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Diagnosis of Overtraining

What Tools Do We Have?

Axel Urhausen and *Wilfried Kindermann*

Institute of Sports and Preventive Medicine, Faculty of Clinical Medicine, University of Saarland, Saarbruecken, Germany

Abstract

The multitude of publications regarding overtraining syndrome (OTS or 'staleness') or the short-term 'over-reaching' and the severity of consequences for the athlete are in sharp contrast with the limited availability of valid diagnostic tools. Ergometric tests may reveal a decrement in sport-specific performance if they are maximal tests until exhaustion. Overtrained athletes usually present an impaired anaerobic lactacid performance and a reduced time-to-exhaustion in standardised high-intensity endurance exercise accompanied by a small decrease in the maximum heart rate. Lactate levels are also slightly lowered during submaximal performance and this results in a slightly increased anaerobic threshold. A reduced respiratory exchange ratio during exercise still deserves further investigation. A deterioration of the mood state and typical subjective complaints ('heavy legs', sleep disorders) represent sensitive markers, however, they may be manipulated. Although measurements at rest of selected blood markers such as urea, uric acid, ammonia, enzymes (creatine kinase activity) or hormones including the ratio between (free) serum testosterone and cortisol, may serve to reveal circumstances which, for the long term, impair the exercise performance, they are not useful in the diagnosis of established OTS. The nocturnal urinary catecholamine excretion and the decrease in the maximum exercise-induced rise in pituitary hormones, especially adrenocorticotropic hormone and growth hormone, and, to a lesser degree, in cortisol and free plasma catecholamines, often provide interesting diagnostic information, but hormone measurements are less suitable in practical application. From a critical review of the existing overtraining research it must be concluded that there has been little improvement in recent years in the tools available for the diagnosis of OTS.

The overtraining syndrome (OTS) or 'staleness' represents one of the most feared complications in competitive athletes and is of real interest in sports medicine and scientific research. Although in recent years the knowledge of central pathomechanisms of the OTS has significantly increased,[1-3] there is still a strong demand for relevant tools for the early diagnosis of OTS.

OTS is characterised by a sports-specific decrease in performance together with disturbances in mood state. This underperformance persists despite a period of recovery lasting weeks or months.[3-5] The definitive diagnosis of OTS always requires the exclusion of an organic disease. OTS is rare; however, short-term over-reaching is planned (or calculated) in training and therefore occurs much more often. Even though some coaches claim it is necessary to induce a state of over-reaching during the training process, a consensus statement concluded that over-reaching should be avoided because of its unpredictable outcome.[6]

In the diagnosis of OTS (or over-reaching), parameters that are inexpensive to measure and may be measured at rest or at least during submaximal exercise, without disturbing the training process, are preferred. Because it is speculated that a continuum exists between training fatigue, over-reaching and OTS,[4] it would be preferable to identify tools which can lead to an early diagnosis of over-reaching/OTS. Therefore, this article also discusses findings from studies defining over-reaching/OTS that have not necessarily assessed a period of recovery.

1. Tools Diagnosing Overtraining Syndrome (OTS) at Rest

1.1 Heart Rate

An increased heart rate at rest has been reported as a sign of OTS.[7-10] However, this was not confirmed in more recent prospective studies.[11-16] An increased heart rate at rest or during submaximal exercise may, however, indicate an infectious disease or glycogen depletion and thus indicate an acutely reduced exercise tolerance that may lead to OTS.

Recently, variability in beat-to-beat changes of the heart rate have been proposed as a tool to help in the diagnosis of OTS. A tempting assumption is that the sympathetic and parasympathetic forms of OTS are reflected by corresponding changes in the low- and high-frequency components of the R-R interval spectral analysis. However, the validity of the heart rate variability in the diagnosis of OTS is not well established at present. The existing heart rate variability findings in overtrained athletes are confusing. In a case report, an increased heart rate variability in the high frequency range suggesting a pronounced parasympathetic modulation was found in an athlete with chronic OTS.[17] A recent study reported a decrease in sympathetic/parasympathetic balance in fatigued athletes after 12 weeks of intense endurance training.[18] After 6-9 weeks of intensive training in nine female endurance athletes, changes in maximal oxygen uptake ($\dot{V}O_{2max}$) were significantly correlated with changes of R-R interval variability during standing; however, the five overtrained individuals of this group showed different individual responses.[19] In another investigation, 6 days of overload training resulting in over-reaching was not accompanied by a change in heart rate variability.[20] In a recent study, we found no change in 15 minutes (5 minutes supine, 5 minutes upright, 5 minutes supine position) heart rate spectral analysis, taken in five fatigued swimmers after a 2-week training camp; in contrast, heart rate variability decreased in the six team mates who did not experience over-reaching.[21]

1.2 Mood State and Subjective Complaints

An impaired mood state and subjective complaints are consistently described as sensitive and early markers of OTS.[16,22-26] Overtrained athletes typically show an inversed 'iceberg profile' in their Profile of Mood State (POMS scale).[23,27] Similarly, the 'self-condition' scale according to Nitsch[28] reveals a reduced 'capacity to act'.[16,25] With persisting OTS, depression increases significantly on both scales. The subjective complaints are dominated by a pronounced feeling of muscular soreness ('heavy legs' in runners, triathletes and cyclists), which usually occurs during low exercise intensities and daily activities. Sleep disorders may also be an early indicator sign of OTS.[16,29]

In practice, the usefulness of these subjective parameters is somewhat restricted because of the difficulty in defining a reference value that indicates OTS. The deterioration in mood state usually starts well before the definitive drop in performance and parallels the increase in training load.[16,23,25] In addition, it should be kept in mind that athletes may manipulate, especially if they fear that coaches will make substitutions for another team mate.

1.3 Enzyme Activities and Metabolic Markers in Blood

Measurements of selected enzyme activities and blood markers under standardised conditions are sometimes proposed to help in the diagnosis of OTS, for example, creatine kinase (CK),[9,30] urea,[9] uric acid[31] and ammonia.[10] However, the validity of these variables, at least in the diagnosis of OTS, is overestimated and systematic studies could not confirm changes in overtrained athletes.[12,13,32,33]

The CK activity mirrors the mechanical-muscular strain of the training in the preceding days and reacts to the intensity and volume of exercise, particularly unaccustomed eccentric forms of exercise. Some athletes are nonresponders and show only very small increases in CK activity. In addition, after a single bout of eccentric exercise, a muscular adaptation lasting several weeks can occur.[34] Elevations of CK activity are not a clear indication of OTS because even after an increase in CK activity to ~1500 U/L, further eccentric strength training did not change CK activity, muscle strength and reports of soreness.[35] It can be speculated, however, that increased CK activity could be used to prevent injuries resulting from the impaired muscular strength or coordination associated with muscle soreness and fatigue.

Increasing serum concentrations of urea are used as a marker of enhanced protein catabolism and stimulated gluconeogenesis resulting from higher training loads, especially longer intensive endurance training.[36,37] Nutritional factors should also be considered, when measuring urea changes.

Follow-up studies of serum concentrations of urea and CK may primarily indicate an acute impairment in exercise tolerance, which may be prophylactic for OTS in the long term. Ammonia has been described to be decreased at rest in athletes with OTS,[3] but this represents an inconsistent finding.[16] Uric acid does not show significant changes in overtrained athletes.[12,13,33] A definitive diagnosis of OTS, however, using these parameters is not possible.[12,25,32,33,38]

1.4 Hormones

A decreased nocturnal urinary excretion of catecholamines has been suggested as a rather late sign of OTS in overtrained athletes[13,39] and has been interpreted as lowered intrinsic sympathetic activity. In contrast, increased resting plasma concentrations of norepinephrine were found in a retrospective observation in three over-reached athletes[40] and a trend was reported in another study.[13] Further investigations have not confirmed decreased nocturnal urine excretion of catecholamines nor the increased plasma levels of free catecholamines.[20,25,33,41]

A decrease in the ratio between testosterone or free testosterone and cortisol has been suggested as a marker of ‘anabolic-catabolic balance’ and as a tool in the diagnosis of OTS.[42] In Finnish weight lifters, the testosterone/cortisol ratio correlated with measurements of strength.[43] However, most of the studies were not able to confirm changes in the testosterone/cortisol ratio in overtrained endurance[13,25,33,39,41] or strength athletes.[44]

Results concerning the behaviour of serum cortisol levels at rest are both variable and equivocal in relation to OTS. While some studies show unchanged values,[25,33,40,41,44] others report increases[22,42] and even decreases,[13] or describe variable responses.[20] Some investigators have suggested that decreased resting serum cortisol is a criterion for the diagnosis of OTS[14] It has been proposed that a decrease in cortisol occurs in the more chronic state of the OTS,[3] while an increase would represent an acute higher physiological strain.[38,45-47]

Overall, the data lead to the conclusion that it is not possible to diagnose an OTS based on single hormonal blood concentrations at rest. Although it can be argued that serial blood samplings of hormones, certain of which show a pulsatile release, may be more helpful, this method would not be practical.

1.5 Immunological Parameters

Up to now, only few data concerning the changes in immunological parameters in athletes diagnosed with OTS have been reported. In one prospective controlled study an enhanced expression of T-cell surface (CD45 RO+) markers were shown in OTS.[48] Others report a reduced concentration of glutamine in peripheral blood in overtrained athletes[32,49] or a decrease in the glutamine/glutamate ratio during training in five athletes.[50] However, a differentiation between intensive training and OTS based on the changes of glutamine could not be made. In another study, lower plasma glutamine was not a consistent finding in overtrained swimmers and glutamine levels did not necessarily decrease during high-intensity phases of training.[51] Increased concentrations of cytokines, as speculated in recent integrative theories about the pathomechanism of OTS,[52] are awaiting confirmation via concrete data in overtrained athletes.

2. Tools Diagnosing OTS during Exercise

2.1 Ergometry

The assessment of a decrement of performance in OTS still represents the gold standard of diagnosis and requires a sports-specific testing procedure that has to be continued until exhaustion. In an incremental graded test procedure the maximal performance or the $\dot{V}O_{2max}$ of overtrained athletes tends to be reduced,[9,13,24,41,53] but unchanged values are not unusual.[11,12,16,54] Speed-endurance or short-term high-intensive endurance exercise tests seem to represent more sensitive tools.[9,11,15,16,20] Especially the so-called 'stress test', which is performed with the intensity of 10% above the individual anaerobic threshold on a cycle ergometer and leads to exhaustion after ~15 to 40 minutes, has repeatedly been successful in diagnosing OTS in controlled prospective studies with reductions in exercise duration of between 14 to 27%.[15,16]

The short-term high-intensity alactacid anaerobic performance was unaffected in most,[9,11,16] but not in all[55] OTS investigations.

An impairment of coordination in overtrained athletes has been commonly reported by coaches in practice[6] and in studies.[10,38] In addition, decrements of the H-reflex[56] or the neuromuscular excitability[57] have been observed, but are difficult to measure from a methodical point of view. In overtrained strength athletes, a decreased 1-repetition maximum strength has been described.[44]

2.2 Blood Lactate

The decrement of maximal performance is paralleled by reduced maximal blood lactate concentrations, as described for example in middle- and long-distance runners,[9] swimmers,[54] cyclists and triathletes,[15,16] judoka[11] and canoeists.[20] However, it is interesting to note that, in OTS, the submaximal lactate concentrations during graded exercise are typically slightly lowered and the resulting calculations of the anaerobic threshold tend to increase. Consequently, a reduced anaerobic threshold points to a training error rather than to OTS. If the athlete has depleted glycogen stores, which is not the case in OTS,[14] decreased maximal and submaximal lactate concentrations can be measured, too. The individual anaerobic threshold, however, will not be influenced by carbohydrate deficiency and maximal heart rate is not reduced.[58,59]

2.3 Ammonia

It has been suggested that ammonia induces exercise-related fatigue by central or peripheral mechanisms.[60] However, the plasma ammonia concentrations did not correlate with the time to exhaustion in corresponding studies[61] and were found to be unchanged or even decreased in OTS.[13,33] However, when monitoring training, it is important to note that at the same absolute workload ammonia concentrations are increased and lactate is decreased in states of glycogen depletion. This increase in submaximal ammonia levels parallels the heart rate response.[59]

2.4 Heart Rate

It has repeatedly been shown that the maximal heart rate is slightly, but significantly reduced in overtrained athletes[13-16,20,41] However, the intra-individual decrease is only ~3 to 5 beats/min and is rather small, which impairs the usefulness of this parameter in practice.

2.5 Respiratory Exchange Ratio

The respiratory exchange ratio during submaximal and maximal exercise has been observed to be lowered in overtrained endurance athletes, which is postulated as an expression of diminished carbohydrate metabolism in OTS.[14-16,54] This finding has been reported in the absence of glycogen depletion in OTS, but the validity of this parameter is not yet well established and requires further investigation.

2.6 Perception of Effort

An increased ratio between the actual exercise intensity expressed in watts or measured by blood lactate concentration and the subjective perception of effort (Borg-scale) has been documented as a criterion for OTS.[24,53] Close analysis of ratings of perceived effort, however, reveals only minor changes in overtrained athletes. Thus the practical usefulness of this tool appears to be limited.

2.7 Hormones

Athletes with OTS also show lower maximal plasma concentrations of free (nor)epinephrine after exhaustive exercise in most,[9,25,41] but not all[13,33] studies. The differences may be due to different forms of OTS (sympathetic or parasympathetic, if such a distinction can be made at all). In one study[13] an increased submaximal concentration of norepinephrine was observed; however, the absolute differences were too small to be used in practice and the result was not replicated.[33,41]

A decreased maximal exercise-induced secretion of pituitary hormones [adrenocorticotropic hormone (ACTH), human growth hormone (HGH)], and to a lesser extent cortisol has been found in several studies of overtrained endurance and strength athletes.[25,33,44] These results corroborate former findings with overtrained marathon runners after insulin-induced hypoglycaemia.[22] Secretion of ACTH and HGH were clearly reduced by 42 to 48%[33] and 24 to 26%, respectively.[25] It is probable that these changes, which can only be observed following maximal exercise, represent an early indicator of 'hormonal over-reaching'. Indeed ACTH and HGH were already lowered by 30 and 36%, respectively, after 2 days of intensive training under laboratory conditions compared with controls, whereas the performance was still unchanged.[62] The ergometry 'stress test' used for these measurements was performed at 10% above the individual anaerobic threshold until exhaustion. In another study, maximal exercise-induced serum cortisol concentrations were reduced after 4 weeks of experimental intensive endurance training without overtraining.[41] Other authors report similar positive results with a repeated testing procedure within 3 hours.[63]

The disadvantages of such hormonal monitoring of training are the necessity of highly standardised conditions and the expensive methods. Furthermore, from a practical standpoint systematic hormonal measurements require the possibility for analysis of small capillary samples.

3. Current Recommendations

The validity of different tools for the diagnosis of OTS is summarised in table I. Before diagnosing OTS, organic diseases that may mimic the symptoms of OTS must be excluded by a physician qualified in sports medicine. The detection of OTS is based on regular assessments of the maximal sports-specific performance (including coordinative skills), which, in some disciplines, may be reproduced by ergometric tests. The only tools available to diagnose OTS under resting conditions are an impaired mood state and subjective complaints such as the feeling of 'heavy legs' and reports of sleep disturbances. Decreased nocturnal urinary catecholamine excretion might also be of value but is not very practical. In the ergometric laboratory, an impaired maximal lactate production and perhaps a reduced maximal heart rate might be indications of OTS. Because reduced maximal performance and lactate concentrations are also the result of muscular glycogen depletion after intensive training, performance testing should be done after at least 2 days of reduced training intensity or rest. Decreased maximal exercise-induced ACTH secretion may be highly suggestive of OTS, but is normally reserved for research study conditions. The lack of validity of some blood variables in diagnosing OTS, however, does not mean that they are not valid in the moni-

Table I. Summary of proposed tools with corresponding changes and suitability for the diagnosis of an overtraining syndrome (OTS)

Tool		Changes in OTS	Suitability
Sports-specific performance	(Sub)maximal exercise	↓	Gold standard; regular testing problematic (in most sports)
Ergometric performance	Anaerobic threshold	(↑)	Does not diagnose OTS, but targets other training errors
	Maximal exercise	↓ or ↔	Incremental graded tests less sensitive than tests-to-exhaustion (or time-trials)
Neuromuscular excitability	At rest	↓	Difficult method; needs more data
Mood profile	At rest	↓	Very sensitive; may be manipulated
Subjective complaints	At rest, submaximal exercise	↑	'Heavy legs': very common; sleep disorders: less common; may be manipulated
Borg-scale	Submaximal exercise	(↑)	Small changes
Heart rate	At rest	↔	↑ may indicate other problems (infection)
	Variability	?	Insufficient data
	Maximal exercise	(↓)	Rather small changes
Respiratory exchange ratio	(Sub)maximal exercise	↓	Limited data
Lactate	Submaximal exercise	(↓)	Does not diagnose OTS, but excludes other training errors
	Maximal exercise	↓	Typical change, but probably not in every sport
CK, urea	At rest	↔	↑ may indicate muscular overuse or prolonged carbohydrate depletion
Testosterone	At rest	↔	↓ may indicate high physiological strain?
Cortisol	At rest	↔	↑ may indicate high physiological strain
	Maximal exercise	(↓)	Differentiation between intensive training and OTS may be questionable
ACTH	Maximal exercise	↓	Very sensitive, differentiation between intensive training and OTS may be questionable
Catecholamines	Excretion (urine)	↓	Marked ↓ as late indicator of OTS
	Maximal exercise (plasma)	↓ or ↔	Parallels changes of lactate

ACTH = adrenocorticotropic hormone; **CK** = creatine kinase; ↓ = decreased; **(**↓**)** = slightly decreased; ↔ = unchanged; ↑ = increased; **(**↑**)** = slightly increased; **?** = not established.

toring of training and thus, in the long term, in the prevention of OTS. *In summary*, a critical review of existing scientific literature leads to the disappointing conclusion that the tools available for OTS diagnosis have not improved much in the last years of overtraining research.

4. Future Studies

Test results always need an individual interpretation and thus the need for initial assessment of individual baseline values. The definition of OTS should be restricted to actual decreases in performance. The assessment and validity of diagnostic tools requires studies including larger numbers of elite competitive athletes and observations over longer time intervals. Future investigations should focus on the causal training factors as well as temporal changes during recovery, which may induce positive adaptations after a preceding period of overload training.

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Correspondence and offprints: *Axel Urhausen*, Institute of Sports and Preventive Medicine, Faculty of Clinical Medicine, University of Saarland, Saarbruecken, 66041, Germany.