Cortisol awakening response, pain and depression in athletes and non-athletes with chronic back pain

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Background/Aims.
Low back pain (LBP) is one of the most prevalent chronic pain syndromes in athletes as well as in non-athletes1. Psychosocial risk factors, such as depression, and an altered basal adrenocortical activity have been shown to modulate pain and pain-related disability in non-athletes2.

Furthermore, physical activity with main emphasis on sports activity revealed a U-shaped relationship to pain in non-athletes3 whereas a main effect has been reported in athletes4. In non-athletes, both, very high and very low levels of activity increase the risk for back pain. In contrast, in athletes primarily high levels of training activity increase the risk of “overreaching” and pain5. Thus, depression on the one hand and specific levels of sports activity on the other may cause an increased level of physiological stress, mirrored by an altered adrenocortical activity. Cumulative burden of stress manifests as physiological dysregulation across multiple systems causing increased levels of “allostatic load”6.

To date, little is known about the relative impact of pain, depression and sports activity on adrenocortical activity in athletes compared to non-athletes with back pain. We expect a cumulative effect of elevated depressive mood and high levels of training activity especially in athletes, while in non-athletes depression x training activity may indicate a stress-reducing effect of training on depression.

Methods
Sample and research design
- 80 patients with LBP (33 athletes, 47 non-athletes) were consecutively recruited from exercise training practices. The participants were divided into an athlete and a non-athlete group, based on their athletic-performance.
- Those who had reported to participate in competitive sports at a high (national or international) or medium (regional) level were assigned to the athlete group, whereas those who were not active or active at a low (local) level were assigned to the non-athlete group.
- Cross-sectional case-control study

Measures
- **CAR**: Cortisol was assessed from saliva, collected with the Salivette sampling device (Sarstedt, Germany). Participants collected five saliva samples (0, 15, 30, 45, 60 minutes after awakening in the morning) on two consecutive days. Free cortisol levels in saliva were measured by employing a time-resolved immunomassay with fluorescence detection. To quantify the CAR of each subject, the mean value of the two days for each sampling time was calculated. Additionally, the “area under the curve with respect to ground” (AUCG) was computed.
- **Pain intensity**: Numerical self-rating scale (NRS, 0-10)
- **Disability**: Von Korff Disability Score (0-100)
- **Depression**: Beck Depression Inventory-Primary Care (BDI-PC)

Statistics
Repeated measurement ANOVAs (RMA) with depression (high/low) and group (athletes, non-athletes) or training frequency (<4h, 4-8h, >8h) as between subjects factors and time of CAR (0, 15, 30, 45, 60 min) as within-subjects factor were performed. Two ANOVAs with depression and group or training frequency as between subjects factors were computed for AUCG. Age and training intensity before LBP started were covariates. Bonferroni post-hoc comparisons were calculated testing single group differences. The level of significance was p < .05.

Results
- **Athletes** and non-athletes did not differ with respect to gender, pain intensity, disability, or depression. Athletes were significantly younger and revealed a higher training frequency per week than the non-athletes.
- **First ANCOVA** revealed a significant main effect group for AUCG, with higher cortisol levels in the athletes compared to the non-athletes (Fig 1a).
- **RMA** showed boundary group and group x dep interaction for CAR 0-60. The high depressive athletes started with lower CAR compared to the non-athletes, showed a higher increase from 0 to 15 min and revealed higher CAR levels from 15 to 60 min (Fig 1 b,c).
- The second ANCOVA indicated a significant main effect for training frequency (F2 = 3.390 p < .05, see Figure 2 a)
- In the second RMA training frequency (p = .06) and training x depression interaction (p = .11) approached significance

Table 1. Descriptives (mean/SD or N%)

<table>
<thead>
<tr>
<th>Patients</th>
<th>Athletes</th>
<th>Non-athletes</th>
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<tbody>
<tr>
<td>Number (%)</td>
<td>33 (41%)</td>
<td>47 (59%)</td>
</tr>
<tr>
<td>Gender (% female)</td>
<td>42.4</td>
<td>57.6</td>
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<tr>
<td>Age</td>
<td>30.48 (10.20)</td>
<td>44.78 (11.99)</td>
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<tr>
<td>Pain intensity</td>
<td>4.09 (1.63)</td>
<td>3.46 (2.12)</td>
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<tr>
<td>Disability (Von Korff)</td>
<td>16.51 (15.25)</td>
<td>24.22 (18.91)</td>
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<tr>
<td>Depression (BDI-PC)</td>
<td>1.69 (1.86)</td>
<td>2.51 (3.08)</td>
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**CAR-AUCG**
- Training frequency pre (h/week*) | 9.17 (9.14) | 2.91 (3.22) |
- Training frequency post (h/week*) | 7.65 (4.93) | 2.98 (2.28) |

* sign group differences p < .05

References
1. Mierswa T et al Pain parameters and correlates in athletes and non-athletes with LBP, submitted

Conclusion
Results of this study indicate that very high and very low training frequency in non-athletes with back pain lower the level of CAR while in the athletes a dose-response relationship was seen: the higher the training frequency the lower the CAR. Thus, impact of training frequency and depression on allostatic load differs between athletes and non-athletes with back pain.