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## Importance of fatiguing, overtraining and chronic fatigue in athletes

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### Abstract

Each training that requires achieving a higher heart rate limit and/or increased concentration in time may cause fatigue, considered to be a natural defence mechanism of a body. In the case of excessive fatigue and insufficient time designated for rest and regeneration, an overtraining syndrome (*OTS*) may develop. The main symptom of overtraining is increased fatigue that fails to become reversed in normal conditions of regeneration. Although authors

are familiar with the overtraining syndrome and associated symptoms, as of today no diagnostic tool has been developed that may form a basis for a final diagnosis, and the diagnosis itself is frequently based on a subjective assessment of the athlete. Possible causes of the band overtraining syndrome are disorders of sodium, inflammatory processes resulting from physical activity and / or disorders of the autonomic nervous system.

**Key words:** OTS, elite sportsmen, supercompensation

### **Introduction**

When performing motor tasks associated with numerous popular sports, the athlete's body is exposed to enormous loads, with muscles generating a significant force and having an impressive power. Other disciplines, particularly speed and/or technical sports, acquire the maximum intensity by multiple repetitions of exercises. Contemporary professional sports are characterised by periodisation of load during the season; however, due to the ease of travelling between continents, the period of rest between starts is shortened, and in extreme cases, eliminated. Each training that requires achieving a higher heart rate limit and/or increased concentration in time may cause fatigue, considered to be a natural defence mechanism of a body. Unfortunately, there are very few available talking about OTS incidence. Among adult athletes, there is a high variability of occurrence depending on the discipline. In the studies evaluating individual seasons the percentage was lower ( $\pm 10\% - 20\%$ ). The higher rates (60%) were related to the evaluation of the entire sports career. In addition, a positive association was observed with OTS with training period.

## Data source

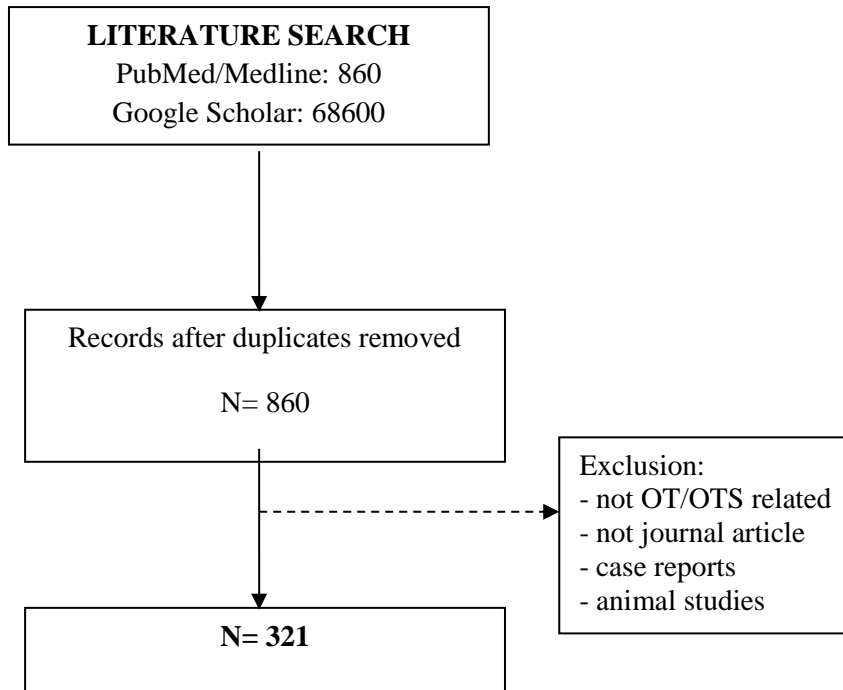


Figure 1. Flowchart of database

Fatigue following a single training is classified as either peripheral or central, on the basis of the structures involved. Peripheral fatigue is associated with the reduced muscle function to generate power and/or speed of the contraction, caused by previous activity. Two components are distinguished here: metabolic fatigue associated with energy conversion processes in the muscle tissue and a reduction in the concentration of energy substrates (phosphocreatine, muscle glycogen) and an increase in the concentration of side products (ADP,  $\text{NH}_3$ , inorganic phosphorus, AMP), and therefore a disruption in ATP resynthesis and non-metabolic fatigue, associated with mechanical damage to actin-myosin connections, particularly in exercises based on multiple repetition of a specific movement. Peripheral fatigue does not have to be uniform in muscles involved in physical activity; in fact, it may vary in the individual fibres of one muscle. It is associated with a theory of hierarchical recruitment of muscle fibres and their specialisation as fast or slow contracting units [12, 15]. Additionally, within 6–12 hours, and up to 24–48 hours after the end of the exercise, a delayed onset of muscle soreness (*DOMS*) occurs, resulting from damages to the morphological structures of the muscle following an intense physical exercise [37]. Central fatigue is possibly located at

supramedullary (changes occurring in the motor cortex and efferent pathways supplying a motoneuron) and medullary (changes in function of muscle receptors and motoneurons) levels [17, 18]. Considering the structure and interactions between centres located close to each other in the motor cortex and responsible for individual body parts, fatigue may spread to centres where the motor potential did not increase, but which are located sufficiently close [17]. Depending on exercise intensity, central and peripheral fatigue occurring after a single training may manifest itself already during the exercise and up to several dozen hours after the end of the training. The commonly applied training model requires exceeding the capacity of the body to adapt itself to physical exercise, thus leading to overload (*OL*) associated with peripheral and central fatigue within the scope of physical exercise physiology. This approach may temporarily deteriorate the achieved results, but combined with sufficient rest and biological regeneration, results in the supercompensation effect (Fig. 1), allowing an athlete to achieve higher performance than at a baseline [21]. The aim of the training is to achieve the best performance within an optimum time. However, when load is not reduced at the moment of *OL*, and the athlete does not spend more time on rest and treatment associated with biological regeneration, functional overreaching (*FO*) may occur during the overload period, characterised by deteriorated sports performance without losing the athlete's readiness for training. A difference between *OL* and *FO* is based on the fact that the former is an element intended to lead to supercompensation, while the latter may disrupt the training microcycle and prolong the period of rest [21, 32]. When *FO* is not captured on time, it may lead to non-functional overreaching (*NFO*). This condition may result in a deterioration of performance and reduced readiness, and in extreme situations affect the athlete's ability to train. It may possibly be caused by exhausting the regeneration capacity of the body, characterised by dominance of catabolic processes over anabolic ones, mainly in the muscle tissue. In the case of excessive fatigue and insufficient time designated for rest and regeneration, an overtraining syndrome (*OTS*) may develop (Figure 2).

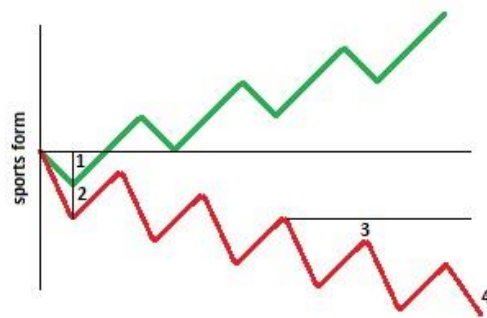


Figure 2. The green line represents a correctly managed macrocycle with an increase in performance, where 1 means OL, and the red line represents incorrect management of the load, where 2 means FO, 3 – NFO, and 4 – OTS.

The meaning of OTS was first regulated at the European College of Sports Science in 2006 [31]. Overtraining is a condition of chronic reduction in the body performance and capacity to train [18]. Restoration of normal adaptation and reversal of changes associated with overtraining may take several weeks, months or, in extreme cases, even years [1, 19]. The main symptom of overtraining is increased fatigue that fails to become reversed in normal conditions of regeneration. Other symptoms are presented in Table 1.

### **OTS diagnosis**

Although authors are familiar with the overtraining syndrome and associated symptoms, as of today no diagnostic tool has been developed that may form a basis for a final diagnosis, and the diagnosis itself is frequently based on a subjective assessment of the athlete. A range of biomarkers have been described that may be useful for the OTS diagnosis (Table 2); however, none of them meets the following criteria:

- objective and sensitive, independent of the current training regimen and other factors, such as diet;
- results reflecting the OTS progress;
- easy to measure and based on the theory for OTS development.

However, other disorders with similar symptoms should be excluded. They include anaemia, magnesium deficiency, autoimmunological diseases, infectious diseases (e.g., Lyme disease), muscle damage, endocrine disorders, dysautonomia, depression, allergies and cardiovascular diseases.

Symptoms of overtraining
<ul style="list-style-type: none"> <li>- persistent fatigue</li> <li>- deteriorated sports performance</li> <li>- infections of the upper respiratory tract</li> <li>- persistent muscle and joint aches</li> <li>- increased heart rate at rest</li> <li>- increased blood pressure at rest</li> <li>- sleep disorders</li> <li>- irritation or depression</li> <li>- neurohormonal changes</li> <li>- intense stress</li> <li>- lower motivation</li> <li>- gastrointestinal disorders</li> <li>- lower libido</li> <li>- reduced concentration</li> <li>- lethargy, apathy</li> <li>- lower appetite</li> <li>- reduced ability for maximum oxygen uptake</li> </ul>

Table 1. Symptoms of overtraining [ 9, 10, 11, 12, 13, 22, 39].

### **Immune system**

The functioning of the innate and the adaptive immune systems is associated with the production of various types of cytokines, whose action may be briefly classified as initiating or inhibiting the inflammatory process. Research conducted by Smith et al. showed that overload and musculoskeletal injuries result in the production of pro-inflammatory cytokines and development of disease response and/or the described overtraining syndrome [38]. The main cytokines having an inflammatory effect and associated with overload are: interleukin-1 beta (IL-1 $\beta$ ), interleukin-6 (IL-6) and tumour necrosis factor (TNF –  $\alpha$ ) (Figure 3). Studies of other authors indicate mediators of inflammatory processes, and IL-6 in particular, as crucial factors in many OTS symptoms, thus suggesting a way for diagnosing OTS [20, 26].

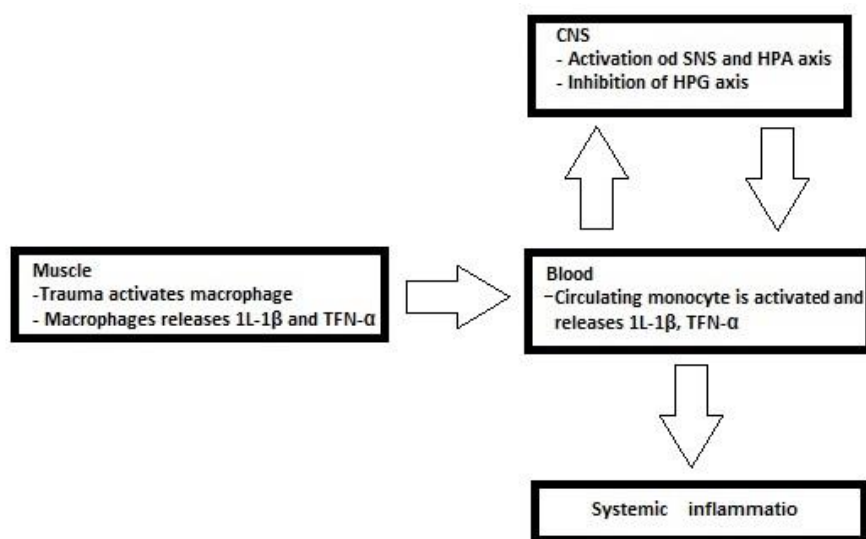


Figure 3. Mechanism underlying the muscle damage influence on triggering infection [12].

### Autonomic nervous system

The autonomic nervous system (ANS), divided into the sympathetic (thoracolumbar) and parasympathetic (craniosacral) sections, regulates the autonomic, unconscious control of the internal organs. According to current knowledge, ANS not only innervates the internal organs, but, more importantly, regulates and integrates their function, including the secretion function of the endocrine system, thus maintaining dynamic balance – homeostasis – in response to changing internal and external conditions. Physical exercise influences the ANS control over the cardiovascular system. Already in the first seconds of the athlete’s exercise, there is a rapid reduction in the vagus nerve tension (parasympathetic effect), leading to increased heart rate (HR). Additionally, physical exercise is accompanied by sympathetic contraction of the vascular bed, influencing control over blood pressure (BP) but not affecting the muscles involved. A significant regulatory role is attributed to the function of baroreceptors (BRS) located in carotid arteries. When physical exercise is stopped, the parasympathetic section of ANS is activated, responsible for HR and BP reduction, and therefore for restoring a balance between antagonistic parts of ANS. It is believed that the overtraining syndrome has an adverse influence on the autonomic control over the cardiovascular system [23]. It is assumed that at the initial stage of OTS there is increased activity of the sympathetic nervous system, and – in more advanced cases – the sympathetic nervous system is then inhibited and the dominant role is undertaken by the parasympathetic section [8]. Yann Le Meur and colleagues

in their evaluation of parasympathetic hyperactivity in the FOR syndrome analysed frequency parameters of the heart rate variability (HRV), confirming the above hypothesis [28]. Amongst other things, HRV serves to reflect the antagonistic mechanisms of autonomic control over cyclically occurring RR intervals between normal QRS complexes. HRV parameters reflect the influence of the autonomic nervous system on the sinus node, and interactions between sympathetic and parasympathetic systems, allowing quantification of that influence [3]. Studies of Baumert and colleagues, aiming at the evaluation of vagal autonomic function in subjects exposed to intense physical exercise by monitoring heart rate (HR), blood pressure (BPV) and baroreceptor sensitivity (BRS), showed that HR and BRS reflect the autonomic nervous system activity in response to intensified training, and therefore may be considered an indicator of OTS [2, 25]. Leti and Bricout noted that the initial increased sympathetic activity after training allows restoring homoeostasis [29].

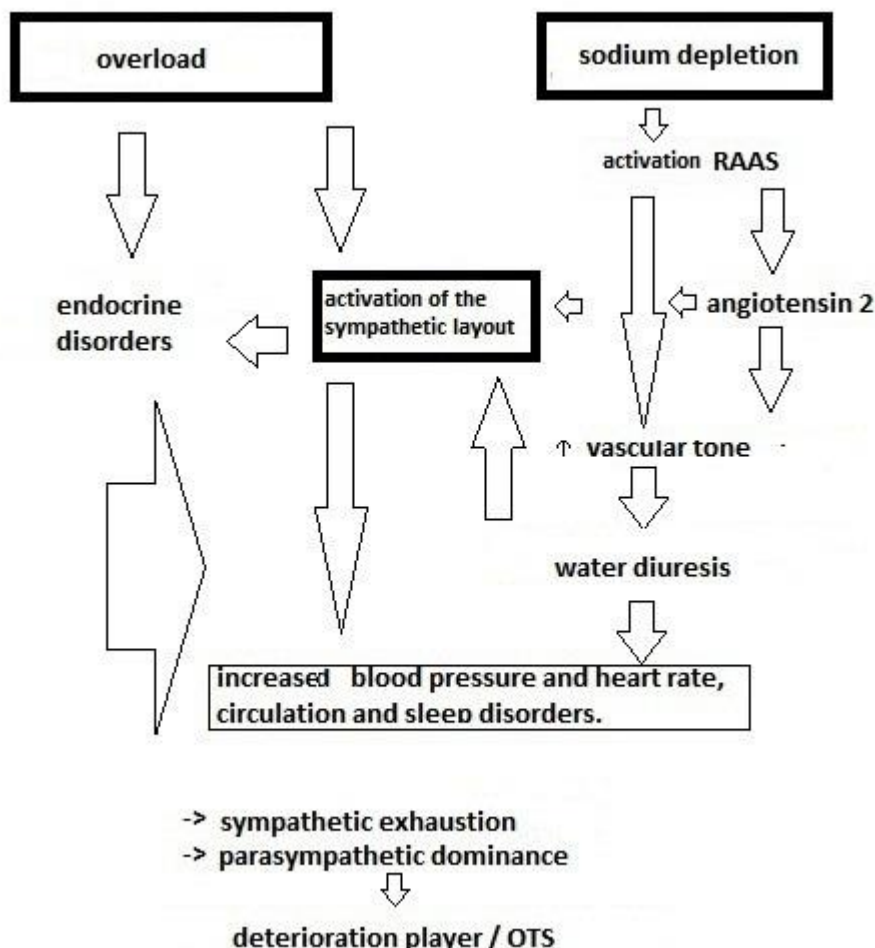


Figure. 4. Review of OR and hyponatremia pathophysiology, taking into account the reduced activity of the sympathetic system. RASS: the renin-angiotensin-aldosterone system [34].



## **Hyponatraemia**

During intense sports training, the excessive sweating results in a significant loss of sodium ions. Popular regimens for biological regeneration – sauna, jacuzzi and swimming in a pool – also contribute to sodium excretion. Additionally, an increasingly popular diet based on fresh ingredients where salt is considered to be a necessary evil, may result in deficits of that element in the athlete's body. Supplementation used by athletes is usually based on carbohydrates, proteins, iron, magnesium or calcium, while sodium is omitted. Symptoms and the underlying mechanism of hyponatraemia pathophysiology resemble those in OR/OTS (Figure. 4) [36]. Blank et al. noted that sodium supply was reversely correlated with OTS symptoms intensity. The symptoms developed several days before the threshold decrease occurred in serum sodium levels. Sleep disorders belonged to significant symptoms of reduced sodium levels. An increased sodium supply without changes in the training load resulted in a weakening of OTS symptoms. In subjects whose diet was supplemented with  $\text{Na}^+$ , symptoms of overtraining did not develop, even after a significant increase in the load [34].

Table 2. Biomarkers that may be used in OR/OTS [34].

Marker	Use in OTS	Direction for research
Mood profile (POMS)	deteriorated mood	Correlation between mood changes and other measures
VO <sub>2</sub> max HR max	↓ parameters	Correlation with other measures describing work performance
Lactates	Reduction in the anaerobic threshold	Levels of glycogen stored in muscles and a liver should be considered
Creatinine kinase iron, urea	Possibility to differentiate OL, FO, NFO from OTS	Confirmation of results
Depletion of glycogen	Basic OR marker	Analyse glycogen levels in diagnosed OTS case
Leukocytosis	In evaluation of acute responses to intense exercise	Are changes characterised by cell redistribution
Neutrophils	Measurement of increase in neutrophil distribution	The tests did not evaluate function in in OR or OTS
IgA in saliva	A marker of mucosal integrity	Evaluate mucosal resistance to bacterial infections OR/OTS
Glutamine	An immune system performance factor	Determine glutamine role in OR/OTS
Cortisol, testosterone	Max. increase in post-exercise levels of cortisol is reduced in OTS	Determine the cortisol value as an OTS marker, determine usefulness of testosterone/cortisol ratio
ACTH	Differences in tests determining plasma hormone levels	Research aiming at its use as OTS marker
Catecholamines	Urine levels drop in OR	Tests aiming at their use as biomarkers
HRV, BRS	Evaluation of the autonomic nervous system influence on the cardiovascular system	Research conducted in fully controlled conditions using standardised methods for HRV and BRS measurements showing a relation with OR or OTS

## Discussion

So far, the mechanisms underlying OTS and methods for its objective diagnosis have not been fully explored. A very important element is the periodisation of trainings and capturing FO and NFO as early as possible so as to prevent the development of the full range of OTS symptoms. To reduce the risk of chronic fatigue, it is recommended to cyclically perform an ergospirometry test before a preparatory mesocycle, and later, as necessary. This way the maximal oxygen uptake, the anaerobic threshold, the AT point and the maximum heart rate limit are determined [27]. Knowledge gained this way will allow to control loads as precisely as possible. Also, knowing mechanisms underlying peripheral and central fatigue, the coach, in cooperation with the physiologist, is able to control loads quite precisely. The authors are of the opinion that peripheral fatigue and OL may be monitored using the creatinine kinase

(CK) test, reflecting the range of non-metabolic peripheral fatigue, thus easily providing information whether an athlete has fully recovered and is ready for another training unit [27]. Modulators that may be used by the coach in charge are also worth mentioning. They include the time, the mean heart rate limit during the exercise, and complexity of sport techniques. Lactic acid measurements conducted before and immediately after the exercise, and then at an interval that clearly indicates a decreasing tendency towards the baseline values, are used for optimal measuring of load and normal progress of regeneration processes during a training [6]. Although some authors are of the opinion that control over training does not result in a better performance [35], yet it is highly possible that it may eliminate the risk of the chronic fatigue syndrome. Other authors point to the method of 24 h HRV measurement as helpful in determining a possibility for OTS development, indicating a reduced flexibility of the heart rate adjustment in subjects with OTS or at risk of OTS development [35]. If the OTS syndrome is associated with a dysfunction of the autonomic nervous system in response to repeated physical exercise, it would be interesting to determine whether this fact influences the circadian rhythm and/or external parameters which, in authors opinion, significantly impact the functioning of the sympathetic and the parasympathetic systems [33,40]. Another interesting aspect is a correlation between OTS and the Chronic Fatigue Syndrome (CFS), as opinions on CFS aetiology have changed significantly. It was suggested that CFS may have genetic, immunologic or psychological background, or may be caused by hormonal disorders or viral infections [4]. Recently, discussion focuses on dysfunctions of the autonomic nervous system, similarly as in OTS, and on neuroinfections [4, 24]. Symptoms described in CFS include sore throat, enlarged lymph nodes, muscle and joint aches, non-localised headaches of nature other than disease-related headaches, memory and concentration loss, and sleep disorders [30]. OTS symptoms can be noted in sportsmen in whom the capacity of the body to adapt to specific training conditions was exceeded. A question may be asked whether CFS develops in people who exhausted adaptation abilities of their body, similarly as in OTS, where all symptoms result from disrupted homeostasis.

## References

1. Armstrong LE, van Heest JL. The unknown mechanism of the overtraining syndrome: clues from depression and psychoneuroimmunology, *Sports Med*, 2002; 32: 185-209,
2. Baumert M, Brechtel L, Lock J, Hermsdorf M, Wolff R, Baier V, Voss A. Heart Rate Variability, Blood Pressure Variability, and Baroreflex Sensitivity in Overtrained Athletes. *Clin J Sport Med.*, 2006; 16: 412–417

3. Bellenger CR, Fuller JT, Thomson RL, Davison K, Robertson EY, Buckley JD. Monitoring Athletic Training Status Through Autonomic Heart Rate Regulation: A Systematic Review and Meta-Analysis. *Sports Med.* 2016 Oct;46(10):1461-86
4. Bitner A, Klawe JJ, Zalewski P, Tafil-Klawe M. Etiology of chronic fatigue syndrome with reference to the autonomic nervous system, *ProblHigEpidemiol*, 2013, 94(1): 6-8
5. Blank M, Bedarf J, Russ M, Grosch-Ott S, Thiele S, Unger J. Total body Na<sup>+</sup>-depletion without hyponatraemia can trigger overtraining-like symptoms with sleeping disorders and increasing blood pressure: Explorative case and literature study. *Medical Hypotheses*, 2012; 79: 799-804
6. Bonen A, Belcastro AN. Comparison of self-selected recovery methods on lactic acid removal rates. *Medicine and Science in Sports*, 1976; 8(3):176, -178
7. Brancaccio P, Maffulli N, Limongelli FM. Creatine kinase monitoring in sport medicine. *Br Med Bull.* 2007; 81-82: 209-30
8. Brechtel LM, Braumann KM, Wolff R. Time course of symptoms during the development of a parasympathetic overtraining syndrome. *Med Sci Sports Exerc.* 1999;31:176.
9. Brukner P, Khan K. *Clinical Sports Medicine*, dbpublishing, 875-885; 2012
10. Budgeh R, Newsholme E, Lehmann M, Sharp C, Jones D, Jones T, Peto T, Collins D, Nerurkar R, White P. Redefining the overtraining syndrome as the unexplained underperformance syndrome. *British Journal of Sports Medicine*. 2000; 1: 67-68
11. Budgett R. Fatigue and underperformance in athletes: the overtraining syndrome. *Br J Sports Med*, 1998; 32 (2): 107-10
12. Carfagno DG, Hendrix JC, Joshua C. Overtraining Syndrome in the Athlete: Current Clinical Practice, *Current Sports Medicine Reports*, 2014; 13: 45-51
13. Celichowski J. Motor units of medial gastrocnemius muscle in the rat during the fatigue test. 11. Changes in the time course of sequential tetani of fatigue test. *Acta Neurobiol. Exp.* 1992, 52: 99-111
14. Cosca DD, Navazio F, Common problems in endurance athletes. *American Family Physioan.* 2007; 76: 237-44.
15. De Luca corresponding C., J, Contessa P. Hierarchical control of motor units in voluntary contractions. *J Neurophysiol.* 2012; 107(1): 178-195.
16. Donatelli R: *Sports-Specific Rehabilitation*, Churchill Livingstone Elsevier, 109-125, 2007
17. Duchateau J, Hainaut K. Effects of immobilization on contractile properties, recruitment and firing rates of human motor units. *J Physiol.* 1990; 422: 55-65.

18. Freude G, Ullsperger P, Eggert S, Ruppe I. Effects of microwaves emitted by cellular phones on human slow brain potentials. *Bioelectromagnetics*. 1998; 19(6): 384-7.
19. Fry AC, Kraemer WJ. Resistance exercise overtraining and overreaching: neuroendocrine responses, *Sports Med*. 1997; 23: 106-129,
20. Hackney AC. Clinical Management of Immuno-Suppression in Athletes Associated with Exercise Training: Sports Medicine Considerations. *Acta Medica Iranica*, 2013; 51(11): 751-756.
21. Halson SL, Jeukendrup AE.: Does overtraining exist? An analysis of overreaching and overtraining research. *Sports MED.*, 2004; 34(14): 967-981
22. Hausswirth Ch, Louis J, Laubry A, Bonnet G, Duffield R, Le Meur Y. Evidence of Disturbed Sleep and Increased Illness in Overreached Endurance Athletes. *Medicine and science in sports and exercise*, 2013; 1036-1045
23. Hedelin R, Wiklund U, Bjerle P, Henriksson-Larsen K. Cardiac autonomic imbalance in an overtrained athlete. *Med Sci Sports Exerc*. 2000; 32: 1531-1533.
24. Institut of medicine, Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome, Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness, Redefining an Illness, 141-179. 2015
25. Kiviniemi AM, Tulppo MP, Hautala AJ, Vanninen A, Uusitalo LT. Altered relationship between R-R interval and R-R interval variability in endurance athletes with overtraining syndrom. *Scand J Med Sci Sports* 2014; 24: e77–e85
26. Main L, Dawson B, Grove R, Landers G. Monitoring training distress: changes in perceived stress and inflammatory cytokines, *Sports Medicine An International Journal*. 2009; 17(2): 121-32.
27. Lambert MI, Borresen J. Measuring Training Load in Sports, *International Journal of Sports Physiology and Performance*, 2010; 5: 406-411
28. Le Meur Y, Pichon A, Schaal K, Schmitt L, Louis J, Gueneron J, Vidal PP, Hausswirth Ch. Evidence of Parasympathetic Hyperactivity in Functionally Overreached Athletes. *American College of Sports Medicine*. 2013.
29. Leti T, Bricout VA. Interest of analyses of heart rate variability in the prevention of fatigue states in senior runners, *Autonomic Neuroscience: Basic and Clinical*, 2013; 173: 14-21
30. McGough A. How to care for patients with chronic fatigue syndrome/ME. *Nurs Times* 2011; 107(40):16.

31. Meeusen R, Duclos M, Gleeson M, Rietjens G, Steinacker J, Urhausen A. Prevention, diagnosis and treatment of the overtraining syndrome: ECSS position statement ‘Task Force.’ *Eur J Sport Sci*, 2006; 6: 1-14.
32. Nederhof E, Lemmink K, Visscher C. Meeusen R., Mulder T. Psychomotor Speed Possibly a New Marker for Overtraining Syndrome, *Sports Med* 2006; 36 (10): 817-828L
33. Pawlak J, Pawlak B, Zalewski P, Klawe JJ, Zawadka M, Bitner A. Praca zmianowa a powstawanie chorób układu sercowo-naczyniowego w kontekście regulacji normatywnej. *Hygeia Public Health* 2013, 48(1): 6-9
34. Purvis D, Gonsalves S, Deuster PA. Physiological and Psychological Fatigue in Extreme Conditions: Overtraining and Elite Athletes, *PM R* 2010; 2: 442-450
35. Schumann M, Botella J, Karavirta L, Häkkinen K. Training Load-Guided Versus Standardized Endurance Training in Recreational Runners. *Int J Sports Physiol Perform*. 2016
36. Sharp RL. Role of sodium in fluid homeostasis with exercise. *J Am Coll Nutr* 2006; 25(Suppl. 3): 231S–9S.
37. Singla N, Desjardins PJ, Cosca EB, Parulan C, Arriaga A, Poole KC, Batz DM, Chang PD. Delayed-onset muscle soreness: a pilot study to assess analgesic study design features. *Pain*. 2015; 156(6): 1036-1045.
38. Smith, LL. Tissue trauma: the underlying cause of the overtraining syndrome? *J Strength Cond Res*. 2004; 18(1): 185-93.
39. Wolanin A, Gross M, Hong E. Depression in Athletes: Prevalence and Risk Factors, *Current Sports Medicine Reports*. 2015; 14: 56-60
40. Zalewski P, Bitner A, Słomko J, Sznajder J, Klawe JJ, Tafil-Klawe M, Newtonc JL. Whole-body cryostimulation increases parasympathetic outflow and decreases core body temperature. *Journal of Thermal Biology*. 2014; 45: 75-80