

Prevention, Diagnosis, and Treatment of the Overtraining Syndrome: Joint Consensus Statement of the European College of Sport Science and the American College of Sports Medicine

Romain Meeusen, Belgium (Chair)
Martine Duclos, France
Carl Foster, United States
Andrew Fry, United States
Michael Gleeson, United Kingdom
David Nieman, United States
John Raglin, United States
Gerard Rietjens, the Netherlands
Jürgen Steinacker, Germany
Axel Urhausen, Luxembourg

ABSTRACT

Successful training not only must involve overload but also must avoid the combination of excessive overload plus inadequate recovery. Athletes can experience short-term performance decrement without severe psychological or lasting other negative symptoms. This functional overreaching will eventually lead to an improvement in performance after recovery. When athletes do not sufficiently respect the balance between training and recovery, non-functional overreaching (NFOR) can occur. The distinction between NFOR and overtraining syndrome (OTS) is very difficult and will depend on the clinical outcome and exclusion diagnosis. The athlete will often show the same clinical, hormonal, and other signs and symptoms. A key-word in the recognition of OTS might be “prolonged maladaptation” not only of the athlete but also of several biological, neurochemical, and hormonal regulation mechanisms. It is generally thought that symptoms of OTS, such as fatigue, performance decline, and mood disturbances, are more severe than those of NFOR. However, there is no scientific evidence to either confirm or refute this suggestion. One approach to understanding the etiology of OTS involves

the exclusion of organic diseases or infections and factors such as dietary caloric restriction (negative energy balance) and insufficient carbohydrate and/or protein intake, iron deficiency, magnesium deficiency, allergies, and others together with identification of initiating events or triggers. In this article, we provide the recent status of possible markers for the detection of OTS. Currently, several markers (hormones, performance tests, psychological tests, and biochemical and immune markers) are used, but none of them meet all the criteria to make their use generally accepted. **Key Words:** OVERTRAINING SYNDROME, OVERREACHING, TRAINING, PERFORMANCE, UNDERPERFORMANCE

The goal in training competitive athletes is to provide training loads that are effective in improving performance. During this process, athletes may go through several stages within a competitive season of periodized training. These phases of training range from insufficient training, during the period between competitive seasons or during active rest and taper, to “overreaching” (OR) and “overtraining” (OT), which includes maladaptations and diminished competitive performance. Literature on “OT” has increased enormously; however, the major difficulty is the lack of common and consistent terminology as well as a gold standard for the diagnosis of OT syndrome (OTS).

In 2006, the European College of Sport Science (ECSS) published its consensus statement on OT (94). We decided to write an update and to ask the American College of Sports Medicine to provide input in this article so that this can be considered as a mutual “consensus statement” of both international organizations. In this “consensus statement,” we will present the current state of knowledge on the OTS going through its definition, diagnosis, treatment, and prevention.

DEFINITION

Successful training must involve overload but also must avoid the combination of excessive overload with inadequate recovery. The process of intensifying training is commonly

used by athletes in an attempt to enhance performance. As a consequence, the athlete may experience acute feelings of fatigue and decreases in performance as a result of a single intense training session or an intense training period. The resultant acute fatigue after an adequate rest period can be followed by a positive adaptation or improvement in performance and is the basis of effective training programs. However, if the balance between appropriate training stress and adequate recovery is disrupted, an abnormal training response may occur and a state of “OR” may develop. Beyond this, the evidence for a supercompensation effect after deliberate periods of intensified training is not abundant.

Many recent articles have referred to the work of Kreider et al. (77) for the definition of OT and OR.

Overreaching—an accumulation of training and/or non-training stress resulting in *short-term* decrement in performance capacity with or without related physiological and psychological signs and symptoms of maladaptation in which restoration of performance capacity may take from *several days to several weeks*.

Overtraining—an accumulation of training and/or non-training stress resulting in *long-term* decrement in performance capacity with or without related physiological and psychological signs and symptoms of maladaptation in which restoration of performance capacity may take *several weeks or months*.

As stated by several authors (13,55), these definitions suggest that the difference between OT and OR is the amount of time needed for performance restoration and not the type or duration of training stress or degree of impairment. These definitions also imply that there may be an absence of psychological signs associated with the conditions. Because it is possible to recover from a state of OR within a 2-wk period (54,66,77,132), it may be argued that this condition is a relatively normal and harmless stage of the training process. However, athletes who are in an “overtrained” state may take months or possibly years to completely recover.

The difficulty lies in the subtle difference that might exist between extreme overreached athletes and those having an

“OTS.” The possibility also exists that these states (OR/OTS) show different defining characteristics and that the OT continuum may be an oversimplification.

To avoid misconception of terminology, we here outline the terms OR, OT, and OTS based on the definitions used by Halson and Jeukendrup (55) and Urhausen and Kindermann (139). In these definitions, “OT” is used as a “verb,” a process of intensified training with possible outcomes of short-term OR (functional OR (FOR)), extreme OR (nonfunctional OR (NFOR)), or OTS. By using the expression “syndrome,” we emphasize the multifactorial etiology and acknowledge that exercise (training) is not necessarily the sole causative factor of the syndrome.

OR is often used by athletes during a typical training cycle to enhance performance. Intensified training can result in a decline in performance; however, when appropriate periods of recovery are provided, a “*supercompensation*” effect may occur with the athlete exhibiting an enhanced performance compared with baseline levels. This process is often used when going on a “training camp” and will lead to a temporary performance decrement, which is followed by improved performance. In this situation, the physiological responses will compensate the training-related stress (133). This form of short-term “OR” can also be called “FOR.” When this “intensified training” continues, the athletes can evolve into a state of extreme OR or “NFOR,” which will lead to a stagnation or decrease in performance that will not resume for several weeks or months. However, eventually, these athletes will be able to fully recover after sufficient rest. “NFOR” emphasizes that not only the evolution on the “OT continuum” is “quantitatively” determined (i.e., by the increase in training volume) but also “qualitative” changes occur (e.g., signs and symptoms of psychological distress and/or endocrine disturbances). This is in line with the classical concept of “sympathetic versus parasympathetic OTS” (65) and recent neuroendocrine findings using a double exercise test (95,96).

In Figure 1, the different stages that differentiate normal training from OR (FOR and NFOR) and from OTS are

PROCESS	TRAINING (overload)	INTENSIFIED TRAINING →		
		FUNCTIONAL OR (short-term OR)	NON-FUNCTIONAL (extreme OR)	OVERTRAINING (OTS)
OUTCOME	ACUTE FATIGUE	FUNCTIONAL OR (short-term OR)	NON-FUNCTIONAL (extreme OR)	OVERTRAINING SYNDROME (OTS)
RECOVERY	Day(s)	Days – weeks	Weeks – months	Months - ...
PERFORMANCE	INCREASE	Temporary performance decrement (e.g., training camp)	STAGNATION DECREASE	DECREASE

FIGURE 1—Possible presentation of the different stages of training, OR and OTS.

presented. Training can be defined as a process of overload that is used to disturb homeostasis, which results in acute fatigue leading to an improvement in performance. When training continues or when athletes deliberately use a short-term period (e.g., training camp) to increase training load, they can experience short-term performance decrement without severe psychological or lasting other negative symptoms. This FOR (or short-term OR) will eventually lead to an improvement in performance after recovery. However, when athletes do not sufficiently respect the balance between training and recovery, NFOR (extreme OR) can occur. At this stage, the first signs and symptoms of prolonged training distress such as performance decrements, psychological disturbance (decreased vigor, increased fatigue), and hormonal disturbances will occur, and the athletes will need weeks or months to recover. Several confounding factors such as inadequate nutrition (energy and/or carbohydrate intake), illness (most commonly, upper respiratory tract infections (URTI)), psychosocial stressors (work, team, coach, and family related), and sleep disorders may be present. At this stage, the distinction between NFOR and OTS is very difficult and will depend on the clinical outcome and exclusion diagnosis. The athlete will often show the same clinical, hormonal, and other signs and symptoms. Therefore, the diagnosis of OTS can often only be made retrospectively when the time course can be overseen. A keyword in the recognition of OTS might be “prolonged maladaptation” not only of the athlete but also of several biological, neurochemical, and hormonal regulation mechanisms.

The borderline between optimal performance and performance impairment due to “OTS” is subtle. This applies especially to physiological and biochemical factors. The apparent vagueness surrounding OTS is further complicated by the fact that the clinical features are varied from one individual to another and are nonspecific, anecdotal, and numerous.

DIAGNOSIS

Although in recent years, the knowledge of central pathological mechanisms of OTS has significantly increased, there is still a strong demand for relevant tools for the early diagnosis of OTS. OTS is characterized by a “sport-specific” decrease in performance together with disturbances in mood state. This underperformance persists despite a period of recovery lasting several weeks or months. Importantly, because there is no diagnostic tool to identify (e.g., rule in) an athlete as experiencing OTS, the solution to the differential diagnosis can only be made by excluding all other possible influences on changes in performance and mood state. Therefore, if no explanation for the observed changes can be found, OTS is diagnosed. Early and unequivocal recognition of OTS is virtually impossible because the only certain sign is a decrease in performance during competition or training. The definitive diagnosis of OTS always requires the exclusion of an organic disease, e.g., endocrinological disorders (thyroid or adrenal gland and diabetes),

iron deficiency with anemia, or infectious diseases (including myocarditis, hepatitis, and glandular fever). Other major disorders or feeding behaviors such as anorexia nervosa and bulimia should also be excluded. However, it should be emphasized that many endocrinological and clinical findings due to OR and OTS can mimic other diseases. The borderline between under- and overdiagnosis is very difficult to judge.

In essence, it is generally thought that symptoms of OTS, such as fatigue, performance decline, and mood disturbances, are more severe than those of OR. However, there is no scientific evidence to either confirm or refute this suggestion. Hence, there is no objective evidence that the athlete is indeed experiencing OTS. In addition, in the studies that induced a state of OR, many of the physiological and biochemical responses to the increased training were highly variable, with some measures in some studies demonstrating changes and others remaining unaltered, most likely because conditions and the degree of OR and OTS differ and were not comparably described. This is also probably because the signs and symptoms of OTS are individual and it is not feasible and certainly unethical to excessively train an athlete in such a way that he/she will develop OTS. Therefore, prospective studies are lacking and only few data exist on OTS.

One approach to understanding the etiology of OTS involves the exclusion of organic diseases or infections and factors such as dietary caloric restriction (negative energy balance) and insufficient carbohydrate and/or protein intake, iron deficiency, magnesium deficiency, allergies, and so on together with identification of initiating events or triggers. One of the most certain triggers is a training error resulting in an imbalance between load and recovery. Other possible triggers might be the monotony of training, too many competitions, personal and emotional (psychological) problems, and emotional demands of occupation. Less commonly cited possibilities are sleep disturbance, altitude exposure, and exercise heat stress. However, scientific evidence is not strong for most of these potential triggers. Many triggers such as glycogen deficiency or infections may contribute to OR or OTS but might not be present at the time the athlete presents to a physician. Furthermore, identifying these possible initiating events has not revealed the causative mechanism(s) of the OTS. Consequently, some scientists have suggested that the OTS be renamed as the unexplained underperformance syndrome (13) that focuses on the key symptom of underperformance in OTS rather than on the mechanisms. This terminology has not been widely adopted outside the UK.

Athletes and the field of sports medicine in general would benefit greatly if a specific, sensitive simple diagnostic test existed for the diagnosis of OTS. At present, no test meets this criterion, but there certainly is a need for a combination of diagnostic aids to pinpoint possible markers for OTS. Especially, there is a need for a detection mechanism for early triggering factors.

Increased training loads as well as other chronic stresses can influence the neuroendocrine system chronically. However, at this time, it is not yet clear which mechanism

eventually leads to OTS. Probably because of this, and because there are several possible hypotheses, some recent review articles have focused on hypothetical explanations for the mechanism behind the OTS. Although these theories have potential, until more prospective studies are carried out where a longitudinal follow-up of athletes (who may develop the OTS) is performed or specific diagnostic tools are developed, these theories remain speculative.

PREVALENCE

It is difficult to give exact prevalence figures on NFOR/OTS merely because not all studies clearly indicate the time frame of data collection. Survey research involving collegiate swimmers and other endurance athletes who completed a training monocycle report a rate of NFOR/OTS of approximately 10% (range, 7%–21%) (118). Higher rates have been reported in other studies, but these values are likely inflated by merging cases of FOR, NFOR, and OTS. The risk of NFOR/OTS becomes compounded over the course of an athlete's career; survey studies of elite runners report 60% of females and 64% of males indicate experiencing at least one previous episode of OTS, with a career rate of 33% in nonelite adult runners (102,103). Similar career rates of OTS have been reported by young athletes, including a 34.6% rate among 231 (age range, 13–18) age-group swimmers from four countries, with OTS being most common among faster performers (117), and a 37% rate in 272 Swedish high school junior national athletes assessed across 16 different sports (72). Retrospective techniques can be prone to bias or inaccurate recall, but a recent longitudinal study of British age-group swimmers found 29% had developed NFOR/OTS at least once, with the risk positively related to skill level (91). These findings reinforce both the growing risk of OTS for young athletes and the utility of retrospective methodologies in OTS research.

Moreover, there is evidence that athletes who have developed OTS are at a heightened risk of relapse. In a study of US collegiate swimmers, it was found that 91% of the swimmers who developed OTS during their first collegiate training season were diagnosed with OTS again in one or more of the following 3 yr of training. In contrast, only 34% of swimmers free of OTS during their first year of collegiate swimming had a later diagnosis of OTS (113).

This interindividual variation in the risk for NFOR/OTS has been observed in athletes who undergo the same overload training. In a study of 13 competitive swimmers who completed 10 d of intensified training at the same volume and relative intensity ($8970 \text{ m}\cdot\text{d}^{-1}$ at 94% $\dot{V}O_{2\text{max}}$), seven swimmers successfully completed the required training regimen but three others had difficulty completing the training requirements, and these athletes had significantly higher levels of POMS mood disturbance (101) and lower levels of muscle glycogen (76). Another three swimmers were so severely affected by the training that they had to be dropped from the study.

It remains unclear whether these findings indicate that some individuals are particularly predisposed to developing the OTS when exposed to overload training or whether succumbing to the OTS raises the risk of relapse. Some tests of potential psychological factors have been conducted and have not found the risk of OTS to be mediated by intrinsic motivation (114), hardiness, or optimism (147).

ASSESSMENT OF OT

OTS reflects the attempt of the human body to cope with physiological and other stressors. Several studies have revealed that OTS represents the sum of multiple life stressors, such as physical training, sleep loss, exposure to environmental stresses (e.g., exposure to heat, high humidity, cold, and high altitude), occupational pressures, change of residence, and interpersonal difficulties. Thus, OTS can be understood partly within the context of the General Adaptation Syndrome of Selye (124). Concomitant to this “stress disturbance,” the endocrine system is called upon to counteract the stress situation. The primary hormone products (adrenaline, noradrenaline, and cortisol) all serve to redistribute metabolic fuels, maintain blood glucose, and enhance the responsiveness of the cardiovascular system. Repeated exposure to stress may lead to altered responsiveness to subsequent stressful experiences depending on the stressor as well as on the stimuli paired with the stressor, either leading to an unchanged or increased or decreased neurotransmitter and receptor function. Behavioral adaptation (neurotransmitter release, receptor sensitivity, receptor binding, etc.) in higher brain centers will certainly influence hypothalamic output (80). Lehmann et al. (86) introduced the concept that hypothalamic function reflects the state of OR or OTS because the hypothalamus integrates many of the stressors. It has been shown that acute stress increases not only hypothalamic monoamine release but also consequently corticotropin-releasing hormone and adrenocorticotrophic hormone (ACTH) secretion (126). Chronic stress and the subsequent chronically elevated adrenal glucocorticoid secretion could play an important role in the desensitization of higher brain centers' response to acute stressors, because it has been shown that in acute and chronic stress, the responsiveness of hypothalamic corticotropin-releasing hormone neurons rapidly falls (6,18,85,137).

The lack of definitive diagnostic criteria for OTS is reflected in much of the “OR” and “OT” research by a lack of consistent findings. There are several criteria that a reliable marker for the onset of the OTS must fulfill: the marker should be sensitive to the training load and ideally be unaffected by other factors (e.g., diet and chronobiological rhythms). Changes in the marker should occur before the establishment of the OTS, and changes in response to acute exercise should be distinguishable from chronic changes. Ideally, the marker should be relatively easy to measure with a quick availability of the result, not too invasive (e.g., repeated venous blood samplings are not well accepted) and

not too expensive. Ideally, the marker should be derived at rest from submaximal or standardized exercise of relatively short duration in order not to interfere with the training process. However, none of the currently available or suggested markers meet all of these criteria.

BIOCHEMISTRY AND HORMONES

Biochemistry. In prolonged training, glycogen stores get close to full depletion, glycogenolysis and glucose transport are down-regulated in muscle and liver as well as the liver production of insulin-like growth factor 1, and catabolism is induced. Although this is one of the likely triggers of OTS, muscle glycogen is typically normal when athletes are examined (130). Blood glucose is also not typically altered (138). Resting blood glucose/insulin ratio may indicate mild insulin resistance (133).

Blood lactate measurements can be dependent on the actual training status of the individual. Other factors that are equally important when discussing changes in blood lactate concentrations are the glycogen status and possible decreases in muscle and liver stores due to increased training. One almost consistent overall finding, at least in endurance and strength–endurance athletes having the OTS, is a diminished maximal lactate concentration while submaximal values remain unchanged or slightly reduced (139).

Individually increased circulating levels of creatine kinase (CK), which especially reacts to eccentric and unaccustomed exercise with elevations lasting from several days to up to a little over 1 wk, and/or urea measured under standardized conditions at rest (140), may provide information concerning an elevated muscular and/or metabolic strain (137), but they are not suitable to indicate an OR or OTS state (137). Under glycogen-depleted compared with carbohydrate-loaded condition, serum urea increases not only during 1 h of cycling at 61% $\dot{V}O_{2max}$ but also before and 4 h after exercise (88). After one single eccentric strength exercise leading to a nearly 10-fold maximal CK increase with a weak significant correlation to the isometric strength loss, the positive response to concentric strength training was significantly delayed for several weeks (33).

After 2 wk of OR with short-term decline of performance and mood state, plasma CK (as well as glutamate) showed a significant and urea a tendency to increase before normalizing after 2 wk of regenerative training in eight moderately well trained cyclists (57).

The concentration of plasma glutamine has been suggested as a possible indicator of excessive training stress (122). However, not all studies have found a fall during periods of increased training and OT (145), and altered plasma glutamine concentrations are not a causative factor of immunodepression in OTS, whereas other authors rather propose the glutamine/glutamate ratio as an indicator of OR (19,128).

Although most of the blood parameters (e.g., blood count, C-reactive protein, erythrocyte sedimentation rate, CK, urea,

creatinine, liver enzymes, glucose, ferritin, sodium, and potassium) are not capable of detecting OR or OTS, they are helpful in providing information on the actual health status of the athlete and therefore useful in the “exclusion diagnosis.”

The problems with biochemistry testing are as follows:

- Lactate differences are sometimes subtle (lying within the measuring error of the apparatus) and depend on the modus of the exercise test used.
- No lactate changes are reported in strength athletes.
- Glutamine may fall with increased training load, but low plasma glutamine concentration is not a consistent finding in OTS.

Hormones. For several years, it has been hypothesized that a hormonal mediated central dysregulation occurs during the pathogenesis of the OTS, and that measurements of blood hormones could help to detect the OTS (40,45,79, 85,96,132,133,136,137). The results of the research devoted to this subject is far from unanimous, mostly because of preanalytical factors; i.e., factors that occur before the final analysis (time of sampling, food intake, time after the end of exercise, gender, age, etc.) may influence the hormonal profile. In addition, measuring methods and/or detection limits of the analytical equipment used may differ between studies. Testing of central hypothalamic/pituitary regulation requires functional tests that are considered invasive and require diagnostic experience, and these tests are time consuming and expensive. Finally, the distinguishing characteristic of endocrine systems is the feedback control of hormone production. Virtually all hormones are under feedback control, some by the peripheral hormones themselves, some by other hormones or cytokines, peripheral metabolites, osmolality, etc. This feedback relationship is the reason why simultaneous assessment of hormone/effector pairs is frequently necessary for the assessment of hormonal status, taking also into consideration the fact that physiological processes related to endocrine regulation are influenced by more than a single hormone in a multilevel integrated way (25).

For a long time, the resting plasma testosterone/cortisol ratio was considered as an indicator of the overtrained state. This ratio decreases in relation to the intensity and duration of training, and it is evident that this ratio indicates only the actual physiological strain of training and cannot be used for diagnosis of OR or OTS (25,84,87,136).

Most of the literature agrees that OR and OTS must be viewed on a continuum with a disturbance, an adaptation, and finally a maladaptation of the hypothalamic pituitary adrenal axis (HPA) and all other hypothalamic axes (85,87,93, 96,136,138). For example, the HPA adaptation to normal training is characterized by increased ACTH/cortisol ratio only during exercise recovery (due to decreased pituitary sensitivity to cortisol) (26,27,85) and by modulation of tissue sensitivity to glucocorticoids (28,30). However, it should be emphasized that during a resting day, in endurance-trained athletes, 24 h of cortisol secretion under nonexercising conditions is normal (28,30,82). Accordingly, morning plasma

cortisol concentration and 24-h urinary free cortisol excretion in resting endurance-trained men are similar to those of age-matched sedentary subjects (29,53,74). Because urinary free cortisol represents an integrated measure of the 24-h cortisol secretion, this is in accordance with the previously reported normal diurnal HPA axis rhythm in endurance-trained men (28,29). Finally, endurance-trained men maintain the seasonal rhythmicity of cortisol excretion; as in sedentary men, the highest concentrations of urinary cortisol, morning plasma cortisol, and saliva cortisol are observed during autumn and winter compared with spring and summer (53). Therefore, it can be concluded that resting cortisol is not a useful measurement.

There is no consensus about plasma, 24-h, or overnight urinary excretion of catecholamines for monitoring the effect of the training load and/or an overload. Some studies report an increase, a decrease, or no change of urinary catecholamine excretion (for a review see Duclos [25]) with successful training, OR or OTS. Factors other than training load influence secretion and could result in variations between studies; these factors include sampling methods, diurnal and seasonal variations of catecholamine excretion, and sex difference effects. Because the relationship between 24 h or nocturnal catecholamine urinary excretion and performance or training monitoring is inconclusive, it is thus inappropriate to use changes in catecholamine excretion as a tool to monitor training status.

In OTS, a decreased rise in pituitary hormones (ACTH, growth hormone (GH), luteinizing hormone, and FSH) in response to a stressful stimulus is reported (6,85,136, 137,148). But behind the seemingly uniform acute hormonal response to exercise, explaining the disturbance to the neuroendocrine system caused by the OTS is not that simple. Whether peripheral metabolic hormones can be used for OR/OTS diagnosis is currently under discussion.

A nutrient-sensing signal of adipose tissue is represented by leptin (127), which, like the glucoregulatory hormone insulin, interleukin-6, and metabolic growth factor insulin-like growth factor 1, has been shown to decrease with training-induced catabolism like in OR. These signaling molecules have profound effects on the hypothalamus and are involved in the metabolic hormonal regulation of exercise and training (133). However, the same molecules respond to chronic energy deficiency, which can be associated with endurance training and/or aesthetic sports (e.g., gymnastics), regardless of the training status (absence or presence of OR/OTS). Chronic energy deficiency (mainly glycogen depletion) certainly amplifies the stress hormone and cytokine responses to exercise and might also be one of the “triggering” factors that can lead to the induction of OTS.

In addition to the need to study different hormonal axes in parallel, it is also important to consider the dynamics of hormonal responses. Indeed, the hormonal responses during exercise influence the hormonal responses during exercise recovery (24,29,68), and it is therefore important to study

both phases of exercise. For this reason, a multiple exercise test that not only gives the opportunity to measure the recovery capacity of the athlete but also can assess the ability to normally perform the second bout of exercise could be useful to detect signs of OTS and distinguish them from normal training responses or FOR.

Meeusen et al. (96) published a test protocol with two consecutive maximal exercise tests separated by 4 h. The use of two bouts of incremental exercise to volitional exhaustion to study neuroendocrine variations showed an exercise-induced increase of ACTH, prolactin, and GH to a two-exercise bout (96). In normal healthy subjects, the test reveals an increase in the circulating concentrations of the hormones after both the first and the second exercise bout. The test could be used as an indirect measure of hypothalamic-pituitary reactivity. Depending on the “training” status of the athlete, hormonal output after the second exercise test will be different. This test has the ability to distinguish a state of NFOR from the OTS. In an FOR stage, a less pronounced neuroendocrine response to a second bout of exercise on the same day is found (96), whereas in an NFOR stage, the hormonal response to a two-bout exercise protocol shows a markedly higher elevation after the second exercise trigger (96). With the same protocol, it has been shown that athletes experiencing OTS have an extremely large increase in circulating hormone concentration after the first exercise bout, followed by a complete suppression in the second exercise bout (95,96). This could indicate a hypersensitivity of the pituitary followed by an insensitivity or exhaustion afterward. Previous reports that used a single-exercise protocol found similar effects (96). In a follow-up study, they could clearly distinguish between NFO and OTS athletes (95). It appears that the use of two-exercise bouts is more useful in detecting OR for preventing OT. Early detection of OR may be very important in the prevention of OTS.

Other hormones such as leptin, adiponectin, and ghrelin, as well as cytokines such as interleukin-6 and tumor necrosis factor- α , have been recently investigated as possibilities for the monitoring of training (67). The authors concluded that although some of these parameters measured in the fasting state or postexercise may provide information about energetic regulatory mechanisms and may change after heavy training or inadequate recovery, there are no studies supporting the possible suitability of these variables as markers of training stress or for the prevention or diagnosis of OR or OTS.

In conclusion, the endocrine system is one of the major systems involved in the responses to acute stress and adaptation to chronic stress. A great diversity of mechanisms is involved in such adaptation, acting at potentially all levels in the cascade, leading to the biological effects of the hormones. However, the current information regarding the endocrine system and OR/OTS shows that basal (resting) hormone measurements cannot distinguish between athletes who successfully adapt to OR and those who fail to adapt

and develop symptoms of OTS. Further studies using multiple exercise tests and/or multiple hormone analyses will be necessary for evaluating the possibility of a hormonal diagnostic test for OR/OTS.

The problems with hormonal data are as follows:

- Many factors affect blood hormone concentrations, and these include factors linked to sampling conditions and/or conservation of the sampling: stress of the sampling, intra- and interassay coefficient of variability;
- Food intake (nutrient composition and/or pre- vs postmeal sampling) can modify significantly either the basal concentration of some hormones (cortisol, Dehydroepiandrosterone-sulphate, and total testosterone) or their concentration change in response to exercise (cortisol and GH);
- Pulsatility of the secretion of some hormones, which modulates the tissue sensitivity to these hormones;
- In female athletes, the hormonal response will depend on the phase of the menstrual cycle;
- Aerobic and resistance protocols typically elicit different endocrine responses;
- Hormone concentrations at rest and after stimulation (exercise = acute stimulus) respond differently;
- Diurnal and seasonal variations of the hormones;
- Stress-induced measures (exercise, pro-hormones, etc) need to be compared with baseline measures from the same individual;
- Poor reproducibility and feasibility of some techniques used to measure some hormones (e.g., free testosterone by radio immunoassay instead of the reference method—reserved to some highly specialized centers—equilibrium dialysis);
- Hormonal responses to exercise can be prolonged during the recovery phase of exercise.

PERFORMANCE TESTING

In athletes who have been diagnosed as having OTS, several signs and symptoms have been associated with this imbalance between training and recovery. However, reliable diagnostic markers for distinguishing between well-trained athletes, OR athletes, and athletes having OTS are lacking. A hallmark feature of OTS is the inability to sustain intense exercise, a decreased sport-specific performance capacity when the training load is maintained or even increased (96,136). Athletes experiencing OTS are usually able to start a normal training sequence or a race at their normal training pace but are not able to complete the training load they are given, or race as usual. The key indicator of OTS can be considered an unexplainable decrease in performance. Therefore, an exercise/performance test is considered to be essential for the diagnosis of OTS (13,136).

It appears that both the type of performance test used and the intensity/duration of the test are important in determining the changes in performance associated with OTS. Debate exists as to which performance test is the most appropriate

when attempting to diagnose OR and OTS. In general, time-to-fatigue tests will most likely show greater changes in exercise capacity as a result of OR and OTS than incremental exercise tests (55,138). Time trials reflect more accurately the sport-specific task of most sports but have only rarely been used to objectively quantify the performance loss in OR (57). In addition, these tests allow that the assessment of substrate kinetics, hormonal responses, and submaximal measures can be made at a fixed intensity and duration. To detect subtle performance decrements, it might be better to use sport-specific performance tests. Tests of high-intensity exercise performance may be appropriate in some sports. For example, isokinetic strength and power were shown to be decreased in seven overreached rugby players (20) but increased after 1 wk of taper.

The problems with performance testing are as follows:

- Baseline measures are often not available, and therefore, the degree of performance limitation may not be exactly determined. Individual comparative values are mandatory.
- The intensity and reproducibility of the test should be sufficient to detect differences (maximum test, time trial).
- Necessity of highly standardized conditions from one test to another and from one laboratory to another.
- Many performance tests are not sport specific.
- Submaximal ergometric test results do not seem to produce significant results (137), but repeated maximal tests, required for assessment of an individual baseline measure, are difficult to obtain in athletes.
- In this regard, because adequate standardization of laboratory tests is problematic, it may be that index training sessions recorded by coaches are better candidates to demonstrate the magnitude, timing, and pattern of performance decrements.

PSYCHOLOGY

The presence of psychological symptoms in cases of OTS has long been acknowledged (22), but systematic study on this topic did not begin until William Morgan's research in the 1980s on college swimmers and athletes in other sports. Using the POMS (100), a questionnaire that measures both general and specific moods, athletes were found to consistently report elevations in negative moods (tension, depression, anger, fatigue, and confusion) and decreases in the positive mood of vigor during periods of rigorous training. More frequent assessments indicated that mood state exhibits a predictable dose-response relationship with training whereby disturbances increase in a stepwise fashion as training loads rise in volume or intensity, with the peak of training and mood disturbance coinciding. Conversely, training tapers usually result in a reduction in negative moods and an increase in vigor such that at the end of a taper, the mood scores return to the positive pattern typically observed at the outset of the season, called the iceberg profile (100,103,116). Dose-response relationships between training

load and mood state have since been observed in studies involving more than 1000 athletes in a variety of endurance and nonendurance sports requiring rigorous training regimens (118).

Research also indicates that mood responses of male and female athletes do not differ except when they are exposed to significantly different training regimens (103,115). Similar dose–response patterns have also been observed using simple self-report measures of muscle soreness, appetite, sleep disturbances, “heaviness,” and perception of effort (72,101,110,118), indicating perceptual responses to increased training are global and systemic in nature, although the magnitude of change differs across measures (102,118).

When conditioning programs involve rapid increases in training load over a course of days, the instructions to complete psychological measures should, if possible, be modified to yield a more transient state measure of mood by having subjects respond according to how they feel “today” or “right now.” Research reveals that as few as 2 d of intensified training can result in significant increases in POMS measures (110) and scores on other psychological scales, which precede changes in commonly used biochemical markers of training stress such as cortisol (19,111). More important for the standpoint of monitoring, athletes with signs of OTS typically exhibit both a greater increase in total mood disturbance compared with athletes undergoing the same training who remain free from symptoms (114). Specifically, among healthy athletes, POMS fatigue and vigor show the largest shifts during peak overload training, and depression increases the least of all POMS factors, whereas in athletes showing signs of OTS, depression increases the most of all POMS variables, with some reports (103) indicating that up to 80% of affected athletes show signs of clinical depression. As shown in Figure 2, mood changes between healthy and overtrained athletes.

The previous findings have led to tests of mood state monitoring as a means to modulate training load with the

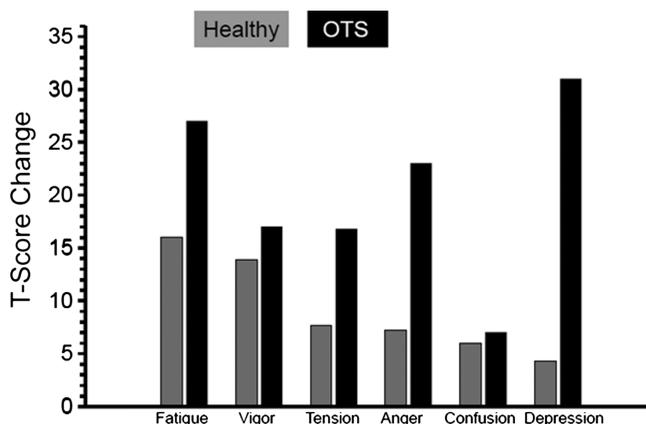


FIGURE 2—Magnitude of changes in POMS mood states from easy to maximal overload training in collegiate varsity swimmers who develop OTS or remain free of symptoms (i.e., “healthy”). (Adapted from Raglin and Morgan [114]).

goal of reducing the incidence of OTS. This intervention paradigm involved reducing the training load of athletes possessing excessively elevated POMS total mood disturbance scores until scores fell within an acceptable range established *a priori* using either off-season baseline of each athlete (7) or the mean value for teammates undergoing the same training regimen (113). Conversely, training loads were increased in athletes exhibiting only minor mood disturbances, and this intervention was nearly as frequent as cases in which training loads were reduced (7). Both studies reported a reduced incidence of OTS compared with previous rates, but replications incorporating involving larger samples and adequate control conditions remain needed.

Although research generally supports the use of psychological assessments for identifying individuals at risk of developing OTS, several potential problems exist that can constrain accuracy. The most serious among these is response distortion, wherein subjects falsely complete psychological questionnaires, particularly those with items of a sensitive or personal nature. The most common form of response distortion involves social desirability or “faking good” in which individuals answer items to present themselves in a uniformly positive light. Factors that can increase the likelihood of response distortion include coercion, the demand characteristics associated with the experimental hypothesis, or in the case of OT studies, “faking bad” to have one’s training load reduced. Administering questionnaires repeatedly over an extended period can sometimes result in a form of response distortion in which participants respond to questions in a random manner. The risk of response distortion can be reduced by including research team members who are trained in the proper administration of psychological questionnaires, providing athletes clear and guaranteed assurances their data will remain confidential and not be used for selection purposes, and by carefully explaining the rationale of using psychological assessments while emphasizing there are no right or wrong ways to respond to the questionnaires.

A separate concern regarding the POMS is the finding that the sensitivity of the mood subscales to training load is not uniform. Some factors, particularly confusion, barely change even after large increases in training load in either healthy or overtrained athletes, whereas other POMS subscales are responsive to non–training-related sport stressors (116). For example, POMS tension scores often remain elevated or even increase during training tapers, most likely because this factor is particularly sensitive to the impending stress of major competitions (116). At a more fundamental level, the POMS was designed for use in general circumstances and samples, and many sport psychologists contend that sport-specific questionnaires should provide greater sensitivity and specificity for assessing athletes in the unique environment of sport. Consequently, several hundred sport-specific psychological measures of personality, motivation, and mood have been developed, including several for NFOR/OTS. In the case of OTS, the decision to use a general or sport-specific measure depends not only on published

evidence of its predictive efficacy and construct validity but also the theoretical orientation of the researcher. If it is believed the risk of OTS is a function of the sum total of stressors an athlete is exposed to—be they training related or not—then a nonspecific questionnaire that captures broad moods, feelings, or perceptions would be most appropriate. Conversely, if non-sport-related stressors (e.g., psychosocial stressors and time zone travel) are viewed as inconsequential or only minor contributors to the OTS, then questionnaires delimited to items particular to the context of training should be used.

For these and other reasons, researchers have developed POMS-based OT scales in the attempt to enhance its sensitivity. Raglin and Morgan (114) created a Training Distress Scale (TDS) based on discriminant function analyses of POMS data from 186 healthy and overtrained college swimmers. A TDS spreadsheet may be accessed at <http://champ.usuhs.mil/choptimize.html>. The seven-item (five depression and two anger items) TDS was more accurate in identifying OT athletes compared with predictions using POMS total mood disturbance scores or depression scores, and subsequent research (72,118) using translations of the scale in several languages found TDS scores to be elevated in young swimmers reporting OTS. Kenttä et al. (73) created a POMS energy index measure by subtracting fatigue from vigor scores to study 11 elite kayakers during an intensive 3-wk training camp. The researchers had athletes complete the entire POMS after practice each day and in the morning before practice to assess mood state after training and recovery. POMS energy index scores were responsive to both training stress and recovery, whereas depression scores were unchanged, suggesting to the authors the index could be a useful tool to reduce NFOR during intense but brief training cycles.

Several sport-specific OTS scales have been developed using theoretical assumptions about what psychological and behavior factors should be associated with OTS. Among them, the most extensively studied has been the Recovery–Stress Questionnaire for Athletes (RESTQ-Sport) (70), a 77-item questionnaire encompassing 19 separate factors that assess both OT and recovery responses in endurance athletes. Monitoring the current levels both of stress and recovery has the possible advantage that problems may be detected before symptoms of OT and staleness (e.g., drowsiness, apathy, fatigue, and irritability) are likely to appear. However, stress and recovery are often different in their time course. Although concerns with its factor structure have been expressed by other researchers (e.g., Ref. [23]), research indicates the RESTQ is responsive to changes in training load, particularly in athletes with signs of OTS (69). Other less well-documented OTS scales include the questionnaire of the Société Française de Médecine du Sport (SFMS), a 54-item forced-choice (i.e., yes–no) questionnaire that assesses whether athletes have experienced mood disturbances and various symptoms of OT during the previous month (cited in Ref. [31]), and the Daily Analyses of Life-Demands in

Athletes, a 50-item scale with two sections assessing general and sport-related stresses (123) experienced over the past day using a three-point Likert format.

In summary, research has provided general support for the efficacy of psychological assessments in both basic and applied research involving athletes undergoing overload training. There remains, however, a need for systematic study of the relative efficacy (i.e., sensitivity and specificity) of promising measures and tests to establish protocols that effectively integrate psychological information with biological assessments to enhance their efficacy.

Psychomotor speed tests. A relatively new but promising tool in the early detection of NFO and therefore a potential preventive tool in developing an OTS is the measurement of psychomotor speed. The advantage of psychomotor speed testing above most other tests lays in the fact that it is easy to use in the (sport) field just by using a simple personal computer. The tests are noninvasive, resistant to conscious manipulation by the athlete, and inexpensive.

It is well described that symptoms such as concentration and memory problems and cognitive complaints are common in patients experiencing chronic fatigue syndrome (45), symptoms also found in people experiencing OTS (85,125). These similarities have led to the use of attention and reaction time tests for early detection of NFO and preventing OTS. Rietjens et al. (120) used a reaction time test (finger precuing test) as a detection tool for NFO. They found a significant decrease in reaction time in a group of seven cyclists after they had doubled their training volume over a period of 3 wk (120), especially on the more difficult conditions in the finger precuing reaction time task, with the more easy conditions being insensitive to OR. This outcome suggests that task complexity is an important mediating variable in the relationship between OR and brain functioning. In line with these findings Nederhof et al. (104) described a decrease in reaction time in five NFO cyclists after a 2-wk training camp. In a later follow-up study, Nederhof et al. (105,106) confirmed these findings.

More recently, Hynynen et al. (64) presented data in which OTS cyclists scored a significantly higher number of mistakes during a STROOP test. All these studies strongly suggest that central fatigue is an early (and maybe the most early) manifestation of OR. This suggestion is ratified by the findings of Tergau et al. (134) who found an intracortical facilitation increase after exercise, indicating motor cortex fatigue.

These findings indicate that reaction and attention tests are promising tools in early detection of NFO and preventing OTS. However, more scientific studies are needed to find out which kind of psychomotor speed tests are the most sensitive for detecting NFO/OTS.

The potential problems with psychological assessments are as follows:

- Mood state and other factors can be influenced by stressors unrelated to training and recovery.

- It remains unclear if intervention paradigms based on psychological information should use off-season baseline mood scores (i.e., intraindividual criterion), team averages (i.e., interindividual) or combinations of baseline and training values would be more effective.
- Psychological measures can be biased or rendered invalid by various forms of faking (e.g., social desirability) or overuse.
- Psychological tests must be administered with the appropriate instructional set (e.g., “right now,” “today,” and “last week including today”) based on the training paradigm. Care must be taken with state (i.e., “right now”) measures of mood because they can be influenced by extraneous factors.
- Care needs to be taken to explain the potential value of psychological measures to coaches and athletes who may be reluctant or skeptical. Researchers should be trained in the administration and interpretation of the measures used.

PHYSIOLOGY

There have been several proposals as to which physiological measures might be indicative of OR or OTS. Reduced maximal heart rates after increased training may be the result of reduced sympathetic nervous system activity, of a decreased tissue responsiveness to catecholamines, and of changes in adrenergic receptor activity or may simply be the result of a reduced power output achieved with maximal effort. Several other reductions in maximal physiological measures (oxygen uptake, heart rate, blood lactate) might be a consequence of a reduction in exercise time and not related to abnormalities *per se*, and it should be noted that changes of resting heart rate are not consistently found in athletes experiencing OTS (139).

Heart rate variability (HRV) analysis has been used as a measure of cardiac autonomic balance, with an increase in HRV indicating an increase in vagal (parasympathetic) tone relative to sympathetic activity (143). Numerous studies have examined the effects of training on indices of HRV, but to date, few studies have investigated HRV in overreached or OTS athletes, with studies showing either no change (3,58,142), inconsistent changes (143), or changes in parasympathetic modulation (59).

Hedelin et al. (58) increased the training load of nine canoeists by 50% over a 6-d training camp. Running time to fatigue, $\dot{V}O_{2\max}$, submaximal and maximal heart rates, and maximal blood lactate production all decreased in response to the intensified training; however, all indices of HRV remained unchanged. On average, there were no significant changes in low-frequency power, high-frequency power, total power, or the ratio of low- to high-frequency power, both in the supine position and after head-up tilt. Similarly, Uusitalo et al. (142) reported no change in intrinsic heart rate and autonomic balance in female athletes after 6–9 wk of intensified training. This involved the investigation of autonomic balance assessed by pharmacological vagal and

β -blockade. In addition, both the time domain and power spectral analysis in the frequency domain were calculated during rest and in response to head-up tilt. Results suggest that HRV in the upright position had a tendency to decrease in response to intensified training in the subjects who were identified as “overtrained” (143). This may indicate vagal withdrawal and/or increased sympathetic activity. However, between-subject variability was high in this investigation. Finally, Hedelin et al. (58) reported increased HRV and decreased resting heart rate in a single “overtrained” athlete when compared with baseline measures. In comparison with normally responding subjects examined during the same period, the “overtrained” subject exhibited an increase in high frequency and total power in the supine position during intensified training, which decreased after recovery. The increase in high-frequency power was suggested to be most likely the result of increased parasympathetic activity (59).

Lamberts et al. (81) proposed that the heart rate return 1 min after high intensity interval exercise could serve to monitor training because it showed some correlation with the evolution of time trial performance after 4 wk in 14 moderately well-trained cyclists, but to date, there are no published results available from athletes in OR or OTS.

In a very recent study (12) in young soccer players, a decrease of submaximal heart rate, a faster return of heart rate after exercise, and an increase of vagal indices of HRV were associated with some positive adaptations to training, but the opposite was not true because “negative” changes of these markers were not indicators of a performance decline.

A meta-analysis (10) concluded that short-term (<2 wk) overload training results in an increased resting heart rate (mean value, +4.5 bpm), decreased maximal heart rate (–7.5 bpm), and a higher ratio between low- and high-frequency HRV. However, this was no longer the case after longer intensified training interventions lasting >2 wk, where the only significant difference remained a decreased maximal heart rate (–3.6 bpm).

Concerning the assumption often claimed in a clinical context that cardiac complications such as arrhythmias or other ECG changes discovered in athletes could be explained by a state of OR or OTS, this hypothesis does not find any support by any study inducing OR or OTS. However, it should be mentioned that an infectious disease—maybe facilitated by the intermittently depressed immunological state—occurring in an athlete engaged in heavy training may expose the individual to a higher risk of cardiac complications including a higher heart rate, extrasystoles, and even myocarditis (38).

The problems with physiological measures are as follows:

- HRV seems to be a tool in theory but does not provide consistent results. One needs to be careful when using HRV as an outcome measure because there are many different ways to record and calculate the data. Currently, there is no consensus regarding the required standardization and the method of measurement.

- The present data do not allow to distinguish between changes in physiological measures resulting from FOR, NFOR, and OTS.

IMMUNE SYSTEM

There are many reports on URTI due to increased training, and also in OR and OTS athletes. It seems feasible that intensified training (leading to OR or OTS) may increase both the duration of the so-called “open window” and the degree of the resultant immunodepression. However, the amount of scientific information to substantiate these arguments is limited. More data are available that each bout of prolonged and intensive exercise has transient but significant, wide ranging effects on the immune system (49,108). Heavy exertion leads to alterations in immunity and host pathogen defense and elevations in stress hormones, pro- and anti-inflammatory cytokines, and reactive oxygen species. The exercise-induced immune perturbations and physiologic stress are associated with an elevated risk of URTI, especially during the 1- 2-wk period after competitive marathon and ultramarathon race events (109). These data imply that chronic immune dysfunction and increased URTI symptomatology may result when exercise training is intensified, leading to OR and OTS, but few well-designed studies have been conducted to verify this hypothesis.

Several studies that have investigated the effects of short periods (typically 1–3 wk) of intensified training on resting immune function and on immunoendocrine responses to endurance exercise indicate that several indices of neutrophil function appear to be sensitive to the training load. A 2-wk period of intensified training in well-trained triathletes was associated with a 20% fall in the bacterially stimulated neutrophil degranulation response (121). In another study, neutrophil and monocyte oxidative burst activity, mitogen-stimulated lymphocyte proliferation, and percentage and number of T-cells producing interferon- γ were lower at rest after 1 wk of intensified training in cyclists (83). Other leukocyte functions including T-lymphocyte CD4⁺/CD8⁺ ratios, lymphocyte antibody synthesis, and natural killer cell cytotoxic activity have been shown to be lower after increases in the training load in already well-trained athletes (144). Several studies have documented a fall in salivary immunoglobulin A (IgA) concentration with intensified training, and some, although not all, have observed a negative relationship between salivary IgA concentration and occurrence of URTI (9,32,47,50,107). Thus, with sustained periods of heavy training, several aspects of both innate and adaptive immunity are depressed. Low levels of salivary IgA concentration or secretion rate and high anti-inflammatory cytokine responses to antigen challenge may predispose to high respiratory illness susceptibility in athletes (32,47,50). Several studies have examined changes in immune function during intensive periods of military training (16,17,135). However, this often involves not only strenuous physical activity but also dietary energy deficiency, sleep deprivation, and psycho-

logical challenges. These multiple stressors are likely to induce a pattern of immunoendocrine responses that amplify the exercise-induced alterations.

Studies that have examined athletes exposed to a long-term training periods (e.g., over the course of a 5- to 10-month competitive season) have shown a general trend of depression of both systemic and mucosal immunity (5,14,47,48,51,52,99). In these studies, depressed immunity was most commonly observed either at the end of the season or after the most intensive periods of training and/or competition. 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. However, it might just be that the increased URTI incidence reflects the increased stress associated with increased training, regardless of the response of the athlete to the increased physical stress. Furthermore, symptoms of respiratory illness reported by some athletes may be due to airway inflammation from noninfectious causes (8,21,146) rather than actual infection with a pathogen.

Whether that immune function is seriously impaired in athletes experiencing OTS is unknown because of insufficient scientific data. However, anecdotal reports from athletes and coaches of an increased infection rate with OTS (129) have been supported by a few empirical studies (75,119). In a cohort study of highly trained athletes before the Olympic Games, more than 50% of the athletes who reported symptoms of “OT” presented with infection compared with none of the athletes in the overreached group (75). In junior rowers, studied during and after a training camp (FOR), 40% of the male subjects had URTI (132). In a study by Reid et al. (119), 41 competitive athletes with persistent fatigue and impaired performance had a thorough medical examination, which identified medical conditions with the potential to cause fatigue and/or recurrent infections in 68% of the athletes. The most common conditions were humoral immune deficiency and unresolved viral infections. Evidence of Epstein–Barr virus reactivation was detected in 22% of the athletes tested. Adventure racing over a 4- to 5-d period has been linked to significant mood state disruption and elevated URTI rates (4). Thus, it seems plausible that a significant number of athletes who are diagnosed as experiencing OTS may experience increased URTI.

There are only a few reports of differences in immune function status in “overtrained” athletes compared with healthy trained athletes (e.g., Refs. [46,89]), and most studies on “overtrained” athletes have failed to find any differences (90,122). Circulating numbers of lymphocyte subsets change with exercise and training. With heavy training, the T-lymphocyte CD4⁺/CD8⁺ (helper/suppressor) ratio falls. However, this has not been shown to be different in athletes diagnosed as experiencing OTS compared with healthy well-trained athletes. One study (46) has shown that the expression of other proteins

on the cell surface of T-lymphocytes does seem to be sensitive enough to distinguish between the majority of “overtrained” athletes and healthy athletes. The expression of CD45RO on T-helper CD4⁺ cells (but not the circulating numbers of CD45RO⁺ T-cells) was significantly higher in athletes experiencing OTS compared with healthy well-trained controls. Using this indicator, “OT” could be classified with high specificity and sensitivity. However, CD45RO is a marker of T-memory cells and activated T-cells. Thus, higher expression of CD45RO on T-cells may merely be indicative of the presence of acute infection, which is, of course, a possible cause of the underperformance. Fry et al. (44) reported a significant increase in activation markers [CD25, human leucocyte antigen receptor test (HLA-DR)] in blood lymphocytes of “overtrained” athletes. Unresolved viral infections are not routinely assessed in elite athletes, but it may be worth investigating this in individuals experiencing fatigue and underperformance in training and competition. Thus, infection might be one of the “triggering” factors that can lead to the induction of OTS, or in some cases, the diagnosis of OTS cannot be differentiated from a state of postviral fatigue such as that observed with episodes of glandular fever. In the OTS diagnostic flowchart (Fig. 3), it is recommended to evaluate for “primary” viral and bacterial infections and systemic inflammatory diseases before proceeding with the diagnostic workup in direction OTS. It is acknowledged in the flowchart that, secondary in the time course of OTS, a reactivation of Epstein–Barr virus can be detected (119), which may contribute to the severity of symptoms. However, despite that this distinction between “primary” and “secondary” infection may be in some cases clinically difficult, it may help in the explanation and treatment of fatigue and underperformance-related diseases.

In conclusion, it is clear that the immune system is sensitive to stress—both physiological and psychological—and thus, potentially, immune variables could be used as an index of stress in relation to exercise training. The current information regarding the immune system and OR confirms that periods of intensified training result in depressed immune cell functions with little or no alteration in circulating cell numbers. However, although immune parameters change in response to increased training load, these changes do not distinguish between those athletes who successfully adapt to OR and those who maladapt and develop symptoms of the OTS. Furthermore, at present, it seems that measures of immune function cannot really distinguish OTS from infection or postviral fatigue states.

The problems with immunological testing are as follows:

- timing of the test (time of the day and time since last exercise session),
- lack of consistency of the data in literature,
- being time consuming and very expensive (for functional measures).

Resistance exercise. Although most research on OT and OR has focused on endurance activities, some research

has shed light on stressful training when using heavy resistance exercise, and it is summarized in several reviews (39,40,42,43). What has become clear is that excessively high volumes or intensities of resistance exercise can present considerably different physiological and performance profiles when compared with OT/OR with endurance activities. When excessive volumes of maximal loads are used for training, maximal muscular strength is one of the last performance measures to be adversely affected. On the other hand, high speed (e.g., sprinting) and power appear to be more sensitive to the stressful resistance exercise training and are the first types of performance to decrease. Although not greatly studied, some data also indicate that psychological variables may be sensitive to resistance exercise OT/OR. From an endocrine perspective, although testosterone concentrations and the testosterone/cortisol ratio may decrease because of resistance exercise OT/OR, these cannot be used to define the presence of an OTS. Rather, these hormonal measures simply indicate the presence of stressful training. When resistance-trained athletes are exposed to a repeated stressful training phase, the decreased hormonal response is lessened, suggesting that repeated training of this type may permit long-term training tolerance. The presence of an elevated acute sympathetic response with excessive resistance exercise loads supports the concept of a sympathetic OTS. This in turn may contribute to down-regulation of β_2 adrenergic receptors in the affected skeletal muscle (41). From a practical standpoint, the actual training program must be carefully monitored to incorporate adequate recovery phases as needed. Finally, it is readily apparent that sport-specific training in addition to the resistance exercise program can add to the training stresses and contribute to OT/OR (98).

The problems with resistance exercise OT/OR research are as follows:

- There are few research studies on resistance exercise OT/OR.
- There are many variations of resistance exercise that make it difficult to study.
- Muscular strength is usually preserved with resistance exercise OT/OR.
- Delayed onset muscular soreness and muscle damage are not necessarily the same as resistance exercise OT/OR.
- Few studies have monitored an adequate recovery period.

Prevention. One general confounding factor when reviewing literature on OTS is that the definition and diagnosis of OR and OTS are not standardized. One can even question if in most of the studies subjects were experiencing OTS. Because OTS is difficult to diagnose, authors agree that it is important to prevent OTS (37,78,141). Moreover, because OTS is mainly due to an imbalance in the training recovery ratio (too much training and competitions and too little recovery), it is of utmost importance that athletes record daily their training load, using a daily training diary or

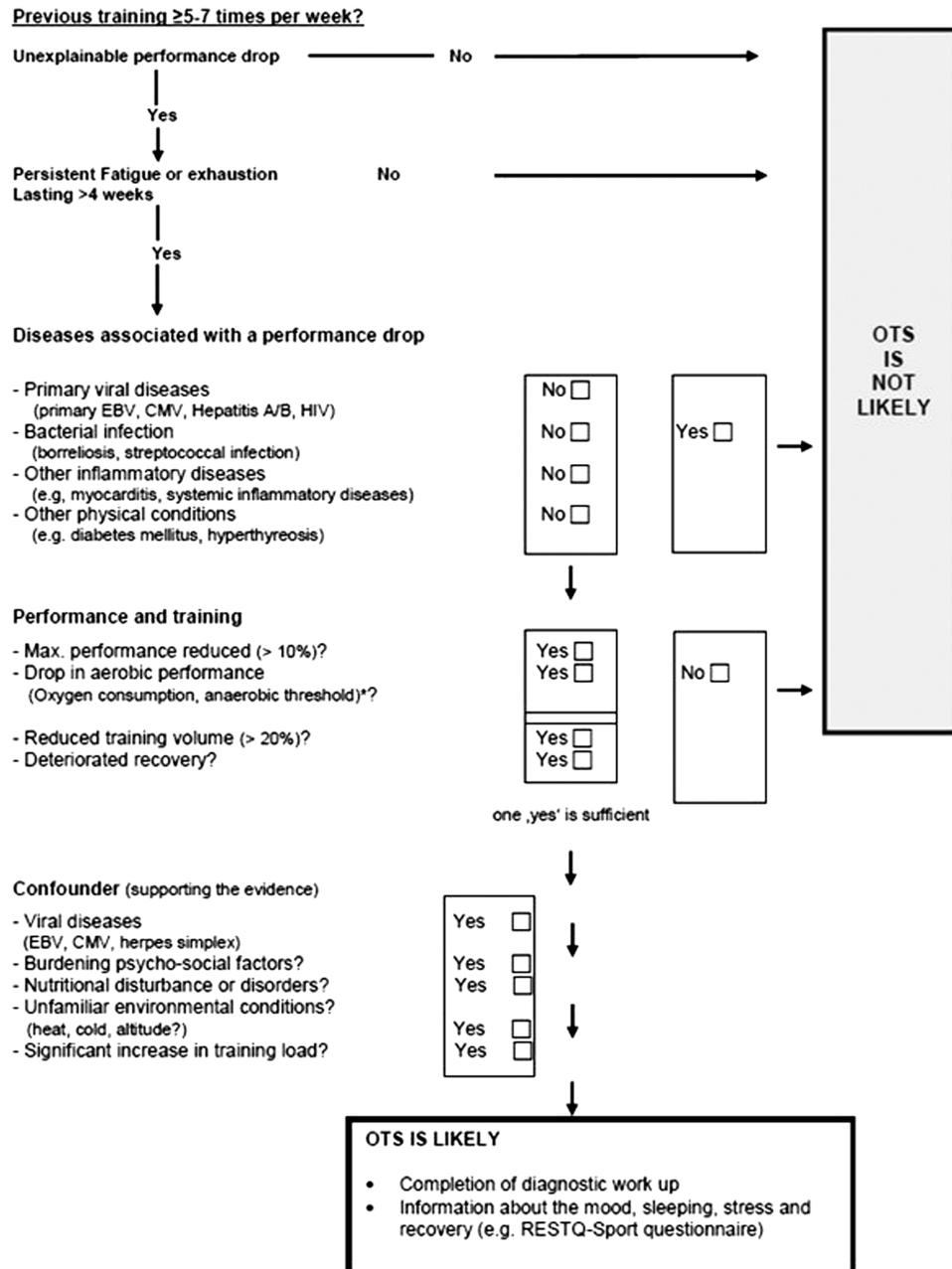


FIGURE 3—Flowchart. Diagnosis of OTS in athletes. When previous training load was high enough, the occurrence of persistent problems in performance creates a suspicion for OTS. The flowchart starts with the key symptoms: decrease in performance; duration of symptoms; then other major diseases that are related to underperformance are ruled out; then performance changes are defined and then possible confounding conditions and diseases are checked. This flowchart is based on the existing evidence together with the expertise/experience of the authors.

training log (34,35,37). The four methods most frequently used to monitor training and prevent OT are as follows: retrospective questionnaires, training diaries, physiological screening, and the direct observational method (62). Also, the psychological screening of athletes (7,60,61,92,101,102, 116,138) and the RPE (1,15,34,35,60,61,71,131) have received more and more attention nowadays.

Hooper et al. (61) used daily training logs during an entire season in swimmers to detect staleness (OTS). The distances swum, the dry-land work time, and the subjective

self-assessment of training intensity were recorded. In addition to these training details, the swimmers also recorded subjective ratings of quality of sleep, fatigue, stress and muscle soreness, body mass, early morning heart rate, occurrence of illness, menstruation, and causes of stress. Swimmers were classified as having OTS if their profile met five criteria. Three of these criteria were determined by items of the daily training logs: fatigue ratings in the logs of more than 5 (scale 1–7) lasting longer than 7 d, comments in the page provided in each log that the athlete was feeling that

he/she responded poorly to training, and a negative response to a question regarding presence of illness in the swimmer's log, together with normal blood leukocyte count.

Foster et al. (35,37) have determined training load as the product of the subjective intensity of a training session using "session RPE" and the total duration of the training session expressed in minutes. If these parameters are summated on a weekly basis, it is called the total training load of an individual. The "session RPE" has been shown to be related to the average percent heart rate reserve during an exercise session and to the percentage of a training session during which the heart rate is in blood lactate-derived heart rate training zones. With this method of monitoring training, they have demonstrated the utility of evaluating experimental alterations in training and have successfully related training load to its performance (35). Foster et al. (36) have demonstrated that athletes often do not perform the same training load prescribed by coaches. In particular, they noted that on days the coaches intended to be "easy," athletes often performed meaningfully longer and/or more intense training. These data fit well with the concept that OTS is a failure of the work-recovery relationship, often in the direction of athletes failing to take appropriate recovery. However, training load is clearly not the only training-related variable contributing to the genesis of OTS. So additional to the weekly training load, daily mean training load as well as the SD of training load was calculated during each week. The daily mean divided by the SD was defined as the monotony. The product of the weekly training load and monotony was calculated as strain. The incidence of simple illness and injury was noted and plotted together with the indices of training load, monotony, and strain. They noted the correspondence between spikes in the indices of training monotony and strain and subsequent illness or injury and thresholds that allowed for optimal explanation of illnesses were computed (34). The data in this study (34) were suggested by earlier data by Bruin et al. (11) in race horses. The horses responded appropriately to progressive increases in the training load until the normal recovery days were made harder (e.g., the monotony of training was increased). At this point, the running performance of the horses deteriorated and the horses demonstrated behavioral signs consistent with an equine version of OTS (e.g., being "off their feed," which included loss of appetite, biting their handlers and kicking their stalls). This finding of a sudden deterioration of performance with loss of normal regeneration is also consistent with the differences in training program design by coaches versus execution by athletes (36).

One of the disadvantages of the traditional "paper and pencil" method is that data collection can be complicated, and that immediate feedback is not always possible. Another problem is that when athletes are on an international training camp or competition, immediate "data computing" is not possible. It might therefore be useful to have an "online" training log that has specific features in detecting not only slight differences in training load but also the subjective

parameters (muscle soreness and mental and physical well-being) that have been proven to be important in the detection of OTS.

Strategies to reduce the symptoms of OR and reduce the risk of developing OTS. Both in the earlier data, reviewed in the ECSS 2006 consensus statement, and in the more contemporary data in this document, there is virtually no evidence suggesting that OTS can be "treated." Like a massive orthopedic injury, OTS (and even NFOR) is just as debilitating and takes a substantial time for recovery to occur spontaneously. Rest and very light training seem to be the only therapeutic agents capable of effecting recovery. The overwhelming impression, particularly in the evidence that has emerged since 2006, is that the emphasis needs to be on prevention of NFOR and OTS (mostly by appropriate periodization of the training program with careful focus on including, and executing, appropriate recovery time into the training program) and on early diagnosis of NFOR and OTS, which at least in principle might shorten the recovery time.

Rest and sleep. One of the most obvious methods for managing fatigue and enhancing recovery is adequate passive rest and obtaining sufficient sleep. It is generally recommended that athletes should have at least one passive rest day each week, because the absence of a recovery day, especially during intensified training periods, is closely related to the onset of signs of OR and underrecovery (11). A passive rest day can also act as a "time-out" period for athletes and prevent them from becoming totally preoccupied with their sport and possibly encourage them to pursue a different (passive) interest. Such distractions from the daily routine of training may alleviate boredom and reduce stress perception.

Sleep is an essential part of fatigue management, because persistent sleep loss can negatively affect the quality of a training session and general well-being. The primary need for sleep has been hypothesized as being neurally based rather than a requirement for restitution of other biological tissues (63). Therefore, with inadequate sleep, cognitive functions are likely to be impaired, especially the ability to concentrate. Individuals have different requirements for sleep, and to prescribe the dose of sleep that a highly trained athlete requires would be erroneous. The general advice is to sleep for the amount of time that is required to feel wakeful during the day, which may vary considerably between individuals.

Nutrition. Because OR is brought about by high-intensity training with limited recovery, it is thought that the fatigue and underperformance associated with OR are at least partly attributable to a decrease in muscle glycogen levels. Decreased glycogen levels can result in disturbances of the endocrine milieu. Glycogen depletion results in higher circulating levels of catecholamines, cortisol, and glucagon in response to exercise while insulin levels are very low. Such hormonal responses will result in changes in substrate mobilization and utilization (for instance, high adrenaline levels in combination with low insulin will increase lipolysis and stimulate the mobilization of fatty acids). Because repeated

days of hard training and carbohydrate depletion seem to be linked to the development of OR, it is tempting to think that carbohydrate supplementation can reverse the symptoms (130). In a group of runners who ran 16 to 21 km on a daily basis for 7 d and treated all those runs as races, performance dropped significantly when a moderate carbohydrate intake of $5.5 \text{ g}\cdot\text{kg}^{-1}$ body mass per day was maintained (2). The runners also displayed a range of symptoms indicating that they were overreached. But when the daily carbohydrate was increased to $8.5 \text{ g}\cdot\text{kg}^{-1}$ body mass per day, the drops in performance were much smaller and OR symptoms were reduced. Recovery from this week of hard training was more complete with the high-carbohydrate treatment. In this study, the dietary intake was strictly controlled and the subjects were fed to maintain energy balance. In a follow-up study, subjects received carbohydrate supplements before, during, and after training sessions, but their dietary intake the rest of the day was recorded but not controlled (56). In this study, a group of well-trained cyclists were required to perform 8 d of intensive endurance training (normal training volume was doubled). This training was performed on two occasions separated by a washout, or recovery, period of at least 2 wk. On one occasion, subjects consumed a 2% carbohydrate solution before, during, and after training (low CHO), and on the other occasion, subjects consumed a 6.4% carbohydrate solution before and during training and a 20% carbohydrate solution after training (high CHO). Total carbohydrate intake was $6.4 \text{ g}\cdot\text{kg}^{-1}$ body mass per day with low CHO and $9.4 \text{ g}\cdot\text{kg}^{-1}$ body mass per day with high CHO. The intensified training protocol induced OR as indicated by a decrease in performance (time to fatigue at approximately 74% of aerobic capacity), although the decrease in performance was significantly less with high CHO, suggesting that high-CHO diets can reduce the severity of OR. Alteration of mood state (assessed by POMS questionnaire) and hormonal disturbances in the response to exercise were also less on high CHO compared with low CHO. By requiring the subjects to consume supplements that contained a large amount of carbohydrate, the total energy intake increased as well (13.0 vs $16.5 \text{ MJ}\cdot\text{d}^{-1}$ for low CHO and high CHO, respectively). Athletes in hard training seem to reduce (or not increase) their spontaneous food intake, and unless they supplement with carbohydrate, they may be in negative energy balance during periods of intensified training. It also appeared that the amount of carbohydrate ingested during training influenced the length of time needed for recovery. After 2 wk of recovery (reduced volume and intensity) from intensified training, performance remained below that of baseline for the low-CHO treatment, whereas performance improved compared with baseline after 2 wk of recovery from intensified training with the high-CHO condition.

Besides carbohydrate depletion, dehydration and negative energy balance can increase the stress response (increased catecholamines, cortisol, and glucagon, whereas insulin levels are reduced), which increases the risk of de-

veloping OR symptoms. Thus, to reduce the symptoms of OR and reduce the risk of developing OTS during periods of intensive training, athletes should be encouraged to increase their fluid, carbohydrate, and energy intake to meet the increased demands. Additional carbohydrate should not be at the expense of reduced protein intake because there is some evidence that insufficient protein can also result in increased risk of OR (75). Supplementation with amino acids (glutamine and branched chain amino acids), however, is not likely to reduce symptoms of fatigue and OR (97).

Considerations for coaches and physicians. Until a definitive diagnostic tool for the OTS is present, coaches and physicians need to rely on performance decrements as verification that an OTS exists. However, if sophisticated laboratory techniques are not available, the following considerations may be useful:

- Maintain accurate records of performance during training and competition. Be willing to adjust daily training intensity/volume or allow a day of complete rest, when performance declines, or the athlete complains of excessive fatigue.
- Avoid excessive monotony of training.
- Always individualize the intensity of training.
- Encourage and regularly reinforce optimal nutrition, hydration status, and sleep.
- Be aware that multiple stressors such as sleep loss or sleep disturbance (e.g., jet lag), exposure to environmental stressors, occupational pressures, change of residence, and interpersonal or family difficulties may add to the stress of physical training.
- Treat OTS with rest. Reduced training may be sufficient for recovery in some cases of OR.
- Resumption of training should be individualized on the basis of the signs and symptoms because there is no definitive indicator of recovery.
- Communication with the athletes (maybe through an online training diary) about their physical, mental, and emotional concerns is important.
- Include regular psychological questionnaires to evaluate the emotional and psychological state of the athlete.
- Maintain confidentiality regarding each athlete's condition (physical, clinical and mental).
- Importance of regular health checks performed by a multidisciplinary team (physician, nutritionist, psychologist, etc.).
- Allow the athlete time to recover after illness/injury.
- Note the occurrence of URTI and other infectious episodes; the athlete should be encouraged to suspend training or reduce the training intensity when experiencing an infection.
- Always rule out an organic disease in cases of performance decrement.
- Unresolved viral infections are not routinely assessed in elite athletes, but it may be worth investigating this in individuals experiencing fatigue and underperformance in training and competition.

Moreover, when OTS is suspected, it is also of utmost importance to standardize the criteria used for diagnosis and/or, at least, as tools for the diagnosis of OTS are lacking, to standardize the criteria of exclusion of OTS (see Fig. 1 for the definition and Tables 1 and 2).

CONCLUSION

A difficulty with recognizing and conducting research into athletes with OTS is defining the point at which OTS develops. Many studies claim to have induced OTS, but it is more likely that they have induced a state of OR in their subjects. Consequently, the majority of studies aimed at identifying markers of ensuing OTS are actually reporting markers of excessive exercise stress resulting in the acute condition of OR and not the chronic condition of OTS. The mechanism of OTS could be difficult to examine in detail maybe because the stress caused by excessive training load, in combination with other stressors, might trigger different “defence mechanisms” such as the immunological, neuro-endocrine, and other physiological systems that all interact and probably therefore cannot be pinpointed as the “sole”

TABLE 1. Diagnosis of OTS—checklist.

Performance - Fatigue
Is the athlete experiencing the following: Unexplainable underperformance Persistent fatigue Increased sense of effort in training Sleep disorders ...
Exclusion criteria Are there confounding diseases? Anemia Epstein-Barr virus Other infectious diseases Muscle damage (high CK) Lyme disease Endocrinological diseases (diabetes, thyroid, adrenal gland, ...) Major disorders of eating behavior Biological abnormalities (increased erythrocyte sedimentation rate, C-reactive protein, creatinine, or liver enzymes, decreased ferritin, ...) Injury (musculoskeletal system) Cardiological symptoms Adult-onset asthma Allergies ...
Are there training errors? Training volume increased (>5%) (h·wk ⁻¹ , km·wk ⁻¹) Training intensity increased significantly Training monotony present High number of competitions In endurance athletes : decreased performance at “anaerobic” threshold Exposure to environmental stressors (altitude, heat, cold, ...) ...
Other confounding factors: Psychological signs and symptoms (disturbed POMS, RESTQ-Sport, RPE, ...) Social factors (family, relationships, financial, work, coach, team, ...) Recent or multiple time zone travel ...
Exercise test Are there baseline values to compare with? (performance, heart rate, hormonal, lactate, ...) Maximal exercise test performance Submaximal or sport-specific test performance Multiple performance tests ...

TABLE 2. Methodological prerequisites for studies of markers for NFOR/OTS.

1. Inclusion of a sufficient number of well-trained subjects
2. Definition of a range of meaningful differences by determination of individual ranges of normal variations including phases of FOR
3. Inclusion of measures showing the decline of sport-specific performance (“gold standard”)
4. Exclusion of medical causes (illnesses) of impaired performance
5. Inclusion of markers measured at rest and/or at submaximal exercise
6. Follow-up after a sufficient duration of recovery before final testing

cause of OTS. It might be that as in other syndromes (e.g., chronic fatigue syndrome or burnout), the psychoneuroimmunology (study of brain-behavior-immune interrelationships) might shed a light on the possible mechanisms of OTS, but until there is no definite diagnostic tool, it is of utmost importance to standardize measures that are now thought to provide a good inventory of the training status of the athlete. A primary indicator of OR or OTS is a decrease in sport-specific performance, and it is very important to emphasize the need to distinguish OTS from OR and other potential causes of temporary underperformance such as anemia, acute infection, muscle damage, and insufficient carbohydrate intake.

The physical demands of intensified training are not the only elements in the development of OTS. It seems that a complex set of psychological factors are important in the development of OTS, including excessive expectations from a coach or family members, competitive stress, personality structure, social environment, relationships with family and friends, monotony in training, personal or emotional problems, and school- or work- related demands. Although no single marker can be taken as an indicator of impending OTS, the regular monitoring of a combination of performance, physiological, biochemical, immunological, and psychological variables would seem to be the best strategy to identify athletes who are failing to cope with the stress of training. We therefore propose a “check list” that might help the physicians to decide on the diagnosis of OTS and to exclude other possible causes of underperformance (Table 2).

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