Recovery training in cyclists: ergometric, hormonal and psychometric findings

O. Faude12, T. Meyer12, A. Urhausen3, W. Kindermann1

1Institute of Sports and Preventive Medicine, University of Saarland, Saarbrücken, Germany, 2Institute of Sports Medicine, University Paderborn, Paderborn, Germany, 3Center of Locomotor System, Sports Medicine and Prevention, Hospital Center of Luxembourg, Luxembourg

Corresponding author: Oliver Faude, PhD, Institute of Sports Medicine, University Paderborn, Warburger Str. 100, 33098 Paderborn, Germany. Tel: +49 05251 60 3587, Fax: +49 05251 60 3188, E-mail: oliver.faude@uni-paderborn.de

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This randomized cross-over study aimed at comparing the recovery effect of 4 days of low-intensity, discipline-specific training of 1 vs 3 h daily. Eleven athletes completed two periods of 13 days intensive cycling training (IT), followed by a recovery period consisting of 4 days of low-intensity cycling for either 1 or 3 h each day. Before IT, after IT and after the recovery period, subjects were tested in the laboratory: venous blood sampling, “profile of mood states” (POMS), graded cycling test and a 30-min time trial (TT). Maximal heart rates and lactate concentrations decreased significantly after IT. Peak power output, maximal heart rates and maximal lactate concentrations changed significantly different during the recovery periods. Whereas these parameters were similar to pre-training values after 1-h daily active recovery, 3-h recovery training (REC) led to further decreases. Power output during TT was neither affected by IT nor by both recovery periods. TT-induced increases in cortisol, adrenocorticotropic hormone and prolactin were reduced only after 3-h REC. Total POMS and subscores fatigue and vigor changed significantly different during the recovery periods, a return to pre-training levels after 1 h active recovery and a further deterioration after 3 h REC. It is concluded that low-intensity training of a 1-h duration each day is more appropriate for recovery after an IT period than 3 h.

Elite endurance sport is characterized by large training volumes. Financial burdens, an increased attention of mass media as well as competitions and training camps distributed all over the world make up the non-training stressors that athletes are exposed to. This may limit the recovery time in the athletes’ training regimens and therefore increase the danger of disrupting the balance between training load and recovery.

Overload is a pre-requisite of successful training and, therefore, is often used by athletes during training camps to enhance performance. Such an intensive training (IT) period may lead to signs of acute fatigue or short-term (functional) overreaching (OR) allowing for full recovery within several days to weeks (Meeusen et al., 2006). Excessive overload in combination with inadequate recovery may lead to extreme (non-functional) OR or even an overtraining syndrome (OTS). At this stage, symptoms of training distress can be observed. This might be performance decrements, psychological as well as hormonal disturbances. Full recovery will need several weeks to months (Meeusen et al., 2006).

Several authors have emphasized the importance of periodizing the training process as a suitable means to prevent an OTS (Fry et al., 1992b; Rowbottom et al., 1998). Intensive training cycles induce both the specific adaptations needed to reach a high performance level as well as exhaustion of certain organ systems. Appropriate recovery after intense training may result in an elevated performance level. Although the importance of active recovery has been emphasized (Fry et al., 1992b; Rowbottom et al., 1998; Foster et al., 1999), recommendations for the design of appropriate recovery training (REC) regimes are vague and almost entirely based on theoretical considerations as well as experiences from practice. Scientific data about the most appropriate conduction of active REC are not available.

Acute recovery between single bouts of intensive exercise may be accelerated when low-intensity exercise is performed in between as compared with passive rest (Ahmaid et al., 1996; Greenwood et al., 2008). The intensity range for active recovery has often been chosen between 30% and 70% of maximal aerobic capacity (Baldari et al., 2004). An intensity corresponding to the first ventilatory threshold (VT) or to the lactate threshold, i.e. the highest velocity before the onset of the curvilinear increase in blood lactate concentrations, seems to be
most effective (Baldaire et al., 2004; Greenwood et al., 2008). These findings indicate that low-intensity training, particularly at an intensity corresponding to the first increase in blood lactate concentrations, is adequate for active recovery.

Previous research has shown that sympathetic tone and stress hormone concentrations – as probable triggers for the OTS – increase with the duration of exercise, even during low-intensity exercise (Schwarz & Kindermann, 1989). Therefore, it seems likely that the duration of REC sessions may affect their efficacy too. The present study focuses on ergometric, psychometric, and hormonal changes during active, discipline-specific training at VT of 1 vs 3 h for four consecutive days after a standardized, strictly controlled IT regime. It was hypothesized that 4 days of REC of 1 h results in more beneficial changes than 3 h/day. A duration of 3 h was chosen because endurance athletes often report using low-intensity training of several hours when coming back from a training camp. Shorter sessions of 1-h duration seemed to represent an appropriate contrast in the context of the study hypothesis.

Materials and methods

The study design and the procedures used were in accordance with ethical standards and the Declaration of Helsinki. The study was approved by the institutional review board. Each athlete gave written informed consent before the start of the study.

Subjects

A total of 15 healthy male subjects were recruited. Four subjects (27%) dropped out of the study because of inadequate compliance with training prescriptions (n = 3) or due to a severe infection (n = 1). Eleven competitive cyclists and triathletes completed the study. Anthropometrical data and performance parameters at baseline are given in Table 1. Subjects had an average training experience of 6.0 (SD 4.4) years. The mean weekly training time was 14.4 (SD 5.9) hours. Subjects had an average training experience of 6.0 (SD 4.4) years. The mean weekly training time was 14.4 (SD 5.9) hours.

General design

The randomized cross-over design included two identical 13-day IT periods, each followed by 4 days of active REC with an intensity corresponding to VT. The duration of the recovery sessions was set in a randomized order at either 1 (REC 1) or 3 (REC 3) h daily. Before and after IT (T1 and T2, respectively) as well as after REC (T3), subjects reported to the laboratory for venous blood sampling, psychometric and ergometric testing. Before both ITs, all subjects performed a 30-min time trial (TT) for familiarization with this procedure. During the following 3–4 days, only low-intensity training of short duration was allowed to ensure that each subject was similarly recovered at the start of the IT period (run-in period). The wash-out period between both training periods was a minimum of 10 weeks and a maximum of 1 year. No order effect of the first and second periods of intensified training on the main physiological outcomes was observed (P > 0.46).

Nutrition and supplementation were controlled by means of a written protocol and were constant in both training cycles. No supplementation was allowed during the recovery period.

Testing days and procedures

On testing days, subjects reported to the laboratory at 8:00 a.m. Before all other procedures, they filled in the “Profile of Mood States” (POMS; German version: 35 items, four subscales, depression, fatigue, anger, vigor). The POMS was additionally determined on the 7th day of IT. Subsequently, athletes were placed in a supine position and rested for about 15 min before venous blood was drawn from an antecubital vein and immediately processed for laboratory testing. Thirty minutes after blood sampling, subjects performed an incremental graded exercise test (GXT). After 2.5 h of the cessation of the GXT, a 30-min all-out cycling TT took place. Subjects were told to keep their nutrition constant and carbohydrate-rich on testing days as well as on the days before each test.

Incremental exercise test (GXT)

The GXT was performed on an electrically braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands). After 3 min at 100 W, power output was increased by 50 W every 3 min. Subjects were verbally encouraged until volitional exhaustion occurred. Capillary whole-blood samples (20 μL) were taken from the hyperemic earlobe before start, at the end of each stage and 1, 3, 5, 7 and 10 min after cessation of exercise and were analyzed for lactate concentrations (automated enzymatic–amperometric method, Greiner BioChemica, Flacht, Germany). The individual anaerobic threshold (IAT; Stegmann et al., 1981) was determined from the lactate–workload plot using a PC routine (software developed by H. Heck, University Bochum, Germany). The IAT is defined as the exercise intensity identified by a tangential drawn to the blood lactate curve during incremental exercise originating at the time that recovery lactate declines to the blood lactate value observed at the cessation of exercise. The IAT has been shown to estimate the maximal lactate steady state during running and cycling (McLellan & Jacobs, 1993; Urhausen et al., 1993) and thus seems to be an adequate means to assess aerobic endurance performance. Heart rate (Polar Electro, Kempele, Finland) was continuously measured and recorded at the end of each stage. Gas exchange measure-

| Table 1. Anthropometric and endurance performance data of the subjects |
|----------------|----------------|----------------|----------------|----------------|----------------|
| Age (years) | Height (cm) | Weight (kg) | Body fat (%) | HV (mL/kg) | VO2peak (mL/min/kg) | IAT (W/kg) |
| 24.8 ± 3.8 | 180 ± 5 | 74.7 ± 6.3 | 10.7 ± 3.3 | 13.7 ± 1.5 | 69.7 ± 9.6 | 3.7 ± 0.5 |

Data as mean ± standard deviation. HV, heart volume; VO2peak, peak oxygen uptake; IAT, individual anaerobic threshold.
ments were carried out throughout the test using a mixing chamber system (MetaMax II, Leipzig, Germany). Peak oxygen uptake (VO\textsubscript{2peak}) was determined as the highest oxygen uptake (VO\textsubscript{2}) averaged over 30 s. Peak power output (PO\textsubscript{peak}) was calculated using the following equation:

\[
PO_{\text{peak}} = PO_{\text{final}} + \frac{t}{T} \times 50 \text{ W}
\]

where PO\textsubscript{final} (W) is the power output during the final stage completed, \(t\) (s) is the amount of time reached in the final uncompleted stage and \(T\) (s) is the duration of each stage (Halson et al., 2002).

**Time trial**

The 30 min TT was carried out on the subjects’ own racing bike frame, which was mounted on the electronic brake of a stationary training device (Cyclus 2, Avantronic, Leipzig, Germany). The bicycle chain over the pinion drives the braking mechanism. The axles are constructed elastically as to allow slight sideward movements of the bicycle frame. This enables similar movements during the TT simulation as cyclists usually use in real outdoor racing situations.

After a 10-min cycling warm-up, subjects were instructed to maintain the highest possible workload for 30 min. Subjects were informed about the elapsed time but blinded for their actual power output. Heart rate was recorded continuously. Capillary blood samples for blood lactate determination were taken before and after warm-up, at 10, 20 and 30 min of TT as well as 2 min after cessation of exercise. Gas exchange measurements were collected between 7 and 10, 17 and 20, and 27 and 30 min. The average of the last 2 min of these periods was used for statistical analysis. For determination of exercise-induced changes in hormonal parameters, a supine venous blood sample was collected before TT as well as 5 min after TT.

**Hormonal parameters**

Blood samples taken at rest, immediately before and after TT were collected into tubes containing K\textsubscript{3}EDTA (2.7 mL) and a clot activator (9 mL). Samples were analyzed for hematological values (hematocrit and hemoglobin) using a Sysmex K-1000 (Sysmex GmbH, Langenfeld, Germany). After they were centrifuged, plasma and serum samples were frozen at \(-40\, ^\circ\text{C}\). Exercise-induced plasma volume changes after TT were estimated using hemoglobin and hematocrit values according to Dill and Costill (1974). Testosterone [intra-assay coefficient of variation (CV) = 2.7%], cortisol (CV = 4.4%), growth hormone (GH; CV = 3.6%), insulin (CV = 2.0%) and prolactin (PRL; CV = 1.4%) were determined from serum samples by ELISA (Access, Beckman Coulter, Krefeld, Germany). Adrenocorticotropic hormone (ACTH; CV = 4.2%) determination was carried out by chemiluminescence immunoassays (Nichols Advantage, Nichols Diagnostika GmbH, Bad Vilbel, Germany) from blood plasma. All samples from each subject were analyzed in the same assay.

**Training monitoring**

All training sessions were performed on the subjects’ own bicycle equipped with a “mobile ergometer” (SRM-System, Schoberer Rad Messtechnik, Jülich, Germany). The transferability of workload measurements between the three ergometers used in the present study (SRM-System, Lode Excalibur Sport, Cyclus 2) has been confirmed earlier by Reiser et al. (2000). Training was scheduled by an experienced exercise physiologist who was familiar with planning the training of elite endurance athletes. It was designed as it is common in training camps of endurance athletes aiming at improving aerobic endurance. It was not necessary to induce non-functional OR, although signs of acute fatigue or (functional) OR were expected. Training intensities were prescribed as workloads relative to the IAT. Subjects trained an average of 20 h each week. This corresponded to an increase of about 40% in relation to their usual amount of training. The first week consisted of mainly extensive training sessions of a long duration, whereas the time spent with interval training of higher intensity was increased during the second week. On the eighth day, no training was scheduled. Twenty percent of the total training load was spent with low-intensity training (66–70% IAT, \(\sim 56\%\) VO\textsubscript{2peak}) of a short duration (1–1.5 h) and 65% at moderate intensities (74–78% IAT, \(\sim 64\%\) VO\textsubscript{2peak}) for a longer duration (2–4 h). Ten percent of the training consisted of long intervals (20 min) at 86% IAT (\(\sim 75\%\) VO\textsubscript{2peak}) and 5% were spent with high-intensity intervals (110% IAT, \(\sim 91\%\) VO\textsubscript{2peak}) of 5-min duration. The adequacy of these training intensities was evaluated by pilot work (unpublished data). A ramp exercise test with spiroergometry was performed on the last day of IT (day 13) to determine VT using the V-slope method (Beaver et al., 1986). VT corresponded on average to 60% VO\textsubscript{2peak} or 72% IAT, respectively. All REC sessions had to be performed in an even landscape.

**Statistics**

All dependent variables were normally distributed (Kolmogorov–Smirnoff test). Data are presented as mean and standard deviation (SD). Global effects over the study period were tested using a 2 × 3 ANOVA (factor 1: 1- vs 3-h regeneration; factor 2: T1 vs T2 vs T3). In case of a significant interaction, a 2 × 2 ANCOVA (factor 1: 1- vs 3-h REC; factor 2: T2 vs T3; covariate: results of T1) was used to evaluate the course of the dependent variable during the recovery period more closely. Post-hoc analyses were carried out using a Scheffé test. The \(\alpha\)-level of statistical significance was set at \(P<0.05\).

**Results**

**Incremental exercise test**

IT caused a significant rightward shift of the lactate curve during GXT that was accompanied by a significant increase in power output at IAT (Table 2). Whereas power output at the IAT was further increased after REC 3, the IAT remained unchanged after REC 1. The change in power output at the IAT from T2 to T3 was significantly different between REC 1 and REC 3.

Maximal heart rate (HR\textsubscript{max}, \(P = 0.005\)) and blood lactate concentrations (L\textsubscript{a}max, \(P<0.001\)) were significantly decreased after IT (Table 2). This decrease was even more pronounced after REC 3 whereas both parameters returned to pre-training values after REC 1. The changes of HR\textsubscript{max}, L\textsubscript{a}max and PO\textsubscript{peak} from T2 to T3 were significantly different between REC 1 and REC 3 (Table 2). For VO\textsubscript{2peak}, no significant effects were observed.
Table 2. Power output and oxygen uptake (VO₂) at the individual anaerobic threshold (IAT) as well as maximal physiological responses during the graded exercise test

<table>
<thead>
<tr>
<th></th>
<th>Recovery (1 h)</th>
<th>Recovery (3 h)</th>
<th>1 vs 3 h</th>
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<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
<td>T3</td>
</tr>
<tr>
<td>IAT (W/kg)</td>
<td>3.71 ± 0.46</td>
<td>3.82 ± 0.44*</td>
<td>3.83 ± 0.43*</td>
</tr>
<tr>
<td>VO₂ (mL/min/kg)</td>
<td>53.8 ± 7.9</td>
<td>55.1 ± 6.0</td>
<td>55.4 ± 5.4</td>
</tr>
<tr>
<td>PO₈ (W/kg)</td>
<td>5.17 ± 0.65</td>
<td>5.03 ± 0.57</td>
<td>5.21 ± 0.53</td>
</tr>
<tr>
<td>VO₂ (mL/min/kg)</td>
<td>56.7 ± 9.6</td>
<td>68.0 ± 6.1</td>
<td>69.1 ± 5.7</td>
</tr>
<tr>
<td>HR (min⁻¹)</td>
<td>192 ± 8</td>
<td>184 ± 10*</td>
<td>188 ± 11</td>
</tr>
<tr>
<td>La max (mmol/L)</td>
<td>11.0 ± 1.9</td>
<td>8.7 ± 1.7*</td>
<td>10.4 ± 2.2</td>
</tr>
</tbody>
</table>

Data as mean ± standard deviation. PO₈, peak power output; VO₂peak, peak oxygen uptake; HRₘₐₓ, maximal heart rate; Laₘₐₓ, maximal blood lactate concentrations.

*Significantly different compared with T1; †Significantly different compared with T2; P-values for 1 vs 3 h are for interaction from the 2 × 2 ANCOVA provided the initial 2 × 3 ANOVA revealed a significant interaction. Thus, P<0.05 indicates a significantly different change of this parameter for 1 vs 3 h active recovery.

Table 3. Average physiological and hormonal responses during the 30-min time trial

<table>
<thead>
<tr>
<th></th>
<th>Recovery (1 h)</th>
<th>Recovery (3 h)</th>
<th>1 vs 3 h</th>
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<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
<td>T3</td>
</tr>
<tr>
<td>PO (W/kg)</td>
<td>3.85 ± 0.53</td>
<td>3.91 ± 0.49</td>
<td>3.88 ± 0.51</td>
</tr>
<tr>
<td>PO (% IAT)</td>
<td>104 ± 7</td>
<td>103 ± 5</td>
<td>101 ± 7</td>
</tr>
<tr>
<td>HR (min⁻¹)</td>
<td>182 ± 8</td>
<td>176 ± 7*</td>
<td>178 ± 9</td>
</tr>
<tr>
<td>La (mmol/L)</td>
<td>5.9 ± 1.7</td>
<td>4.7 ± 1.4*</td>
<td>5.3 ± 2.0</td>
</tr>
<tr>
<td>VO₂ (mL/min/kg)</td>
<td>56.3 ± 6.8</td>
<td>57.3 ± 6.8</td>
<td>56.1 ± 7.0</td>
</tr>
<tr>
<td>VO₂ (% VO₂peak)</td>
<td>81.0 ± 3.9</td>
<td>84.8 ± 7.1</td>
<td>80.6 ± 7.4</td>
</tr>
<tr>
<td>RER</td>
<td>0.94 ± 0.02</td>
<td>0.94 ± 0.02</td>
<td>0.94 ± 0.02</td>
</tr>
<tr>
<td>Testosterone (nmol/L)</td>
<td>0.68 ± 1.15</td>
<td>0.88 ± 0.34</td>
<td>1.07 ± 0.76</td>
</tr>
<tr>
<td>GH (µg/mL)</td>
<td>36.2 ± 14.4</td>
<td>33.8 ± 12.2</td>
<td>32.8 ± 11.6</td>
</tr>
<tr>
<td>Insulin (µg/mL)</td>
<td>−1.9 ± 8.9</td>
<td>−3.0 ± 4.2</td>
<td>−7.3 ± 10.1</td>
</tr>
</tbody>
</table>

Data as mean ± standard deviation. PO, power output; HR, heart rate; La, blood lactate concentrations; VO₂, oxygen uptake; RER, respiratory exchange ratio; GH, growth hormone.

*Significantly different compared with T1. †P-values for 1 vs 3 h are for interaction from the 2 × 2 ANCOVA provided the initial 2 × 3 ANOVA revealed a significant interaction. Thus, P<0.05 indicates a significantly different change of this parameter for 1 vs 3 h active recovery.

Time trial

The mean power output during the time trial (POₜₜ) was not significantly affected by either IT or REC. This was also true when POₜₜ was expressed as a percentage of the IAT (Table 3). Oxygen uptake and respiratory exchange ratio during TT remained unchanged after IT and REC. Heart rates and lactate concentrations were significantly lower after IT compared with the pre-training values. This was also true for heart rate after REC 3. No significant interactions between REC 1 and REC 3 were observed (Table 3).

TT increases in cortisol (P = 0.01), ACTH (P = 0.07) and prolactin (P = 0.001) after REC 3 were reduced at T3 compared with T1 (Fig. 1). The change of the exercise-induced cortisol increase from T2 to T3 was significantly different between REC 1 and REC 3 (P = 0.04). Exercise-induced changes of the other hormone concentrations showed no significant effects (Table 3).

Resting measurements

A significant interaction for the change from T2 to T3 between REC 1 and REC 3 was observed for resting GH concentrations with a decrease after REC 1 and an increase after REC 3. None of the remaining hormonal parameters showed significant effects over time or during the different recovery periods (Table 4).

After both IT periods, a significant increase in the POMS subscore fatigue (P = 0.04) was observed. The change in the total POMS score (P = 0.03) as well as the subscores fatigue (P = 0.01) and vigor (P = 0.02) from T2 to T3 was significantly different between REC 1 and REC 3 (Fig. 2). No significant effects
were observed for the subscores anger and depression, respectively (data not presented).

Discussion

The present study was conducted to evaluate the effectiveness of 4 days of low-intensity cycling training of two different durations with regard to the recovery from 2 weeks of intensified training. The training-induced changes in some ergometric, hormonal and psychometric parameters indicating fatigue were further deteriorated by cycling 3 h each day with an intensity corresponding to the VT. In contrast, active REC of 1 h each day switched those fatigue markers back to pre-training levels. This is in accordance with the previously published results of this study showing a significantly different change of immunological parameters during the recovery periods (Meyer et al., 2004). Therefore, low-intensity training of a short duration (about 1 h) appears to be more appropriate than daily training of a 3-h duration when active recovery is the aim after periods of intensified training (e.g., training camps).

In the present study, power output during the 30-min TT was not significantly affected by IT as well as by REC. In contrast, other training studies with cyclists showed an ~4% reduction in TT performance after an intensified training period of a similar duration (Jeukendrup et al., 1992; Halson et al., 2002; Rietjens et al., 2005) and a recovery – with TT performance exceeding pre-training values – during the following REC period (Jeukendrup et al., 1992). However, training in these studies consisted of several highly intensive interval sessions. This may have caused a more pronounced exhaustion than in the present study. Training in our investigation was designed to improve basic endurance as it is typical for early stages of the preparation period in high-level athletes.

Table 4. Resting hormone values at the different testing days during both training cycles

<table>
<thead>
<tr>
<th>Recovery (1 h)</th>
<th>Recovery (3 h)</th>
<th>1 vs 3 h</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
</tr>
<tr>
<td>T1</td>
<td>T2</td>
<td>T3</td>
</tr>
<tr>
<td>Testosterone (nmol/L)</td>
<td>18.1 ± 6.5</td>
<td>16.8 ± 6.5</td>
</tr>
<tr>
<td>Cortisol (nmol/L)</td>
<td>462 ± 131</td>
<td>415 ± 110</td>
</tr>
<tr>
<td>T/C</td>
<td>0.043 ± 0.021</td>
<td>0.043 ± 0.018</td>
</tr>
<tr>
<td>GH (µU/mL)</td>
<td>0.142 ± 0.080</td>
<td>0.160 ± 0.084</td>
</tr>
<tr>
<td>Insulin (µU/mL)</td>
<td>9.9 ± 5.8</td>
<td>10.0 ± 4.7</td>
</tr>
<tr>
<td>PRL (µU/mL)</td>
<td>260 ± 78</td>
<td>210 ± 50</td>
</tr>
<tr>
<td>ACTH (pmol/L)</td>
<td>7.39 ± 2.97</td>
<td>5.85 ± 2.77</td>
</tr>
</tbody>
</table>

Data as mean ± standard deviation. T/C, testosterone–cortisol ratio; GH, growth hormone; PRL, prolactin; ACTH, adrenocorticotropic hormone. P-values for 1 vs 3 h are for interaction from the 2 × 2 ANCOVA provided the initial 2 × 3 ANOVA revealed a significant interaction. Thus, P<0.05 indicates a significantly different change of this parameter for 1 vs 3 h active recovery.
Heart rate and blood lactate concentrations during TT were significantly reduced after IT. This is in accordance with other studies that reported similar decreases after IT periods (Jeukendrup et al., 1992; Halson et al., 2002). These results might be interpreted as an enhanced aerobic performance capacity so that the organism is less reliant on lactacid pathways for maintaining the same average power output during TT. Depleted glycogen stores seem unlikely, because RER remained unchanged during all TTs and elevated levels of glycolytic hormones (e.g., cortisol, GH) were not observed. In contrast, after REC 3 reduced exercise-induced increases of cortisol and other pituitary hormones (ACTH, prolactin) were observed. This is in line with earlier observations suggesting a major role of pituitary and adrenal hormones in the development of fatigue (Barron et al., 1985; Urhausen et al., 1995, 1998a). An impaired hypothalamic and pituitary function with a consecutive functional limitation of the adrenal glands is hypothesized as one pathomechanism of the OTS (Urhausen et al., 1995, 1998a; Lehmann et al., 1997). Additionally, reduced sensitivity of ACTH receptors of the adrenal cortex cannot be ruled out (Lehmann et al., 1997), particularly because changes in cortisol were more pronounced than those of ACTH.

It is unlikely that subjects in the present study were non-functionally overreached after IT, because it was possible to reverse fatigue within 4 days of active recovery (Meeusen et al., 2006). Therefore, significant changes in hormonal responses to TT were not expected. Otherwise, Meeusen et al. (2004) have shown that exercise-induced increases in hormones of the pituitary–adrenal axis were inhibited in overreached subjects when one maximal exercise bout was performed 4 h before the second one. This is similar to the procedure used in the present study, with the GXT being conducted 2.5 h before TT. Therefore, the suppressed responses of cortisol, ACTH and prolactin after REC 3 might be interpreted as early signs of non-functional OR (Meeusen et al., 2006).

A rightward shift in lactate curves as well as slight but significant increases in the IAT were observed after IT. These changes were augmented after 3 h of daily low-intensity training and could be interpreted in terms of an improved aerobic endurance capacity. Urhausen et al. (1998b) reported lower submaximal lactate values in overreached athletes. This may lead to an overestimation of aerobic endurance capacity. Therefore, the further rightward shift of the lactate curve after REC 3 might be interpreted as indicating an inhibited anaerobic-glycolytic energy production. This is in line with the lower exercise-induced excretion of hormones of the pituitary and adrenal cortex after REC 3.

Previous findings with regard to maximal ergometric performance are not consistent. While some authors observed significantly reduced values after an intensified training period (Fry et al., 1992a; Jeukendrup et al., 1992; Hedelin et al., 2000; Bosquet et al., 2001; Halson et al., 2002), others found no significant effects (Lehmann et al., 1992; Urhausen et al., 1998b; Rietjens et al., 2005). Similarly, the findings regarding VO2peak were inconsistent in the above-cited studies. VO2peak showed no significant changes during the intervention periods in the present study either. Nevertheless, reduced maximal heart rates and blood lactate concentrations as indicators of cardiocirculatory/metabolic effort after an exhaustive training period were detected in most of these studies (Jeukendrup et al., 1992; Lehmann et al., 1992; Urhausen et al., 1998b; Hedelin et al., 2000;
Bosquet et al., 2001; Halson et al., 2002). Such a reduced ability to reach maximal cardiocirculatory/metabolic capacity may be interpreted as a fatigue marker.

The different changes of maximal power output, heart rate and blood lactate concentrations during the recovery periods suggest that low-intensity cycling of a short duration supports the recovery process after IT, whereas exhaustion is intensified by long-duration training at the same intensity.

In contrast to the exercise-induced changes of hormone concentrations, the resting levels of these hormones remained unchanged. Only GH concentrations changed significantly different during both recovery periods. Meeusen et al. (2004) similarly found higher resting GH levels in overreached cyclists. To what extent this behavior is relevant for the recovery process cannot be answered conclusively – particularly when considering the relatively large interindividual variability present. Nonetheless, the scientific literature regarding resting hormone concentrations is controversial. In an attempt to summarize, Urhausen et al. (1995) pointed out that most studies showed no relevant changes after intensified training or when subjects were overreached.

Negative changes in mood states are often described as early markers of OR. Morgan et al. (1987) and O’Connor (1997) reported considerable changes in total POMS scores as well as in the subscores fatigue and vigor during training phases that were characterized by large differences in total training volume. The mood disturbances in the present study are not as distinct as in the cited studies, but they are comparable to those observed by Rietjens et al. (2005), who analyzed training-induced fatigue over a 2-week IT period. The relatively slight mood disturbances are explained by the short intervention period as well as by the large interindividual variability. However, noticeable differences were found between the two recovery periods. Active recovery of 1 h each day reversed the training-induced mood disturbances, whereas 3 h of daily training intensified them.

Up to now, there exists no established physiological background that might explain the underlying mechanisms of the recovery process in high-level athletes. In a recent review (Barnett, 2006), several possible influences on post-training recovery were discussed. Accordingly, possible candidates might be seen in the reversal of exercise-induced metabolic disturbances or muscle damage, adequate rehydration and glycogen re-synthesis, influencing inflammatory processes in muscle repair or the imbalance between stress and recovery. However, from the present results, no conclusive statement with regard to these mechanisms is possible.

Active recovery in this study influenced the hormonal changes within the hypothalamic–pituitary–adrenal axis and the consecutive inhibition of carbohydrate metabolism. Barnett (2006) suggested that early changes in carbohydrate metabolism as well as deteriorations in mood state, which may reflect an imbalance between stress and recovery, might be used to assess the efficacy of recovery modalities. The recovery process could be seen as a reversion of fatigue, which was induced by intensified training. The present findings after REC 3 (decrease in exercise-induced hormone concentrations, decreased maximal heart rate and blood lactate concentrations, deteriorated mood state) can be interpreted as insufficient recovery. In contrast, the reversion of those effects toward pre-training levels after REC 1 can be seen as adequate recovery. However, the underlying mechanisms still need to be addressed in future research.

**Limitations of the study**

A problem in controlled training studies is to recruit a sufficient number of elite athletes, because study participation represents a considerable intervention in their training process. Therefore, it is difficult to reach sufficient statistical power to generalize the study results obtained. However, the number of athletes in the present study is higher than in comparable training studies, where sample sizes have ranged from five to nine subjects (Fry et al., 1992a; Jeukendrup et al., 1992; Lehmann et al., 1992; Halson et al., 2002; Rietjens et al., 2005). The subjects in the present study were cyclists and triathletes of regional to national level. Their aerobic capacities were comparable to those of previous studies (Jeukendrup et al., 1992; Halson et al., 2002; Rietjens et al., 2005). However, there is evidence that differences exist between amateur and professional endurance athletes. Any direct transfer of the present results to professional athletes should therefore be made with caution. It may be speculated that high-level athletes cope better with a longer duration of REC sessions because their usual training amount exceeds that of the subjects in the present study. However, an increase of stress hormone activity with time, as a possible pathomechanism of OR, may also be present in professional cyclists.

A further limitation might be that there exists no scientific research comparing an active recovery regimen with passive rest after an intensified training period. Although such an approach might be limited by the compliance (to several days of total rest) of appropriately well-trained athletes, future research might address this issue.

**Perspectives**

This randomized cross-over study aimed at comparing the effectiveness of 4 days of low-intensity,
discipline-specific training of 1 vs 3 h daily with regard to the recovery process after 2 weeks of intensified training. The results obtained indicate that daily training of 1-h duration at an intensity corresponding to VT supports recovery from 2 weeks of intensified training. In contrast, 3 h of daily training at the same intensity augment the training-induced changes, and may even lead to signs of non-functional OR. Therefore, long-lasting training sessions of several hours should be avoided in active REC, even when they are of low intensity. It seems useful to evaluate the transferability of the present results to high-level athletes by means of single- or few-subject studies with professional endurance athletes.

There are only scarce scientific data regarding the design of adequate regeneration strategies in high-level endurance sports. Further research is needed to analyze, for instance, passive rest, variations in training intensity or the value of cross-training with regard to the recovery process after IT periods. Additionally, scientific efforts are needed to analyze the underlying physiological background of the recovery process in high-level athletes.

Key words: cycling, endurance training, active regeneration, fatigue, over-reaching.

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