and exercise. J Appl Physiol 103: 693–699, 2007; doi:10.1152/japplphysiol.00008.2007.—Regular moderate exercise is associated with a reduced incidence of infection compared with a completely sedentary state. However, prolonged bouts of strenuous exercise cause a temporary depression of various aspects of immune function (e.g., neutrophil respiratory burst, lymphocyte proliferation, monocyte antigen presentation) that usually lasts ~3–24 h after exercise, depending on the intensity and duration of the exercise bout. Postexercise immune function dysfunction is most pronounced when the exercise is continuous, prolonged (>1.5 h), of moderate to high intensity (55–75% maximum \( \text{O}_2 \) uptake), and performed without food intake. Periods of intensified training (overreaching) lasting 1 wk or more may result in longer lasting immune dysfunction. Although elite athletes are not clinically immune deficient, it is possible that the combined effects of small changes in several immune parameters may compromise resistance to common minor illnesses, such as upper respiratory tract infection. However, this may be a small price to pay as the anti-inflammatory effects of exercise mediated through cytokines and/or downregulation of toll-like receptor expression are likely mediators of many of the long-term health benefits of regular exercise.

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The circulating numbers and functional capacities of leukocytes may be decreased by repeated bouts of intense, prolonged exercise. The reason is probably related to increased levels of stress hormones during exercise (26) and entry into the circulation of less mature leukocytes from the bone marrow (13). Falls in the blood concentration of glutamine have also been suggested as a possible cause of the immunodepression associated with heavy training, although the evidence for this is less compelling (13). Also, during exercise, there is an increased production of reactive oxygen species, and some immune cell functions can be impaired by an excess of free radicals (31). During exercise, exposure to airborne pathogens is increased due to the higher rate and depth of breathing. An increase in gut permeability may also allow increased entry of gut bacterial endotoxins into the circulation, particularly during prolonged exercise in the heat. Hence the cause of the increased incidence of infection in athletes is likely to be multifactorial: a variety of stressors (physical, psychological, or environmental, nutritional) can suppress immune function (13), and these effects, together with increased exposure to pathogens, can make the athlete more susceptible to infection.

ACUTE EFFECTS OF EXERCISE ON IMMUNE FUNCTION

A single, acute bout of prolonged, strenuous exercise has a temporary depressive effect on immune function, and, in a few studies involving rather extreme exercise (marathons and ultramarathons), this has been associated with an increased incidence of infection in the weeks following the event. For example, several studies have described a substantially higher (two- to sixfold) frequency of self-reported symptoms of URTI in athletes who completed long-distance foot races compared with control runners who did not compete in the events (30, 34–36). An acute bout of physical activity is accompanied by responses that are remarkably similar in many respects to those induced by infection, sepsis, or trauma (32): there is a substantial increase in the number of circulating leukocytes (mainly lymphocytes and neutrophils), the magnitude of which is related to both the intensity and duration of exercise. There are also increases in the plasma concentrations of various substances that are known to influence leukocyte functions, including inflammatory cytokines, such as TNF-α, macrophage inflammatory protein-1, and IL-1β; anti-inflammatory cytokines IL-6, IL-10, and IL-1-receptor antagonist (IL-1ra); and acute phase proteins, including C-reactive protein (CRP). The large increase in plasma IL-6 concentration observed during exercise can be entirely accounted for by release of this cytokine from contracting muscle fibers (51). However, IL-6 production by monocytes (49) and IL-2 and IFN-γ (but not IL-4) production by T lymphocytes are inhibited during and for several hours after prolonged exercise (21, 32).

Hormonal changes also occur in response to exercise, including increases in the plasma concentration of several hormones [e.g., epinephrine (adrenaline), cortisol, growth hormone, and prolactin] that are known to have immunomodulatory effects. Muscle-derived IL-6 appears to be at least partly responsible for the elevated secretion of cortisol during prolonged exercise. Infusion of recombinant human IL-6 into resting humans to mimic the exercise-induced plasma levels of IL-6 increases plasma cortisol in a similar manner (50). In contrast, the same recombinant human IL-6 infusion does not change plasma catecholamine or insulin levels in resting healthy young subjects. Therefore, muscle-derived IL-6 may be partly responsible for the cortisol response to exercise, whereas other hormonal changes cannot be ascribed to IL-6. Stimulation of cortisol secretion by IL-6 may be due to an effect of IL-6 on the hypothalamus, stimulating the release of ACTH from the anterior pituitary gland, or by a direct effect of IL-6 on cortisol release from the adrenal glands; evidence for both mechanisms exists. In addition, it was recently demonstrated that relatively small increases in plasma levels of IL-6 induce the two anti-inflammatory cytokines IL-1ra and IL-10, together with CRP (50). During exercise, the increase in IL-6 precedes the increase in these two cytokines, arguing circumstantially for muscle-derived IL-6 to be the initiator of this response.

Whether humoral or cell-mediated immunity will dominate depends largely on the type of cytokines that are released by the activated T helper cells. T lymphocytes can be classed as type 1 or type 2 cells, depending on which cytokines they predominantly produce. Type 1 T cells produce mainly IFN-γ and tumor necrosis factor, and their actions activate macrophages and induce killer mechanisms, including T-cytotoxic cells, thus driving the immune system toward cell-mediated immune responses, which primarily provide protection against intracellular pathogens such as viruses. Type 2 cells mainly produce IL-4, IL-5, IL-10, and IL-13, which are necessary for promotion of humoral immunity, IgE-mediated allergic reactions, and activation of potentially tissue-damaging eosinophils. IL-4 and IL-13 primarily drive B-cell differentiation to antibody production, while IL-5 stimulates and primes eosinophils. Together with IL-4, IL-10 (which is also produced by monocytes and B cells) can inhibit type 1 T-cell cytokine production. Interestingly, it appears that exercise can influence the type 1/type 2 cytokine balance.

In accordance with the elevations of circulating IL-6, IL-10, and IL-1ra, strenuous exercise decreases the percentage of type 1 T cells in the circulation, whereas the percentage of type 2 T cells does not change (21). Both cortisol and epinephrine suppress the type 1 T-cell cytokine production, whereas IL-6 directly stimulates type 2 T-cell cytokine production. Another important action of IL-6 is that it suppresses production of TNF-α, which is a potent activator of inflammation (48). As type 1 T cells promote cell-mediated immune responses, which primarily provide protection against viruses, exercise, possibly working through muscle-derived IL-6, may decrease virus protection in the host and thus may account for why athletes appear to be more prone to acquire URTI. However, it is very important to stress that the shift toward type 2 T-cell dominance might be beneficial, because it also suppresses the ability of the immune system to induce tissue damage and inflammation. Blood markers of inflammation are strongly associated with cardiovascular and metabolic disease in the middle-aged and elderly population, and inflammation has been implicated in the pathology of several chronic diseases. Thus elevated systemic levels of IL-6 during and following exercise could be one of the mechanisms by which regular exercise provides protection against the development of chronic diseases (Fig. 1). However, it could be argued that the relative importance of IL-6 in this context is likely to be rather small, as significant health benefits of regular exercise are apparent even when the exercise is of light-moderate intensity.
Acute exercise temporarily increases the number of circulating lymphocytes from athletes, but following exercise, NK cell counts drop to less than half of normal levels for a couple of hours. Normal resting values are usually restored within 24 h. NK-cell cytolytic activity (per cell) falls after exercise, and, if the activity is both prolonged and strenuous, the decrease in NK cell counts and cytolytic activity may begin during the exercise session. During recovery from exercise, lymphokine-activated killer cell numbers and activity also fall below preexercise levels. Acute exercise has been shown to diminish the proliferative response of lymphocytes to mitogens and decrease the expression of an early activation marker (CD69) in response to stimulation with mitogen. When the exercise bout is strenuous and very prolonged, the decrease in NK cell counts and cytolytic activity may begin during the exercise session. During recovery from exercise, lymphokine-activated killer cell numbers and activity also fall below preexercise levels. Acute exercise has been shown to diminish the proliferative response of lymphocytes to mitogens and decrease the expression of an early activation marker (CD69) in response to stimulation with mitogen. When the exercise bout is strenuous and very prolonged (≥1.5 h), the number of circulating lymphocytes may be decreased below preexercise levels for several hours after exercise, and the T-lymphocyte CD4⁺/CD8⁺ ratio is decreased.

Antigen-presenting cell function is also affected by exercise. Exercise-induced reductions in macrophage major histocompatibility complex class II expression and antigen-presenting capacity have been documented. Both T-memory (CD45RO⁺) and T-naive (CD45RA⁺) cells increase temporarily during exercise, but the CD45RO⁺/CD45RA⁺ ratio tends to increase due to the relatively greater mobilization of the CD45RO⁺ subset. The functional significance, if any, of this temporary shift is unclear at present. Following prolonged, strenuous exercise, the production of immunoglobulins by B lymphocytes is inhibited, and delayed-type hypersensitivity responses to subdermal antigen injection (a marker of the cell-mediated immune response) are diminished. These changes during early recovery from exercise would appear to weaken the potential immune response to pathogens and have been suggested to provide an "open window" for infection, representing the most vulnerable time period for an athlete in terms of their susceptibility to contracting an infection.

Numerous studies report effects of exercise on functions of isolated leukocytes when these cells are stimulated in vitro by added antigens or mitogens. However, it is difficult to extrapolate from the in vivo stimulated response of isolated cells to how these same cells would respond in the far more complex in vivo environment. In addition to the presence of antigens, leukocyte function is also influenced by endogenous chemicals, including hormones, neurotransmitters, and cytokines, and the plasma concentration of these may change during exercise. The pH and temperature of the blood also change during exercise, but these factors are often ignored in experiments on isolated cell types. Thus separating cells from their in vivo environment is somewhat artificial and to a large degree excludes the effects of exercise-induced chemical changes in the blood that will undoubtedly modify leukocyte function. The closest one can get to the in vivo condition is by performing measurements on whole blood, in which the proximity between the leukocytes and the extracellular milieu is retained. Another limitation in the interpretation of such studies, even where multiple parameters are assessed, is that presently no instruments are available to predict the cumulative effects of several small changes in immune system parameters on host resistance. Furthermore, it should be borne in mind that only 0.2% of the total leukocyte mass is circulating at any moment; the remainder is in lymphoid tissue, the bone marrow, and other tissues. It may thus be more important to assess the status of leukocytes in the skin, mucosa, and lymph nodes rather than in the blood.

**CHRONIC EFFECTS OF EXERCISE TRAINING ON IMMUNE FUNCTION**

Following an acute bout of exercise, changes in circulating leukocyte numbers and functions normally return to preexercise values within 3–24 h. Cross-sectional studies that have compared leukocyte numbers and functions in blood samples taken from athletes more than 24 h after their last training session with those of sedentary individuals have generally reported very few differences. Thus, in the true resting state, immune function appears to be broadly similar in athletes compared with nonathletes. There is a weak suggestion of a slightly elevated NK cell count and cytolytic action in trained individuals, but these effects are small and unlikely to be of any clinical significance. Levels of secretory immunoglobulins by B lymphocytes are inhibited, and delayed-type hypersensitivity responses to subdermal antigen injection (a marker of the cell-mediated immune response) are diminished. These changes during early recovery from exercise would appear to weaken the potential immune response to pathogens and have been suggested to provide an "open window" for infection, representing the most vulnerable time period for an athlete in terms of their susceptibility to contracting an infection.

Fig. 1. Possible mechanisms by which exercise increases susceptibility to infection but reduces inflammation and risk of developing chronic disease. TLR, Toll-like receptor; TH1, T helper 1; IL-1ra, IL-1 receptor antagonist. The encircled minus sign represents an inhibitory action of IL-6 on TNF production.
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lins, such as salivary IgA (s-IgA), vary widely between indi-
viduals, and, although some early studies indicated that s-IgA
concentrations are lower in endurance athletes compared with
sedentary individuals (54), the majority of more recent studies
indicate that s-IgA levels are generally not lower in athletes
compared with nonathletes, except when athletes are engaged
in periods of very heavy training (12). Furthermore, moderate
exercise training in healthy young adults does not appear to
have an effect on the initiation of a specific antibody response
to vaccination or delayed-type hypersensitivity responses, as
measured by the swelling that arises 48 h after injecting antigens into the skin (4). These in vivo measures of immune
function are probably more meaningful than in vitro individual
cell-type functional measures, as they represent the whole
system response to challenge.

Athletes commonly intensify their training for a few days or
weeks at certain stages of the season. This may induce a state
of overreaching in which performance is temporarily reduced,
but, following a period of taper with only light training, results
in supercompensation and an increase in performance. Several
studies in recent years have investigated the effects of short
periods of intensified training on resting immune function and
on immunoenocrine responses to endurance exercise. These
studies indicate that several indexes of leukocyte function,
including neutrophil and monocyte oxidative burst, T-lympho-
cyte CD4+/CD8+ ratios, mitogen-stimulated lymphocyte prolif-
eration and antibody synthesis, and NK-cell cytotoxic activ-
ity, are sensitive to increases in the training load in already
well-trained athletes (15, 20, 21, 41, 55). Even following
relatively short periods (1–3 wk) of intensified training, marked reductions in neutrophil function, lymphocyte prolif-
eration, s-IgA, and the circulating number of T cells producing
IFN-γ have been observed (12, 20, 21). Thus, with chronic
periods of very heavy training, several aspects of both innate
and adaptive immunity are depressed, but athletes are not
clinically immune deficient. In other words, exercise-induced
immune dysfunction does not put athletes in danger of serious
illness, but it could be sufficient to increase the risk of picking
up common infections such as URTI or influenza should the
dreaded outbreak occur.

Several longitudinal studies have monitored immune func-
tion in high-level athletes such as cyclists (1), swimmers (12,
15), and footballers (5, 8, 39) over the course of a competitive
season. In a recent study of American football players, the
incidence of URTI was increased during intense training, and
it was reported that the secretion rate of s-IgA (which repre-
sents the amount of s-IgA available on the mucosal surfaces for
protection against pathogens) was significantly and inversely
related to URTI incidence (8). In an earlier, much cited study,
the impact of long-term training on systemic and mucosal
immunity was assessed prospectively in a cohort of elite
Australian swimmers over a 7-mo training season in prepara-
tion for the national championships (12, 15). The results
indicated significant depression of resting serum and salivary
immunoglobulin concentrations in athletes, associated with
long-term training at an intensive level. Furthermore, resting
s-IgA concentrations at the start of the training period showed
significant inverse correlation with infection rates, and the
number of infections observed in the swimmers was predicted
by the preseason and mean pretrained s-IgA levels. These
studies on mucosal immunity in athletes are representative of a
very small number of studies that have established a relation-
ship between some surrogate measure of immune function and
infection incidence in athletes. A few studies on soldiers during
intensive periods of military training have also reported a
negative relationship between s-IgA concentration and occur-
rence of URTI (6), although others have not (17, 53). In one
recent study, an increased URTI incidence during 4 wk of
intense military training was significantly correlated with
decreased numbers of circulating NK cells (17). However,
in these situations, the training often involves not only strenuous physical activity, but also dietary energy defi-
ciency, sleep deprivation, and psychological challenges.
These multiple stressors are likely to induce a pattern of
immunoendocrine responses that amplify the exercise-in-
duced alterations.

THE POSTEXERCISE SORE THROAT: AIRWAY INFECTION OR INFLAMMATION?

Self-reporting of symptoms of URTI using questionnaires
has been used in a number of studies designed to evaluate the
effects of acute, prolonged exercise (e.g., running a marathon)
or periods of intensified training on infection incidence. How-
ever, this approach leaves such studies open to the criticism
that the reporting of symptoms (e.g., sore throat, runny nose,
congestion, fever) is subjective and that factors other than
infection (e.g., allergies, inhalation of air pollutants, airway
inflammation) could also cause some of these symptoms.
Recently, a surveillance study was conducted over a 5-mo
summer/autumn competition season to identify the pathogenic
etiolo and symptomatology of upper respiratory illness
(URI) in highly trained elite athletes (n = 32), recreationally
competitive athletes (n = 31), and untrained sedentary controls
(n = 20) (47). Nasopharyngeal and throat swabs were collected
on subjects presenting with two or more defined URI symp-
toms. Swabs were analyzed using microscopy, culture, and
polymerase chain reaction testing for bacterial, viral, chlamyd-
ial, and mycoplasmal respiratory pathogens. The Wisconsin
Upper Respiratory Symptom Survey (WURSS-44) question-
naire was administered to assess the daily symptomatology and
functional impairment. A total of 37 URI episodes in 28
subjects were reported (9 controls, 7 recreationally competitive
exercisers, and 21 elite athletes). The overall distribution mim-
icked the “J”-shaped curve with rate ratios for illness higher in
both the control (1.93, 95% confidence interval: 0.72–5.18) and
elite (4.50, 95% confidence interval: 1.91–10.59) cohorts than
the referent recreationally competitive athlete cohort. How-
ever, of these 37 episodes, infectious agents were identified
in only 11 (30%) (2 control, 3 recreationally competitive
exercisers, and 6 elite athletes). No pathogens were identi-
fied in 26 episodes of URI. Specific global symptom, total
symptom, and functional impairment severity scores were
higher in subjects with an infectious URI episode, particu-
larly on illness days 3–5. These findings strongly suggest
that URI in elite athletes are seldom infectious, and the
symptomatology is distinct between infectious and nonin-
fec tious episodes. In future research, noninfectious causes
of URI should be considered and investigated to identify
alternative mechanisms and mediators.
CAN EXERCISE-INDUCED IMMUNODEPRESSION BE PREVENTED, AND DO WE WANT TO PREVENT IT?

Studies from Bente Pedersen’s group in Copenhagen indicate that the release of IL-6 from contracting muscle can be attenuated by long-term antioxidant supplementation. In a recent single-blind, placebo-controlled study (8), it was reported that 4 wk of oral supplementation, with a combination of vitamin C (500 mg/day) and vitamin E (400 IU/day), markedly attenuated the release of IL-6 from active muscle and the plasma IL-6 and cortisol response to 3 h of dynamic two-legged, knee-extensor exercise at 50% of maximal power output compared with placebo. High levels of circulating IL-6 stimulate cortisol release, and this study provides some strong evidence that the mechanism of action of the antioxidant supplementation was via a reduction in IL-6 release from the muscle fibers of the exercising legs. Attenuating the IL-6 and cortisol response would be expected to limit the exercise-induced depression of immune function, and this may be the mechanism that could explain the reported lower incidence of URTI symptoms in ultramarathon runners supplemented with vitamin C (alone or in combination with other antioxidants) compared with placebo (35, 36).

Consumption of carbohydrate during exercise also attenuates increases in plasma IL-6, catecholamines, ACTH, and cortisol (27, 29). Carbohydrate intake during exercise also attenuates the trafficking of most leukocyte and lymphocyte subsets, including the rise in the neutrophil-to-lymphocyte ratio, prevents the exercise-induced fall in neutrophil function, and reduces the extent of the diminution of mitogen-stimulated T-lymphocyte proliferation following prolonged exercise (16). Recently, it was shown that consuming 30–60 g of carbohydrate per hour during 2.5 h of strenuous cycling prevented both the decrease in the number and percentage of IFN-γ-positive T lymphocytes and the suppression of IFN-γ production from stimulated T lymphocytes observed on the placebo control trial (22). IFN-γ production is critical to antiviral defense, and it has been suggested that the suppression of IFN-γ production may be an important mechanism leading to an increased risk of infection after prolonged exercise bouts (32).

Pedersen’s group, however, have argued that the reduction in the IL-6 response to exercise may be a double-edged sword, as IL-6 has several metabolic effects and shared mechanisms exist regarding immune impairment and training adaptation (48). Attenuating the IL-6 response to exercise will also inhibit lipolysis (48), reduce the anti-inflammatory effects of exercise, and attenuate the expression of a number of metabolic genes in the exercised muscle (37). In other words, it is possible that antioxidant supplementation and/or carbohydrate ingestion during exercise sessions could limit adaptation to training. However, it can also be argued that carbohydrate intake during training allows the athlete to work harder and longer, and as yet there is no evidence that physiological and performance adaptations are impaired by carbohydrate intake during training sessions. Further research is needed to determine how nutrient intake might affect the transcriptional regulation of metabolic genes in skeletal muscle and what, if any, consequences this has for training adaptation or even long-term health benefits.

The concern for athletes is that, although these nutritional interventions may reduce their risk of infection, another effect may be to limit their hard-earned adaptation to training.

A SMALL PRICE TO PAY FOR LONG-TERM HEALTH BENEFITS?

Blood markers of inflammation are strongly associated with chronic disease in the older population, and inflammation has been implicated in the pathiology of several cardiovascular and metabolic diseases. Evidence is now emerging that exercise has anti-inflammatory effects. Individuals who are physically active on a regular basis have a reduction in the levels of biomarkers that are used to assess systemic inflammation. For example, higher levels of habitual physical activity are associated with lower mitogen-stimulated inflammatory cytokine production, lower skeletal muscle inflammatory protein content, lower adipokine production, and lower serum levels of cortisol (27, 29).

Pathogen molecules e.g. LPS
Endogenous danger signals e.g. HSP

Toll-like receptors

Inflammatory cytokine production e.g. IL-1 & TNF-α

Antigen

EPRR

MHCII

TCR

Activation and Differentiation

T cell

Cytokine production e.g. IL-2 & IFN-γ

Antigen Presenting Cell

CD80/86

CD28

Fig. 2. Simplified summary of the results of activating the TLR signaling pathway. Binding of certain pathogen molecules (e.g., LPS) or endogenous danger signal molecules, such as heat shock proteins (HSPs), to TLRs leads to activation of the antigen-presenting cell and subsequent activation of T cells that it interacts with. Antigen-presenting cells take up antigen via endocytic pattern recognition receptors (EPRRs) and process (degrade) it to immunogenic peptides, which are displayed to T-cell receptors (TCRs) in the polymorphic groove of major histocompatibility complex (MHC) class II molecules after their appearance at the cell surface (which is upregulated by TLR activation). An interaction occurs between the antigen-presenting cell and the T cell, as indicated (involving cell surface expression of costimulatory molecules such as CD80 and CD86, which are also upregulated by TLR activation), usually resulting in cellular activation and differentiation. Cytokines produced by antigen-presenting cells (stimulated by TLR activation) and T cells result in inflammation and activation of other immune components.
CRP (14). Thus, although extreme exercise stress may impair immune function and increase susceptibility to infection, this may not be entirely detrimental to the host and may, by reducing immune activation and subsequent inflammation, be one of the mechanisms through which regular exercise benefits long-term health (Fig. 1).

An accumulation of chronic, low-grade inflammation is common in individuals who live a sedentary lifestyle; however, the mechanism underlying this connection is not fully understood. A new and potentially important finding is that, following a prolonged bout of strenuous exercise, the expression of Toll-like receptors (TLRs) 1, 2, and 4 on monocytes is decreased for at least several hours (14). TLRs are highly conserved transmembrane proteins that play an important role in the detection and recognition of microbial pathogens, and they can also be activated by endogenous danger signals of tissue damage, such as heat shock proteins. The key product of TLR signaling in antigen-presenting cells is the production of inflammatory cytokines and proteins, and thus the TLR pathway plays an important role in mediating whole body inflammation (Fig. 2). Evidence is now emerging that TLRs may be involved in the link between a sedentary lifestyle, inflammation, and disease. Recent studies have shown that both acute aerobic and chronic resistance exercise bouts result in decreased monocyte cell-surface expression of TLRs (14, 23, 52). Prolonged exercise also results in a decreased induction of costimulatory molecules and cytokines following stimulation with known TLR activators (23). Furthermore, a period of chronic exercise training decreases both inflammatory cytokine production and the cell-surface expression of TLR4 on monocytes (52). Although these effects may contribute to postexercise immunodepression and the reported higher susceptibility to infection in athletes, this may be a small price to pay as, over the long term, a decrease in TLR expression may represent a beneficial effect, because it decreases the inflammatory capacity of leukocytes, thus altering whole body chronic inflammation and possibly reducing the risk of developing chronic disease. The precise physiological stimulus mediating an exercise-induced decrease in cell-surface TLR expression is not known; however, a number of possible signals have been implicated, including anti-inflammatory cytokines, stress hormones, and heat shock proteins (14).

SUMMARY

In summary, acute bouts of exercise cause a temporary depression of various aspects of immune function (e.g., neutrophil respiratory burst, lymphocyte proliferation, monocyte TLR, and major histocompatibility complex class II protein expression) that lasts ~3–24 h after exercise, depending on the intensity and duration of the exercise bout. Postexercise immune function depression is most pronounced when the exercise is continuous, prolonged (>1.5 h), of moderate to high intensity (55–75% maximum O2 uptake), and performed without food intake. Periods of intensified training (overreaching) lasting 1 wk or more can result in longer lasting immune dysfunction. Although elite athletes are not clinically immune deficient, it is possible that the combined effects of small changes in several immune parameters may compromise resistance to common minor illnesses such as URTI. Protracted immune depression linked with prolonged training may determine susceptibility to infection, particularly at times of major competitions. This is obviously a concern because of the potential impact of an infectious episode on exercise performance. It is not really a concern for the general population, as individuals do not need to indulge in heavy training loads to obtain the health benefits of exercise that may well be proven to be due, in large part, to its anti-inflammatory effects.

Hundreds of studies have now been conducted that confirm both acute and chronic effects of exercise on the immune system, yet there are still very few studies that have been able to show a direct link between exercise-induced immune depression and increased incidence of confirmed illness in athletes. This is an important issue that needs to be addressed in future studies, although it must be recognized that this is a difficult task. Even among the general population, we do not know the impact of small changes in specific immune parameters on risk of infection. Most clinical studies have only been concerned with the risk of life-threatening illness in immunodeficient patients, not with the risks of picking up common infections such as colds and flu.

REFERENCES

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