Training and overtraining markers in selected sport events

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ABSTRACT

HARTMANN, U., and J. MESTER. Training and overtraining markers in selected sport events. Med. Sci. Sports Exerc., Vol. 32, No. 1, pp. 209-215, 2000. Purpose: Varieties of symptoms are supposed to detect overtraining (OT). Besides the problems of diagnosis and analysis in elite athletes, a daily monitoring of training status takes place with measurement of the parameters serum urea (SU) and serum creatine kinase (CK); therefore, their meaningfulness will be examined, with special respect inter- and intra-individually. Methods: Data were obtained from determinations during training from athletes in rowing and athletes of international level. Results: For 6981 SU determinations (male, N = 717; female, N = 285), a slightly asymmetric normal distribution was found (male, 80%, 5–7). $mmol\cdot L^{-1}$; female, 75%, 4–6 $mmol\cdot L^{-1}$). Values for women were approximately 1.5 $mmol\cdot L^{-1}$ lower. Individual variability was enormous; there seems little point in setting fixed value as 8.3 mmol· L^{-1} for men and 7.0 mmol· L^{-1} for women as a critical limit for OT. CK has also been measured and evaluated in sports as an essential parameter for determination of muscular stress. Frequency distributions of CK in 2790 samples (male, N = 497; female, N = 350) presented an asymmetric normal distribution with distinct trend toward higher values being evident for the range between 100 and 250 U·L $^{-1}$. Conspicuously elevated values occurred in the ranges 250-350 U·L⁻¹ and 1000-2000 U·L⁻¹. Men's maximal values were 3000 U·L⁻¹ and 1150 U·L⁻¹ for women. Individual variability was enormous. Athletes with chronically low CK exhibited mainly low variability; those with chronically higher values exhibited considerable variability. Conclusions: Establishment of both parameters should be useful to determine individual baselines from a large number of samples. Determinations should be made at least every 3 d in standardized conditions. If a large increase is observed in combination with reduced exercise tolerance after a phase of exertion (2-4 d), then the possibility of a catabolic/metabolic activity or insufficient exercise tolerance becomes much more likely. Key Words: BIOCHEMICAL MARKERS, OVERREACHING, EXER-CISE INTENSITY, GENDER, UREA, CREATINE KINASE

ccording to the *Oxford Dictionary of Sport Science* and Medicine (29), overtraining is a complex syndrome, and it is described as "a combination of signs and symptoms of overtraining which typically causes the sufferer to feel mentally fatigued in absence of physical fatigue and causes deterioration of performance. The sufferer's basal metabolic rate is elevated, there is usually a loss of body weight associated with a negative nitrogen balance, and the rate of return of exercise pulse-rate to resting pulse-rate is delayed. The overtraining syndrome involves changes in the neuron and endocrine systems, particularly the hypothalamus."

Following Lehmann et al. (33), "overtraining" (OT) is understood as a "long-term" form of overloading, whereas "overreaching" is "short-term overtraining." Foster et al. (13) suggest two stages of the adaptation process related to the average load of training (volume \times intensity). In the beginning, the performance increases with a load up to a plateau, and beyond this, there is a slight decrease of per-

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formance named "overreaching" followed by a dramatic loss due to the stages of OT.

However, this division is only a rough guideline because the transitions between the points listed above are fluent. However, the causes, symptoms, and treatments of OT are not sport-specific, and the results of a study of OT are transferable to many other sports.

EXPECTED MARKERS FOR OVERTRAINING

In the given literature a variety of symptoms of OT, which may also serve as diagnostic parameters, are presented for an overview (33).

Most of the examined parameters are dealing with more specific judging criteria and cannot be used during the routine training monitoring process. The use of specific biochemical markers for the diagnosis of OT are still in discussion (12,28,33–35).

However, according to Karvonen (28), serum prealbumin, serum creatine kinase, urine 3-methylhistidine, and saliva testosterone/saliva cortisol ratio give inconsistent results in classifying OT. Wilmore and Costill (53) state that the endocrine changes mentioned above simply may reflect the stress of training, rather than a breakdown in the adaptive process. Martin and Coe (39) concluded that "metabolic indicators, as measured by changes in the values reported



Figure 1—Influencing factors on serum urea level.

for blood chemistry variables, are at present, imprecise indicators for the actual onset of OT because no consistently identifiable threshold level exists for this condition".

According to Israel (25), OT can be either sympathetic (or basedoid) or parasympathetic (or addisonoid) because the effects of autonomic nervous system may be predominant. In both forms of OT, there is a marked decrease of maximal performance. However, the causes, symptoms, and treatments of OT are not sport-specific, and the results of a study of OT are transferable to many other sports.

Regardless of the problems of diagnosis and explanations concerning the symptoms of OT, one tries to register and identify corresponding forms of appearance by means of parameters that can be measured and interpreted easily and quickly in training practice (i.e., the documentation of performance and training, changes in body weight, changing heart rate or heart rate variability). In many top elite athletes, a daily monitoring of the training status takes place with a measurement and interpretation of the parameters of serum urea (SU) and serum creatine kinase (CK); therefore, in the following, the meaningfulness of SU and CK will be examined. In this context, special consideration is attributed to the inter- and intra-individual variability of the measuring quantities that may contradict a supposedly simple interpretation.

SERUM-UREA IN BLOOD

Urea is an end product of the degradation of nitrogenous or protein materials. Measurements recorded in the field as a component of the training program represents the concentration of SU (can be regarded as equal to plasma), i.e., balance of urea synthesized in the liver and urea excreted renally. Many authors report that it is possible to draw conclusions about the extent of protein degradation (7–9,22,42). Normally, the concentration is between 1.7 and 8.3 mmol·L⁻¹ (10–50 mg·dL⁻¹) (Fig. 1). The influencing factors on SU level are depicted in Figure 1.

Changes in SU occurring in connection with physical effort have been described in a number of publications (1,3,18,26,41,43). The endogenous SU pool can be increased by up to 100% as result of physical exertion (43).

Figure 2 shows the percentage of distribution of 6981 SU determinations recorded in male (N = 717) and female (N = 285) competitive rowers during training. A slightly asymmetric normal distribution was found; around 80% of the male values were between 5 and 7 mmol·L⁻¹, and 75% of the female values between 4 and 6 mmol·L⁻¹. It should be noted that the right side of the distribution curve being somewhat more strongly pronounced is the part of the SU curve used for interpretation within the framework of measures aimed at the regulation of training schedules. Values for female athletes exhibited a similar distribution to those of the men but were approximately 1.5 mmol·L⁻¹ lower.

Figure 3 illustrates the individual variability of the SU values in the sample of measurements in 12 male and 12 female athletes. Individual mean values, standard deviations, and maximal values were calculated for a group of 19 subjects with a large number of individual measurements (>100).

Athletes with a large number of individual measurements (>100) were divided arbitrarily into three groups, with chronically low (women, <4.5 mmol·L⁻¹; men, <6 mmol·L⁻¹), chronically middle-range (around 5.0 mmol·L⁻¹ and 6.5 mmol·L⁻¹), and chronically high (>5.5 mmol·L⁻¹ and >7.0 mmol·L⁻¹) mean SU values. Athletes with high mean values exhibited considerable variation, whereas the opposite is true for those with low values. What is striking here is that as the mean values increased so did the range of variation of the corresponding standard deviations; there was particularly an increase in the variation of the maximal values.

In light of these findings, there is little value in setting some fixed numerical value (e.g., 8.3 mmol·L⁻¹ for men and 7.0 mmol·L⁻¹ for women) as a critical limit indicative of a state of OT. It would be far more useful to specify a certain range for each athlete based on individual variation and gender. According to Lorenz et al. (36), a comparative evaluation of SU changes should be based not only on the



Figure 2—Frequency distribution (%) of a high number of urea samples in female and male top athletes.



Figure 3—Individual variability of the serum urea $(\text{mmol}\cdot\text{L}^{-1})$ in 12 male and 12 female athletes (for further explanations see text)

absolute values but also on increases in the values; they believe that the basal values must always be taken into account.

Thus, although a marked increase in exercise can be associated with an increase in the SU value, the conclusion of a catabolic/metabolic activity does not automatically result from such elevated values. However, if a large increase in the SU values is observed in combination with a reduced exercise tolerance after a longer phase of intense exertion (2-4 d; Fig. 4), then the possibility of a catabolic/metabolic activity or insufficient exercise tolerance becomes much more likely. This is also in line with the results obtained by other authors (3,16,18,19). In our own data, correlation between a training regimen regulated according to physiological aspects and the SU level measured on the following day were not significant in general, only in individual cases. Similar results were obtained by Janssen et al. (26).

Gottert et al. (16) suggested that the determination of SU can serve as a means of regulating training in the short term. However, according to our own findings and the literature, it is appropriate to speak of a catabolic/metabolic activity only after elevated SU levels have been measured for 2–3 d. Preliminary balance estimates suggest that elevated SU values maintained for 3–5 d may lead to a massive loss of protein. However, in competitive sport, a retrospective diagnosis of this kind falls well short of the ideal of an optimal scientifically based accompaniment to training oriented to warrant a positive prognosis of performance.

In summary, it should be useful to establish an individual baseline level from a large number of samples. Blood samples for the determination should be taken at least every 3 d, they should be taken at standardized times and conditions. A distinct increase in exertion can be associated for a short time with an increase in SU, and this should not automatically be interpreted as a sign of a catabolic/metabolic activity. Only after elevated SU levels have been measured for 2–3 d can one conclude that a catabolic/metabolic activity or an inadequate exercise tolerance is present (Fig. 4).

According to a preliminary balance estimate, a massive protein loss may have occurred after 3–5 d of elevated SU values. However, from the point of view of acting as a guide to further training, a retrospective diagnosis of this kind is far from ideal.

The dependence of SU on physical exertion varies considerably from one individual to the next, i.e., a direct correlation between training and SU is seemingly no more than a random occurrence. This leads to the problem of individual exercise tolerance.

Provided that all foregoing points are taken into account, SU determinations can be recommended during training within the framework of justifiable diagnostic workloads and blood sampling programs for optimal development of athletic performance. SU measurement can be more reliably interpreted if coupled with measurements of hematocrit or hemoglobin. In addition, the dietary situation and the water balance should be known and standardized.

SERUM CREATINE KINASE IN BLOOD

Serum CK has for years been measured and evaluated in exercise science as an essential parameter for the determination of muscular stress. The literature data on the variation of CK as a function of physical exertion in athletes and nonathletes have been reviewed by Hortobagyi and Denahan (24). Apart from this work, however, little detailed information is available on the level or range of enzyme in blood occurring in conjunction with the stress associated with competitive sports (10,11,24), and what information there is was obtained from relatively small groups of subjects (21,40,45,49).

As the data for SU, the following results are also based within the framework of routine performance-diagnostic investigations and determinations performed during training. All athletes were participating at national and international level. Frequency distributions of the CK values in 2790 blood samples (male, N = 497; female, N = 350) are presented in Figure 5.

An asymmetric normal distribution of the values can be observed in both groups, a distinct trend toward somewhat



Figure 4—Example of an increase in serum urea $(\text{mmol}\cdot L^{-1})$ above the athlete's individual average value plus his individual standard deviation over a 10 d period during a training camp



Figure 5—Frequency distributions of the CK $(U\cdot L^{-1}$ during routine training monitoring in various sports groups (for further information see text)

higher values being evident for the range between 100 and 250 U·L⁻¹. Conspicuously elevated values occurred in the ranges 250-350 U·L⁻¹ and 1000-2000 U·L⁻¹, though this was partly due to the scaling.

An absolute maximum of up to 3000 $U\cdot L^{-1}$ was measured in men, as compared with an absolute maximum of up to 1150 $U\cdot L^{-1}$ in women.

Spitler et al. (46) determined CK levels in men and women of comparable fitness levels and found significantly lower CK in women than in men. This is in agreement with our own findings (Fig. 6). Sex-specific hormonal influences, higher resistance to cell damage, or simply a lower enzyme content compared with the men have been put forward as possible explanations. The individual variability in CK values determined in the sample of 12 male and 12 female subjects is illustrated in Figure 6.

Using athletes for whom a large number of individual measurements were available (women, N > 45; men, N > 55), the individual mean values, standard deviations, and maximal values were plotted. For this purpose, the athletes were divided arbitrarily into three groups with chronically low (women, $<45 \text{ U}\cdot\text{L}^{-1}$; men, $<65 \text{ U}\cdot\text{L}^{-1}$), medium (women, $70-80 \text{ U}\cdot\text{L}^{-1}$; men, $95-110 \text{ U}\cdot\text{L}^{-1}$), and high (women, $>80 \text{ U}\cdot\text{L}^{-1}$; men $>150 \text{ U}\cdot\text{L}^{-1}$) CK levels. As the figure shows, athletes with chronically low CK exhibited mainly low variability, whereas those with chronically higher values exhibited considerable variability of this parameter.

A particularly important consideration relating to the use and the interpretation of CK values in the sports sector is the dependence of this parameter on nature of the stress. Whereas in older publications (47,48), the amount of exercise was considered the most important factor, according to Tiidus and Ianuzzo (51), it is the exercise intensity that really matters the most.

According to our observations, considerable CK increases in a large number of athletes were recorded after moderate to intensive maximum strength or endurance training.

This statement must be qualified in that stress intensity does not depend solely on the duration of exercise. Thus,

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distinct CK increases were likewise recorded in individual athletes after predominantly extensive training (LA < 2 mmol·L⁻¹), prolonged forms of exercise (120 min/training unit), and after high-level training over several days. Distinct changes in CK behavior were also observable after a reduction in training.

In light of the above, we find ourselves in agreement with the conclusions formulated by Thompson et al. (50), i.e., that both the intensity and the volume of exercise are important, as they have a bearing on the reduction in the high-energy phosphates in the muscle cell.

Jones et al. (27) speak in this connection of a "duration activity," which occurs when the oxygen uptake is between 70% and 90% of the maximal oxygen uptake (\dot{VO}_{2max}). Hortobagyi and Denahan (24) also conclude that the interplay between the extent and the intensity of exercise merits considerable attention. Hecht et al. (20), Hoppeler (23), and Kuipers and Keizer (32) reported on the microdamage to skeletal muscle possibly associated with sustained exertion of this kind.

A number of authors (4-6,14) also arrived at the conclusion of interdependence between the form of muscle contraction and subsequent CK activity, insofar as higher CK values were determined after eccentric type of exercise.

Figure 7 illustrates the behavior of CK in a male athlete in a longitudinal study over 5 yr. In the course of a 3-yr period, divided into blocks A–F, the mean CK value was $87 \pm 34 \text{ U}\cdot\text{L}^{-1}$. After a 1-yr break, the athlete resumed performance training (block G), which resulted in a dramatic rise in the CK (mean value 294 \pm 176 U·L⁻¹). The only way in which further training overloads could be avoided was by introducing considerable modifications in the training program.

Robbins (44) speculated about the existence of an adaptation effect in athletes, as a result by which the active tissue complex may be stabilized by years of training. There is also evidence that a hormonal improvement takes place, insofar as a change in the cell membrane permeability takes place (15,31).



Figure 6—Individual variability of CK values (U·L⁻¹ determined on the example of 12 male and 12 female subjects, four of each type (low, medium, high; for further information see text).



Figure 7—Creatine kinase levels (U·L⁻¹ of an athlete in longitudinal study over 5 yr (male rower, 36 values; average CK = $138 \text{ U} \cdot \text{L}^{-1}$; for further information see text).

To summarize, with regard to the behavior of CK in athletes in a training camp, where exercise takes place several times a day, it may be assumed that an accumulation of CK activity can occur. Diagnostics based on the determination of CK appear a sensible and useful means of evaluating any increase in muscle stress or individual tolerance to muscular exertion.

In light of the aforementioned frequency distributions and findings, a mean CK of approximately $100-150 \text{ U}\cdot\text{L}^{-1}$ and approximately $200-250 \text{ U}\cdot\text{L}^{-1}$ can be regarded as normal in female and male athletes, respectively. However, supported by our own findings, we must assume that athletes with chronically low CK exhibit a predominantly low variability (women up to 100 U·L⁻¹ and men up to 200 U·L⁻¹), whereas athletes with chronically higher values (women up to 200 U·L⁻¹ men up to 400 U·L⁻¹) exhibit considerable variability of this parameter. This is particularly important within the framework of individual training regimens.

No definite or statistically significant relationship between given training loads and the behavior of CK could be established. There is an obvious need for regular CK determinations within the framework of a program of measures designed to accompany training.

GENERAL REMARKS AND CONCLUSIONS

The causal mechanisms of OT are still unclear, although there are some indications that faulty hormonal regulations of the stress response at central level or a neuroendocrine/ hypothalamic dysfunction (2,17) play an important role in this context (25,30). It is certain, however, that OT is almost the result of a disparity between load and load tolerance

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(25); according to this aspect, OT should not only be discussed under the clinical aspect (30), but more under the aspect of the training content.

According to the fundamental questions of adaptation, calculations at a simulation model (37,38) demonstrate that at a stage of hypertrophy, despite an increased total protein turnover proportional to the existing protein mass, the relative increase of protein turnover according to the existing hyperfunction is far too low to be detected in humans (52). Following this observation, the determination of SU could be useless.

About the use of routine parameters SU and CK to interpret or to detect a tendency to OT or "overreaching," the following summary can be given:

No (simple or single) parameter is available to predict OT. All findings indicate events that already took place. This retrospective diagnosis of markers for OT is far from ideal; the influence of the training load is a key factor. Thus, a differentiated training protocol is necessary. Daily weight checks, dietary situation, and water balance must be known and should be standardized. For most of the parameters, an establishment of a "baseline" level from a large number of samples should take place. Blood samples for further determination should be taken at standardized times and conditions, at least every third day. Elevated levels over more than 2-3 d in conjunction with reduced exercise tolerance point to a catabolic situation or an inadequate exercise tolerance. Dependence of the given parameters (SU, CK) on physical exertion varies considerably from one individual to the next; a direct correlation between training and those parameters is seemingly no more than a random occurrence. All this leads to the problem of an individual exercise tolerance where the influence of training is the key factor.

As far as treatment of OT is concerned, most authors agree that those factors that lead to obvious OT (in both daily life and training) should be determined and eliminated as soon as possible. The intensity and amount of training should be reduced for a while. Monotonous training should be avoided (change of load, and environment, sometimes even of event). A good training schedule is characterized by the alternation of load and regeneration phases. Regular sleep is very important. Adequate quantity and quality of nutrition should be ensured regardless of any loss of appetite connected with OT.

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