Testosterone, cortisol and anxiety in elite field hockey players

Raúl Aguila a,⁎, Manuel Jiménez b, José R. Alvero-Cruz c

a Facultad de Psicología, Universidad de Málaga, Campus Teatinos s/n, 29071, Málaga, Spain
b Facultad de Educación, Universidad Internacional de la Rioja, Gran Vía Rey Juan Carlos I, 26002, Logroño, Spain
c Facultad de Medicina, Universidad de Málaga, Campus Teatinos s/n, 29071, Málaga, Spain

HIGHLIGHTS

• Winning experiences elicit slight testosterone rises.
• Losing experiences elicit testosterone drops together with peak cortisol levels.
• Anticipatory somatic anxiety positively correlates with pregame cortisol levels.
• Physical exertion negatively correlates with the change in testosterone levels.

ARTICLE INFO

Article history:
Received 24 May 2013
Accepted 5 December 2012
Available online 3 June 2013

Keywords:
Winner–loser effect
Dominance motivation
Testosterone
Anxiety
Cortisol
Lactate

ABSTRACT

The aim of the present study was to assess the change in the levels of testosterone and cortisol after victory and defeat in male field hockey players during an important tournament. In the beginning of the game series, the players were ranked very closely to achieve (for the first time) the championship rising to The Honor Division-A, the highest status national category. The first game resulted in a 7–4 victory, the second game resulted in a 6–1 victory, and the third game resulted in a 1–2 defeat. As expected, there were changes in testosterone levels after the competition, dropping in the game which ended in defeat, and rising slightly in the two games which ended in victory; there were also changes in cortisol levels, rising in the game which ended in defeat, and showing no variations in the games which ended in victory; correlational analyses congruently showed that defeat led to rises in cortisol whereas victory led to rises in testosterone; anticipatory somatic anxiety was related to cortisol levels prior to games, and physical exertion during competition was related to the change in testosterone levels (suggesting an inhibitory effect) but not to the change in cortisol levels. Hence, this pattern of hormonal responses to a real-life dominance challenge complied with Mazur’s (1985) biosocial model of status and dominance motivation, by showing that testosterone and cortisol are linked to victory and defeat in a theoretically predictable fashion.

1. Introduction

According to current neuropsychological theories of emotion, when a social situation is appraised as threatening or challenging by orbitofrontal and limbic regions, a cascade of nervous physiological processes orchestrated by the central nucleus of the amygdala begins, which engage three neuroendocrine systems contributing to prepare the body for action: the hypothalamic–pituitary–gonadal axis, the sympathetic–adrenal–mediullary axis and the hypothalamic–pituitary–gonadal axis (e.g. [24]). As a part of this cascade the hypothalamus discharges the corticotropin-releasing factor to the anterior lobe of the pituitary gland which in turn releases the adrenocorticotropic hormone into the bloodstream. Then the adrenocorticotropic hormone reaches the cortex of the adrenal glands provoking a rise in circulating cortisol, the end product of this hypothalamic–pituitary–gonadal stress response.

Another important part of this cascade involves the hypothalamic–pituitary–adrenal stress response, whereby the catecholamine adrenaline and noradrenaline are released from the medulla of the adrenals, which act on target tissues provoking bodily changes such as increased heart rate. Likewise, the hypothalamic–pituitary–gonadal axis contributes to bodily arousal, by way of the gonadotrophin releasing factor in the anterior pituitary and then by the luteinizing hormone through the bloodstream, which acts on the reproductive and adrenal glands to rise circulating testosterone. The role of testosterone in the stress response is more related to situations of stress threat that demand vigorous actions (e.g. [1,2,18,26,28]); and this relationship is reciprocal in that basal testosterone not only influences dominant behaviors, but positive outcomes reinforce those dominant behaviors and increase subsequent testosterone levels (e.g. [17,18,21,22,27]). In addition to being triggered by stimuli evaluated as threats and social challenges, these neuroendocrine systems are sensitive to physiological irritants such as peripheral infection, pain and...
physical exertion (e.g. [7,11,23]), making it difficult to tease apart the portion of the hormonal variance that is psychological in origin rather than physiological.

Here, we carried out a follow-up study on elite field hockey players across the last three games of the national league, by measuring the levels of testosterone and cortisol prior to games, as well as the change in the levels which follow both winning and losing experiences. Provided that physical exertion is a potent stimulus to secretion of testosterone and cortisol (e.g. [11,31]), the levels of lactate and parallel perceptions of physical exertion were also measured, in an attempt to disentangle whether the changes observed in hormone levels could be attributable to competition outcome specifically. In addition, the players responded to the Competitive State Anxiety Inventory–2 (CSAI-2; [15]), which has many items for assessing perceived autonomic arousal and worries about the outcome, in order to have an experiential measure of the anticipatory stress response to competition (e.g. [5,8–10]). To our knowledge, there exist no prior studies on field hockey players that have jointly taken these hormonal, exertion and anxiety measures using a repeated measures (within-subject) design, probably because this particular team sport is characterized by frequent substitutions (of variable duration) of the players, and so it is difficult to obtain comparable measures from different subjects, or from the same subjects over competition days. The design has the conceptual advantage (when circumventing that inherent difficulty) to set the stage for dissociating the measures being stable over days from those showing changes. Thereby, if all of the measures were relatively stable over days, and only postgame changes in testosterone and cortisol were observed, then this observation could be taken as clear evidence that winning and losing experiences modulate hormonal responses.

Based on the biosocial hypothesis of status (that testosterone increases to cope with status threat, increases a bit further or maintains its levels after victory, decreases after defeat, and reinforces dominant behaviors; [16]), and in congruence with the challenge hypothesis (that testosterone increases to cope with social challenges; [30]), we made the following predictions: first, there should be a rise (or no change) in testosterone levels when the games end in victory and a drop when ending in defeat; second, there should be a rise in cortisol levels when the games end in defeat and no change when ending in victory; and third, there should be increased levels of testosterone, cortisol and anxiety prior to games. In line with prior research, there should be positive correlations between pre-competitive anxiety and cortisol, defeat and cortisol changes, victory and testosterone changes, lactate and ratings of perceived exertion (e.g. [5,8–10,13]), and between these exertion measures and playing position, to the extent that the players’ role could be differentially related to the psychophysiological demands of physical exertion (e.g. a defender runs less than a forward and thus should have a lower lactate level; [14]).

2. Methods

2.1. Participants

For the selection of participants, it was agreed with the coach of the Club Hockey Polideportivo Benalmádena (the target team of this study), that the same subjects played the last three games of the championship, and that none of them were inactive more than 2 min (or substituted) during each game. In doing so, the differences among participants in the time spent playing and the resulting variations in physical exertion were minimized. As a consequence, the sample used was 7 male field hockey players (mean age ± SEM = 28.71 ± 1.71 years; height = 180.71 ± 4.07 cm; mass = 78.87 ± 2.54 kg; body mass index = 24.22 ± 0.84 kg m²) representing all court positions: goalkeeper, 2 defenses, 2 midfielders and 2 forwards. They were professional players in The Honor Division-B national league, from Benalmádena town in the southernmost region of Spain (Andalucía). Meetings were held with representatives of their club in the pre-season to explain the procedure that was going to be followed. Medical records were taken to exclude metabolic or endocrine diseases, drug consumption or psychiatric disorders with the informed consent signed by each participant. This protocol was approved by the Committee of Ethics of the University of Málaga in accordance with the Declaration of Helsinki.

2.2. Procedure

Anxiety was measured 45 min prior to competition with the CSAI-2, a 27-item scale composed of three sub-scales of 9 items measuring somatic anxiety, cognitive anxiety and self-confidence [15]. The items were self-rated on a 5-point Likert format from “not at all” to “very much so”, the scoring range for each sub-scale being 9–45, the higher the score the higher the perceived intensity.

Saliva samples (5–10 ml) for hormone determination were taken in plastic tubes (SalCap) 45 min before and after competition, with an average interval between samples of about 165 min. Since the pre-competition warm-ups began after taking the first saliva sample, their influence on steroid hormone levels was ruled out. The players had been instructed not to eat any food or brush their teeth 30 min prior to the sample time. The samples were frozen at —30 °C 15 min after being collected and conserved until assayed using an enzyme immunoassay equipment (Grifols Triturus) from the Hematology Laboratory of the Virgen de la Victoria University Hospital of Málaga. Samples were assayed in duplicate within the same assay. The intra-assay and inter-assay coefficients of variation were (respectively) 5.5 and 9.3% for testosterone and 8.0 and 14.1% for cortisol. The lower limits of detection for testosterone and cortisol kits were 3.5 pg/ml and 0.05 ng/ml.

Psychophysiological measures of physical exertion were assessed by ratings of perceived exertion and capillary lactate concentration. Perceived exertion was assessed with a Likert scale ranging from 0 to 10 according to perceived intensity, from “very, very light” on the left side of the scale to “maximum” on the right side [3,6]. Lactate was obtained from 20 μl of capillary blood (Dr. Lange LP20 Miniphotometer, Berlin, Germany), by lobular puncture of the earlobe during the first 60 s after game cessation. Lactate levels increase according to requirement for higher metabolic demands during physical exertion, and thus lactate is considered a good index of the effort exerted in anaerobic exercise [29]. Both perceived exertion and lactate measures were taken twice, in half-time and after competition.

In the beginning of the game series the target team was ranked in second position, so that they were very close to achieve the championship rising to The Honor Division-A, the highest status national category. This real-life dominance challenge could make them be strongly motivated to win, for this was the first time they strove for winning such an important tournament. Also importantly, the home advantage could be helpful to their performance since the games assessed always took place at the home venue (e.g. [4,5,12,20]). The games had two halves of 35 min each, with a resting period of 5 min in-between. The first game, resulting in a 7–4 victory over Pedralbes H.C. (2–2 in half-time), began at 12:30 pm, ended at 13:45 pm, and took place on April 11; the second game, resulting in a 6–1 victory over C.H. Natació Linia (2–0 in half-time), began at 12:30 pm, ended at 13:47 pm, and took place on April 25; and the third one, resulting in a 1–2 defeat against Vallès Esportiu C.H. (1–1 in half-time), began at 12:30 pm, ended at 13:48 pm, and took place on May 16. By defeating these rivals, the target team was able to win for the first time the national league, a good measure of the importance of the games assessed. Even though they lost the last game and finished in second place in the national league, their rise to The Honor Division-A was eventually achieved, because one of the favorite teams (F.C. Junior) did not win a critical game that was taking place simultaneously. This outcome was unknown when the players gave the second saliva sample, since that information was delivered shortly afterward. This implied that hormonal responses to defeat in the last game were probably accompanied by strong stress/
frustration responses, as the participants had lost (at least apparently) an unique opportunity in their sports careers.

2.3. Statistical analyses

In order to analyze the levels of testosterone, cortisol and anxiety prior to games, as well as the change in the levels of cortisol and testosterone which follow both winning and losing experiences, a repeated measures design over three consecutive games was used. Pregame and postgame hormonal levels were assessed over days by a two-way analysis of variance (ANOVA), with measurement time as a between-subject factor and with days as a within-subject factor. CSAT-2 ratings prior to games were assessed over days by a one-way ANOVA with days as a within-subject factor. The effect sizes $r^2$ were also calculated. The homogeneity of the variances of the dependent variables was assessed by means of the Shapiro–Wilk Normality Test, showing that pregame and postgame hormones, perceived exertion and lactate were not normally distributed. Pregame and postgame hormonal levels were log-transformed to fulfill the homogeneity assumption (non-transformed data were depicted in the figures to facilitate comparison with prior studies), whereas perceived exertion and lactate did not fulfill this assumption even after log or square-root transformations, so that Wilcoxon signed rank test for related samples were applied. Relationships between the main variables were tentatively explored by Spearman’s correlation coefficient ($\rho$), using all of the observations taken from the subjects, a total of 21 (7 subjects per 3 games). These analyses were applied by using an SPSS statistical package.

3. Results

Pregame and postgame testosterone levels over days are shown in Fig. 1A. Results showed that testosterone levels changed with measurement time in some days, as indicated by a marginal main effect of days: $F(2,24) = 2.84, p = 0.078$, $r^2 = 0.19$, and a days x time interaction: $F(2,24) = 3.47, p = 0.047$, $r^2 = 0.22$. Wilcoxon test for related samples confirmed a marginal (though expected) difference between pregame and postgame testosterone levels on the day of Defeat May 16 ($z = -1.69, p = 0.091$). This drop in testosterone after defeat jointly with the apparent (but statistically non-significant) slight rise after the two victories, relative to unchanged pregame levels, thereby accounted for that interaction. As shown in Fig. 1B, cortisol levels also changed with measurement time in some days, this being indicated by a main effect of days $F(2,24) = 10.46, p < 0.001$, $r^2 = 0.47$, and a days x time interaction: $F(2,24) = 5.98, p = 0.008$, $r^2 = 0.33$. Wilcoxon test for related samples confirmed a pregame-postgame rise in cortisol levels on the day of Defeat May 16 ($z = -2.37, p = 0.018$).

In Table 1 are shown both anxiety and exertion-related measures over days. Pregame ratings of somatic anxiety, cognitive anxiety and self-confidence did not vary over days: $F(2, 12) = 1.08, p = 0.37$; $F(2, 12) = 2.19, p = 0.16$; $F(2, 12) = 3.92, p = 0.068$ (respectively), suggesting that players coped with pregame anxiety at consecutive home games in a similar way. With regard to half-time ratings of perceived exertion, there was a significant difference between the day of Victory April 25 and the day of Defeat May 16 ($z = -2.03, p = 0.042$); for postgame ratings of perceived exertion, there were significant differences between the two victory days ($z = -2.39, p = 0.017$); for half-time lactate levels, there were significant differences between the two victory days ($z = -2.37, p = 0.018$), and between the day of Victory April 25 and the day of Defeat May 16 ($z = -2.37, p = 0.018$); for postgame lactate levels, there was a marginal difference between the two victory days ($z = -1.86, p = 0.063$), and a significant difference between the day of Victory April 25 and the day of Defeat May 16 ($z = -2.03, p = 0.043$); finally, there was a significant increase in perceived exertion from half-time to postgame on the day of Defeat May 16 ($z = -2.07, p = 0.038$).

In Table 2 the correlations between the main variables are shown. Pregame cortisol positively correlated with half-time lactate ($\rho = 0.44, p = 0.047$) and playing position ($\rho = 0.46, p = 0.036$); somatic anxiety positively correlated with postgame lactate ($\rho = 0.57, p = 0.007$) and playing position ($\rho = 0.61, p = 0.003$); playing position positively correlated with half-time lactate ($\rho = 0.51, p = 0.018$) and postgame lactate ($\rho = 0.48, p = 0.029$), and negatively with testosterone change ($\rho = -0.55, p = 0.010$); half-time perceived exertion positively correlated with half-time lactate ($\rho = 0.62, p = 0.003$), and postgame perceived exertion positively correlated with postgame lactate ($\rho = 0.73, p < 0.0001$). This pattern of correlations mainly indicated that a greater role in offensive positions was accompanied by higher levels of pregame cortisol and somatic anxiety, higher postgame lactate levels and lower postgame testosterone changes; it also indicated that lactate levels served as an index of perceived exertion (and vice versa). Consistent with prior studies, one-tailed (expected) positive correlations were found between pregame cortisol and somatic anxiety ($\rho = 0.41, p = 0.069$; e.g. [8–10]), and between pregame cortisol and pregame testosterone ($\rho = 0.40, p = 0.074$; e.g. [19]). In addition, a negative correlation between outcome and cortisol change ($\rho = -0.70, p < 0.0001$), and a positive correlation between outcome and testosterone change ($\rho = 0.47, p = 0.033$) were found, indicating that defeat led to rises in cortisol and victory led to rises in testosterone. Asymmetrical game, that is, the degree of easy to win (see Table 2 for a more detailed description), negatively correlated with postgame lactate ($\rho = -0.44, p = 0.047$), postgame perceived exertion ($\rho = -0.54, p = 0.011$) and cortisol change ($\rho = -0.59, p = 0.005$), as well as positively with outcome ($\rho = 0.87, p < 0.0001$), thus indicating that to compete with an easy...
rival required less exertion, both physical and mental, and was accompanied by lower postgame cortisol changes. Hence, the fact that asymmetrical game and outcome highly correlated with each other does not necessarily mean that they reflected the same thing, in that they correlated with effort-related measures in a different way.

4. Discussion

We have shown here that the pattern of hormonal responses displayed by elite hockey players in a series of decisive home games, complied with Mazur's [16] biosocial hypothesis of status: there were changes in testosterone levels, with a drop in the game which ended in defeat and a slight rise in the two games which ended in victory; and there were changes in cortisol levels, with a sizable rise in the game which ended in defeat, and no changes in the games which ended in victory. Some caution is in order though before discussing in depth our results, for the design of the present study has several limitations. One limitation is the small number of participants used, what could have been compensated to some extent by having taken from them repeated measures over days. A second limitation is that the participants were not asked to report their affective state after competition, what is a relevant factor in the biosocial model, in that affective valence arising from winning and losing was originally thought to mediate hormonal changes. Moreover, it could be argued (given the lack of baseline measures) that the stability over days in pregame levels of anxiety and hormones was due to a floor effect, for the time elaps ed from pregame measurement to the time competition was too long to trigger noticeable preparatory stress responses. In an attempt to circumvent this possibility it could be useful to make results with the comparisons of a similar study carried out by Carré et al. [5], who also took hormonal and CSAI-2 measures 45 min prior to games from elite men's ice hockey: when compared to them, our players showed higher cortisol levels and higher scores in the three sub-scales of the CSAI-2 (after converting our raw scores from a 5-point scale to a 4-point scale like theirs), as did when compared to the norm samples provided by Martens et al. [15]. As expected from the challenge hypothesis, our players appeared therefore physiologically aroused and worried, at least to the same extent than Carré et al.'s ice hockey players. Thereby, these pregame levels of anxiety and hormones could reflect preparatory stress responses triggered when arriving at the competition venue (i.e. conditioned somatic responses to a social environment replete with classically conditioned stimuli), and could be related to arousal demands to mobilize energy resources in advance (accordingly to such demands) as the time to compete neared.

Owing partly to pregame stability over days in hormone levels, a clear winner–loser effect was obtained by a days × time interaction. This effect was obtained for testosterone because postgame levels slightly rose after the two games which ended in victory, and dropped after the game which ended in defeat, relative to unchanged pregame levels; and for cortisol because postgame levels dramatically rose after defeat, and there were no changes after the two victories, relative to unchanged pregame levels; in addition, these effects were consistent with the results from correlational analyses, showing that defeat led to rises in cortisol whereas victory led to rises in testosterone. It must be noted that lactate levels (our main index of physical exertion) did not differ from half-time to postgame measures, and were similar in the first game which ended in victory and in the game which ended in defeat. Hence, these results could have at least three implications: first, that the rise in cortisol was something more than a mere response to inflammatory processes after great

Table 1
Mean (±SEM) for the average scores in the three sub-scales of the CSAI-2 prior to competition, and for ratings of perceived exertion and lactate in half-time and after competition, taken from the first victory on April 11, the second victory on April 25, and the defeat on May 16. Scores ranged from 9 to 45 in each sub-scale of the CSAI-2 and from 0 to 10 in perceived exertion. The game played on April 11 resulted in a 7–4 victory (2–2 in half-time); the game played on April 25 resulted in a 6–1 victory (2–0 in half-time); and the game played on May 16 resulted in a 1–2 defeat (1–1 in half-time).

<table>
<thead>
<tr>
<th></th>
<th>Somatic anxiety</th>
<th>Cognitive anxiety</th>
<th>Self-confidence</th>
<th>Half-time rating of perceived exertion</th>
<th>Postgame rating of perceived exertion</th>
<th>Half-time lactate (mmol/l)</th>
<th>Postgame lactate (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Victory April 11</td>
<td>24.43 (2.31)</td>
<td>32.57 (1.36)</td>
<td>36.14 (2.02)</td>
<td>5.71 (0.42)</td>
<td>6.71 (0.57)</td>
<td>5.08 (0.74)</td>
<td>4.75 (0.75)</td>
</tr>
<tr>
<td>Victory April 25</td>
<td>22.14 (1.50)</td>
<td>30.29 (1.15)</td>
<td>37.29 (1.78)</td>
<td>4.00 (0.76)</td>
<td>4.14 (0.77)</td>
<td>3.60 (0.44)</td>
<td>3.64 (0.45)</td>
</tr>
<tr>
<td>Defeat May 16</td>
<td>23.43 (2.06)</td>
<td>29.57 (0.92)</td>
<td>37.00 (1.21)</td>
<td>5.86 (0.34)</td>
<td>6.86 (0.34)</td>
<td>5.13 (0.55)</td>
<td>5.68 (0.65)</td>
</tr>
</tbody>
</table>

a Difference between Victory April 25 and Defeat May 16.
b Difference between Victory April 25 and the rest of the days.

Table 2
Correlation matrix among the measures taken 45 min before, in the half-time and 45 min after the competition. Correlations > 0.43 (in bold) are statistically significant with a p value less than 0.05, and correlations < 0.43 (in bold) are one-tailed statistically significant, with a p value less than 0.10. OUT, outcome: defeat or victory (1); ASYM, asymmetrical game: the degree of ease to win as inferred from the difference between teams in the goals scored: 1–2, 2–4 or 6–1 (2); PPOS, playing position: goalkeeper, defense, midfield or forward (3); SC, self-confidence (4); SOM, somatic anxiety (5); COG, cognitive anxiety (6); T-PRE, pregame testosterone (7); C-PRE, pregame cortisol (8); T-POST, postgame testosterone (9); C-POST, postgame cortisol (10); T-DIFF, testosterone difference (11); C-DIFF, cortisol difference (12); L-HALF, half-time lactate (13); RPE-HALF, half-time rating of perceived exertion (14); L-POST, postgame lactate (15); RPE-POST, postgame rating of perceived exertion (16).

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>OUT</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>ASYM</td>
<td>0.87</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>PPOS</td>
<td>0.00</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>SC</td>
<td>0.02</td>
<td>0.03</td>
<td>1.11</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>SOM</td>
<td>−0.02</td>
<td>−0.13</td>
<td>0.61</td>
<td>−0.26</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>COG</td>
<td>0.24</td>
<td>0.38</td>
<td>0.04</td>
<td>−0.01</td>
<td>0.27</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>T-PRE</td>
<td>−0.11</td>
<td>0.04</td>
<td>0.15</td>
<td>0.42</td>
<td>0.17</td>
<td>−0.05</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>C-PRE</td>
<td>−0.05</td>
<td>0.00</td>
<td>0.46</td>
<td>−0.19</td>
<td>0.41</td>
<td>−0.08</td>
<td>0.40</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>T-POST</td>
<td>0.50</td>
<td>0.48</td>
<td>−0.28</td>
<td>0.03</td>
<td>−0.16</td>
<td>0.06</td>
<td>0.09</td>
<td>−0.31</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>C-POST</td>
<td>0.43</td>
<td>0.20</td>
<td>0.57</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>T-DIFF</td>
<td>0.47</td>
<td>0.41</td>
<td>−0.55</td>
<td>0.17</td>
<td>−0.40</td>
<td>0.05</td>
<td>−0.45</td>
<td>−0.58</td>
<td>0.79</td>
<td>−0.25</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>C-DIFF</td>
<td>−0.70</td>
<td>−0.59</td>
<td>−0.20</td>
<td>0.35</td>
<td>−0.03</td>
<td>−0.11</td>
<td>−0.26</td>
<td>−0.30</td>
<td>−0.17</td>
<td>0.90</td>
<td>0.02</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>L-HALF</td>
<td>−0.25</td>
<td>−0.42</td>
<td>0.51</td>
<td>−0.01</td>
<td>0.41</td>
<td>−0.04</td>
<td>0.01</td>
<td>0.44</td>
<td>−0.47</td>
<td>0.26</td>
<td>−0.54</td>
<td>0.12</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>RPE-HALF</td>
<td>−0.25</td>
<td>−0.40</td>
<td>0.32</td>
<td>0.00</td>
<td>−0.09</td>
<td>−0.21</td>
<td>−0.22</td>
<td>0.37</td>
<td>−0.12</td>
<td>0.25</td>
<td>−0.11</td>
<td>0.15</td>
<td>0.62</td>
<td>1.00</td>
</tr>
<tr>
<td>15.</td>
<td>L-POST</td>
<td>−0.38</td>
<td>−0.44</td>
<td>0.48</td>
<td>−0.02</td>
<td>0.57</td>
<td>0.01</td>
<td>0.13</td>
<td>0.41</td>
<td>−0.36</td>
<td>0.28</td>
<td>−0.49</td>
<td>0.23</td>
<td>0.52</td>
<td>1.00</td>
</tr>
<tr>
<td>16.</td>
<td>RPE-POST</td>
<td>−0.32</td>
<td>−0.54</td>
<td>0.22</td>
<td>−0.18</td>
<td>0.34</td>
<td>0.16</td>
<td>−0.05</td>
<td>0.32</td>
<td>−0.24</td>
<td>0.29</td>
<td>−0.27</td>
<td>0.23</td>
<td>0.57</td>
<td>0.54</td>
</tr>
</tbody>
</table>
physical exertion, for postgame lactate levels were similar in the first and the third games, with the rise in cortisol being observed after defeat only; second, that postgame lactate levels were not responsive to competition outcome; and third, that physical exertion (physiological or perceived) did not contribute to winning or losing distinctively. Following this reasoning, if physical exertion had no relation to competition outcome then the most parsimonious account would be that afforded by Mazur’s [16] biosocial model of status, that is to say, that the slight rise in testosterone was triggered by status attainment, in turn leading to a rise or maintenance in perceived dominance, where as the rise in cortisol was triggered by the failure to get such a highly valuable goal, in turn leading to a state of frustrative nonreward or stress (as argued in Jiménez et al. [13]).

Perhaps correlational data could yield additional insight into the role of physical exertion in hormonal responsiveness. For example, pregame cortisol positively correlated with half-time lactate and playing position, somatic anxiety positively correlated with postgame lactate and playing position, playing position positively correlated with half-time lactate and postgame lactate, and negatively with testosterone change. This pattern of correlations mainly indicated that a greater role in offensive positions was accompanied by higher levels of pregame cortisol and somatic anxiety, higher postgame lactate levels and lower testosterone changes. Should we expect there to be a positive correlation, like that observed here, between a greater role in offensive positions and lactate levels? The response appears to be yes. Although studies focused on playing position in field hockey players are still scanty, it has been recently shown in women that forwards spend a greater percentage of time running, fast running and sprinting than defenders (who spend more time walking), as well as have less time to recover after these activity bouts of moderate-to-high intensity [14]. However, both half-time and postgame lactate levels negatively correlated with testosterone changes, a result that is not consistent with the idea that the players having higher lactate levels in response to acute bouts of physical exertion should have higher hormonal changes, apparently contradicting a large amount of evidence showing that physical exertion has a stimulatory effect on hypothalamic–pituitary–adrenal/gonadal axes (e.g. [11,31]). So, it is as if physical exertion could have an inhibitory influence on postgame testosterone, to the point of abolishing the winner effect in those players with higher lactate levels. In fact, exploratory analyses with non-parametric tests (data not shown), dividing the sample into two groups, one with three players in defensive positions (goalkeeper and defenses) and other with four players in offensive positions (midfielders and forwards), showed that in those offensive positions (higher in lactate levels than defenses) did not display the expected rises in testosterone after victory. Along the same lines, Suay et al. [29] failed to obtain testosterone changes after victory in judoists with apparently high lactate levels, and lactate levels were not different between winners and losers. As noted by Salvador [25], also based on the results of a study with judo wrestlers, that the team “who ended up winning displayed the highest decreases in testosterone, contrary to what was expected, suggests that the physical effort made during the combat could be the cause of these reductions, which compensated for the increases associated with the victory” (p. 198).

In conclusion, we assessed here hormonal, anxiety and physical exertion responses to competition from an unusual sample of elite field hockey players, when living the decisive steps toward a historical event for themselves and their club: the championship rising to The Honor Division-A of the Spanish national league. Results showed a good fit with what was expected from current hypotheses relating hormones to dominance; this fit had probably much to do with the mode of inducing the participants’ motivational state, that is, a significant real-life dominance challenge with palpable consequences for their status. In addition to showing that winning experiences elicited slight testosterone rises, and losing experiences elicited testosterone drops together with peak cortisol levels, results showed that anticipatory somatic anxiety positively correlated with pregame cortisol levels, and that physical exertion during competition negatively correlated with the change in testosterone levels: this negative correlation suggested therefore that physical exertion could have an inhibitory influence on post-exertion testosterone levels, a possibility that could help to explain the null findings previously reported in some studies on the winner–loser effect in sport competition research.

References