Journal of Strength and Conditioning Research, 2004, 18(1), 185–193

Brief Review

TISSUE TRAUMA: THE UNDERLYING CAUSE OF OVERTRAINING SYNDROME?

LUCILLE LAKIER SMITH

Tshwane University of Technology, Pretoria, South Africa.

ABSTRACT. Smith, L.L. Tissue trauma: the underlying cause of overtraining syndrome? *J. Strength Cond. Res. 18(1):184–191. 2004.*—An athlete who trains intensely, yet consistently underperforms, is considered to be suffering from overtraining syndrome (OTS). OTS is a complex state that involves a large variety of signs and symptoms. Symptoms include changes in mood or behaviour, decreases or increases in concentration of different blood molecules, and alterations in immune function. Although several hypotheses have been proposed, each only explains a selective aspect of OTS. Presently, the sole agreement is that OTS is associated with excessive training and insufficient rest and recovery. The hypothesis proposed in this paper suggests that excessive training/competing causes repetitive tissue trauma, either to muscle and/or connective tissue and/or to bony structures, and that this results in chronic inflammation. It is further proposed that traumatized tissue synthesizes a group of inflammatory molecules, cytokines. Cytokines have been shown to coordinate the different systems of the body to promote recovery. Suggestions are made to detect, prevent, and rehabilitate the overtrained athlete.

KEY WORDS. burnout, staleness, interleukins, tumor necrosis factor, depression

PURPOSE

The purpose of this paper is to propose an underlying mechanism that drives the condition referred to as overtraining syndrome (OTS). This will be achieved by integrating information from a variety of different areas of research and proposing a unifying hypothesis that will attempt to explain many of the biochemical, psychological, physiological, hormonal, and immune changes that have previously been associated with OTS. For the sake of brevity, where possible, review articles are referenced.

Although a majority of studies have demonstrated OTS in endurance-trained athletes (27, 29, 35, 38), this condition may equally impact anaerobically/resistance-trained athletes, with some variations (28, 27, 64, 73), although this issue remains uncertain (1, 29, 35, 73). However, since a typical conditioning program incorporates many different aspects of training, including high-intensity resistance exercise, high-volume resistance exercise, interval/sprint training, and endurance training, a distinction will not be drawn between different aspects of training/competing and the induction of OTS. Additionally, it is proposed that OTS occurs in subelite as well as in lay athletes, but, for obvious reasons, is not closely monitored in these cases.

INTRODUCTION

OTS, also known as "staleness" or "burnout" is a serious condition that afflicts many athletes, often in the prime of their athletic careers. By definition, it occurs when an athlete is training intensely, but, instead of improving, shows a deterioration in performance, even after an extended rest period (1, 26, 27, 29, 47, 55, 74). This sports-specific decrease in performance, be it in individual or team sports (56), is regarded as the gold standard, and may extend over a period of weeks or months (39), while in some instances, the athlete may never recover (1, 27, 55).

There are numerous signs and symptoms associated with OTS (Table 1) (1, 29, 55, 73, 75). These may be grouped into four categories: psychological, physiological, biochemical, and immunological. However, athletes manifest different combinations of these signs and symptoms, with varying degrees of severity (56). Presently, there do not appear to be specific patterns of signs and symptoms that can be associated with specific sporting events (56). However, it has been suggested that there may be distinct differences between sports regarding which signs and symptoms predominate, although to date this has not been clarified (27, 73). Generally, the first indication of impending OTS is a change in mood (1, 45, 47, 73), although it is the decline in performance that usually captures the attention of the athlete and coach.

OTS should be distinguished from the condition of short-term overtraining, typically referred to as "overreaching" or "supercompensation training" (27, 39, 73). "Overreaching" describes an initial temporary deterioration in performance, usually lasting a few days (27). With sufficient rest, athletic performance is regained and frequently there is improvement (26, 27, 73). "Overreaching" is regarded by many as part of the training stimulus/adaptation (27, 73).

CAUSES OF OVERTRAINING SYNDROME?

Notwithstanding the seriousness and prevalence of OTS, and despite the fact that this condition has been recorded as far back as the 1920s (quoted in 55, 75), the underlying causes of OTS remain unclear. A number of hypotheses have been proposed, but for the most part each explains only one aspect of OTS. For example, the glycogen hypothesis suggests that general fatigue and complaints of 'heavy legs' are due to reduced muscle glycogen (10, 70). The central fatigue hypothesis suggests that general complaints of fatigue are due to an excess of a specific amino acid, tryptophan, in the central nervous system (36). The glutamine hypothesis suggests that the prevalence of immune-related disorders is due to reduced blood levels of glutamine, an amino acid crucial to optimal functioning of immune cells (51, 76). There are additional hypotheses concerning the causes of OTS, including involvement of the sympathetic and parasympathetic nervous system

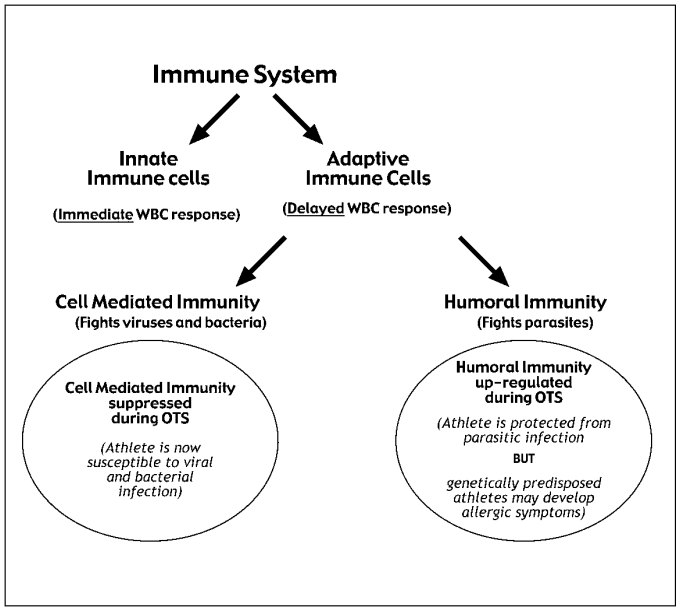

FIGURE 1. Proposed continuum of injury that may occur in response to training. This Figure suggests that mild injury is adaptive and part of the "normal" training response, while severe injury is undesirable.

(37), involvement of the hypothalamic pituitary axis (2), and emphasis on the problem of performing similar workouts on a daily basis, which induces a condition of "monotony" (24). It is proposed in this paper that most of these hypotheses are pertinent, but that none explain the totality of the condition.

At present, it appears that the only thing that most investigators agree upon is that OTS is related to an increase in volume and/or intensity of training, or a consistently high volume of training/competing over an extended period of time, with insufficient time for recovery (26, 27, 54, 55). Thus, it is frequently the conscientious athlete who suffers from OTS, and not the athlete who is casual about his or her training schedule.

As was stated earlier, the purpose of the present paper is to present a hypothesis that attempts to integrate many of the signs and symptoms that have previously been associated with OTS and ascribe them to one underlying cause. It is proposed that this underlying cause is some form of tissue trauma (67). A number of investigators have previously described an association between injury and OTS (14, 17, 26, 28, 40, 55, 57). However, none have specifically identified tissue trauma as the primary driving force in this condition. This possibility will now be explored.

EXERCISE AND TISSUE TRAUMA

It is well established that mild tissue trauma, followed by recovery, is an integral part of the training process (1, 67). Thus, when tissue trauma is mild and sufficient time is allowed for recovery/adaptation to occur, athletic performance frequently improves; this has been referred to as adaptive microtrauma (AMT) (67) (Figure 1). However, when the athlete dramatically increases volume and/or intensity, often abruptly, and does not allow sufficient time for recovery, it is possible that what was initially a mild, self-limiting form of tissue trauma develops into a more chronic, severe form of tissue trauma. This trauma/injury may be to skeletal muscle tissue, to bone, to connective tissue such as tendons or ligaments, and/or to connective tissue invaginated within muscle. It is also suggested that this tissue trauma differs from an acute, specific injury, such as injury to a hamstring muscle or to the Achilles tendon, which is obvious and requires clear-cut attention. It is proposed that the underlying injury implied to be involved in OTS is a more diffuse, widespread, low-grade trauma, not as easily identified as is an acute overt injury, and may be more similar to an overuse injury (26) or possibly to a repetitive-motion injury resulting from high-volume training.

There is in fact a considerable amount of evidence that would support the notion that an athlete who is experiencing OTS has sustained some form of tissue trauma, since it is well established that heavy training is associated with muscle damage (12, 26, 28, 32, 55, 71). Furthermore, in OTS there are distinct signs and symptoms that may be directly ascribed to injury/trauma. These include complaints of "heavy legs", of muscle and joint aches and pains, of persistent muscle soreness that increases from one session to the next, and of muscle weakness (28, 29, 54). There have also been reports of increased levels of blood markers of muscle damage and inflammation, such as elevated creatine kinase and C-reactive protein, respectively (27–29, 55, 75). Recently, several scientists, using muscle biopsy techniques, have confirmed the presence of muscle trauma in athletes who train/compete excessively (12, 63, 71). Therefore, direct and indirect information is suggestive of the presence of muscle/tissue trauma in overtrained subjects.

Importantly, with regard to injury and OTS, it is suggested that tissue trauma/injury may be the factor that directly impacts on performance (28). It is well documented that with muscle microtrauma, there is a reduction in strength (77) and a reduction in range of motion, most likely due to swelling of the injured area (9); these factors may result in an alteration in execution of specific skills, due to compensatory movements that protect the injured area. Fry et al. (28) suggest that with high-intensity resistance exercise overtraining, the primary site of maladaptation appears to be in the periphery, as indicated by decreases in 1 repetition maximum (1RM) and in voluntary and stimulated isometric leg extension torque. So injury, of even a subclinical nature, may well account for the primary sign of OTS, a decrease in performance (5, 28).

ACUTE AND CHRONIC INFLAMMATION AND OVERTRAINING SYNDROME

The body's response to any form of tissue damage is the up-regulation of acute inflammation, irrespective of the cause of injury or the type of tissue that has been traumatized (6, 61, 69). Acute inflammation is a well-orchestrated response, involving such diverse systems as the blood clotting cascade, complement activation, and the immune system, and is the only mechanism capable of promoting healing/regeneration. Up-regulation of acute inflammation, as would occur after an acute injury such as a sprained ankle, usually resolves within approximately 72 hours, although healing may not be complete for up to 6 weeks (61). If acute inflammation does not

resolve, for any variety of reasons (such as insufficient time for recovery, or repeated application of injurious stimulus), then acute inflammation may be transformed into an ongoing, undesirable condition of chronic inflammation (4).

A major player in acute and chronic inflammation is the infiltration of specific white blood cells (WBC) into the traumatized tissue at specific intervals after the injury. Factors that assist these cells in localizing to the appropriate site, and in the precise sequence, are several families of molecules, collectively referred to as cytokines (65).

INJURY, CYTOKINES, AND OVERTRAINING SYNDROME

Cytokines may be regarded as "emergency" molecules in that they are generally not detectable in the circulation under normal, "healthy" conditions. However, if the body is exposed to some kind of trauma, either an injury or illness, then a variety of different cells begin producing these cytokine molecules (42).

Cytokines are also regarded as "communication" molecules. They are crucial for transmitting information from one cell to another (paracrine action), and when found in increased concentrations in the blood, they transmit information around the whole body (endocrine action). In this manner, different organs of the body are "informed," for example, of a possible injury in the musculature, and thus the brain, liver, kidneys and immune cells can make necessary adjustments to accommodate the injury and promote healing/recovery (42).

There are several different families of cytokines and many different cytokines within a family. This paper will be dealing primarily with the pro-inflammatory cytokines: interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α), as well as interleukin-6 (IL-6), with the latter cytokine possessing pro- and anti-inflammatory properties (13).

The basis of the hypothesis presented in this paper is that high-volume/intensity training with insufficient rest/recovery, may result in chronic injury and thus chronic inflammation. This in turn results in the production of elevated levels of circulating cytokines (IL-1β, TNF-α, and/or IL-6), which interact with various systems of the body, and which, it will be argued, may account for most of the signs and symptoms that have previously been associated with OTS. It is further suggested that these signs and symptoms may be adaptive, in that, if an individual is injured, it is in the best interests of the organism to rest and redirect all resources to regaining the state of wellness. Therefore, it is proposed that many of the signs and symptoms seen in OTS may represent the body's attempt to heal itself (67). The role of injury and cytokines will now be examined with regard to psychological changes, biochemical changes, and alterations in immune function.

MOOD STATE IN OVERTRAINING SYNDROME AND CYTOKINES

One of the first signs of OTS is a change in mood and behavior. The athlete may be transformed from an outgoing, enthusiastic, sociable, highly competitive individual, to being constantly tired, depressed, and uninterested in training and competing (1, 4, 25, 48, 55, 73–75), and possibly angry and hostile (73). Frequently, changes in mood and behavior are evident before the drop in performance (1, 47, 48), but, as long as the athlete is performing well, these factors may be ignored.

In an attempt to explain these changes in mood and behavior, one needs to keep in mind that modern medicine is a recent phenomenon in terms of our evolutionary history. Until recently, healing and recovery from illness and injury were heavily dependent on natural environmental phenomena, including the individual's behavior. This is clearly illustrated by Hart's (31) description of how a sick or injured animal behaves in the wild, in order to increase its chance for recovery. The wounded animal becomes lethargic (akin to human depression), loses its appetite, has a reduced thirst drive and a reduced libido, and is less sociable. All these factors encourage rest and protect it from exposure to predators, thus increasing its chances of survival. This cluster of behaviors is now referred to as "sickness" or "recuperative" behavior. Many overtrained athletes display similar behavioral changes, including reduced appetite, thirst, libido, and desire to socialize, and many appear to be clinically depressed (29, 48, 73). Although this behavior may seem inappropriate for an athlete, if, in fact, the athlete is suffering from a chronic injury, such behavior should optimize recovery.

What factors could activate these behavioral changes in OTS? There is now a growing body of evidence that suggests that elevated blood levels of proinflammatory cytokines (IL-1β, IL-6, and TNF-α) are able to communicate with the brain and induce these behaviors commonly referred to as "sickness behavior" (11). It appears that cytokines represent the link between the body (circulation) and the brain (42), inducing behavioral changes that are more appropriate for an injured or sick individual (11, 16).

Cytokines access the brain via several routes, and act on several different areas of the brain (42). One important area is the hypothalamus, which is regarded as the control center for many basic drives such as hunger, thirst, and libido. The action of cytokines in these areas could undoubtedly produce many mood and behavioral changes that have been seen in OTS (Table 1). In particular, cytokines have been closely linked to the development of depression (41). Cytokines may also act on other areas of the brain such as the hippocampus, which could disrupt memory and learning (15, 42). Inability to concentrate and impaired academic performance (55), are all factors that have been noted in OTS. Thus, cytokines produced due to tissue trauma and chronic inflammation could account for previous reports of mood or behavior changes and for changes in cognitive functioning associated with OTS.

In addition to changing behavior by acting on specific brain structures, cytokines have also been shown to alter the levels of certain hormones in the peripheral circulation. Specific cytokines have been shown to inhibit the release of certain hormones in the hypothalamus (43), which would be necessary for the release of testosterone in the periphery of the body; this could account for the reduced levels of testosterone seen in OTS (27, 72, 75). Cytokines may also be responsible for stimulating an increase in blood levels of certain other hormones such as cortisol (62, 72); increased blood cortisol levels have also been reported in OTS (27, 72, 75). Changes in circulating levels of testosterone and cortisol could further exacer-

bate mood or behavior changes (72). In summary, mood and behavior changes, as well as changes in levels of circulating hormones, seen in OTS could be accounted for by the action of cytokines on the brain. It is suggested that injury-related cytokines access the brain and cause changes in mood and behavior and hormonal patterns that are not conducive to optimal athletic performance, but rather are conducive to rest and subsequent recovery.

BLOOD MARKERS IN OVERTRAINING SYNDROME AND CYTOKINES

Many investigators have noted changes in the concentration of different circulating molecules in OTS, with some increasing (such as C-reactive protein) and others decreasing (such as albumin) (29) (Table 1). Many of these changes appear to be disassociated. However, if one considers the athlete to be injured and suffering from chronic inflammation, these changes may be viewed as reflecting part of the process termed the acute phase response. An important aspect of the acute phase response is the production of "new" proteins, which will assist in recovery from illness/injury. These new proteins are collectively referred to as "acute phase proteins" and will now be discussed. (The word "acute" is extremely confusing, since many of these "acute" phase proteins may also be associated with chronic conditions [58]).

In response to any illness or injury, the body is able to rapidly begin synthesizing these "new" acute phase proteins due to altered liver functioning (58). Generally, the liver may be regarded as a molecule-producing factory, synthesizing many different molecules essential for normal daily living. However, when illness or injury occurs, with a concomitant increase in blood levels of proinflammatory cytokines (IL-1β, IL-6, and TNF-α), circulating cytokines act on liver cells (hepatocytes) to alter the typical day-to-day production of molecules and stimulate the synthesis of different recovery-related molecules—acute phase proteins. The ill or injured individual who experiences local trauma now develops a systemic (circulatory) counterpart to this local trauma, which again represents part of the body's self-healing arsenal. It is proposed in this paper that many of the changes seen in the blood chemistry of overtrained athletes reflect alterations in the synthesis of acute phase proteins (Table 1).

Increased synthesis of acute phase proteins may also explain, in part, reductions in muscle mass, as well as a negative nitrogen balance, frequently associated with OTS (29, 73). In a sick or injured individual with a reduced food intake, there is a reduced availability of amino acids to synthesize all the necessary proteins to assist with recovery and healing. However, the body's muscle mass represents an abundant store of proteins/amino acids (44). Thus, during illness or injury, there are frequently increased blood levels of the catabolic hormone cortisol, as well as certain cytokines such as IL-6 and TNF-α which act to degrade muscle protein, resulting in the release of amino acids into the circulation (7). Cytokines then stimulate liver cells to absorb these amino acids (20), thus making amino acids available to synthesize new acute phase proteins (44).

This increased uptake of amino acids from the blood into liver cells, driven by the presence of elevated blood cytokines, would account for the reduced levels of amino acids in the blood, such as reduced blood glutamine (21).

Table 1. Signs and symptoms associated with overtraining syndrome (based on 29, 55, 73, 75).

Performance parameters
Decreased performance
Inability to meet previous standards
Prolonged recovery
Reduced toleration of loading
Decreased muscular strength
Decreased maximum work capacity
Physiological
Changes in blood pressure
Changes in heart rate at rest, during exercise, and during recovery
Increased frequency of respiration
Increased oxygen consumption at submaximal exercise intensities
Decreased body fat
Decreased lean body mass
Elevated basal metabolic rate
Psychological/behavioral
Constant fatigue
Reduced appetite
Change in sleep patterns (hyper- or hyposomnia)
Depression
General apathy
Decreased self-esteem
Emotional instability
Fear of competition
Easily distracted
Gives up when the going gets tough
Information processing
Loss of coordination
Reappearance of previously corrected mistakes
Difficulty in concentrating
Decreased capacity to deal with large amounts of information
Reduced capacity to correct technical faults
Biochemical parameters
Rhabdomyolysis
Elevated C-reactive protein
Elevated creatine kinase
Negative nitrogen balance
Increased urea concentration
Increased uric acid production
Hypothalamic dysfunction
Depressed muscle glycogen levels
Decreased hemoglobin
Decreased free testosterone
Increased serum cortisol
Decreased serum iron and ferritin
Immunological parameters
Constant fatigue
Complaints of muscle and joint aches and pains
Headaches
Nausea
Gastrointestinal disturbances
Increased aches and pains
Muscle soreness/tenderness
Increased susceptibility to and severity of illnesses, colds, and allergies
Reactivation of herpes viral infection
Bacterial infections
One-day colds
Swelling of lymph glands

Reduced blood glutamine has frequently been noted in OTS (3, 50). So, the use of blood glutamine for the synthesis of acute phase proteins would help account for low blood glutamine in OTS.

Besides the amino acids needed as building blocks for acute phase proteins, additional ingredients such as iron and zinc are required as cofactors (44). Reduced levels of these minerals have been noted in OTS (29). Thus, synthesis of acute phase proteins could help account for reductions in the levels of certain minerals (zinc and iron) which act as cofactors.

In summary: if the overtrained athlete is suffering from injury, and the liver is stimulated to synthesize acute phase proteins, this could account for increases in blood levels of certain proteins (e.g. C-reactive protein), decreases in blood levels of other proteins (e.g. albumin), decreases in blood levels of certain minerals such as iron and zinc, and decreases in levels of certain amino acids such as glutamine, as well as loss of muscle mass.

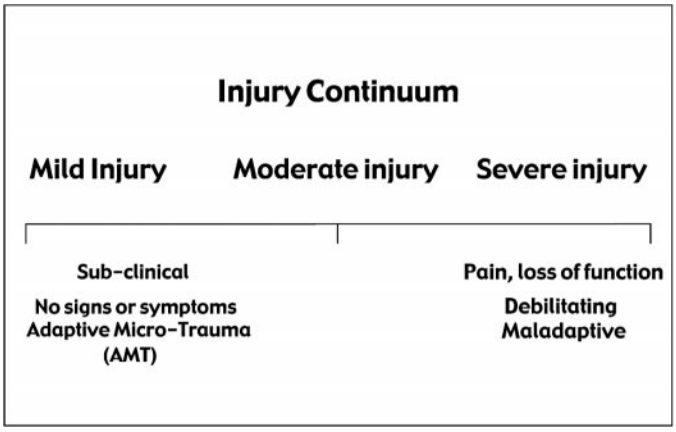

FIGURE 2. Divisions of the immune system and possible relationship to OTS. It is proposed here that humoral immunity is up-regulated while cell-mediated immunity is suppressed in the overtrained athlete.

IMMUNOSUPPRESSION IN OVERTRAINING SYNDROME AND CYTOKINES

The overtrained athlete displays numerous signs and symptoms suggestive of altered immune function. These include increased susceptibility to colds and allergies, increased incidence of infections, and swollen lymph glands (29, 53, 54). This, too, may be explained by the presence of tissue trauma.

The primary purpose of the immune system is to monitor the internal environment for the presence of foreign pathogens such as viruses, bacteria, and parasites, which could ultimately cause extreme harm, including death, to the organism (64). The immune system operates using specific immune cells, white blood cells (WBC), to survey the internal environment, and also receives support from additional immune-related systems such as the cytokine network and the complement system (33).

Academics have artificially separated the immune system (and associated WBCs) into two sections: the innate immune system and the acquired immune system. The innate immune system is the arm of the immune system that reacts immediately to any foreign invaders; although usually extremely effective, it acts in an unrefined fashion, not having specific "instructions" concerning the invading pathogen. The adaptive immune system is slower to react, but attacks foreign invaders in a more focused, sophisticated fashion (Figure 2).

The adaptive arm of the immune system is again divided into two branches. The one branch is referred to as cell-mediated immunity; this branch deals predominantly with elimination of intracellular viruses and bacteria. The other branch, humoral immunity, focuses on eliminating extracellular pathogens, such as the malaria parasites, operating in the fluid medium of the body, such as blood and extracellular fluid.

Until recently, it was believed that both arms of the adaptive immune system (cell-mediated immunity and humoral immunity) were up-regulated simultaneously, in response to any foreign invader (8). However, since 1986 (49), it has been clear that either one or the other branch of the adaptive immune system is up-regulated in response to a foreign invader. Furthermore, not only is one branch of the adaptive immune system activated at a time, but, simultaneously, the other branch is deliberately suppressed (46). This new perspective on how the adaptive immune system functions has had a profound effect on how scientists are redefining the functioning of the immune system (18). This new information will now be applied in an attempt to better understand altered immune function in OTS (66, 68).

Because the present hypothesis focuses on excessive training/competing and resulting tissue trauma, it seems appropriate to examine what happens to the two arms of the adaptive immune system when the body sustains tissue trauma. In the research literature pertaining to severe trauma (such as in response to a major surgical procedure), it has been shown that subsequent to severe tissue trauma, humoral immunity is up-regulated and consequently cellular immunity is suppressed (19). Since cell-mediated immunity, which protects against viral and many bacterial infections, is now suppressed, an individual experiencing severe bodily trauma is at an increased risk of developing a viral or bacterial infection. This is in fact the case in hospitalized settings, where a secondary infection may prove more life-threatening than the initial body trauma (19). It is proposed here that a similar scenario exists in OTS. If the overtrained athlete is suffering from tissue trauma and chronic inflammation, albeit of a milder degree, this may result in up-regulation of humoral immunity and suppression of cell-mediated immunity, rendering the athlete susceptible to infection. Such a scenario would help explain the increased incidence of illness and infection in OTS (34, 66, 68).

The possible up-regulation of humoral immunity in OTS (68) may also explain the increased incidence of allergies noted in some overtrained athletes (55). Although up-regulation of humoral immunity provides specific types of protection for the organism, it is associated with the production of immunoglobulin E, which is closely aligned with the development of allergies in genetically predisposed individuals (59). Thus, increased incidence of allergies in OTS could be attributed to injury and the upregulation of humoral immunity (66, 68).

During the last decade, an important question has been: how can one determine whether humoral immunity or cell-mediated immunity is up-regulated? Are there specific markers on the surface of immune cells that could

identify whether humoral immunity or cell-mediated immunity is predominant? The answer is no. It is now widely accepted that, when humoral immunity is up-regulated, a specific pattern of cytokines are produced, including IL-6, IL-4, and IL-10. Conversely, if cell-mediated immunity is dominant, a different pattern of cytokines is evident; these include IL-12 and interferon-γ (18, 46, 59, 68). Blood markers from acute, strenuous bouts of exercise, such as after a marathon, support the notion of an upregulation of humoral immune-related cytokines (52, 68). Furthermore, if alterations in immune function associated with OTS is regarded as the cumulative effect of repeated strenuous bouts of exercise (40), then athletes suffering from OTS may display a more persistent humoral response (68); this supposition awaits confirmation.

In summary, it is proposed that excessive training may induce chronic injuries that may up-regulate the humoral arm of the adaptive immune system and simultaneously suppress the cell-mediated arm of the adaptive immune system. With suppressed cell-mediated immunity the overtrained athlete is at increased risk of contracting an infection. Concurrently with up-regulation of humoral immunity, the overtrained athlete may also be at increased risk of developing allergies.

OVERALL SUMMARY

The overall hypothesis presented in this paper suggests that OTS, which occurs in a response to excessive training/competing with insufficient time for rest and recovery, results in some form of tissue trauma and associated chronic inflammation, with the resulting release of a group of molecules, cytokines. It is further proposed that increased blood cytokine levels are capable of accessing the central nervous system and stimulating specific brain areas, resulting in behaviors such as depression, loss of appetite, and sleep disturbances, to name a few. These behaviors have been noted in OTS. Cytokines are also capable of stimulating liver cells to reduce production of certain molecules (such as albumin), and increase the production of other molecules (such as C-reactive protein); these molecules are referred to as acute phase proteins, and play a role in recovery. The presence of these acute phase proteins may explain, in part, changes in certain biochemical markers seen in OTS. Additionally, it was suggested that, in the overtrained athlete, the immune system up-regulates one arm of the adaptive immune system, humoral immunity, which renders the individual susceptible to developing an infection; concurrently, genetically predisposed individuals may experience an increased incidence of allergies (Figure 3). Finally, it was suggested that if these mood, behavior, physiological, biochemical, and immune changes occur in response to tissue trauma and elevated cytokine levels, then most of the changes seen in OTS should be regarded as being adaptive, in that they promote withdrawal from daily training activities and encourage rest, an element crucial to healing and recuperation.

PRACTICAL APPLICATIONS

Prevention is the best strategy (60). This entails extreme vigilance on the part of the coach and athlete. Individualizing training and "listening to your body" are fundamental to training appropriately. To prevent full-blown OTS, the reader is referred to several excellent articles and books (22, 26, 30, 35, 55, 60, 73, 75).

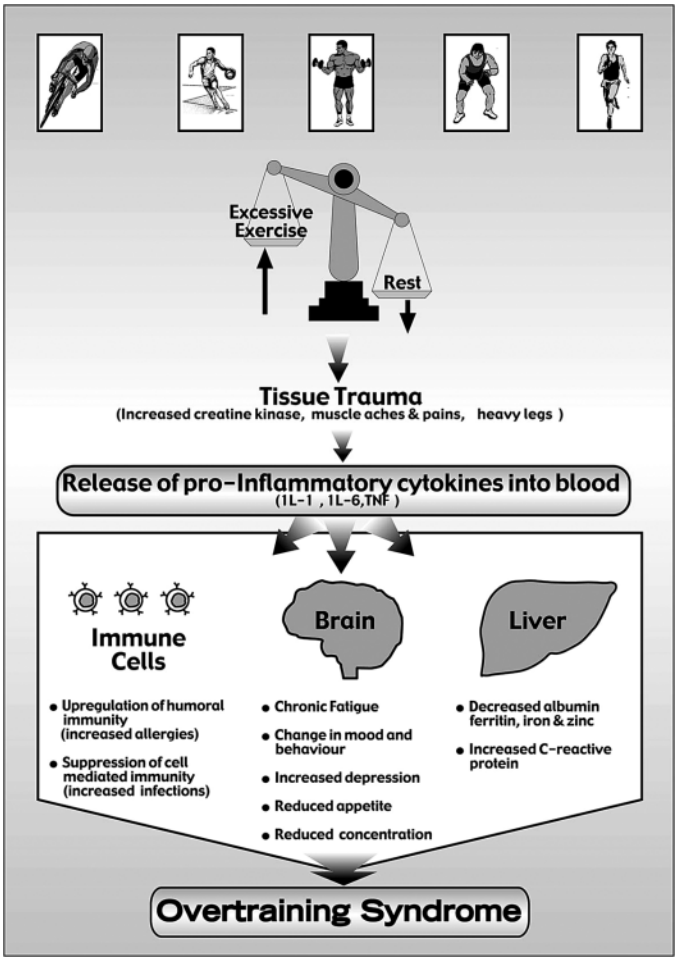

FIGURE 3. Proposed mechanism of the sequence of events that may result in the overtraining syndrome.

It has been proposed that how the athlete "feels" during training and competing is probably the most important thing (55). If the same workout feels harder or the athlete takes longer to recover, this should be noted. If heart rate or rating of perceived exertion is higher for the same exercise intensity, take note. It has also been suggested that if the athlete recovers by 24–48 hours after a hard workout, then all is well. If not, the athlete may be heading toward a state of OTS and continued training could be counterproductive. Recovery from OTS usually takes about 6 to 12 weeks, although OTS may be a chronic relapsing condition and some people may never recover completely (12). An athlete with OTS should start training only when the desire returns, and then start slowly (55).

SPECIFIC RECOMMENDATIONS

For the most part, these recommendations are for resistance-trained as well as for endurance-trained athletes. For more specialized recommendations, the reader is again referred to the above listed articles and books.

- To monitor performance, maintain meticulous records of training and competition. Resistance trained athletes should keep records of choice of exercise, order of exercise, load or resistance used, volume of exercise, and

rest between sets (22). Endurance athletes should record training details, time trials, and racing results (60).
- Don't increase exercise intensity abruptly. Use periodization and a graded approach to training (22, 30, 55). Don't increase weekly training loads by more than 10% (30).
- Have at least one complete day of rest per week (30,35,55). Also, be aware of the amount of rest needed between sets and exercises during resistance training (35).
- Have variety in daily training schedule. Even if doing the same amount of work on a weekly basis, vary the load from day to day (24). It may also be useful to utilize cross-training (23).
- Vary hard and light days (24, 30, 55).
- On heavy days, split loads if possible, and avoid extremely long training sessions (30, 55).
- Include seasonal variety in training, including utilizing of macrocycles, mezocycles and microcycles (56).
- Avoid too many competitions (30, 55).
- Eat a well balanced diet. Include a large variety of food that contains adequate carbohydrate, protein and micronutrients. It may be advisable to use a vitamin supplement and to increase the intake of antioxidants (30).
- The effects of psychological stress may be additive to the physical stress of training. Psychological stressors could include competition, work and family pressure, selection pressure, international travel, funding, and other life events (30). If external personal life stresses are high, reduce training load (55).
- Avoid merely treating symptoms, such as using antidepressants to reduce depression (1). Rather attempt to treat the underlying cause of the depression, which may be related to excessive training.
- Rest, active or passive, is probably the most important strategy. Rest withdraws the athlete from exposure to the harmful stimulus (training/competing) and simultaneously allows time for healing of any injured tissue. Rest/recovery should be an integral part of an effective training program (35). Make rest more appealing and determine exactly how much is needed (23).

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Acknowledgments

Thanks to Brian Switala and Monica Van der Westhuyzen, Publication and Design Services, Technikon Pretoria, for assistance with all diagrams. Thanks to Stuart Semple, M.Tech., Department of Sport and Physical Rehabilitation Sciences, Technikon Pretoria, for editorial suggestions.

Address correspondence to Lucille Lakier Smith, smithl@techpta.ac.za