

Overload and injuries in young competitive athletes: fate or avoidable

Sobrecarga y lesiones en deportistas jóvenes de competición: ineludibles o evitables

Peter H. Schober

Medical University of Graz, Graz, Austria.

doi: 10.18176/archmeddeporte.00212

Introduction

In recent years, the developmental conditions for our children and adolescents have deteriorated significantly, particularly in industrialized countries. Sensory experiences and perceptions are increasingly restricted, and movement and physical experiences are reduced to a minimum. These changes impact youth sports, but also the competitive sports of young athletes, where overload, performance drops and drop outs are common. This age group is also particularly vulnerable to the multiple stresses of training, competition, and instruction, which can lead to physical and psychological problems. Individual performance capacity and thus the ability to regenerate are often disregarded in load management. Furthermore, too little attention is paid to recovery as part of the training plan. This can prevent adequate, optimal, age-appropriate performance improvement. This poses a major challenge for sports medicine and sports science¹. However, since the problem of overtraining is common, some authors even consider the state of overload to be a "normal" part of continuous performance development². However, I cannot accept this and strongly disagree with it. Nor can we adapt the health-relevant norm values of blood lipids (cholesterol, LDL, etc.), which are becoming increasingly higher due to the lifestyle of the population, to this phenomenon and thereby accept health damage such as heart attacks or strokes.

The physiology of "functional" overload

The principle of so-called stress-recovery-supercompensation has become established in competitive sports. Adequate stimuli adapted

to the training status are applied to induce the desired adaptations for improved performance. These stresses are catabolic and lead to temporary fatigue of the organism with depleted glycogen stores, increased resting heart rate, increased testosterone-cortisol levels, and an increase in creatine kinase levels, with subsequent training adaptations in the sense of supercompensation. However, the prerequisite for this is that the organism is allowed to undergo the regenerative processes that then lead to an increase in individual performance³⁻⁵.

In principle, however, the physiological processes involved in "functional" overtraining represent an essential component of training to improve performance but are reversible with sufficient regeneration and adaptation of the organism. "The dose makes the poison" - When exercise is performed without adequate regeneration, catabolism predominates, which can lead to chronic overload and overtraining. However, the specialist literature uses inconsistent terms for one and the same phenomenon. However, the terms (functional) overreaching, overtraining and overtraining syndrome (OTS) seem to be gaining ground^{4,6}.

The definition of overtraining

"The overtraining syndrome is a condition of fatigue and underperformance, often associated with frequent infections and depression which occurs following hard training and competition. The symptoms do not resolve despite two weeks of adequate rest, and there is no other identifiable medical cause"⁷.

The cause is a long-term catabolic recovery imbalance due to training and competition, which then leads to stagnation or a decline in general athletic performance as well as sport-specific performance.

Correspondence: Peter H. Schober
E-mail: peter.schober@medunigraz.at

This decline in performance usually occurs gradually and often “despite” continued or even intensified training. This decline in performance is often accompanied by pronounced physical or mental health problems². Generally, however, overtraining syndrome is a diagnosis of exclusion, as despite the assumption of adequate regenerative measures in training methods, a persistent decline in performance occurs without any detectable organic pathological causes, such as viral or bacterial infections. This can lead to painful changes in the musculoskeletal system, changes in the blood-forming system, and endocrine disorders, especially of the thyroid or adrenal glands. Disturbances of the sleep-wake rhythm and psychological disturbances are very common. Especially at the onset of overtraining, a reliable diagnosis is often difficult to make^{4,8}. These parameters usually only become noticeable when the clinical condition of overtraining is also evident⁹.

The pathophysiology of overtraining

Glycogen depletion leads to glycogen deficiency in the stressed muscles, which leads to “hard muscles,” shortened fascia, and injuries¹⁰. A neuroendocrine imbalance affects both motor function and mental health. The regeneration deficit leads to an amino acid imbalance with inadequate nutritional supply to muscles, leukocytes, and erythrocytes¹¹. The consequences also affect the autonomic nervous system, leading to an imbalance and dysfunction of stress hormones. This reduces neuromuscular excitability and inhibits alpha motor neuron activity. These neurophysiological phenomena lead to fatigue of the muscular structures, reduced speed of action, and increased coordination deficits. This reduces the opportunity for movement correction and increases the injury rate.

This results in a disruption of the stress axis (HPA - hypothalamic-pituitary-adrenal axis) with reduced adrenal sensitivity to ACTH and increased pituitary sensitivity to growth hormone releasing hormone (GHRH) for growth hormone GH release, which leads to a counter-regulatory shift in anabolic endocrine action and a regeneration deficit. Overall, the “stress response” is diminished, which negatively impacts athletic performance and reduces performance.

To counteract cellular stress, the organism reacts by increasing beta-adrenoreceptor density and hypersensitivity to catecholamines. This results in reduced intrinsic sympathetic activity, which reflects impaired cell function and impaired regeneration. In addition, intracellular protective mechanisms are ramped up with increased synthesis of heat shock proteins (HSP 70) to stabilize cell structure. These phenomena are expressions of strategies against impending overload-induced cell damage.

Causes of overtraining

The causes and mechanisms of overtraining are diverse and can be divided into endogenous and exogenous factors. What they all have in common, however, is reduced anabolic function of the organism due to inadequate regeneration resulting from very extensive and/or intensive training sessions. Excessively frequent, high training intensities in the anaerobic-lactic (high intensity interval training, HIIT) or high intensity

endurance range, especially over a longer period, can lead to exhaustion of the regenerative, anabolic system and a state of exhaustion in the organism. Particularly in junior athletes, high training volumes that increase within a short period of time, and often an excessive number of competitions with a lack of time for regeneration, play a significant role and can exacerbate this problem. Training monotony with constant, uniform, monotonous exertion is also more prone to exhaustion. Particular attention must be paid to ensuring that training or the amount of training is not inappropriately initiated or increased after infections (viral, bacterial) or longer training breaks due to injury⁴.

Especially in youth sports, there are stress factors that are often overlooked in training and competition planning that lie outside the realm of sport but are often of significant importance. These include academic stress, exam situations, relationship problems, constant bottlenecks in daily time management, frequent travel, inadequate altitude adaptation, or climate change. However, an inadequate, overly one-sided diet with insufficient nutrient density can also be the cause of overtraining⁹. With all these possibilities and risks, the risk of overtraining always depends on an athlete’s training experience, anthropometry, and genetics. In any case, the decisive factor for training tolerance and recovery capacity is individual oxygen uptake ($\text{VO}_2/\text{min}/\text{kg}$ body weight)^{4,12}. This is also the measure of recovery capacity in non-endurance-dominated sports. This means that the scope and intensity of training require an adapted oxygen uptake for adequate recovery and performance development. The same training volume and intensity may already constitute an overload for one athlete with lower oxygen uptake, but may be adequate for another athlete in terms of their physical and mental performance.

As a rule, coaches and athletes should be aware of the effects of overtraining. Nevertheless, a lack of knowledge about how to categorize their individual training control seems to be a common cause of overtraining. Other reasons can include excessive ambition or insufficient self-assessment. The result is overtraining syndrome with persistent or even progressive fatigue and, above all, reduced performance. Despite this, or perhaps because of this, the training workload is increased instead of the necessary recovery time, out of fear of not achieving the desired performance goal^{4,12}. This is also the measure of recovery capacity in non-endurance-dominated.

General symptoms of overtraining

Overtraining is usually multifactorial and manifests itself in changes to various organic subsystems. These can include cardiocirculatory, metabolic, hormonal, and immunological changes, or impairments of the autonomic nervous system^{13,14}. Above all, however, problems with the musculoskeletal system can also occur with symptoms of overtraining. In addition to changes in organic subsystems, psychological changes can also occur. These signs are often the first to appear when overload begins³. As for the symptoms of overtraining, stagnation and a decline in performance are always the main symptoms. Fatigue, heavy legs, or signs of depression are often ignored until performance is permanently reduced for an extended period^{4,12}.

The diagnosis of overtraining is made based on several objective parameters and forms the basis for the decision of sports physicians,

sports scientists, trainers and therapists as to whether and to what extent training loads should be reduced.

Clinical symptoms

The most important and easily recognizable symptom is a stagnation or decline in athletic performance. Coordination deficits are particularly noticeable, which massively increase the risk of injury. The drop in VO_2max is not an early symptom.

There is a reduced subjective perception of chronic fatigue with persistently high fatigue ratings, triggered by abnormal sleep-wake rhythms caused by reduced secretion of growth hormone (GH) and melatonin. Persistent muscle soreness is present, and muscle and tendon tears, as well as stress fractures, can subsequently occur. Athletes also complain of loss of appetite and eating disorders, resulting in weight loss (Relative Energy Deficiency in Sport RED-S). The persistent stress leads to loss of libido and, in women, changes or cessation of the menstrual cycle. However, the prolonged absence of menstruation can also lead to severe disturbances in bone metabolism, possibly leading to osteoporosis. Psychologically, overtraining can lead to nervousness, emotional instability, burnout, depression and the risk of suicide!^{2,12}.

Biochemical changes

- Decreased hemoglobin levels, as well as serum iron and ferritin (consumption, intestinal absorption, inadequate nutrition).
- Negative nitrogen balance - catabolism (urea, uric acid).
- Decreased glutamine levels (leukocytes, muscle, intestine).
- Increased creatine kinase levels.
- Trace element deficiencies (Zn, Co, Al, Mn, Se, Cu, etc.).
- Lack or only a slight increase of stress hormones after exercise due to dysfunction of the Hypothalamic-pituitary-adrenal axis (HPA).
- Low free testosterone (catabolism).
- Decrease in the ratio of free testosterone to cortisol.

Cellular changes

The disruption of the stress axis (HPA) with reduced adrenal sensitivity to ACTH and increased pituitary sensitivity to Growth Hormone Releasing Hormone (GHRH) for Growth Hormone GH release leads to a regeneration deficit with:

- Increased beta-adrenoreceptor density with hypersensitivity to catecholamines and cellular stress.
- Decreased intrinsic sympathetic activity with impaired cellular function (reduced cell regeneration).
- Activation of intracellular protective mechanisms through the synthesis of cell structure-stabilizing heat shock proteins (HSP 70), which compensate for overload-induced cellular damage.

Immunological changes

High levels of stress hormones (cortisol) and cytokines also lead to increased susceptibility to infections, including:

- Increased susceptibility and severity of bacterial infections.

- Reactivation of herpes virus infections.
- Decreased neutrophil function (bacterial infections).
- Decreased lymphocyte counts (viral infections).
- Decreased immunoglobulin production and secretion (viral and bacterial infections).

Cardiovascular changes

Cardiovascular changes are characterized by abnormally high sympathetic tone:

- Increased resting heart rate (HR) and decreased maximum heart rate.
- Increased heart rate and blood pressure.
- "Systemic Stress" with deterioration of heart rate variability HRV.

Changes in performance

The causes of the deterioration in performance lie in the catabolic-anabolic imbalance due to a lack of regeneration, triggered by endocrine Pathomechanisms^{5,12,13}:

- Reduced neuromuscular excitability due to inhibition of alpha motor neuron activity, resulting in reduced reaction speed (increased risk of injury).
- Fatigue of muscular structures, resulting in reduced reaction speed and coordination deficit (increased risk of injury).
- Reduced adrenal sensitivity to ACTH due to increased cortisol secretion (regeneration deficit).
- Increased sensitivity of the pituitary gland to growth hormone releasing hormone (GHRH) for growth hormone GH release, resulting in a counter-regulatory shift in the anabolic, endocrine effect and a regeneration deficit. Overall, this leads to a reduced "stress response" and a deterioration in performance (regeneration deficit).

Preventive measures against overload, overtraining and injuries

To prevent overload, overtraining, injuries, and dropouts in young athletes, appropriate measures must be taken to detect overuse syndromes early. These must be suitable so that negative consequences can be prevented. Documented, standardized load measurements are particularly important for this purpose. The focus is on a training plan adapted to individual performance (VO_2max)^{5,9,11,15-17}.

External stress measurement

- Training diary (athlete).
- Power meters (time, watts, km/h, kg).
- Time-motion analyses (h/wk).
- Functional testing (Ergometry).
- Sport-specific field tests.

Internal stress measurement

- Perception of exertion, e.g., BORG Scala.
- Heart rate (resting HR, maximum HR, training HR).

- Training impulse TRIM.
- Sleep measurement, autonomic nervous system ANS (HR, HRV, Respiration).
- Oxygen uptake (VO₂ ml/min/kg), Watts/kg.
- Neuromuscular function (sports motor tests, force plate).
- Biochemical data (Lactate, CPK, HST, Hb, Fe).
- Hormonal data (Cortisol, Testosterone) - specific indication for OTS
- Immunological data (CRP, Ferritin, IL-6, IL-8) - specific indication for OTS.

Bibliography

1. Enoksen E. Drop-out rate and drop-out reasons among promising Norwegian track and field athletes: a 25-year study. *Scandinavian Sport Studies Forum*. 2011;2:19-43.
2. Israel S. Zur Problematik des Übertrainings aus internistischer und leistungsphysiologischer Sicht. *Medizin und Sport*. 1976;16:1-12.
3. Güllich A, Michael M. *Sport–Das Lehrbuch für das Sportstudium 2022*; Hrsg. Springer Spektrum.
4. Fendl, V. Das Entstehen von Übertraining. *Stresssyndrome im Sport*. München. GRIN Verlag. 2019; 9783346237712 (ISBN).
5. Ithaca EJ. *Toward an understanding of human performance movement*. Publications N.Y. 1977.
6. Pichot V, Busso T, Roche F, Garet M, Costes F, Duverney D, *et al*. Autonomic adaptations to intensive and overload training periods: a laboratory study. *Med Sci Sports Exerc*. 2002;34(10):1660-6.
7. Budgett, R. Overtraining Syndrome. *Br J Sp Med*. 1990;4:231-6. doi: 10.1136/bjism.24.4.231
8. Heck BVH, Bartmus U. *Sportphysiologie*. Sportverlag Strauß; 2017..
9. Vogel R. Übertraining. Begriffserklärungen, ätiologische Hypothesen, aktuelle Trends und methodische Limits. *Schweizerische Zeitschrift für Sportmedizin und Sporttraumatologie*. 2001;49:154-162.
10. Cheng AJ, Jude B, Lanner JT. Intramuscular mechanisms of overtraining Redox. *Biology*. 2020;35:101480.
11. Heikura IA, Uusitalo ALT, Stellingwerff T, Bergland D, Mero AA, Burke LM. Low energy availability is difficult to assess but outcomes have large impact on bone injury rates in elite distance athletes. *Int J Sport Nutr Exerc Metab*. 2018;28:403-411.
12. Lehmann MJ, Lormes W, Opitz-Gress A, Steinacker JM, Netzer N, Foster C, *et al*. Training and overtraining: an overview and experimental results in endurance sports. *J Sports Med Phys Fitness*. 1997;37:7-17.
13. Halson S, Jekendrup A. Does overtraining exist? an analysis of overreaching and overtraining research. *Sports Med*. 2004;34:967-81.
14. Uusitalo AL, Tahvanainen KU, Uusitalo AJ, Rusko HK. Non-invasive valuation of sympathovagal balance in athletes by time and frequency domain analyses of heart rate and blood pressure variability. *Clin Physiol*. 1996;16:575-88.
15. Hedelin R, Wiklund U, Bjerle P, Henriksson-Larsén K. Cardiac autonomic imbalance in an overtrained athlete. *Med Sci Sports Exerc*. 2000;32:1531-3.
16. Uusitalo AL, Uusitalo AJ, Rusko HK. Endurance training, overtraining and baroreflex sensitivity in female athletes. *Clin Physiol*. 1998;18:510-20.
17. Urhausen A, Kindermann W. Diagnosis of overtraining: what tools do we have? *Sports Med*. 2002;32:95-102.

Analizador Instantáneo de Lactato Lactate Pro 2

arkray
LT-1730

- Sólo 0,3 µl de sangre
- Determinación en 15 segundos
- Más pequeño que su antecesor
- Calibración automática
- Memoria para 330 determinaciones
- Conexión a PC
- Rango de lectura: 0,5-25,0 mmol/litro
- Conservación de tiras reactivas a temperatura ambiente y
- Caducidad superior a un año



Importador para España:



c/ Lto. Gabriel Miro, 54, puerta 9
46008 Valencia. Tel: 963857395
Móvil: 608848455 Fax: 963840104
info@bermellelectromedicina.com
www.bermellelectromedicina.com

 Bermell Electromedicina

 @BermellElectromedicina

 Bermell Electromedicina



Monografías Femede nº 12
Depósito Legal: B. 27334-2013
ISBN: 978-84-941761-1-1
Barcelona, 2013
560 páginas.



Dep. Legal: B.24072-2013
ISBN: 978-84-941074-7-4
Barcelona, 2013
75 páginas. Color



Índice

Foreward
Presentación
1. Introducción
2. Valoración muscular
3. Valoración del metabolismo anaeróbico
4. Valoración del metabolismo aeróbico
5. Valoración cardiovascular
6. Valoración respiratoria
7. Supuestos prácticos
Índice de autores

Índice

Introducción
1. Actividad mioeléctrica
2. Componentes del electrocardiograma
3. Crecimientos y sobrecargas
4. Modificaciones de la secuencia de activación
5. La isquemia y otros indicadores de la repolarización
6. Las arritmias
7. Los registros ECG de los deportistas
8. Términos y abreviaturas
9. Notas personales

Información: www.femede.es