



The Overtraining Syndrome: A Meta-Analytic Review

Frank B. Wyatt¹, Alissa Donaldson¹, Elise Brown²

¹Midwestern State University, Department of Athletic Training and Exercise Physiology, Wichita Falls, Texas, USA, ²The University of the West of Scotland, Institute of Exercise and Health Science, Hamilton, Lanarkshire, Scotland.

ABSTRACT

Wyatt FB, Donaldson A, Brown E. The Overtraining Syndrome: A Meta-Analytic Review. *JEPonline* 2013;16(2):12-23. The overtraining (OT) syndrome has been studied extensively with little agreement as to reliable markers for detection. The purpose of this meta-analysis was to provide summary quantitative findings of markers (e.g., blood, physiological, and psychological) associated with the OT syndrome. A meta-analytic research design was utilized to investigate selected studies allowing for a coding process to record data. Thirteen studies met the inclusion/exclusion criteria. Markers included samples taken with the subjects in the normal (N) condition and during the OT. The biomarkers consisted of glutamine (μm), glutamate (μm), cortisol ($\text{nmo}\cdot\text{L}^{-1}$), IL-6 (nm), testosterone ($\text{mg}\cdot\text{dL}^{-1}$), total cholesterol ($\text{mg}\cdot\text{dL}^{-1}$), glucose ($\text{mg}\cdot\text{dL}^{-1}$), leptin ($\text{ng}\cdot\text{mL}^{-1}$), hematocrit (%), hemoglobin ($\text{g}\cdot\text{L}^{-1}$), norepinephrine ($\text{pg}\cdot\text{mL}^{-1}$), epinephrine ($\text{pg}\cdot\text{mL}^{-1}$), and creatine kinase ($\text{u}\cdot\text{L}^{-1}$). Cardiovascular variables included resting heart rate (RHR, $\text{beats}\cdot\text{min}^{-1}$), and resting diastolic and systolic blood pressure (DBP and SBP, mmHg). Psychological measures included tension, anger, fatigue, confusion, depression, vigor, sleep, stress, and self-perceptions of physical status. Selected variables (i.e., anger, depression, etc.) were noted in terms of direction (+, -) of change in the OT state compared to the N state. To determine magnitude of difference between N and OT, the effect size calculation of $M_2 - M_1 / SD_1$ was used where M_2 is the mean of the OT sample, M_1 was the mean of the N sample and SD_1 is the standard deviation of the N sample. Combined sample size (N) was 238 subjects with the mean time in OT of 6.6 wks. The following are mean (SD) of the combined subject demographics: height (cm) 175.4 (2.4); weight (kg) 71.7 (2.6); body fat (%) 11.8 (0.9); age (yrs) 23.5 (2.03); VO_2 max ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) 55.4 (0.8). Mean (SD) biomarker changes from the N state to the OT state

were glutamine -56.3 (-2); glutamate 49.7 (2); cortisol -89.7 (-12.2); IL 6 -0.52 (0.12); testosterone -88.9 (-30); cholesterol 4.6 (-1.6); glucose -13.3 (1.9); leptin 0.15 (-0.11); hematocrit -0.83 (-0.4); hemoglobin -20; norepinephrine 36 (-4.1); epinephrine -2.2 (-3.5); creatine kinase 29.2 (8.5). Effect size calculations for the above biomarkers were considered large for the following: glutamine (-4.02), glutamate (8), cortisol, (-1.4), IL 6 (-5.2), glucose (-1.1). Mean (SD) cardiovascular changes and direction of change (+, -) were the following: RHR, -1.9 (-0.22); resting SBP, -2 (-2); resting DBP, 0 (1). Effect size calculations for the above measures were -0.95, -0.33 and 0, respectively. Three articles reported decreases in tension at OT, and one noted increases at MW. Fatigue increased at OT in 6 studies and showed no change in a separate study. Confusion did not change in two studies, increased at OT in another, and increased at MW and, then, declined at OT in a final article. Vigor reportedly remained stable in two studies and decreased in two other studies. Anger did not change in 2 articles, decreased in another, and increased in a different study with its peak at MW. There was no change in depression in three studies, but a decrease was reported in a separate article at OT with an increase at MW. Studies reported impaired sleep patterns, increased wakefulness, and decrements and stability in sleep quality. Two studies indicated increased levels of stress with one specifying stress related to training, sleep, and health. Findings showed a decreased perception of strength, decreased perception of recovery, and no change in perception of muscle soreness. From this analysis, the noted biomarker changes and direction of change (+, -) indicates considerable immune-suppression and increased stress with athletes experiencing the OT syndrome. A negative effect size for HR and SBP indicates questionable alterations from N to OT in subjects. Increased sympathetic and/or decreased sympathetic influence may be affected in the OT state. However, low effect size calculations allow for non-determinant conclusions related to cardiovascular indicators of the OT syndrome. Lastly, athletes in the OT state are likely to experience disturbances in sleep, self-perception, and mood factors.

Key Words: Biomarkers, Effect Size, Mal-adaptation

INTRODUCTION

Overtraining (OT) syndrome has been investigated extensively with little agreement as to reliable detection using non-invasive cardiovascular (CV) markers, biomarkers (i.e., blood) or physiological (i.e., maximal oxygen consumption), and psychological variables for detection. Yet, there seem to be many definitions describing the overtraining syndrome (OTS). In their study on OTS as a multi-contextual assessment, Meehan and colleagues (17) concur in their difficulties of defining OTS while stating that it is often characterized by reduced athletic performance (9). Armstrong and VanHeest (1) also acknowledged the complexities of diagnosing OTS (1). Commonalities in the athlete include a lack of recovery from training, reduced performance, and chronic maladaptive responses. In addition, they pointed out that objective biomarkers for identifying underlying mechanisms for OTS are lacking in the research literature.

In fact, Halson and Jeukendrup (9) questioned the validity of distinguishing between the OTS and overreaching. Their reasoning is specific to the multiple methodological errors in past research. Polman and Houlahan (24) distinguish between overtraining and overreaching as the first being a condition of excessive training and inadequate recovery leading to underperformance while the second is excessive training with adequate recovery enabling an athlete to reach a higher level of performance. These authors also suggest a misunderstanding in the literature related to adaptation, maladaptation, and questions pertaining to what is considered an adequate disruption of homeostasis (24). Further difficulties associated with detection of the OTS in athletes pertain to timelines. An

athlete may experience a loss of performance in a transient sense (i.e., overreaching) or as a result of OTS. Diagnosis then becomes problematic as to when the occurrence of OTS began and under what conditions (i.e., overtraining, under-recovery, inadequate diet, etc.)

The procedure of meta-analytic review is designed to compile studies in a specific area in hopes of reaching a consensus view (28). It differs from a general literature review in that the quantitative values of the reviewed studies are compiled to provide statistical review of the combined studies (28). In general, the findings of a specific topic from several studies allow for enhanced statistical power through the combined sample sizes of the included studies. This is especially true for studies in Exercise Physiology involving humans as subjects. Many studies in this venue are weak in sample size. There is also the difficulty in obtaining original research on the topic that fit the inclusion criteria. Of particular concern is the fact that many "Research Reviews" were done on the topic of OTS without the additional follow-up of quantitative analysis afforded by a meta-analysis. Studies that met the inclusion criteria reported biomarkers and psychological consequences of OTS, but only a few reported on performance variables such as maximal oxygen consumption ($\text{VO}_2 \text{ max}$), one repetition maximal (1 RM) strength, and power (watts) output changes. Thus, the purpose of this meta-analysis was to provide summary quantitative findings of non-invasive physiological measures, biomarkers (i.e., blood), and psychological aspects associated with the overtraining syndrome.

METHODS

A meta-analytic research design was used to investigate selected studies that allowed for a coding process to record data (28). Twelve studies met the inclusion/exclusion criteria, which included: (a) human studies; (b) peer-reviewed research; (c) determination of OTS; and (d) pre and post measures before and after OTS detection. Biomarkers included samples taken with subjects in normal (N) condition and during the OT state. They included glutamine (μm), glutamate (μm), cortisol ($\text{nmo}\cdot\text{L}^{-1}$), IL-6 (nm), testosterone ($\text{mg}\cdot\text{dL}^{-1}$), total cholesterol ($\text{mg}\cdot\text{dL}^{-1}$), glucose ($\text{mg}\cdot\text{dL}^{-1}$), leptin ($\text{ng}\cdot\text{mL}^{-1}$), hematocrit (%), hemoglobin ($\text{g}\cdot\text{L}^{-1}$), norepinephrine ($\text{pg}\cdot\text{mL}^{-1}$), epinephrine ($\text{pg}\cdot\text{mL}^{-1}$), and creatine kinase ($\text{u}\cdot\text{L}^{-1}$). Cardiovascular (CV) variables included measures taken with subjects in the normal (N) condition and during the OT condition. They included resting heart rate (RHR, $\text{beats}\cdot\text{min}^{-1}$), resting systolic blood pressure (SBP, mmHg), and resting diastolic blood pressure (DBP, mmHg). Recorded psychological variables included tension, anger, fatigue, confusion, depression, vigor, sleep, stress, and self-perceptions of physical status. The participants were measured during a normal (N) phase, midway phase (MW), and OT phase. In the review, selected variables (i.e., anger, depression, etc.) were noted in terms of direction (+, -) of change in the OT state compared to the N state.

Statistical Analyses

Means ($\pm\text{SD}$) were determined for descriptive data and group changes from N to OT. To determine the magnitude of difference between N and OT, the effect size was calculated using the following equation:

$$\sqrt{\frac{[\text{SD}_2^2(N-1) + \text{SD}_1^2(N-1)]}{(N+N-2)}}$$

where SD is the standard deviation of M_2 and M_1 , respectively and n = sample size. Effect size is a standardized value that determines the level of significance. Noted by Thomas and Nelson (2005), effect size significance is the following: Large = 0.8 or greater; Moderate = 0.5; Small = 0.2 (18).

Additionally, rates of change were compared for each variable (i.e., blood, physiological) for variance with an analysis of variance (ANOVA). Statistical significance was established *a priori* at $P < 0.05$.

RESULTS

The combined sample size of the 12 coded studies was $n = 238$. A summary of the 12 studies used for analysis can be seen in Table 1. Descriptive means (\pm SD) are in Table 2.

Table 1-A Summary of Coded Studies Utilized in the Meta-Analysis.

Study (Year)	Subjects	Training Period	Assessment
Maso et al. (2004)	25 international rugby players	15 hr·wk ⁻¹ plus one match a week for 2 mth. Training consisted of: running intervals, strength training with loads at 80-90% of 1 RM, technical training, and rugby specific training.	Investigation of cortisol and testosterone, using radioimmunological methods with saliva samples and French Society of Sports Medicine's Overtraining Questionnaire.
Hooper et al. (1993)	5 male and 9 female elite swimmers	10 to 12 workouts·wk ⁻¹ and 6 d·wk ⁻¹ during a 6-mth season.	Monitoring of sympathetic activity by measuring plasma epinephrine, norepinephrine concentrations and cortisol levels. Tracking of mood states, training loads with daily logs and Profile of Mood States (POMS) questionnaire.
Hooper et al. (1995)	5 male and 9 female elite swimmers	10 to 12 workouts·wk ⁻¹ and 6 d·wk ⁻¹ during a 6-mth season.	Measurement of norepinephrine, neutrophil number, self-assessment logs of fatigue, stress, muscle soreness, emotional strain, illness, menstruation, swim distance, and training intensity.
Callister et al. (1990)	8 male and 7 female elite judo athletes	10 wk: resistance (3 d·wk ⁻¹), interval (2 d·wk ⁻¹), judo training (2.5 hr session, 5 d·wk ⁻¹) during weeks 1-4; 50% increase in resistance and interval training; no increase in judo training during weeks 5-8; return to baseline resistance/interval training and 100% increase in judo training during weeks 9-10.	Measurement of isokinetic strength, interval/sprint times, vertical jump, body weight, body fat %, submaximal and maximal VO ₂ and HR, resting HR and BP, and blood lactate.
Parry-Billings et al. (1992)	40 male international level athletes participated in cycling, distance running, sprint running, race-walking rowing,	3 wk	Measurement of OTS affect on plasma amino acid concentrations, affect of different exercises, intensities, and durations on plasma concentrations of specific amino acids.

	squash, and swimming		
Booth et al. (2006)	38 male, 5 female army recruits	45 d in an Army Common Recruit Training course	Measurement of fatigue using the Multi-Dimensional Fatigue Symptom Inventory-Short Form, weekly self-assessment of quality of sleep, symptoms of fatigue, sickness, mood. Physiological markers included serum free testosterone/cortisol ratio, ferritin, C-reactive protein, in vivo inflammatory cytokine tissue necrosis factor, ratio of neutrophil to lymphocyte, iron status. Physical fitness: aerobic endurance, strength/muscular endurance, explosive power. Mood status using the POMS.
Bosquet et al. (2003)	9 experienced endurance athletes	4-wk intervals, continuous fast running, continuous slow running; increased intensity by 33%, 66%, and 100%.	Assessment of heart rate variability to study changes in SNS and PNS activity using an ECG, blood lactate levels, and daily log of fatigue; and the French Society of Sports Medicine Questionnaire.
Fry et al. (1994)	17 weight-trained males randomly divided into an overtraining and control group.	2 wk maximal relative intensity (100% 1 RM) and a low volume of exercise (10 sets x 1 rep) using a squat resistance exercise machine.	Measurement of quadriceps strength, voluntary isokinetic, isometric, and stimulated isometric quadriceps torque; 70% of 1 RM with maximal repetitions on squat machine to fatigue. Blood sampling of CK, blood lactate, anthropometric measures, dietary reports, and profile of mood states questionnaire POMS.
Pichot et al. (2002)	6 sedentary males	2 mth of intensive physical training (3 d·wk ⁻¹) at re-evaluated 90% VO ₂ peak, 1 mth of overload training (5 d·wk ⁻¹) on a cycle ergometer.	Assessment of heart rate variability to measure automatic nervous system activity. Measurement of physiological markers: VO ₂ max and nocturnal mean heart rate.
Halsen et al. (2003)	8 male cyclists	6 wk: 2 wk normal (7 ± 2 h·wk ⁻¹), 2 wk intensified (14 ± 5 h·wk ⁻¹), 2 wk recovery (3.5 ± 2.5 h·wk ⁻¹)	Monitored plasma cytokines, glutamines, glutamate, tumor necrosis factor-α, IL-6, salivary IgA, ammonia, urea, creatine kinase activity, hematological measures. Monitored mood state with questionnaires: Daily Analysis of Life Demands of Athletes and POMS.
Ishigaki et al. (2005)	13 male collegiate distance runners	8 d and ran 284.1 ± 48.2 km, which was almost twice the amount of km in normal	Monitored plasma glucose, total cholesterol, testosterone, plasma leptin, serum non-ester free fatty

		training.	acids, hematocrit levels, and dietary intake for leptin changes in runners.
Filaire et al. (2004)	12 national level male cyclists	8 mth with increasing intensity: T0 1 mth 60% VO ₂ max and 150 km·wk ⁻¹ ; T1 1 mth 70% VO ₂ max and 240 km·wk ⁻¹ ; T2 2 mth 80% VO ₂ max for 150 km·d ⁻¹ and 100% VO ₂ max for 35 km·d ⁻¹ ; T3 4 mth racing 600 to 750 km weekly.	Measured urinary concentrations of MHPG-sulphate, nor-metanephrine, and metanephrine, salivary concentrations of cortisol and testosterone, assessment of mood status using POMS, an overtraining questionnaire, and muscle soreness ratings.

Table 2. Descriptive Mean (\pm SD) Values for 238 Subjects in 12 Coded Studies.

Variables	Mean	Standard Deviation
Age (yrs)	23.5	2.03
Height (cm)	175.4	2.4
Weight (kg)	71.7	2.6
VO ₂ Max (mL·kg ⁻¹ ·min ⁻¹)	55.4	0.8
BF (%)	11.8	.9

The overall group mean time for subjects in an overtrained state was 6.6 wk. Mean (\pm SD) biomarker changes from N to OT are in Table 3. Figure 1 is a graphic representation of these biomarkers and their direction of change.

Table 3. Mean (SD) Biomarker Changes from N to OT and Effect Size Calculations.

Variables	Mean	Standard Deviation	Effect Size
Glutamine	-56.3	-2	-4.02*
Glutamate	49.7	2	8*
Cortisol	-89.7	-12.2	-1.4*
IL-6	-0.52	0.12	-5.2*
Testosterone	-88.9	-30	-0.7
Cholesterol	4.6	-1.6	.23
Glucose	-13.3	1.9	-1.1*
Leptin	0.15	-0.11	.51
Hematocrit	-0.83	-0.4	-.25
Hemoglobin	-20	---	---
Norepinephrine	36	-4.1	.44
Epinephrine	-2.2	-3.5	-.37
Creatine Kinase	29.2	8.5	.67

*Significant Effect Size

The effects size calculations for glutamine, glutamate, cortisol, IL-6, and glucose were significant ($P < 0.05$). The mean (\pm SD) physiological changes and direction of change (+, -) between N and OT, respectively, were: RHR, -1.9 (-0.22); resting SBP, -2 (-2); and resting DBP, 0 (1). Effect size calculations for the above measures were -0.95, -0.33, and 0, respectively. In regards to the psychological alterations associated with the OTS, the following findings were recorded. Three

articles reported decreases in tension at OT. One article reported increases at MW. Fatigue increased at OT in 6 studies and showed no change in a separate study. Confusion did not change in two studies, increased at OT in another, and increased at MW, then, it declined at OT in a final article. Vigor reportedly remained stable in 2 studies and decreased in 2 other studies. Anger did not change in 2 articles, decreased in 1 article, and increased in a different study with its peak at MW. There was no change in depression in 3 studies, but a decrease was reported in a separate article at OT with an increase at MW. Studies reported impaired sleep patterns, increased wakefulness, and decrements and stability in sleep quality. Two studies indicated increased levels of stress with one specifying stress related to training, sleep, and health. The findings showed a decreased perception of strength, decreased perception of recovery, and no change in perception of muscle soreness.

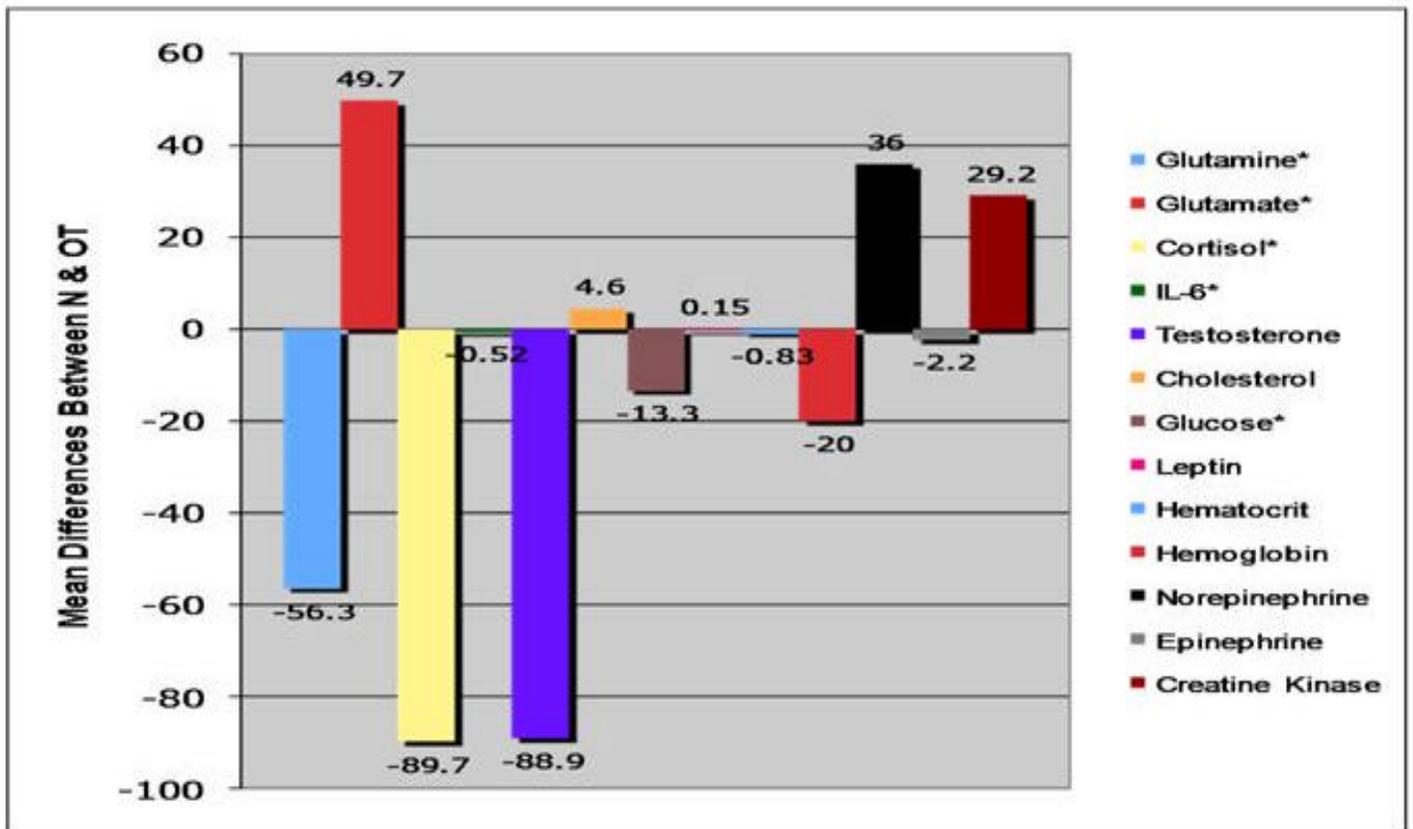


Figure 1. Graph of Mean (SD) Biomarker Changes from N to OT. Note: order of bar graph (left to right) matches order of variable (top to bottom)

DISCUSSION

Biomarker Summary

As noted in Figure 1, several biomarkers indicate change between normal (N) and those in an overtrained (OT) state. Glutamine is the mono-amide of aminoglutaric acid. It is present in the juices of many plants, and is essential in the hydrolysis of proteins. During exercise, L-glutamine facilitates urea production. It is released by the skeletal muscles, thus allowing for a vital role in maintenance of the key process of glutamine utilization in the immune cells. Also, it is one of two amino acids (along with alanine) for nitrogen delivery from the peripheral tissues to the liver. Under intense physical duress, the demand for glutamine is such that the lymphoid system is forced into a glutamine debt (8). The factors that influence glutamine synthesis or the release may also influence the function of

lymphocytes and monocytes (8). Reduced glutamine with the OTS sets the stage for a reduced immune function via altered lymphocytes (T lymphocyte immune response) and monocytes (defense in the inflammatory response)(9,10).

As indicated in Figure 1, glutamate showed an increase in effect size between N and OT states. Glutamate is the salt of glutamic acid; an amino acid formed in the hydrolysis of proteins. It is the only amino acid metabolized by the brain. In the central nervous system, glutamate is the predominant excitatory neurotransmitter driving neuronal activity. It is important to note that glutamate signaling is central to mediating up-regulation of Brain Derived Neurotrophic Factor (BDNF), which is a growth factor that promotes neuronal survival, health, and activity. Moreover, neuronal activity stimulates BDNF gene regulation and protein release. The release of BDNF at the synapses enhances synaptic transmission and neuronal excitability (8). Physical activity has been shown to increase BDNF. Thus, while OTS shows a decline in several biological and physiological markers, in relation to glutamate the OT state may facilitate brain and nervous system function (9,10).

Cortisol is a much studied hormone in relation to stress and specifically physical stress. Cortisol is released during exercise in relation to duration and, in general, short term exercise does not facilitate cortisol release. During stressful periods, however, it is of primary importance for its regulatory action in the metabolism of fats, carbohydrates, sodium, potassium, and proteins. Any reduction in cortisol reduces the regulation of metabolic factors associated with adenosine triphosphate (ATP) formation. It is also seen as an anti-inflammatory agent. This may explain why OTS is reported more in endurance athletes than in non-endurance athletes. The increase in cortisol during exercise is mediated by a lymphokine, interleukin-6 (IL-6) (8). Reduced cortisol levels noted in the OT athlete may also lead to reduced anti-inflammatory reaction (12,16).

Interleukin-6 (IL-6) showed a small effect size decline in the OT state. IL-6 is a lymphokine that works with IL-2 to stimulate the growth and maturation of T lymphocytes. Research during recent years suggests IL-6 plays an important role in metabolism. Also, IL-6 is produced in larger amounts than any other cytokine in relation to exercise. IL-6 levels seem to be elevated in response to short-intense work, low intensity long duration work, and post exercise (8). Research indicates a strong correlation between IL-6 levels and the intensity of exercise (10,16,27). Previous work suggested IL-6 was related to muscle damage, but recent studies demonstrate marked elevations with muscle contractions without muscle damage (22,25). It is suggested that the increases in IL-6 are linked to sympatho-adrenal responses (19). Important to note is that IL-6 mRNA is present in small amounts in resting skeletal muscle, but is enhanced up to 100 times in contracting muscle. Thus, IL-6 gene induction is facilitated by exercise. Moreover, IL-6 mRNA, nuclear transcriptional activity and protein release from skeletal muscle are augmented by muscle glycogen reduction. It may serve as a mediator in glucose uptake during exercise and as a novel lipolytic hormone. In addition, by stimulating cortisol production, IL-6 together with chemokines appears to play an important role in exercise-induced immune regulation. In relation to the OTS, reduced IL-6 may lead to reduced metabolism, reduced recovery to high intensity exercise bouts or, perhaps, an altered response to catecholamine release. In fact, IL-6 is negatively affected by chronic glycogen depletion that seems common in OT athletes. Thus, reductions in IL-6 may lead to altered carbohydrate (CHO) and free fatty acid (FFA) use in the formation of ATP for muscle contraction. Additionally, a reduction may lead to immune system dysfunction (26).

Another highly reduced biomarker for the OTS was seen in the hormone, testosterone. Aside from its role as an anabolic agent that accelerates growth in tissues and blood flow, testosterone also affects many metabolic activities (8) including the stimulation of protein synthesis and red cell production. Reduced testosterone may lead to reduced protein synthesis, decreased red blood cell production,

and a delay in overall recovery (19) from exercise. With aforementioned alterations in metabolic (i.e., energy production) components, it is not surprising that cholesterol was shown to increase slightly in the comparison of N and OT athletes. Cholesterol serves as an important component in metabolism, serving as a precursor of various steroid hormones. Many of the metabolic changes noted in OTS may be linked to reduced substrate availability (i.e., glycogen), which suggests an over-reliance on the gluconeogenic process (11). This point is supported by the reduced glucose levels during OTS.

Another noted increase was seen in the circulating protein hormone, Leptin. This hormone acts on the hypothalamus to decrease food intake and increase energy consumption. There are indications that leptin may facilitate bone loss. Leptin receptors are found in various peripheral tissues as well as the brain. In rats, it has been associated with depressed thyroid function and increased glucocorticoid secretion. There is evidence that leptin increases the activity of uncoupling proteins and, therefore, produces a direct peripheral increase in energy expenditure. The slight increase shown in this study may indicate an altered food intake while at the same time facilitating metabolic activity (14). This stimulation of the athletes' metabolism may promote excess weight loss in the OT state as well as an accelerated bone loss.

While the change in hematocrit (Hct) was small, hemoglobin (Hg) showed considerable alteration in the OT state. Reduced Hct and Hg may lead to reduced oxygen (O_2) carrying capacity of the blood, thus reducing the metabolic formation of adenosine triphosphate (ATP) at the cell level and overall reduction in energy (8,19) for muscle contraction.

Adrenal production of norepinephrine (NE) was shown to increase while epinephrine (E) was reduced. These catecholamines are secreted during periods of stress allowing for enhanced vasoactivity and altered myocardial function. Interestingly, a change in NE has been implicated in affective disorders. Since, the NE response with the OT state may involve altered brain reasoning capabilities. The decrease in E may reduce cardiac function, vascular response, and bronchial activity in the pulmonary system (12,13).

An increase in creatine kinase (CK), an enzyme present in skeletal, cardiac, and brain tissue was shown with overtraining. This enzyme plays an important metabolic role in that it catalyzes the reversible transfer of high-energy phosphate between creatine and phosphocreatine and between ADP/ATP (8). Additionally, elevated levels of CK in specific isoforms are seen in the blood following cellular damage (i.e., skeletal muscle, cardiac, and brain) (6). Thus, increased levels of CK may be indicative of cellular disruption and may facilitate reductions in skeletal muscle ATP regeneration (7).

Psychological Summary

The reported psychological changes in athletes were not consistent across the studies. This of course may be the psychological outcome of athletes in the OT state in that the responses are individualistic in nature (5,7,20). Another issue associated with the psychological alterations is that the marker may be physiological rather than psychological. An example of this is the reported increase in fatigue. Based on the methodology of data collection fatigue may be seen as a psychological response to OT (17). However, the components for establishing this feeling by the athletes is physiological (1,5,15). Other criteria for psychological disturbances were not consistently established across the studies. Suffice it to say, it seems evident through this meta-analysis that psychological issues may be evident while an athlete is in the OT state, but does not necessarily mean that the athletes will experience psychological changes. Thus, using psychological measures does not seem prudent in establishing if an athlete is truly overtrained.

Physiological Summary

The physiological measures addressed in the research articles that were examined in this study were either lacking or conflicting in information. In particular, it was noted in the articles that resting heart rate and systolic blood pressure were reduced in the overtrained athlete. This finding conflicts with the past reviews that have indicated an increase in RHR in the OT state (3). Other physiological measures that were taken to identify the level of athlete included maximal oxygen consumption (4). However, this measure was generally taken prior to the OT state and no post measures of VO_2 max were reported. As a result, the resting measures of heart rate and systolic blood pressure do not provide a reliable measure for determination of being overtrained.

CONCLUSION

Altogether, the data presented in this meta-analysis indicate considerable immune-suppression and increased stress in athletes who experience the OT syndrome (1,5,6,7,10,12,16,21,22,25,26,27). From this analysis, a negative effect size for the cardiovascular markers of HR and SBP indicates questionable alterations from N to OT in subjects (18,23,24). Increased sympathetic and/or decreased sympathetic influence may be affected in the OT condition. However, low effect size calculations allow for non-determinant conclusions related to cardiovascular indicators of the OT syndrome (3,13,15). Lastly, athletes in the OT state are likely to experience disturbances in sleep, self-perception, and mood factors (11).

Address for correspondence: Wyatt, FB, EdD, Department of Athletic Training and Exercise Physiology, Midwestern State University, Wichita Falls, TX, USA, 76308. Phone (940) 397-6229; FAX: (940)397-4901; Email. frank.wyatt@mwsu.edu.

REFERENCES

1. Armstrong LE, VanHeest JL. The unknown mechanism of the overtraining syndrome: clues from depression and psychoneuroimmunology. *Sports Med.* 2002;32(3):185-209.
2. Booth CK, Probert B, Forbes-Ewan C, Coad RA. Australian Army recruits in training display symptoms of overtraining. *Military Med.* 2006; 171(11):1059.
3. Bosquet, L, Papelier, Y, Leger, L, Legros, P. Night heart rate variability during overtraining in male endurance athletes. *J Sports Med Phys Fitness.* 2003;43(4):506-512.
4. Callister R, Callister RJ, Fleck SJ, Dudley DA. Physiological and performance responses to overtraining in elite judo athletes. *Med Sci Sports Exerc.* 1990;22(6):816-824.
5. Filaire E, Legrand B, Lac G, Pequignot J-M. Training of elite cyclists: effects on mood state and selected hormonal responses. *J Sports Sci.* 2004;22:1025-1033.
6. Finaud J, Lac G, Filaire E. Oxidative stress: Relationship with exercise and training. *Sports Med.* 2006;36(4):327-358.
7. Fry RW, Grove JR, Morton AR, Zeroni PM, Gaudieri S, Keast D. Psychological and immunological correlates of acute overtraining. *Br J Sports Med.* 1994;28(4): 241-246.

8. Ganong WF. **Review of Medical Physiology**. (22nd Edition). San Francisco, CA: Lange Medical Books/McGraw-Hill, 2005.
9. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. **Sports Med**. 2004;34(14):967-981.
10. Halson SL, Lancaster GI, Jeukendrup AE, Gleeson M. Immunological responses to overreaching in cyclists. **Med Sci Sport Exerc**. 2003;35(5):854-861.
11. Hawley CJ, Schoene RB. Overtraining syndrome: A guide to diagnosis, treatment, and prevention. **Physician Sportsmed**. 2003;31(6):25-32.
12. Hooper SL, MacKinnon LT, Gordon RD, Bachmann AW. Hormonal responses of elite swimmers to overtraining. **Med Sci Sports Exerc**. 1993;25(6):741-747.
13. Hooper SL, MacKinnon LT, Bachmann AW, Howard A, Gordon D. Markers for monitoring overtraining and recovery. **Med Sci Sports Exerc**. 1995;27(1):106-112.
14. Ishigaki T, Koyama K, Tsujita J, Tanaka N, Hori S, Oku Y. Plasma leptin levels of elite endurance runners after heavy endurance training. **J Physiol Anthr Applied Hum Sci**. 2005;24(6):573-578.
15. Johnson MB, Thiese SM. A review of overtraining syndrome-recognizing the signs and symptoms. **J Athl Train**. 1992;27(4):352-354.
16. Maso F, Lac G, Filaire E, Michaux O, Robert, A. Salivary testosterone and cortisol in rugby players: Correlation with psychological overtraining items. **Br J Sports Med**. 2004; 38(3):260-263.
17. Meehan HL, Bull SJ, Wood DM, James DVB. The overtraining syndrome: A multicontextual assessment. **Sport Psychol**. 2004;18:154-171.
18. Meeusen R, Piacentini MF. Exercise and Neurotransmission: A window to the future? **Eur J Sport Sci**. 2001;1(1).
19. Mooren FC, Volker K. (Editors). **Molecular and Cellular Exercise Physiology**. Champaign, IL: Human Kinetics, 2005.
20. Nederhof E, Lemmink APM, Visscher C, Meeusen R, Mulder T. Psychomotor speed: Possibly a new marker for overtraining syndrome. **Sports Med**. 2006;36(10):817-828.
21. Ogonovszky H, Sasvari M, Dosek A, Berkes I, Kaneko T, Tahara S, Nakamoto H, Goto S, Radak, Z. The effects of moderate, strenuous, and overtraining on oxidative stress markers and DNA repair in rat liver. **Can J Appl Physiol**. 2005;30(2):186-195.
22. Parry-Billings M, Budgett R, Koutedakis Y, Blomstrand E, Brooks S, Williams C, Calder PC, Pilling S, Baigrie R, Newsholme EA. Plasma amino acid concentrations in the overtraining syndrome: possible effects on the immune system. **Med Sci Sport Exerc**. 1992; 24(12):1353-1358.

23. Pichot V, Busso T, Roche F, Garet M, Costes F, Duverney D, Lacour JR, Barthelemy JC. Autonomic adaptations to intensive and overload training periods: a laboratory study. **Med Sci Sport Exerc.** 2002;34(10):1660-1666.
24. Polman R, Houlahan K. A cumulative stress and training continuum model: a multidisciplinary approach to unexplained underperformance syndrome. **Res Sports Med.** 2004;12:301-316.
25. Ring C, Carroll D, Hoving J, Ormerod J, Harrison LK, Drayson M.. Effects of competition, exercise, and mental stress on secretory immunity. **J Sports Sci.** 2005;23(5):501-508.
26. Smith LL. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? **Med Sci Sport Exerc.** 2000;32(2):317-331.
27. Smith LL. Overtraining, excessive exercise, and altered immunity: is this a T helper-1 T helper-2 lymphocyte response? **Sports Med.** 2003;33(5): 347-364.
28. Thomas JR, Nelson JK, Silverman SJ. **Research Methods and Physical Activity.** (5th Edition). Champaign, IL: Human Kinetics, 2005.

Disclaimer

The opinions expressed in **JEPonline** are those of the authors and are not attributable to **JEPonline**, the editorial staff or the ASEP organization.