Introduction

Maintaining a healthy balance between training load and recovery by prescribing the most appropriate amount of recovery time after a training session is one of the most challenging aspects of attaining peak performance. In most cases, these decisions are based on the subjective feedback of an athlete and the behavior of the athletes that an experienced coach or trainer perceives. As training load increases and competition events become longer – such as triathlons, multiday bike races or ultramarathons – the difficulty in quantifying the impact of a race and/or training session and the subsequent determination of the appropriate amount of recovery time required after these events become more complex.

The assessment of blood markers [12, 13] and hormones [28] have shown to be promising in quantifying the effects of training. In support of this, Hecksteden et al. [12] recently reported increased urea levels and decreased insulin-like growth factor 1 lev-
els with increased fatigue after a 6-day training camp in endurance cyclists, team sport players and strength athletes. In addition, Muijka et al. [23] reported that a professional triathlete required 5–8 days to recover from a long-distance triathlon race for creatine kinase levels to return to baseline levels. Although these measurements show great potential in contributing to monitoring athletes more accurately [13], these tests are generally perceived as too invasive to be conducted on a weekly or even daily basis. Therefore, a noninvasive submaximal performance test that could be used on a daily or weekly basis could potentially function as an initial screening tool to monitor athletes and/or determine which athletes require additional screening with invasive techniques.

Many noninvasive submaximal tests have been developed, validated and can be performed on a regular basis, such as the Heart-rate Interval Monitoring System (HIMS) [18], the Lamberts and Lambert Submaximal Cycle Test (LSCT) [20] and the Submaximal Ergometer Rowing Test (SERT) [25]. Some of these were developed for use in interval sports (such as soccer and rugby) and others for the use of sporting equipment like in cycling and rowing. Various studies have shown that these submaximal tests are reliable and have good correlations with maximal performance parameters [18, 20, 25]. In addition, several papers have shown that these types of tests are able to monitor changes in training status and reflect maladaptation to training [4, 9, 19].

In 2009 Lamberts [19] reported increased ratings of perceived exertion (RPE) levels with higher submaximal power outputs (at the same submaximal heart rate) and a faster 60-second heart rate recovery (HRR60S) rate, after a strong increase in training load in a world-class cyclo-cross cyclist. These findings were counterintuitive, because one would expect lower submaximal heart rate and a slower HRR after an increased training load, which resulted in increased fatigue levels. However, in 2015 a part of these counterintuitive responses to a high training load and fatigue were confirmed by Aubrey et al. [1], who reported a faster HRR in 22 triathletes after a 3-week overload training camp. In addition, Mann et al. [21] in 2016 reported increased RPE levels and faster HRR60S rates in 10 runners after an 87 km ultramarathon. However, no differences were found in submaximal heart rate during the run, which can possibly be explained by the fact that the run was clamped at about 70% of VO2 max and runners were tested 2–4 days after the ultramarathon.

Otter et al. [24] studied the effects of a sudden change in perceived psychosocial stress and recovery (negative life events) in 16 runners. As part of her study, she used a submaximal running test, which was based on the LSCT and during which submaximal heart rate and running economy were captured. Otter [24] reported a significant decrease in running efficiency after a negative life event, whereas no changes in submaximal heart rate were found.

However, the effects of fatigue on heart rate and RPEs at different submaximal running intensities in combination with HRR have not yet been studied together. Therefore, the aim of the study was to determine if submaximal heart rates and RPEs (at different submaximal running intensities) and HRR60S changed after a fatigue-inducing 56 km ultramarathon. All testing was scheduled exactly 7 days before and 2 days after the ultramarathon and at the same time of the day to improve the research design and minimize the effect of confounding factors in contrast to the study by Mann et al. [21].

### Materials and Methods

#### Subjects

The sample size for this study was determined by the day-to-day variability of submaximal HR and HRR60S, with the smallest meaningful difference established as 9 beats per minute and a standard deviation of 6 beats per minute [16]. Based on these parameters, the minimum number of runners required to achieve a statistical power of 80% and a significance level of 5% was n = 7.

Due to the nature of an ultramarathon and the risk of runners dropping out of the study, 15 endurance runners (4 female and 11 male) were recruited to participate in this study. All runners were experienced, completed multiple marathons and trained 35 to 100 km wk^-1 on average. All participants were required to be injury-free for at least 6 months, to have completed at least one marathon within the last 12 months and must have entered the 56 km Old Mutual 2 Oceans Ultra Marathon in Cape Town, South Africa. Prior to study participation, all runners signed an informed consent form, and ethical approval for the study was given by the local Human Research Ethics Committee. The study was conducted according to international standards [10] and the 2013 principles of the Declaration of Helsinki were followed.

#### Experimental overview

All participants visited the laboratory on 3 different occasions, once for familiarisation purposes and a maximal performance test (visit 1) and twice for a submaximal run and the assessment of muscle soreness and overall well-being (visit 2 and 3). Visit 1 was scheduled 2 weeks before the 56 km ultramarathon, and tests 2 and 3 were scheduled 7 days before and 2 days after the ultramarathon, respectively. During visit 1, all runners were asked to complete a short training history questionnaire, and their weight, height and 7 skinfolds (triceps, biceps, subscapular, abdomen, sub-iliac, thigh, calf) were also captured. In addition, after being familiarised with the treadmill, runners completed a peak treadmill running speed (PTRS) test. Exactly 7 days before (visit 2) and 2 days after the ultramarathon (visit 3), runners performed the Lamberts Submaximal Running Test (LSRT). Before the runners started the LSRT, they first completed the Daily Analysis of Life Demands of Athletes (DALDA) questionnaire [27] and scored their delayed onset muscle soreness (DOMS) on a visual analogue scale (VAS) (DOMS-VAS) [21].

Runners were requested to refrain from any strenuous exercise 24 h prior, maintain the same eating habits and refrain from any caffeine in the last 3 h before the test, and testing was scheduled at the same time of day (within 30 min). All tests were performed in the same laboratory on the same treadmill with the participants wearing the same outfit and running shoes; the ambient temperature (22.5 ± 1.0 °C) and humidity (41.7 ± 2.4%) were kept constant.

#### Peak treadmill running speed (PTRS) test

After a 5-min self-paced warm-up, during which the runners familiarised themselves with the motorized treadmill (Viasys, LE-500-CE, Nussdorf-Traunstein, Germany), the runners were fitted with a heart rate monitor (Suunto T6D, Vantaa, Finland), and encouraged to try to perform to the best of their abilities. The peak treadmill running (PTRS) tests was started at a speed of 10 km · h^-1 for 1 min,
after which the speed was progressively increased by 0.5 km·h⁻¹ every 30 s, as previously described [21]. During the test the runners were strongly encouraged to perform to their maximal effort. Heart rate was captured continuously, and predicted maximal oxygen uptake (VO₂max) was calculated based on the linear relationship between PTRS and VO₂max measurements in our laboratory database (unpublished data) and the following equation:

\[ \text{Predicted VO}_2\text{max} = (\text{PTRS} \times 2.849) + 8.553 \]

**Lamberts Submaximal Running Test (LSRT)**
The Lamberts Submaximal Running Test (LSRT) is based on Lamberts and Lambert Submaximal Cycle test (LSCT), which also exists for 3 intensity stages. Within the LSRT, runners are asked to run for 6 min at 60 % and at 70 % and 3 min at 85 % of their personal PTRS (Fig. 1). In contrast to Otter et al. [24], metabolic gas analysis was not captured during the LSRT, while the intensity of stage 1 was slightly adapted and the measurement of heart rate recovery was added to the protocol. Heart rate was captured continuously throughout the test, and runners were asked to rate their RPE on a 6–20 RPE Borg scale. Upon completing the third stage of the LSRT, subjects were asked to stand still and not speak for 60 s so that the HRR₆₀s could be captured [5, 14, 15]. As part of this process, runners were asked to side-step (one leg each side) off the treadmill belt while holding onto the treadmill support bars, after which the treadmill was stopped, and the runner could step back on and recover for the remainder of the 60s.

HRR₆₀s was calculated as the difference between mean heart rate of the last 16 s of stage 3 and the last 16 s of the end of the 60-second recovery period [5, 15]. Mean submaximal heart rate was calculated over the last 5 min of each stage 1 and 2 and over the last 2 min of stage 3 (Fig. 1), and RPE for each stage was captured 30 s before the end of each stage [2].

**Delayed Onset Muscle Soreness (DOMS)**
Delayed onset muscle soreness (DOMS) before and after the 56 km ultramarathon was measured with a VAS [21]. Runners were asked to rate the DOMS-VAS score of their quadriceps, hamstrings and calf muscles while ‘in rest’, ‘during daily activities’, ‘during passive stretching’ and ‘during applying pressure’ on a 10 cm VAS scale, which ranged from 0 (‘no pain’) to 10 (‘unbearable pain’) [21].

**Daily Analysis of Life Demands of Athletes questionnaire (DALDA)**
In contrast to other questionnaires, the Daily Analysis of Life Demands of Athletes (DALDA) was specifically developed for athletes and with the aim of reflecting sources and symptoms of perceived stress [27]. The DALDA has 34 items and consists a ‘sources of stress’ part with 9 questions (Part A) and a ‘symptoms of stress’ part with 25 questions (Part B). All subjects completed the questionnaire by rating each question/term by “worse than normal” score (‘a’), a “same as normal” score (‘b’) or a “better than normal” score (‘c’). The DALDA has shown to be able to reflect levels of fatigue and stress in athletes [8, 17, 26].

**Statistical analysis**
The data was analyzed with STATISTICA 13.0 (StatSoft Inc., Tulsa, OK, USA). The homogeneity of variance of the data was assessed with Kolmogorov-Smirnov and Lilliefors test. Data are expressed as mean ± standard deviation (SD) for all parametric data (height, weight, body fat percentage, HR and HRR values during the LSRT), while nonparametric data (VAS scores relating to DOMS and DALDA scores) were expressed as median (interquartile ranges). The DALDA was assessed by the number of ‘a’ scores that were scored before and after the ultramarathon. Differences in HR and HRR before and after the ultramarathon were analyzed with a t-test for dependent samples, whereas differences in DOMS-VAS and DALDA scores were analyzed with a Wilcoxon t-test for paired observations. Statistical significance was accepted at P < 0.05. Cohen’s effect sizes were also calculated and described as trivial = < 0.2, small = 0.2 to < 0.5, moderate = ≥ 0.5 to < 0.8, or large = ≥ 0.8. The meaningful difference for magnitude-based inferences was set at Cohen’s level 0.3 with a standard deviation times 0.2, as recommended by Hopkins [29]. In addition, magnitude-based inferences were used to calculate the probability percentages and were given the appropriate qualitative descriptor. These analyses were performed calculated using a spreadsheet obtained from http://sportsci.org [29].

**Results**
All 15 runners completed the 56 km ultramarathon. However, one runner could not participate in post ultramarathon testing due to an injured ankle. All the data for this runner was excluded from further analysis. The descriptive and performance characteristics of the remaining 14 runners are shown in Table 1.

**Lamberts Submaximal Running Test (LSRT)**
Submaximal heart rate responses, ratings of perceived exertion and heart rate recovery rates during the LSRT 7 days before and 2 days after the ultramarathon are shown in Table 2.

The individual responses of submaximal heart rate, RPE, and HRR₆₀s before and after the ultramarathon are shown in Fig. 2.
Individual responses in submaximal heart rate (▶ Fig. 2a,b,c), showed that 13 runners (92.8%) had a lower heart rate during stage 2, while 9 runners had a lower (64.3%), 2 had an unchanged (14.3%) and 3 runners had a higher (21.4%) submaximal heart rate during stage 3 of the LSRT (▶ Fig. 2a,b,c). Individual RPE responses (▶ Fig. 2d,e,f) showed that 13 runners (92.8%) scored a higher RPE during stage 1, while during stage 3, 9 runners reported a higher (64.3%), 4 runners reported no change (28.6%) and 1 runner (7.1%) reported a lower RPE. Although no significant differences were found in overall RPE scores during stage 2, individual responses showed that 9 runners reported a higher RPE (64.3%), 1 runner reported an unchanged (7.2%) and 4 runners reported a lower RPE (28.6%). Individual HRR<sub>60s</sub> responses (▶ Fig. 2g) showed that all runners had a faster HRR after the ultramarathon (100%).

Delayed Onset of Muscle Soreness (DOMS) questionnaire

The DOMS-VAS scores 7 days before and 2 days after the 56 km ultramarathon are shown in ▶ Table 3. Significantly higher DOMS-VAS scores were found in all muscle groups in every different condition (P < 0.01).

#### Table 1 Descriptive and performance characteristics of the 14 runners that completed the study. Data is expressed as mean ± SD.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Male (n = 11)</th>
<th>Female (n = 3)</th>
<th>All (n = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Descriptive characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>37 ± 8</td>
<td>41 ± 4</td>
<td>38 ± 8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179 ± 7.0</td>
<td>167 ± 4.0</td>
<td>176 ± 8.0</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>75.5 ± 7.1</td>
<td>61.1 ± 11.6</td>
<td>71.7 ± 9.9</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>13.5 ± 4.1</td>
<td>20.6 ± 1.7</td>
<td>15.0 ± 4.8</td>
</tr>
<tr>
<td><strong>Training and performance characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-reported best marathon time (hh:mm:ss)</td>
<td>03:36:56 ± 0:34:32</td>
<td>03:59:00 ± 0:01:00</td>
<td>03:44:04 ± 0:26:37</td>
</tr>
<tr>
<td>Total training distance (km · wk&lt;sup&gt;−1&lt;/sup&gt;)</td>
<td>62.3 ± 22.1</td>
<td>49.3 ± 12.5</td>
<td>59.5 ± 20.7</td>
</tr>
<tr>
<td>Peak Treadmill Running Speed (PTRS) (km · h&lt;sup&gt;−1&lt;/sup&gt;)</td>
<td>19.1 ± 1.1</td>
<td>16.2 ± 1.0</td>
<td>18.4 ± 1.6</td>
</tr>
<tr>
<td>Predicted VO&lt;sub&gt;2max&lt;/sub&gt; (ml · min&lt;sup&gt;−1&lt;/sup&gt; · kg&lt;sup&gt;−1&lt;/sup&gt;)</td>
<td>62.8 ± 3.2</td>
<td>54.6 ± 3.0</td>
<td>61.1 ± 4.6</td>
</tr>
<tr>
<td>56 km ultramarathon time (hh:mm:ss)</td>
<td>05:04:03 ± 0:45:48</td>
<td>06:28:05 ± 00:31:32</td>
<td>05:43:45 ± 0:48:23</td>
</tr>
</tbody>
</table>

**Table 2** Parameters measured as part of the LSRT before (pre) and after (post) 56 km ultramarathon.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre</th>
<th>Post</th>
<th>Mean difference (95 % CI)</th>
<th>p-value</th>
<th>Cohen's Effect Size (95 % CI)</th>
<th>Cohen's Descriptor</th>
<th>Percentage Change positive/trivial/negative (qualitative descriptor)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 1–60 % of PTRS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>135 ± 11</td>
<td>134 ± 12</td>
<td>-0.7 (-4.4–3.0)</td>
<td>0.681</td>
<td>0.1 (0.0–0.4)</td>
<td>Trivial</td>
<td>5/75/19 likely trivial</td>
</tr>
<tr>
<td>RPE (units)</td>
<td>10 ± 2</td>
<td>12 ± 1</td>
<td>2.0 (0.8–3.2)</td>
<td>0.002</td>
<td>1.0 (0.4–1.5)</td>
<td>Large</td>
<td>99/1/0 very likely positive</td>
</tr>
<tr>
<td><strong>Stage 2–70 % of PTRS</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>161 ± 9</td>
<td>157 ± 9</td>
<td>-3.4 (-4.8–-1.9)</td>
<td>&lt; 0.001</td>
<td>0.4 (0.0–0.5)</td>
<td>Small</td>
<td>0/2/98 very likely negative</td>
</tr>
<tr>
<td>RPE (units)</td>
<td>14 ± 1</td>
<td>15 ± 2</td>
<td>0.6 (-0.4–1.6)</td>
<td>0.189</td>
<td>0.4 (0.0–1.1)</td>
<td>Small</td>
<td>76/21/3 likely positive</td>
</tr>
<tr>
<td><strong>Stage 3–85 % of PTRS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>177 ± 8</td>
<td>175 ± 8</td>
<td>-2.1 (-3.6–-0.6)</td>
<td>0.002</td>
<td>0.2 (0.0–0.4)</td>
<td>Small</td>
<td>0/29/71 possibly negative</td>
</tr>
<tr>
<td>RPE (units)</td>
<td>17 ± 1</td>
<td>18 ± 1</td>
<td>0.9 (0.3–1.5)</td>
<td>0.006</td>
<td>0.8 (0.3–1.3)</td>
<td>Large</td>
<td>98/1/0 very likely</td>
</tr>
<tr>
<td><strong>Recovery</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR&lt;sub&gt;end of test&lt;/sub&gt; (bpm)</td>
<td>177 ± 8</td>
<td>177 ± 9</td>
<td>-0.5 (-2.3–1.3)</td>
<td>0.566</td>
<td>0.1 (0.0–0.3)</td>
<td>Trivial</td>
<td>1/91/8 likely trivial</td>
</tr>
<tr>
<td>HRR&lt;sub&gt;60s&lt;/sub&gt; (beats)</td>
<td>42 ± 8</td>
<td>48 ± 7</td>
<td>6.8 (5.2–8.4)</td>
<td>&lt; 0.001</td>
<td>0.8 (0.6–1.0)</td>
<td>Large</td>
<td>100/0/0 most likely positive</td>
</tr>
</tbody>
</table>

Data is expressed as raw data (mean ± SD), mean differences (95 % confidence intervals (95 % CI), standardized mean difference (Cohen's unit and descriptors), and as the percentage change that the difference is clinically relevant (percentages and descriptors).
Daily Analysis of Life Demands of Athletes (DALDA) questionnaire
No significant differences in the number of ‘sources of stress’ were found before (1.5(0.0–2.0)) and after (1.5(1.0–2.0)) the ultramarathon (P = 0.76). However, the runners experienced more ‘symptoms of stress’ after the marathon (7.5(4.0–9.0)) than before (3.0(1.0–3.0)) the marathon (P = 0.028).

Discussion
The current study aimed to determine which submaximal parameters, measured during the LSRT, would change with fatigue after completing a 56 km ultramarathon. Significantly increased DOMS-VAS scores confirmed the overload stimulus of the ultramarathon, while DALDA scores confirmed that our runners were fatigued after the ultramarathon.

The main findings of this study are that submaximal heart rate was lower during stage 2 (on average 3 beats) and 3 (on average 2 beats) after the ultramarathon, while RPE levels were higher during stage 1 (on average 2 units) and stage 3 (on average 1 unit). In addition, HRR_60s was significantly faster (7 beats) after the ultramarathon.

Magnitude-based inferences indicated that the lower submaximal heart rate during stage 2 seems to be very likely clinically relevant (98% change), while the lower heart rate during stage 3 of the LSRT was possibly clinically relevant (77% change). The increase in the RPE during stage 1 of the LSRT was very likely to be clinically relevant (99% change), while the increase in RPE during stage 2 (76% change) was likely and during stage 3 (98% change) was very likely...
to be relevant. The faster heart rate recovery after stage 3 of the LSRT had the most likelihood of being clinically relevant (100 % change).

Interesting to note is that alongside the fatigue after the ultramarathon, lower submaximal heart rates at the same running intensity and a faster HRR_{60s} were observed. These findings are counterintuitive, because an improvement in training status will also be reflected by lower submaximal heart rates and a faster HRR [14]. However, the response in RPE is different between a change in training status and a stage of fatigue. While RPEs remain unchanged at the same exercise intensity with a change in training status [14], RPEs increased at the same exercise intensity with fatigue and/or overreaching. This was most clearly seen during stage 1 and 3 of the LSRT.

However, more importantly, there findings emphasized that a multiparameter approach needs to be used when monitoring athletes because interpreting only one can possibly lead to drawing the wrong conclusions and/or training prescription.

In line with our findings, Mann et al. [21] reported increased RPE levels and a faster HRR after an 87 km ultramarathon. Runners in this study completed a 20-min steady-state run at 70 % of VO_{2max} before and 3 to 4 days after completing an 87 km ultramarathon. In contrast to our study and although submaximal heart rate decreased on average from 153 to 148 bpm, submaximal heart rate was not significantly lower during the steady-state run after the 87 km ultramarathon. Possible explanations for not finding a significantly lower submaximal heart rate might be clamping the exercise intensity to 70 % of VO_{2max}, which may have resulted in running speed variations, and/or not testing all the subject at the same time interval after the ultramarathon (3–4 days vs. exactly 2 days).

Another study that supports our finding is a study by Hammes et al. [9], who studied 23 cyclists after a 6-day high-intensity interval training period. They used the LSCT to monitor the cyclists, which clamps cyclists at 60, 80 and 90 % of heart rate maximum while monitoring submaximal power output, RPE and HRR. After the overload training camp, RPEs were higher during stage 2 (95 % likelihood of being clinically relevant) and stage 3 (100 % likelihood of being clinically relevant) of the LSCT, while HRR was faster (91 % likelihood of being clinically relevant). In addition, Hammes et al. [9], reported higher power outputs during stage 2 (100 % likelihood) and stage 3 (92 % likelihood) of the LSCT. These increases in power output are in line with lower submaximal heart rates at the same exercise intensity (LSRT), as more power will be needed within the LSCT to elicit the same submaximal heart rate.

The findings by Hammes et al. [9] are similar to the findings by Ducroix et al. [6], who studied 6 elite female professional cyclists during an 8-day overload training camp. After the camp, Ducroix reported increased power outputs during stage 2 (99 % likelihood of being clinically relevant) and stage 3 (90 % likelihood), with increased RPE levels during stage 2 (100 % likelihood) and stage 3 (99 % likelihood) of the LSCT. Although HRR could not accurately be measured after day 8 of the training camp because the target HR could not be reached during stage 3 of the LSCT, HRR was significantly faster 5 days into the training camp (90 % likelihood of being clinically relevant).

Possible mechanisms that can explain a lower submaximal heart rate and a faster HRR vary from increased plasma volume after an exercise bout to reduced β-adrenergic receptor sensitivity and autonomic neuromodulation (increased parasympathetic activity with decreased sympathetic activity) [3, 7, 11, 28].

Buchheit et al. [3] reported a faster HRR, increased heart rate variability indices (HRV) and a 4.8 % increase in plasma volume after a supramaximal exercise session. It was hypothesized that the increased higher blood volume would have resulted in a higher stroke volume that affects the cardiovascular mechanoreceptors, which are interlinked with the regulation of the autonomic nervous system [3]. Although we did not observe changes in weight that may have been indicative of large changes in plasma volume, it is plausible that a change in plasma volume may have contributed to our findings.

In addition, Fry et al. [7] reported a decrease in β2 adrenergic receptor density, resulting in a decreased sensitivity of the β2 adrenergic receptors after a 2-week overload training camp. Similarly, Sagnol et al. [28] reported a decrease in free plasma catecholamine levels after a 10-h triathlon, which contributed to depleted adrenaline reserves and a suppressed adrenal medulla response. Both these mechanisms may have influenced the observed lower submaximal heart rate and faster HRR in this study.

The amount of perceived stress, which we used as a marker of fatigue, was determined through the DALDA questionnaire and increased significantly after the ultramarathon. These findings are in line with Halson et al. [8], who reported increased stress symptoms in eight elite cyclists after a 2-week intensified training block. The increased symptoms of stress were accompanied by a decline in performance, lower maximal heart rate and increased RPE levels, while no changes were found in substrate utilization, cycling effi-

### Table 3

Perceived muscle soreness levels before (pre) and after (post) the 56 km ultramarathon. The data is expressed as median and interquartile ranges.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre</th>
<th>Post</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadriceps</td>
<td>4.5 (3.0–5.0)</td>
<td>22.5 (8.0–51.0)</td>
<td>0.004</td>
</tr>
<tr>
<td>During daily activities</td>
<td>4.0 (3.0–5.0)</td>
<td>26.5 (10.0–50.0)</td>
<td>0.002</td>
</tr>
<tr>
<td>During passive stretching</td>
<td>4.0 (3.0–5.0)</td>
<td>41.5 (11.0–57.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>When applying pressure</td>
<td>4.0 (3.0–5.0)</td>
<td>35.5 (12.0–62.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>4.0 (3.0–6.0)</td>
<td>11.0 (7.0–31.0)</td>
<td>0.004</td>
</tr>
<tr>
<td>During daily activities</td>
<td>4.0 (3.0–5.0)</td>
<td>17.0 (9.0–32.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>During passive stretching</td>
<td>4.0 (3.0–5.0)</td>
<td>15.0 (10.0–33.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>When applying pressure</td>
<td>4.5 (3.0–5.0)</td>
<td>20.0 (10.0–37.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>Calf muscles</td>
<td>In rest 4.0 (3.0–4.0)</td>
<td>9.0 (4.0–25.0)</td>
<td>0.004</td>
</tr>
<tr>
<td>During daily activities</td>
<td>4.0 (3.0–5.0)</td>
<td>17.0 (10.0–43.0)</td>
<td>0.002</td>
</tr>
<tr>
<td>During passive stretching</td>
<td>4.0 (4.0–8.0)</td>
<td>30.0 (10.0–55.0)</td>
<td>0.007</td>
</tr>
<tr>
<td>When applying pressure</td>
<td>4.5 (4.0–11.0)</td>
<td>39.0 (10.0–73.0)</td>
<td>0.002</td>
</tr>
</tbody>
</table>
ciency, lactate, urea, ammonia and catecholamines. Based on these findings, Halson [8] concluded that the DALDA is an effective monitoring tool to detect early overreaching. In support of this statement, Robson-Ansley et al. [26] also reported increased DALDA scores (symptoms of stress) in 8 endurance-trained athletes after 2 weeks of intense interval training. In contrast to Halson, [8] Robson-Ansley [26] reported significantly elevated creatine kinase and plasma interleukin-6 levels while total leukocyte and neutrophil counts, plasma cortisol and salivary IgA remained unchanged.

Conclusions
The LSRT shows good potential to be a practical and noninvasive monitoring tool to monitor fatigue. Fatigue and/or a state of over-reaching after an ultramarathon was reflected within the LSRT by lower submaximal heart rates at the same running intensities, by higher rating of perceived exertion at the same running intensity and a faster HRr. From a standpoint of clinical significance, a lower submaximal heart rate during stage 2 (98 %), a higher RPE during the stage 3 (98 %) and faster heart rate recovery after the LSRT were most sensitive at reflecting this status of fatigue and/or overreaching.

As the individual interpretation of these parameters might be counterintuitive and possibly can lead to incorrect interpretation, a multifactorial approach should always be used when monitoring athletes over time. In addition, we recommend that a subjective fatigue questionnaire, such as the DALDA questionnaire, is used in combination with a submaximal monitoring test.

We would like to emphasize that both the LSRT and LSCT were developed as a practical, noninvasive monitoring tools with the aim to detect early symptoms, fatigue and/or maladaptation. This means that additional performance tests, such as the double PPO protocol [22] and/or blood and hormone markers [12, 13, 23] might be needed to confirm a status of overreaching and/or identify any underlying pathologies that may have contributed to the development of the overreaching status.

Acknowledgements
We would like to thank all runners that participated in this study. In addition, we would like to thank Ian Darragh, Inge Stoter, Jana Burger, Jereme Outerleys and Nikhil Divekar for assisting us with the data collection, as without their help it would not have been possible to conduct this study.

Conflicts of interest
The authors have no conflict of interest to declare.

References


