Time course of performance changes and fatigue markers during intensified training in trained cyclists

SHONA L. HALSON, MATTHEW W. BRIDGE, ROMAIN MEEUSEN, BART BUSSCHAERT, MICHAEL GLEESON, DAVID A. JONES, AND ASKER E. JEUKENDRUP

1Human Performance Laboratory, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, B15 2TT, United Kingdom; 2School of Human Movement Studies, Queensland University of Technology, Kelvin Grove, Queensland 4059, Australia; and 3Department of Human Physiology and Sportsmedicine, Vrije Universiteit Brussel, B-1050 Brussels, Belgium

Received 26 November 2001; accepted in final form 3 April 2002

Halson, Shona L., Matthew W. Bridge, Romain Meeusen, Bart Busschaert, Michael Gleeson, David A. Jones, and Asker E. Jeukendrup. Time course of performance changes and fatigue markers during intensified training in trained cyclists. J Appl Physiol 93: 947–956, 2002.—To study the cumulative effects of exercise stress and subsequent recovery on performance changes and fatigue indicators, the training of eight endurance cyclists was systematically controlled and monitored for a 6-wk period. Subjects completed 2 wk of normal (N), intensified (ITP), and recovery training. A significant decline in maximal power output (N = 338 ± 17 W, ITP = 319 ± 17 W) and a significant increase in time to complete a simulated time trial (N = 59.4 ± 1.9 min, ITP = 65.3 ± 2.6 min) occurred after ITP in conjunction with a 29% increase in global mood disturbance. The decline in performance was associated with a 9.3% reduction in maximal heart rate, a 5% reduction in maximal oxygen uptake, and an 8.6% increase in perception of effort. Despite the large reductions in performance, no changes were observed in substrate utilization, cycling efficiency, and lactate, plasma urea, ammonia, and catecholamine concentrations. These findings indicate that a state of overreaching can already be induced after 7 days of intensified training with limited recovery.

cycling; overtraining; overreaching; overload

THE BALANCE BETWEEN TRAINING and overtraining is often a very delicate one. Many athletes incorporate high training volumes and limited recovery periods into their training regimes. This may disrupt the fragile balance, and the accumulation of exercise stress may exceed an athlete’s finite capacity of internal resistance. Often this can result in overreaching, defined as an accumulation of training and/or nontraining stress resulting in a short-term decrement in performance capacity, in which restoration of performance capacity may take from several days to several weeks (17). It is generally believed that if the imbalance between training and recovery persists, this may result in an accumulation of training and/or nontraining stress resulting in a long-term decrement in performance capacity, in which restoration of performance capacity may take several weeks or months. This condition is termed overtraining (17).

Increased exercise stress is manifested in physiological and biochemical changes and is often in conjunction with psychological alterations, all of which result from an imbalance in homeostasis (10). However, the quantity of training stimuli that results in either performance enhancement or a chronic fatigue state is presently unknown. Of the current information regarding training regimes and protocols, most is derived from conjectural or experiential sources and has little research support. Because it is difficult to ascertain the volume of training that will result in overreaching or overtraining, it is necessary to identify markers that distinguish between acute training-related fatigue and overreaching.

Similarly, much of our knowledge about overtraining is derived from cross-sectional studies and anecdotal information (2, 14). Although a number of studies have used a longitudinal approach (8, 19, 22, 24, 27), in many cases failure to adequately monitor performance means we know little about the time course of changes of potential indicators of overreaching and early phases of the overtraining syndrome.

The aim of this investigation was to identify the time course of changes in selected physiological, biochemical, and psychological parameters during 2 wk of intensified training and 2 wk of recovery in trained cyclists. To ascertain the time course and fluctuations of these changes, repeated performance tests were conducted. To our knowledge, this is one of the first attempts to systematically induce a state of overreaching while monitoring training stress and performance in a supervised and highly controlled environment. We hypothesize that the intensified training program employed will result in a state of overreaching, identified by a reduction in performance and an increase in global mood disturbance. In addition, we hypothesize that the

Address for reprint requests and other correspondence: A. E. Jeukendrup, Human Performance Laboratory, School of Sport and Exercise Sciences, Univ. of Birmingham, Edgbaston, B15 2TT Birmingham, UK (E-mail: A.E.Jeukendrup@bham.ac.uk).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
intensified training will result in a decrement in performance during the initial period of training; however, consistent elevations in mood disturbance will not occur until later during the intensified training period. It is hypothesized that laboratory-assessed performance will continue to decline throughout the intensified training period and will return to baseline or above baseline levels on completion of a recovery period. Changes in performance will occur along with changes in maximal physiological parameters [i.e., heart rate, oxygen consumption (\(\dot{V}O_2\)); however, substrate utilization, cycling efficiency, and other biochemical indicators will remain unchanged.

**METHODS**

**Subjects.** Eight endurance-trained male cyclists volunteered for this study. All subjects had competed for at least 2 yr and were training a minimum of 3 days/wk. The study was approved by the South Birmingham Local Research Ethics Committee. Before participation, and after both comprehensive verbal and written explanations of the study, all subjects gave written, informed consent. Subject characteristics are presented in Table 1.

**Experimental protocol.** Subjects were familiarized to the test procedures by completing both a maximal cycle ergometer test (MT) and a time trial (TT) in the week preceding the study commencement. No subjects exhibited signs or symptoms of overreaching or overtraining on the basis of the previously mentioned definitions (18); i.e., during the 2 wk of baseline training maximum power output was unchanged and mood state was within normal ranges for athlete populations.

The training of each subject was then controlled and monitored for a period of 6 wk in total, which was divided into three distinct phases each of 2-wk duration (Fig. 1). The first phase consisted of moderate training with a small number of exercise testing sessions. Subjects completed their normal or usual amount and type of training (Fig. 1).

The second phase consisted of an increase in training volume and intensity (Fig. 1) as well as the number of exercise tests performed. Subjects trained 7 days/wk for these 2 wk in addition to the laboratory tests. A similar protocol has previously been shown to induce a state of overreaching in the time frame specified (15). The third phase of the study was one of reduced training (Fig. 1) and aimed to provide subjects with a period of recovery.

To describe the time course of the changes in performance and potential indicators of overreaching, subjects performed three different exercise tests at regular intervals during the examination period (Fig. 1). Each individual test was performed at the same time of day. A total of 20 exercise tests were performed per subject, 10 of which were in the overtraining phase. In total, subjects underwent six MTs, six TTs, and eight intermittent tests (ITs).

**Training quantification.** Each subject received a Polar Vantage NV heart rate monitor (Polar Electro, Kempele, Finland) for the duration of the study. Each subject was given a training diary to record duration of training, distance covered, average heart rate, maximal heart rate, and weather conditions. Subjects recorded all training sessions, which were downloaded to a computer using the Polar Interface (Polar Electro). From this information, average heart rate, maximal heart rate, and time spent in each of the heart rate zones could be calculated and verified against the training diary.

The majority of subjects performed their training outdoors; however, on occasion, subjects trained inside the laboratory if weather conditions prevented them from training outdoors. Subjects were encouraged to consume a carbohydrate-rich diet and to remain euhydrated during the entire experimental period.

After each maximal test, subjects’ training zones were calculated from their individual lactate and heart rate curves. Lactate threshold was determined by using the maximal distance (Dmax) method as described elsewhere (4). Five training zones were calculated and expressed as percentages of individual maximum heart rate. The training zones for the eight subjects before the intensified training period were, on average, as follows: zone 1, \(69\%\) maximal heart rate; zone 2, \(69\%\)–\(81\%\) maximal heart rate; zone 3, \(82\%\)–\(87\%\) maximal heart rate; zone 4, \(88\%\)–\(94\%\) maximal heart rate; zone 5, \(>94\%\) maximal heart rate.

Subjects training programs for the intensive training weeks were based on their current amount of training in the 2-wk baseline period. In the 2 wk of intensive training, the researchers aimed to increase the amount of time the subjects trained in zones 3, 4, and 5. This was achieved by designing individual training programs that doubled normal

<table>
<thead>
<tr>
<th>Week 1</th>
<th>MT</th>
<th>IT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 2</td>
<td>MT</td>
<td>TT</td>
</tr>
<tr>
<td>Week 3</td>
<td>TT</td>
<td>IT</td>
</tr>
<tr>
<td>Week 4</td>
<td>TT</td>
<td>IT</td>
</tr>
<tr>
<td>Week 5</td>
<td>MT</td>
<td>IT</td>
</tr>
<tr>
<td>Week 6</td>
<td>MT</td>
<td>TT</td>
</tr>
</tbody>
</table>

**Fig. 1.** Study design. MT, maximal cycle ergometer tests (maximal oxygen uptake); TT, time trial; IT, intermittent test; shaded areas, moderate- to high-intensity training.
training volumes. The majority of the increase in training volume was in the form of high-intensity training, i.e., above the lactate threshold (Fig. 2).

**MT.** Subjects arrived at the laboratory after an overnight fast, and a Teflon catheter (Becton-Dickinson, Quickcath) was inserted into an antecubital vein. After this, the subjects performed an incremental test to exhaustion on an electrically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) to determine maximal power output (W_max), submaximal and maximal VO_2, and heart rate throughout the test.

Resting data were collected before subjects began cycling at 95 W for 3 min. The load was increased by 35 W every 3 min until volitional exhaustion. Expiratory gases were collected and averaged over a 10-s period, by using a computerized on-line system (Oxycon Alpha, Jaeger, Bunnik, The Netherlands). W_max was determined using the equation

\[ W_{\text{max}} = W_{\text{final}} + (t/T) \cdot W_{\text{inc}} \]  

where W_{final} (W) is the power output during the final stage completed, t (s) is the amount of time reached in the final uncompleted stage, T (s) is the duration of each stage, and W_{inc} (W) is the workload increment.

Heart rate was recorded throughout the exercise test using a heart rate monitor (Polar Vantage NV). Rating of perceived exertion (RPE) was recorded at the end of each stage, by using the modified Borg scale (3).

Blood samples were collected at rest, in the last 30 s of each stage, and immediately after the cessation of the test for the determination of blood lactate, plasma urea, plasma ammonia, and catecholamine concentrations. Blood samples were immediately analyzed for lactate (YSI 2300 STAT Plus, Yellow Springs Instruments, Yellow Springs, OH). Heparinized blood samples were centrifuged for 10 min at 1,500 g. Plasma was stored at −20°C and analyzed for ammonia (model 171-UV, Sigma Chemical, Poole, UK) and urea (model 640, Sigma Chemical).

Plasma epinephrine, norepinephrine, and dopamine were measured by HPLC with electrical detection (BioRad, Nazareth, Belgium). Interassay coefficients of variation for epinephrine and norepinephrine were 7.8 and 4.2%, respectively.

From carbon dioxide production and VO_2, rates of carbohydrate and fat oxidation were calculated by using stoichiometric equations (6).

Gross efficiency (GE) was calculated by using the formula (11)

\[ GE = \frac{\text{power}}{\text{EE}} \cdot 100\% \]  

where EE is energy expenditure.

Cycling economy was calculated by using the formula (28)

\[ \text{Economy} (J/l) = \frac{\text{power}}{V_{O_2}} \]  

**TT.** After a 5-min warm-up at 50% W_max, subjects performed a simulated TT in which a target amount of work was to be completed in as short a time as possible. The amount of work to be performed was calculated by assuming that subjects could cycle at 75% of their W_max for ~60 min, and thus these TTs lasted ~60 min for all subjects. The formula for this is described elsewhere (16) and is as follows

Total amount of work = \( 0.75 \cdot W_{\text{max}} \cdot 3,600 \) s

For each of the TTs, the ergometer was set in the pedaling-dependent mode so as to replicate as accurately as possible a TT in a field setting. Thus power varies with cadence (rpm) and is represented by the following formula

\[ W = L \cdot (\text{rpm})^2 \]  

Hence, the work rate (W) measured in watts is equal to the cadence squared [(rpm)^2] multiplied by the linear factor (L). L was based on each subject’s W_max and calculated so that 75% W_max was produced at a pedaling rate of 90 rpm. With use of Eq. 5, L could be calculated by W/(rpm)^2.

A computer was connected to the ergometer, and work, power, and time were recorded. However, subjects received little information other than the amount of work performed and the present amount of work relative to the total to be completed. Subjects received no feedback on time, W, cadence, or heart rate. Any changes in TT performance in response to the intensified training were determined by examining changes in time taken to complete the set amount of work for each subject.

Subjects were required to fast for at least 3 h before each test. Blood samples were taken before and immediately after each test and were analyzed for lactate. Heart rate was monitored continuously throughout the test (Polar Vantage NV).

**IT.** Unlike the TT, the IT was of a set duration and a change in work production was assessed. Subjects completed a 5-min warm-up at 50% W_max followed by two 10-min bouts of maximal exercise. Each subject was given a 5-min rest between bouts.

Subjects were asked to cycle as “hard” as possible for each of the 10-min bouts. Similar to the TT protocol, the ergometer was set in the pedaling-dependent rate mode. However, in this case the W was set as 90% W_max. The ergometer was again connected to a computer as in the TTs; however, subjects only received information on time and power indicated graphically. Heart rate was recorded continuously throughout the test (Polar Vantage NV).

Any changes in IT performance over time were analyzed by examining both work and power for each of the bouts. Subjects again fasted for at least 3 h before testing. Blood samples were taken before the test and on completion of the second exercise bout and were assayed for lactate concentration.

**Questionnaires.** Every day for the duration of the study, subjects completed both the Daily Analysis of Life Demands of Athletes (DALDA) (29) and the short form of the Profile of
Mood States (POMS) questionnaire (POMS-22) (25). The DALDA is divided into parts A and B, which represent the sources of stress and the manifestation of this stress in the form of symptoms, respectively. Subjects were asked to complete these questionnaires at the same time of each day before training. Subjects also completed the 65-question version of the POMS (25) once a week on the morning of the MT. Global mood state was determined by using the method described by Morgan et al. (26).

Additional measurements. Once per week a number of additional measurements were taken. Skinfold measurements were made to estimate percent body fat, and body weight was also measured. Subjects also recorded their morning resting heart rate every day for the duration of the study with the heart rate monitor (Polar Vantage NV).

Statistical analysis. One-way analysis of variance with repeated measures was used with least significant difference comparison performed to identify significant differences between the individual means. The level of significance was set at 0.05.

RESULTS

Subjects completed 2 wk of normal training (7 ± 2 h/wk), 2 wk of intensified training (14 ± 5 h/wk), and a final 2 wk of recovery training (3.5 ± 2.5 h/wk) (Fig. 2). The laboratory tests were included during the calculation of total hours of training completed in each period. The intensified training period predominantly consisted of high-intensity interval training, with significant increases in training in zones 3, 4, and 5 (450, 200, and 147%, respectively). The previous criteria for the detection of overreaching set by Jeukendrup et al. (15) were used to determine whether the training protocol resulted in overreaching. The criteria to be met were 1) a reduction in performance in the laboratory tests and 2) increased affirmative responses to questionnaires that assessed impaired general health status, negative mood, psychological status, and feelings of fatigue.

All eight subjects completed the intensified training period and met the criteria for overreaching at the conclusion of the 2-wk intensified training phase. Some of the observed responses to intensified training included reduced performance in the form of reduced \( W_{\text{max}} \) during the MT, increased time taken to complete the TT, and a reduction in average work produced during the IT. Maximal heart rate was reduced in all three tests and responses to all questionnaires completed were altered.

MT. After the 2 wk of intensified training, \( W_{\text{max}} \) significantly declined (Fig. 3A) and at completion of the recovery phase was unchanged from baseline values. After 1 wk of intensified training, \( W_{\text{max}} \) during the MT had declined in six of the eight subjects. In the other two subjects, \( W_{\text{max}} \) remained unchanged. On average \( W_{\text{max}} \) declined 3.3% during week 1 of intensified training and 5.4% during week 2 of intensified training. Maximal \( V_{\text{O2}} \) (l/min) significantly declined (4.5%) after the intensified training period; however, it was unchanged after week 1 of intensified training. There were no changes in submaximal \( V_{\text{O2}} \) at 200 W (Table 2).

There was a 15 beats/min decline in maximal heart rate during the MT as a result of the intensified training (Fig. 3B); however, submaximal heart rate (at 200 W) remained unchanged (Table 2). Maximal lactate concentrations from the MT were lower in the intensified training period; however, this did not reach statistical significance (Table 2). There were also no significant changes in resting or submaximal concentrations of plasma lactate (Table 2). RPE scores reported at 200 W were significantly increased during intensified training and after 2 wk of recovery were significantly lower than baseline scores (Table 2).

TT. Time taken to perform the TT significantly increased by 9.8% from 59.4 min during N to 65.3 min at
the end of the first week of intensified training (Fig. 4). Subjects on average took 4 min and 30 s longer to complete the given amount of work. Correspondingly, average power declined during intensified training and slightly but nonsignificantly increased after recovery training (Table 3). A decline in maximal heart rate and average heart rate were noted in the TT after intensified training (Table 3). Resting and maximal blood lactate concentrations from the TT did not change significantly; however, maximal lactate concentrations were unaffected by the intensification period (Table 4). Submaximal plasma epinephrine, norepinephrine, and dopamine concentrations were also not different during the 6-wk training period (Table 4).

Substrate oxidation and cycling efficiency. Carbohydrate and fat oxidation were unchanged over the 6-wk period. Fat oxidation had a tendency to be increased during the intensified training period; however, this was not statistically significant (Table 4). Submaximal efficiency, submaximal economy, and maximal economy were unaffected by the intensified training (Table 4).

Additional measures. Body weight and percent body fat significantly declined throughout the testing period and were lowest after recovery training (Table 4). During the intensified training period, resting heart rate was not different than during normal training; however, it was slightly, but significantly, than lower during recovery training (Table 4).

Questionnaire responses. Global mood state scores on the POMS-65 were significantly increased from 90.4 during normal training to 116.4 during the intensified training period (Fig. 3C). On completion of recovery training, scores returned to 91.5. From this questionnaire, the subscales of tension, fatigue, and confusion were also significantly elevated, whereas vigor significantly declined. No changes were evident in the depression or anger subscales. Altered mood states were also identified by the POMS-22, with significantly elevated total scores.

Parts A and B of the DALDA were both increased during the intensified training period; however, only part B was significantly higher than during normal training (Fig. 6). The most common changes in sources of stress, as identified by part A of the DALDA, were related to sport training, sleep, and health. Part B of the DALDA showed the greatest changes during the the intensified training period, with the majority of subjects showing changes on many of the items. The most common alterations in responses were increased problems associated with the following areas: need for

---

Table 2. Selected changes in maximal test variables over the course of the study period

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>ITP</th>
<th>R</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maximal responses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO₂ max, ml/min</td>
<td>4,271 ± 187</td>
<td>4,372 ± 197</td>
<td>4,391 ± 196</td>
<td>4,078 ± 198†</td>
</tr>
<tr>
<td>VO₂ max, ml·kg⁻¹·min⁻¹</td>
<td>58.0 ± 1.73</td>
<td>59.0 ± 1.49</td>
<td>60.0 ± 1.88*</td>
<td>55.5 ± 1.50†</td>
</tr>
<tr>
<td>Maximal lactate, mmol/l</td>
<td>7.2 ± 0.6</td>
<td>7.8 ± 0.7</td>
<td>7.2 ± 0.7</td>
<td>6.7 ± 0.6</td>
</tr>
<tr>
<td><strong>Submaximal responses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO₂ at 200 W, ml/min</td>
<td>2,852 ± 72</td>
<td>2,766 ± 46</td>
<td>2,873 ± 39</td>
<td>2,821 ± 75</td>
</tr>
<tr>
<td>HR at 200 W, beats/min</td>
<td>152 ± 7</td>
<td>146 ± 8</td>
<td>147 ± 7</td>
<td>145 ± 6</td>
</tr>
<tr>
<td>Lactate at 200 W, mmol/l</td>
<td>1.2 ± 0.2</td>
<td>1.3 ± 0.3</td>
<td>1.1 ± 0.3</td>
<td>1.3 ± 0.2</td>
</tr>
<tr>
<td>RPE at 200 W</td>
<td>9.4 ± 0.8†</td>
<td>9.2 ± 0.9</td>
<td>10.0 ± 1.3</td>
<td>10.9 ± 1.2*</td>
</tr>
</tbody>
</table>

Values are means ± SE. N, normal training; ITP, intensified training; R, recovery; VO₂, oxygen consumption; HR, heart rate; RPE, rating of perceived exertion. *Significantly different from N, P < 0.05. †Significantly different from R, P < 0.05.
Table 3. Selected changes in time trial and intermittent test variables over the course of the study period

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>ITP</th>
<th>R</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time trial</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximal HR, beats/min</td>
<td>179±3</td>
<td>173±3†</td>
<td>168±2†</td>
<td>0.001</td>
</tr>
<tr>
<td>Average HR, beats/min</td>
<td>162±3</td>
<td>161±3</td>
<td>156±2†</td>
<td>0.002</td>
</tr>
<tr>
<td>Power, W</td>
<td>261.7±18.5</td>
<td>240.0±16.2†</td>
<td>239.5±17.1†</td>
<td>0.005</td>
</tr>
<tr>
<td>Resting lactate, mmol/l</td>
<td>1.32±0.23</td>
<td>1.29±0.24</td>
<td>0.93±0.12</td>
<td>0.292</td>
</tr>
<tr>
<td>Maximal lactate, mmol/l</td>
<td>8.06±0.83</td>
<td>5.87±0.24</td>
<td>5.87±1.39</td>
<td>0.198</td>
</tr>
<tr>
<td><strong>Intermittent test</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average work, kJ</td>
<td>181.3±10.1</td>
<td>171.2±10.3</td>
<td>169.8±10.6*</td>
<td>0.001</td>
</tr>
<tr>
<td>Average HR, beats/min</td>
<td>168±5</td>
<td>166±4</td>
<td>160±6*</td>
<td>0.048</td>
</tr>
<tr>
<td>Resting lactate, mmol/l</td>
<td>1.33±0.25</td>
<td>1.22±0.16</td>
<td>1.30±0.30</td>
<td>0.105</td>
</tr>
<tr>
<td>Maximal lactate, mmol/l</td>
<td>10.39±1.25</td>
<td>8.52±1.45</td>
<td>8.35±1.26†</td>
<td>0.017</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significantly different from N, P < 0.05. †Significantly different from R, P < 0.05.

a rest, recovery, irritability, between-session recovery, general weakness, and training effort.

**DISCUSSION**

It is generally assumed that overtraining results from chronic exercise stress in the presence of an inadequate regeneration period. However, there are nondistinct phases in the development of overtraining, which has been termed the overtraining continuum (9). The first phase along this continuum relates to the fatigue experienced after an isolated training session. Further intense training with insufficient recovery can lead to overreaching and increased complexity and severity of symptoms (9). Finally, if high training loads are continued with insufficient recovery from the overreached state, the overtraining syndrome may develop. Currently, there is no literature or information available to discriminate between the early and late phases of this continuum (9). To our knowledge, this is the first study to attempt to identify the changes that occur in the transition from acute fatigue to overreaching.

A number of examinations have measured performance before and on completion of increased volume and/or intensity training (5, 24, 33), whereas few have examined changes in performance during increased training (15, 23). Lehmann et al. (19) reported a number of performance changes before, during, and on completion of 4 wk of increased volume training in runners. Subjects showed a decline in total running distance during incremental ergometric exercise after 2 wk of training; however, performance was not significantly reduced until after 4 wk of training was completed. This may, in part, be explained by the graded increase in volume throughout the 4-wk period. Jeukendrup et al. (15) included examinations of TT performance before, at the midpoint, and after a 2-wk period of intensified training in cyclists. Similar to the present investigation, TT performance had declined significantly after 1 wk of training, although it declined further after an additional week.

The present study incorporated an increased number of performance assessments, including four TTs and four ITs during the increased training period, in addition to initial and recovery assessments. From the information on the time course of performance changes, it appears that overreaching may be induced after a period of 7 days. Although TT performance was decreased during the first several days of increased

---

**Fig. 5.** Time course of changes of the exercise tests, expressed as a percentage of baseline, during normal training, intensified training, and recovery. • and dashed line, MT; ■ and solid line, TT; ▲ and dotted line, IT.
training, subjects could not be considered overreached because mood state was unaltered. On the first day of the intensified training period subjects completed a long-duration, high-intensity ride, and thus the initial decline in performance most likely reflected fatigue from the previous training session. Therefore, although performance was significantly lower than baseline, subjects were acutely fatigued as opposed to overreached. It is likely that complete recovery would have occurred within a few days. However, the continual exercise stress, without regeneration, results in failing adaptation and altered biochemical, physiological, and psychological states (10), which identify the athletes as reaching a chronic as opposed to acute fatigue state.

To determine whether a reduction in performance is the result of acute fatigue from previous exercise, or from overreaching, the DALDA questionnaire may be effective and practical. As described by Rushall (29), a period of baseline assessment should occur, with the recognition that scores may oscillate because of fatigue from isolated training sessions. However, if scores remain elevated for >4 consecutive days, a period of rest should occur. As can be seen from Fig. 6, scores from the questionnaire oscillate during normal training. However, scores are continually and consistently elevated above baseline for the minimum of 4 consecutive days, approximately midway through the intensified training period. Thus, with the use of psychological questionnaires, it is possible to discriminate acute from chronic or excessive fatigue in the presence of uniform performance decrements. Figure 6 suggests that in this particular investigation it took 3–7 days of intensified training before overreaching developed.

Continual intensive training after 7 days does not result in further performance decrements; however, performance is still significantly below that of baseline values. It appears that subjects become somewhat tolerant to the increased training, and this was expressed by a number of the subjects. A number of subjects stated that they had reached a level of maximal fatigue and lethargy after the first week of training. Continued training, therefore, did not result in a decline in performance because of the already high levels of fatigue. Interestingly, mood state continued to decline in the final week of intensified training. All exercise tests employed in this study showed a similar time course of change (Fig. 5); however, the two performance tests (TT and IT) showed a similar magnitude of decline in performance when expressed as change from baseline. It is important to consider the training status of the individuals who participated in this study and to appreciate possible differences in responses between these cyclists and elite or world-class athletes. It is possible that the physiological, biochemical, and psychological responses observed in the present group of moderately trained cyclists may be similar to those of more highly trained cyclists. However, it is impossible

### Table 4. Selected changes in additional measures over the course of the study period

<table>
<thead>
<tr>
<th>Additional Measures</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
<th>Week 5</th>
<th>Week 6</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal plasma epinephrine, nmol/l</td>
<td>4.61 ± 3.36</td>
<td>2.71 ± 2.20</td>
<td>4.67 ± 1.74</td>
<td>1.45 ± 0.70</td>
<td>5.57 ± 3.76</td>
<td>4.97 ± 2.55</td>
<td>0.568</td>
</tr>
<tr>
<td>Maximal plasma norepinephrine, nmol/l</td>
<td>24.82 ± 16.37</td>
<td>18.72 ± 12.71</td>
<td>34.15 ± 15.70</td>
<td>20.48 ± 11.52</td>
<td>33.44 ± 9.90</td>
<td>31.85 ± 13.65</td>
<td>0.541</td>
</tr>
<tr>
<td>Maximal plasma dopamine, nmol/l</td>
<td>12.18 ± 6.69</td>
<td>13.79 ± 6.76</td>
<td>14.91 ± 1.14</td>
<td>17.11 ± 1.23</td>
<td>14.99 ± 1.52</td>
<td>15.25 ± 1.47</td>
<td>0.035</td>
</tr>
<tr>
<td>Maximal economy, J/L</td>
<td>4.7 ± 0.1</td>
<td>4.7 ± 0.1</td>
<td>4.6 ± 0.1</td>
<td>4.7 ± 0.1</td>
<td>4.7 ± 0.1</td>
<td>4.6 ± 0.1</td>
<td>0.361</td>
</tr>
<tr>
<td>Economy at 200 W</td>
<td>4.2 ± 0.1</td>
<td>4.3 ± 0.1</td>
<td>4.2 ± 0.1</td>
<td>4.3 ± 0.1</td>
<td>4.2 ± 0.1</td>
<td>4.4 ± 0.1</td>
<td>0.456</td>
</tr>
<tr>
<td>GE at 200 W</td>
<td>18.0 ± 0.4</td>
<td>19.1 ± 0.3</td>
<td>18.5 ± 0.4</td>
<td>19.1 ± 0.5</td>
<td>18.1 ± 0.4</td>
<td>19.0 ± 0.6</td>
<td>0.223</td>
</tr>
<tr>
<td>CHO oxidation at 200 W, g/min</td>
<td>3.12 ± 0.09</td>
<td>2.75 ± 0.21</td>
<td>2.63 ± 0.22</td>
<td>2.61 ± 0.21</td>
<td>2.84 ± 0.16</td>
<td>2.43 ± 0.19</td>
<td>0.303</td>
</tr>
<tr>
<td>Fat oxidation at 200 W, g/min</td>
<td>0.34 ± 0.04</td>
<td>0.35 ± 0.09</td>
<td>0.46 ± 0.07</td>
<td>0.44 ± 0.07</td>
<td>0.34 ± 0.06</td>
<td>0.41 ± 0.05</td>
<td>0.061</td>
</tr>
<tr>
<td>Resting HR, beats/min</td>
<td>52 ± 1</td>
<td>47 ± 1</td>
<td>54 ± 1†</td>
<td>53 ± 1†</td>
<td>51 ± 1</td>
<td>49 ± 1</td>
<td>0.004</td>
</tr>
<tr>
<td>POMS-22</td>
<td>−0.3 ± 0.7</td>
<td>0.3 ± 0.7</td>
<td>2.0 ± 0.8</td>
<td>2.8 ± 1.3‡</td>
<td>−0.2 ± 0.2</td>
<td>−0.1 ± 0.6</td>
<td>0.019</td>
</tr>
<tr>
<td>Plasma ammonia, μmol/l</td>
<td>38.6 ± 7.5†</td>
<td>60.2 ± 14.4</td>
<td>45.1 ± 15.3</td>
<td>60.8 ± 14.4</td>
<td>40.5 ± 14.9</td>
<td>48.1 ± 12.9</td>
<td>0.067</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>73.7 ± 2.5†</td>
<td>74.0 ± 2.5†</td>
<td>73.2 ± 2.4†</td>
<td>73.3 ± 2.3</td>
<td>72.7 ± 2.2*</td>
<td>72.2 ± 2.3*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>14.6 ± 1.1†</td>
<td>14.3 ± 1.1†</td>
<td>13.8 ± 1.2*</td>
<td>13.2 ± 1.0*</td>
<td>13.1 ± 1.1*</td>
<td>13.3 ± 1.2*</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are means ± SE. GE, gross efficiency; CHO, carbohydrate; POMS-22, short form of the Profile of Mood States questionnaire; DALDA, Daily Analysis of Life Demands of Athletes.

![Fig. 6. Changes in Daily Analysis of Life Demands of Athletes (DALDA) part B “a” scores, indicating that symptoms are “worse than normal” during normal training, intensified training, and recovery.](http://japplphysiol.org/)

---

**J Appl Physiol • VOL 93 • SEPTEMBER 2002 • www.jappl.org**
to speculate on the time course of changes and the volume of training necessary to induce similar alterations in performance in elite cyclists.

At the end of the first week of the intensified training period, it took the subjects 9.8% longer to complete the simulated TT. Given that the daily variation of this test is <3% (16), the major decline in performance can be attributed to the effects of the intensified training protocol. Similarly, Jeukendrup et al. (15) reported a 5% increase in time taken to complete a TT. This increase in time taken to complete an 8.5-km TT (15) is somewhat lower than the 9.8% found in this investigation. The TT performed by the subjects in the present study was of longer duration (to approximate a 40-km TT), which may explain the different results from those of the earlier study (15).

Lehmann et al. (19) reported an 8% decline in total running distance during incremental ergometric exercise in eight middle- and long distance runners who performed 3 wk of increased volume training, from 85 to 174 km/wk. A 29% reduction in time to fatigue was found in a group of five elite soldiers after 10 days of increased-intensity running training (8). A similar decline in time to fatigue on a cycle ergometer (27%) was reported by Urhausen et al. (33) after an undefined individual increase in intensive training. From the information derived from the present study and those mentioned above, it appears that the longer the duration of the exercise bout the larger the impact of overreaching on performance.

Changes in mood state as assessed by the DALDA and POMS occurred alongside the performance reductions. Continued elevated “a” scores, indicating symptoms are “worse than normal” can be a useful tool in determining early overreaching. The subscales of the POMS may be useful to identify psychological aspects that may be disturbed in individual athletes. Changes in mood state are highlighted in the overtraining literature, and the POMS has been found to show significant changes during overreaching and overtraining in other studies as well as the present investigation (7, 13, 27).

Presently, the mechanisms behind the reduction in performance are relatively unknown. Glycogen depletion has been previously suggested as a cause of the underperformance characteristic of overtraining (5). Intensive training may result in a decline in glycogen stores and an increased reliance of fat metabolism. However, the results of this study suggest that substrate metabolism was unchanged after intensified training. Other evidence to suggest that subjects in the present study were not glycogen depleted includes unchanged resting, submaximal, and maximal blood lactate concentrations.

It has also been suggested that some of the performance changes that occur with overtraining and overreaching may be due to reduced efficiency (1). Inadequate recovery of cellular homeostasis can lead to fatigue of motor units and thus additional, less efficient motor units may need to be recruited in a bid to maintain performance (9). To our knowledge, this is the first study to systematically investigate either gross efficiency or economy after a period of intensified training. No changes in either of these variables were noted in this investigation, and thus there is presently no evidence to suggest that changes in gross efficiency or economy can explain the performance deterioration that occurs with overreaching or overtraining. Other suggested mechanisms have included changes in metabolic enzyme concentrations and chronic dehydration (31). However, it would be expected that chronic dehydration would result in an increase in submaximal heart rate to maintain cardiac output. This was not evident in this study or other overtraining investigations (15, 19, 30, 33).

The mechanism(s) for the reduced maximal performance appears to be related to the generation of fatigue before the maximal engagement of the cardiorespiratory and/or metabolic systems. The underlying cause(s) of fatigue is not clear. However, from this study it appears that subjects demonstrate an increased perception of exertion, identified by significantly higher submaximal RPE scores.

During the intensified training period, maximal heart rate was decreased in all three performance tests. Jeukendrup et al. (15), Lehmann et al. (19), and Urhausen et al. (33) all reported reduced maximal heart rates after increased training. This may possibly be the result of a reduced power output observed during maximal exercise. At this stage, however, it is not clear whether the decreased maximal heart rate and possibly a decreased cardiac output are the cause or the consequence of premature fatigue. There have been suggestions that disturbances in the autonomic nervous system are responsible for the altered heart rate during overtraining (20). Decreased sympathetic influence and/or increased parasympathetic influence, decreased β-adrenoreceptor number or density, increased stroke volume, and plasma volume expansion are all possible mechanisms for the reduction in maximal heart rate (34). However, strong evidence for any of these mechanisms is lacking.

Lehmann et al. (19) reported a tendency toward increased stroke volume after an increase in training volume in middle- and long-distance runners. This was in conjunction with a decreased maximal heart rate. A recent study by Hedelin et al. (12) reported increased plasma volume and reduced maximal heart rates after a 50% increase in training volume in elite canoeists. Although performance was not assessed after recovery and therefore it could not be determined whether the athletes were fatigued or overreached, there was no relationship between the changes in maximal heart rate and changes in blood volume.

Decreases in maximal heart rate may also be the result of a downregulation of the sympathetic nervous system or changes in parasympathetic/sympathetic tone. A number of investigations have examined changes in plasma and urinary catecholamine production during periods of intensified training that resulted in overreaching or overtraining (12, 19, 32). Lehmann et al. (19) reported decreased nocturnal urinary norepineph-
rine and epinephrine excretion and increased submaximal plasma norepinephrine concentration after an increase in training volume. Submaximal and maximal heart rates significantly declined along with the changes in catecholamines. However, the findings of unchanged catecholamine concentrations and significantly decreased maximal heart rates, as evidenced during the present study, have also been reported (12, 32). Unchanged resting, submaximal, and maximal free epinephrine and norepinephrine concentrations were described by Urhausen et al. (32) in underperforming cyclists and triathletes over a 15-mo period. Although catecholamine concentration remained stable, maximal heart rate was significantly reduced. Finally, Hedelin et al. (12) also reported decreased maximal heart rates, yet no changes in resting catecholamine production were observed. Thus there does not appear to be a consistent relationship between changes in heart rate and changes in catecholamine concentration. However, downregulation of β-adrenoceptors, or a decrease in receptor number, may occur as a result of the prolonged exposure to catecholamines that can occur as a result of intensified training and/or psychological stress (20, 34). This may be one unexplored alteration that could explain the reduction in maximal heart rate observed in overtrained athletes. The lack of change in maximal lactate concentration at the end of MT is in contrast to other investigations (15, 19, 21). Although maximal lactate concentrations fell from 7.2 to 6.7 mmol/l, this was not statistically significant. The reduction in maximal lactate concentration observed in the overtraining literature has previously been suggested to result from reduced glycogen stores (5). However, both carbohydrate and fat oxidation were unchanged during the intensified training period, and resting lactate concentration was also unaltered.

Changes in morning heart rate, body weight, and percent body fat have previously been suggested as markers of overtraining. However, many studies have failed to show changes in these variables as a result of intensified training (8, 15, 19, 33). Individual variation may partly explain the lack of changes in resting heart rate from baseline.

Conclusion. Decreased performance was observed almost immediately after the onset of increased training, which is likely the result of acute fatigue from the initial training sessions. Successive training stimuli resulted in further fatigue, reductions in performance, and increased mood disturbance in the group of subjects studied. After 7 days of intensified training, a state of overreaching developed. Maximum heart rate was dramatically reduced and perception of exertion was increased. Changes in substrate utilization and cycling efficiency and economy were unrelated to performance changes associated with overreaching and thus cannot explain the increased fatigue and decreased performance.

We thank Chris Dewaele and Luc Vanmelckebeke for assistance in analyzing plasma catecholamines.

REFERENCES


