Psychoneuroendocrinology 191 (2026) 107934

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

Modulation of neuroendocrine and autonomic responses to competitive stress by slow cortical potentials training in golfers: An exploratory randomized controlled trial

Eugenio Lizama [a], Bastian Carcamo-Herrera [b], Gonzalo López [c], Carolina Pereira [c], Miguel A. Serrano [d,e,*]

[a] Sport Brain Center, 1000 Pembroke Rd, Hallandale Beach, FL 33009, USA
[b] Centro de Investigación para la Resiliencia a Pandemias, Facultad de Ciencias de la Vida e Instituto de Salud Pública, Universidad Andrés Bello, Santiago, Chile
[c] Centro de Entrenamiento Neuropsicológico, Avenida Italia 1883, Santiago de Chile, 7770385, Chile
[d] Department of Psychobiology, Universitat de València, València 46010, Spain
[e] Behavioral, Affective and Cognitive Neuroscience Laboratory, University of Valencia, Spain

ARTICLE INFO

Keywords:
Neurofeedback
Competitive stress
Heart rate variability
Cortisol
DHEA

ABSTRACT

Performance in precision sports such as golf depends on the athlete's ability to regulate psychophysiological responses under competitive pressure. Heart rate variability (HRV), cortisol, and DHEA provide complementary indices of autonomic and endocrine adaptation to competitive stress, and SCP neurofeedback represents a potential training approach for cortical self-regulation, although evidence in athletes remains limited. This study evaluated a standardized SCP training protocol in golfers by examining cortisol, DHEA and HRV responses during simulated competitions and putting performance. In this exploratory parallel-group randomized controlled trial, 42 (21 per group) amateur and semi-professional golfers (mean age = 43.00 years, SD = 13.13) were stratified by handicap and assigned to an SCP neurofeedback group or a wait-list control group. The SCP group completed an 8-week neurofeedback program. A first simulated tournament was performed before training and a second after the intervention. During both competitions, salivary cortisol and DHEA were collected pre- and post-task (including DHEA/Cortisol ratio), and heart rate variability (HRV) was assessed at baseline and during gameplay (HR, LF, HF, LF/HF, total power, RMSSD). Hormonal responses showed a re-exposure pattern in both groups, with lower cortisol and higher DHEA during the second tournament. Exploratory autonomic analyses during gameplay indicated higher HRV magnitude and RMSSD in the trained group than in controls post-intervention, although these findings should be interpreted cautiously. Putting performance did not improve significantly following the intervention. Overall, these findings suggest that SCP training may be associated with a relative preservation of autonomic modulation during repeated competitive exposure. However, this interpretation remains preliminary, and causal attribution is limited by the exploratory design and the absence of an active sham-control condition.

1. Introduction

Golf is a precision sport requiring technical consistency under environmental, cognitive, and emotional variations, making psychophysiological self-regulation a critical performance determinant (Stöckl and Lamb, 2018). Practiced by approximately sixty million people worldwide, the sport's competitive demands—integrating fine motor control, emotional stability, and attentional regulation under pressure—have driven increasing interest in optimizing performance from physical, psychological, physiological, and environmental perspectives (Bangsbo, 2015; Carson et al., 2020; Fronso et al., 2017; Gladdines et al., 2022). The precision required in each stroke and the need to maintain consistency under changing conditions make this self-regulation especially relevant in critical moments where small variations can significantly alter performance (Bortoli et al., 2012; Lu et al., 2021).

From a psychophysiological perspective, competitive stress emerges

* Correspondence to: Department of Psychobiology, Universitat de València,Avd. Blasco Ibañez, 21, València 46010, Spain.
E-mail addresses: eugenio.lizama@bpgchile.cl (E. Lizama), b.crcamoherrera@uandresbello.edu (B. Carcamo-Herrera), gonzalo.esloga@gmail.com (G. López), cpereirahinke@gmail.com (C. Pereira), m.angel.serrano@uv.es (M.A. Serrano).

https://doi.org/10.1016/j.psyneuen.2026.107934
Received 9 February 2026; Received in revised form 5 June 2026; Accepted 5 June 2026
Available online 7 June 2026

when perceived demands exceed an athlete's available resources, triggering coordinated cognitive, emotional, autonomic, and neuroendocrine responses that threaten homeostatic balance (Tsigos et al., 2000; James et al., 2023). In sport, this threat appraisal manifests as anxiety and tension perceived as exceeding coping capacity, accompanied by activation of the sympathetic-adrenomedullary axis and the hypothalamic-pituitary-adrenal (HPA) axis—mechanisms that prepare the organism to meet perceived demands (Smith and Vale, 2006; Godoy et al., 2018; Hertting et al., 2020; Thielmann et al., 2022). These adjustments are captured by complementary biomarkers. Cortisol reflects early HPA-axis engagement and energy mobilization around competition (MacDonald and Wetherell, 2019; Smith and Vale, 2006), with anticipatory increases documented in precision sports including golf (Filaire et al., 2009; Van Paridon et al., 2017) and recently in elite cross-country runners, where pre- and post-race cortisol correlated with race time and predicted competitive success (Aydemir et al., 2025); parallel evidence from triathlon, volleyball, and golf further confirms its value as a marker of competitive pressure (Balthazar et al., 2012; Costa et al., 2022; Doan et al., 2007). DHEA counteracts cortisol-related catabolic effects and supports physiological adaptation to effort, and the cortisol/DHEA ratio has been proposed as a sensitive index of the balance between activation and recovery with implications for performance quality (Dutheil et al., 2021; Gerra et al., 2001; Lee et al., 2020; Lennartsson et al., 2013; Morgan et al., 2009; Wang et al., 2009). Complementarily, heart rate variability (HRV) indexes autonomic flexibility—the dynamic balance between sympathetic and parasympathetic modulation—and in golf specifically allows evaluation of how efficiently athletes regulate arousal, maintain emotional stability, and recover between high-demand moments (Laborde et al., 2017; Shaffer and Ginsberg, 2017; Thayer et al., 2012). The core HRV indices for characterizing competitive autonomic adjustment include HR, total power, LF, HF, and LF/HF ratio as frequency-domain markers, and RMSSD as a time-domain measure sensitive to vagal modulation and central autonomic control under pressure (Damoun et al., 2024; Quigley et al., 2024; Schaeuble and Myers, 2022). Collectively, these autonomic and endocrine responses differ across challenge and threat appraisals in ways that predict performance outcomes (Meijen et al., 2020; Schaeuble and Myers, 2022).

Performance in precision sports further depends on the central nervous system's ability to efficiently regulate cortical excitability—a process crucial for attentional stability and motor preparation under demand (Pearce and Kidgell, 2009). Expert athletes demonstrate more stable and economically modulated cortical activation patterns than novices, a phenomenon described by neural efficiency theory (Bertollo et al., 2016; Wang et al., 2020). Both biofeedback and neurofeedback approaches have been developed to train this capacity: multimodal biofeedback protocols providing real-time access to HRV, galvanic skin response, and peripheral temperature have improved cognitive function and reduced state anxiety in competitive athletes (Makaracı et al., 2024; Mirifar et al., 2017), while EEG-based neurofeedback further extends self-regulation training to specific cortical parameters (Brito et al., 2022). Among neurofeedback modalities, slow cortical potential (SCP) training targets slow polarization shifts reflecting neuronal excitability—negative SCPs linked to preparation and activation, positive SCPs to inhibitory regulation—and has produced stable improvements in attentional control and cortical self-regulation in multicenter clinical trials (Elbert, 1993; Mayer et al., 2016; Strehl, 2009; Strehl et al., 2017) as well as in healthy adults (Gevensleben et al., 2014), supporting its application in sport contexts (Hasslinger et al., 2022; Kimura et al., 2024). Using this same standardized SCP protocol in competitive golfers, our group previously reported neurophysiological effects consistent with improved cortical self-regulation and neural efficiency under ecologically valid competitive conditions (Lizama et al., 2025). A theoretical rationale for examining SCP training in relation to autonomic and endocrine responses comes from neurovisceral integration models, which link cortical regulatory systems with autonomic and HPA-axis activity (Thayer et al., 2012; Matusik et al., 2023). Within this neurovisceral integration framework, enhanced prefrontal inhibitory tone would support higher vagal activity and attenuated HPA reactivity—suggesting that SCP-induced improvements in cortical self-regulation may propagate through this cortical-to-autonomic-to-endocrine cascade to influence HRV and cortisol/DHEA responses under competitive pressure (Schaeuble and Myers, 2022).

Despite growing interest in competitive stress physiology and neurofeedback applications in sport, direct evidence examining how standardized SCP training influences HRV and stress-related hormones in athletes under ecologically relevant competitive conditions remains limited. The present exploratory study addresses this gap by examining whether SCP training is associated with different autonomic, endocrine, and performance-related patterns across repeated competitive exposure in golfers, compared with controls, using HRV, cortisol, DHEA, and putting performance measures collected during simulated competition. Based on the theoretical framework outlined above, we explored whether: (1) golfers who completed SCP training show different HPA-axis-related responses under competitive conditions, reflected in cortisol levels and the cortisol/DHEA ratio, compared with controls; (2) SCP training would be associated with different HRV patterns during competition, particularly in RMSSD, HF power, and LF/HF ratio; and (3) these autonomic and endocrine patterns would be accompanied by differences in performance outcomes under competitive pressure.

Table 1
Baseline demographic and performance characteristics of the sample.

Variable	Control group (n = 21)	Training group (n = 21)	*p*	Cohen's *d*
Age, years, M ± SD	46.48 ± 13.41	40.57 ± 13.18	.16	0.44
Age range, years	25–67	17–64	—	—
Male sex, n (%)	16 (76.19%)	15 (71.43%)	.73	—
Female sex, n (%)	5 (23.81%)	6 (28.57%)	—	—
Handicap, M ± SD	9.71 ± 7.88	7.91 ± 9.57	.51	0.22
Handicap range	0–26	−3–35	—	—

Note. Values are presented as mean ± SD unless otherwise indicated. Sex differences were tested using a chi-square or Fisher's exact test, as appropriate. Age and handicap were compared using independent-samples *t*-tests. Cohen's d is reported for continuous variables only. SD = standard deviation.

2. Methods

2.1. Participants

A total of 42 amateur golfers aged 22–64 years participated in the study (mean age = 43.00 years, SD = 13.13). All were active players from golf clubs in Santiago, Chile, training at least once per week. The sample covered a wide range of competitive levels, with handicaps spanning from PRO and low negative values (−3, −1) to high handicaps (up to 35), ensuring representation of elite, advanced, and amateur golfers. To balance group characteristics, participants were evenly assigned to the intervention or control group through stratified randomization based on handicap, resulting in 21 participants per group: the control group had a mean age of 46.48 years (SD = 13.41) and a mean handicap of 9.71 (SD = 7.88), whereas the training group had a mean age of 40.57 years (SD = 13.18) and a mean handicap of 7.91 (SD = 9.57), with no significant between-group differences in age (p = .16, Cohen's d = 0.44) or handicap (p = .51, Cohen's d = 0.22) (Table 1).

Sample size was calculated with a priori analysis using G-Power 3.1.9.7. The primary comparison of interest was the difference between control and intervention groups at the post-intervention assessment, for which a two-tailed independent samples test was conducted with α = 0.05, statistical power of 70%, and an expected effect size of Cohen's d = 0.8 (Panjeh et al., 2023). Based on these parameters, the required

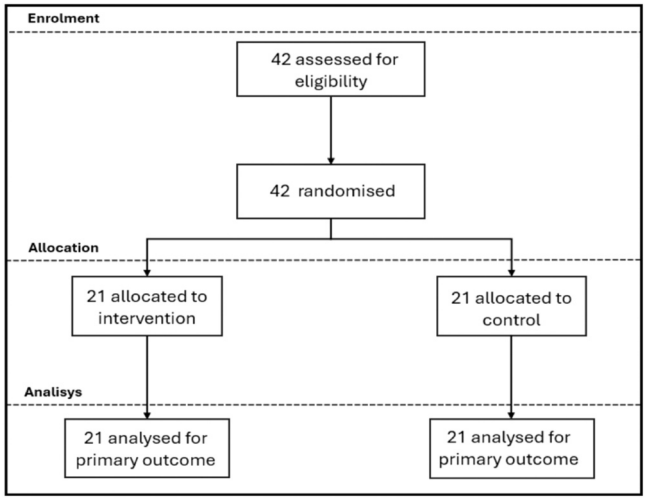

Fig. 1. CONSORT-style flowchart showing participant screening, stratified randomization of 42 golfers, allocation to intervention and control groups, and final inclusion of 21 participants per group in the analysis.

sample size was estimated to be 21 participants per group (42 in total). Although the power is lower than the conventional 80% benchmark, it was selected due to the exploratory nature of the study and was considered sufficient to address the primary research question.

Eligibility criteria included being an active golf player and providing informed consent. Exclusion criteria followed previous guidelines and included (Gamaiunova et al., 2022): recent changes in psychoactive medication, use of benzodiazepines or hypnotic agents, current treatment for major psychiatric disorders (e.g., bipolar, schizophrenia), significant medical conditions interfering with participation, and the use of medications affecting central nervous system or HPA axis function.

This study followed a two-group, parallel randomized controlled trial design with a superiority framework. Both groups completed pre- and post-intervention assessments over an eight-week period, including characterization of psychological, physiological, and neurobiological parameters, SCP evaluation, and sports performance under simulated competition. One participant was evaluated per day (Monday to Friday, 8:00–11:00 a.m., Santiago, Chile) at the designated golf sports facility. The control group followed the same assessment schedule without receiving SCP training and was offered the intervention after the study was completed.

2.2. Randomization and allocation procedures

Random allocation was performed using a computer-generated sequence created with Randomizer.org by a researcher who was not involved in participant assessment or intervention delivery. The allocation sequence was kept by this researcher and was not available to the team responsible for enrolling participants or conducting baseline evaluations. Group assignment was disclosed only after each participant completed baseline procedures, ensuring that the sequence remained concealed until allocation. Participants were enrolled by the assessment team, who remained blinded to the allocation sequence, and the independent researcher who generated the sequence implemented the final group assignments. Stratified randomisation based on handicap was used to maintain comparable levels of competitive experience across groups Fig. 1.

Data analysts were not formally blinded to group allocation during HRV and hormone processing, and full allocation concealment could not be completely ensured because of the nature of the neurofeedback intervention and the wait-list control design; this represents a methodological limitation, as it may have introduced expectancy- or assessment-related bias.

2.3. Neuropsychological status

The neurophysiological assessment focused on slow cortical potentials (SCPs), which provide a measure of participants' ability to voluntarily regulate cortical excitability. SCPs were recorded at Cz, a central scalp location commonly used in SCP neurofeedback protocols, to assess whether participants could intentionally shift brain activity in two directions: toward cortical activation, reflected by negative SCP shifts, and toward cortical inhibition, reflected by positive SCP shifts. At both pre- and post-intervention assessments, participants completed a brief set of activation and deactivation trials designed to evaluate this self-regulatory capacity outside the training sessions. Thus, these recordings were used as neurophysiological outcome measures of learned cortical regulation, whereas the full training protocol, feedback procedures, and session parameters are described in 2.6.

2.4. Physiological status

Salivary cortisol and dehydroepiandrosterone (DHEA) were assessed before and after the intervention to characterize endocrine responses to competitive exposure. In each evaluation session (pre- and post-intervention), saliva was collected twice: (1) immediately after completing the psychological questionnaires (pre-task sample), and (2) immediately after completing the sports performance assessment (post-task sample). Thus, each participant provided four saliva samples across the study (two per session). Samples were collected in 5 mL tubes, centrifuged and stored at −80 °C until analysis. Hormone concentrations were quantified using commercial ELISA kits (Thermo Fisher Scientific; cortisol Catalog # EELR004, DHEA Catalog # EIADHEA). The intra- and inter-assay coefficients of variation were 8.8% and 8.1% for cortisol, and 7.0% and 8.4% for DHEA, respectively. The DHEA/cortisol ratio was computed as an additional index reflecting the balance between stress-related activation and adaptive hormonal modulation.

Heart rate variability (HRV) was recorded using a Polar H10 chest-strap sensor and processed with the Elite HRV platform. A 5-min resting baseline was obtained before the simulated tournament under seated and quiet conditions, providing a pre-competition autonomic reference. During the tournament, HRV was recorded continuously, and R–R interval series were exported for subsequent analysis. Prior to HRV calculation, raw R–R interval data were visually inspected and processed using the platform's automated artifact-detection and correction procedures. The Elite HRV preprocessing pipeline identifies potential movement artifacts, ectopic beats, and physiologically implausible intervals using a software-native threshold-based filtering algorithm. Flagged intervals were corrected using the platform's interpolation procedures in order to preserve the continuity of the R–R interval series required for time- and frequency-domain analyses. Recordings or segments classified by the platform as poor quality or showing excessive signal noise or unstable recording quality upon visual inspection, were not retained for HRV computation.

To further minimize the influence of extreme values on statistical analyses, secondary outlier screening was conducted after extraction of HRV variables. This screening was performed dynamically according to the distribution of each variable: normally distributed variables were screened using a Z-score threshold of ±3 SD, whereas non-normally distributed variables were screened using the interquartile range method, with values outside 1.5 × IQR considered potential outliers. When an outlier was identified in a longitudinal comparison, the corresponding participant was excluded from that specific paired analysis to preserve the integrity of the repeated-measures design. Normality was assessed using the Shapiro–Wilk test.

To characterize autonomic regulation during performance, HRV indices were calculated for the competitive task period using an averaged in-game segment across the four distance blocks, with a mean segment duration of approximately 5 min. When applicable, exploratory analyses were also conducted for the distance-specific blocks of the

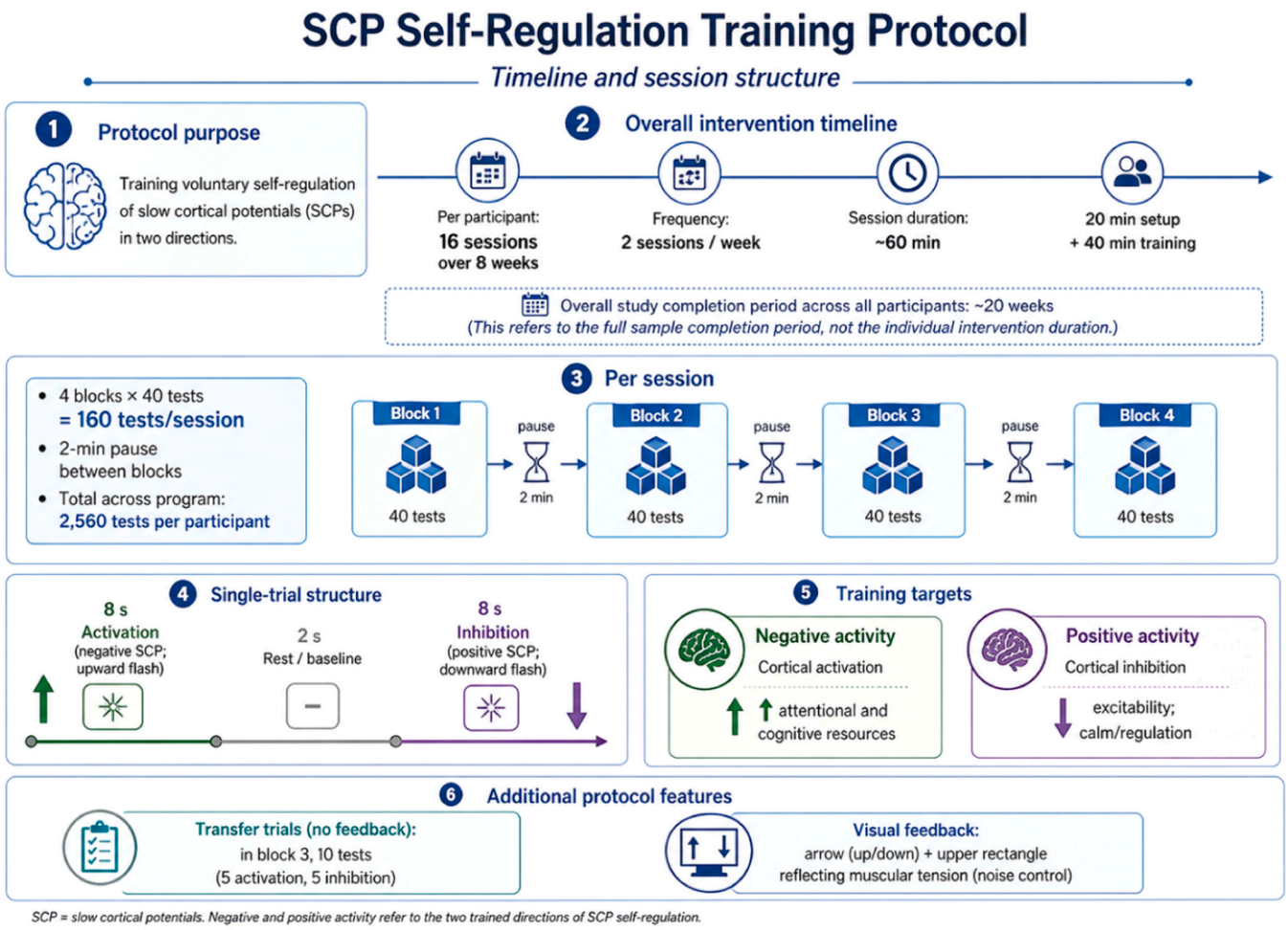

Fig. 2. Diagram summarizing the structure of the SCP neurofeedback protocol, including session design, activation/inhibition tasks, transfer trials, and visual feedback configuration.

putting protocol, using the full available R–R interval segment corresponding to each block, up to a maximum duration of 5 min per distance. The analyzed HRV variables included mean heart rate (HR), total power (TP), low-frequency power (LF), high-frequency power (HF), the LF/HF ratio, and RMSSD. These indices provided complementary information on overall cardiovascular activation, global variability magnitude, frequency-domain HRV components, and short-term vagal-related dynamics. Because spectral HRV indices are particularly sensitive to preprocessing decisions and respiration, LF, HF, and LF/HF were interpreted cautiously.

2.5. Sports performance assessment

Sports performance was evaluated through a standardized simulated putting task in which each participant executed 120 putts per session. Putts were divided into four distances (4 m, 3 m, 2 m, and 1 m; 30 putts per distance) and were performed on the same natural green under identical conditions in both the pre- and post-intervention evaluations. Each participant attended an individual one-hour session to complete the full task. The task was designed as a standardized competitive exposure rather than a head-to-head contest. Performance was operationalized as the number of successful putts holed on the first attempt (total score, 0–120), yielding one performance score in Tournament 1 (pre-intervention) and one in Tournament 2 (post-intervention) (Dias et al., 2021). Group effects were evaluated by comparing pre-post changes between the training and control groups.

2.6. Slow cortical potentials neurofeedback intervention

The SCP neurofeedback intervention followed the standardized protocol previously reported by our group in competitive golfers, where its implementation and neurophysiological effects on neural efficiency and beta activity modulation were described in detail (Lizama et al., 2025). In the present study, the intervention is summarized to clarify how the training was embedded within the broader randomized protocol and how it preceded the endocrine, autonomic, and performance assessments shown in the timeline figure Fig. 2.

Briefly, participants in the training group completed 16 SCP neurofeedback sessions over 8 weeks, with two sessions per week. Training was recorded at Cz using Ag/AgCl electrodes, with vertical and horizontal EOG channels included to monitor ocular artifacts; electrode impedances were kept below 5 kΩ. Each session lasted approximately 60 min including preparation and setup, with around 40 min of active training. Participants completed four blocks of 40 trials per session, resulting in 160 trials per session and 2560 trials across the full intervention. Trials required participants to learn two complementary forms of cortical self-regulation: producing negative SCP shifts, associated with increased cortical activation, and positive SCP shifts, associated with cortical inhibition. Most trials included visual feedback, whereas transfer trials without feedback were interleaved to promote generalization of self-regulation beyond the training context.

All sessions were conducted in the same facility and supervised by a certified neurofeedback practitioner. The control group followed the same assessment schedule but did not receive neurofeedback during the

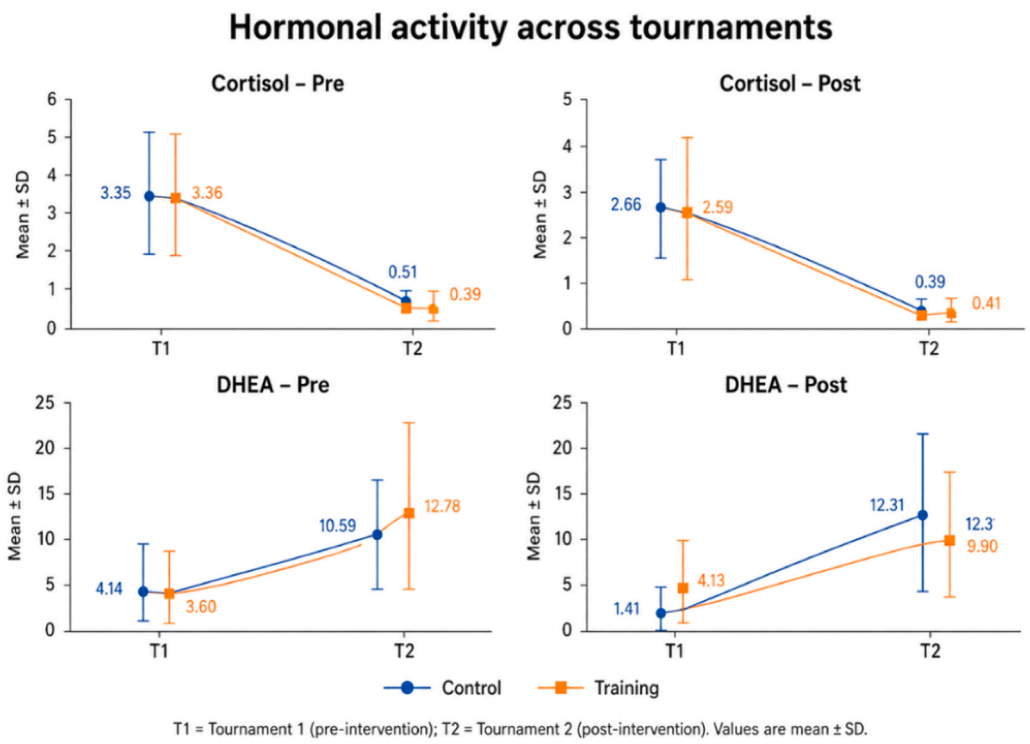

Fig. 3. Cortisol and DHEA responses across tournaments and groups. Data are presented as mean ± SD.

study period, serving as a wait-list control group. Participants in the control group were offered the intervention after completing the study.

2.7. Statistical analysis

All variables were tested for normality prior to analysis. When data met parametric assumptions, group comparisons were performed using Student's *t*-tests (paired *t*-tests for within-group pre–post comparisons and independent *t*-tests for between-group comparisons). When assumptions were violated, non-parametric alternatives were applied (Wilcoxon signed-rank tests for within-group changes and Mann–Whitney U tests for between-group comparisons). Effect sizes were quantified as Cohen's d for parametric tests and rank biserial correlation (RBC) for non-parametric tests. Associations between hormonal and HRV variables were examined using Spearman's rank correlation (ρ). Results are reported as mean ± SD or median [IQR], depending on the distribution of each variable. Statistical significance was set at $p < 0.05$. All analyses were conducted using Python®. All participants were included in the statistical analyses according to their originally assigned groups, and no missing data were present, as all participants completed every assessment.

It is important to note that given the exploratory nature of the study and the number of physiological and correlational analyses performed, false discovery rate (FDR) corrections using the Benjamini–Hochberg procedure were calculated and are reported in the Appendices to provide a transparent assessment of the robustness of the findings after adjustment for multiple comparisons.

3. Results

Results section report the uncorrected analyses to describe the exploratory pattern of results; however, FDR-corrected analyses are provided in the Appendices (1 & 2), and the principal group-related physiological and correlational findings did not remain statistically significant after correction for multiple comparisons.

Table 2
Hormonal activity across tournaments.

Hormone	Sample	Tournament	Control	Training
Cortisol	Pre	T1	3.35 ± 1.33	3.36 ± 1.26
			3.15 [2.11]	3.48 [1.91]
		T2	0.51 ± 0.34	0.39 ± 0.28
			0.45 [0.57]	0.26 [0.29]
	Post	T1	2.66 ± 0.91	2.59 ± 1.47
			2.56 [1.27]	2.57 [2.03]
		T2	0.39 ± 0.23	0.41 ± 0.26
			0.29 [0.20]	0.32 [0.22]
DHEA	Pre	T1	4.14 ± 3.92	3.60 ± 3.46
			2.81 [5.68]	3.04 [5.90]
		T2	10.59 ± 5.68	12.78 ± 9.79
			10.98 [7.94]	11.32 [11.79]
	Post	T1	1.41 ± 1.63	4.13 ± 4.37
			0.71 [1.86]	2.90 [7.05]
		T2	12.31 ± 8.67	9.90 ± 6.50
			9.45 [10.48]	9.80 [9.43]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

3.1. Hormonal responses

Hormonal levels were assessed before and after each tournament to characterize endocrine responses to competitive stress. Detailed descriptive statistics for hormonal outcomes are provided in Fig. 3 and Table 2. Cortisol levels decreased from Tournament 1 to Tournament 2 at both sampling points in both groups. Specifically, pre-task cortisol decreased from Tournament 1 to Tournament 2 in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = −1.00) and in the control group (Wilcoxon signed-rank test, $p < .001$; RBC = −1.00) (Fig. 3). Similarly, post-task cortisol decreased from Tournament 1 to Tournament 2 in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = −0.98) and in the control group (Wilcoxon signed-rank test, $p < .001$;

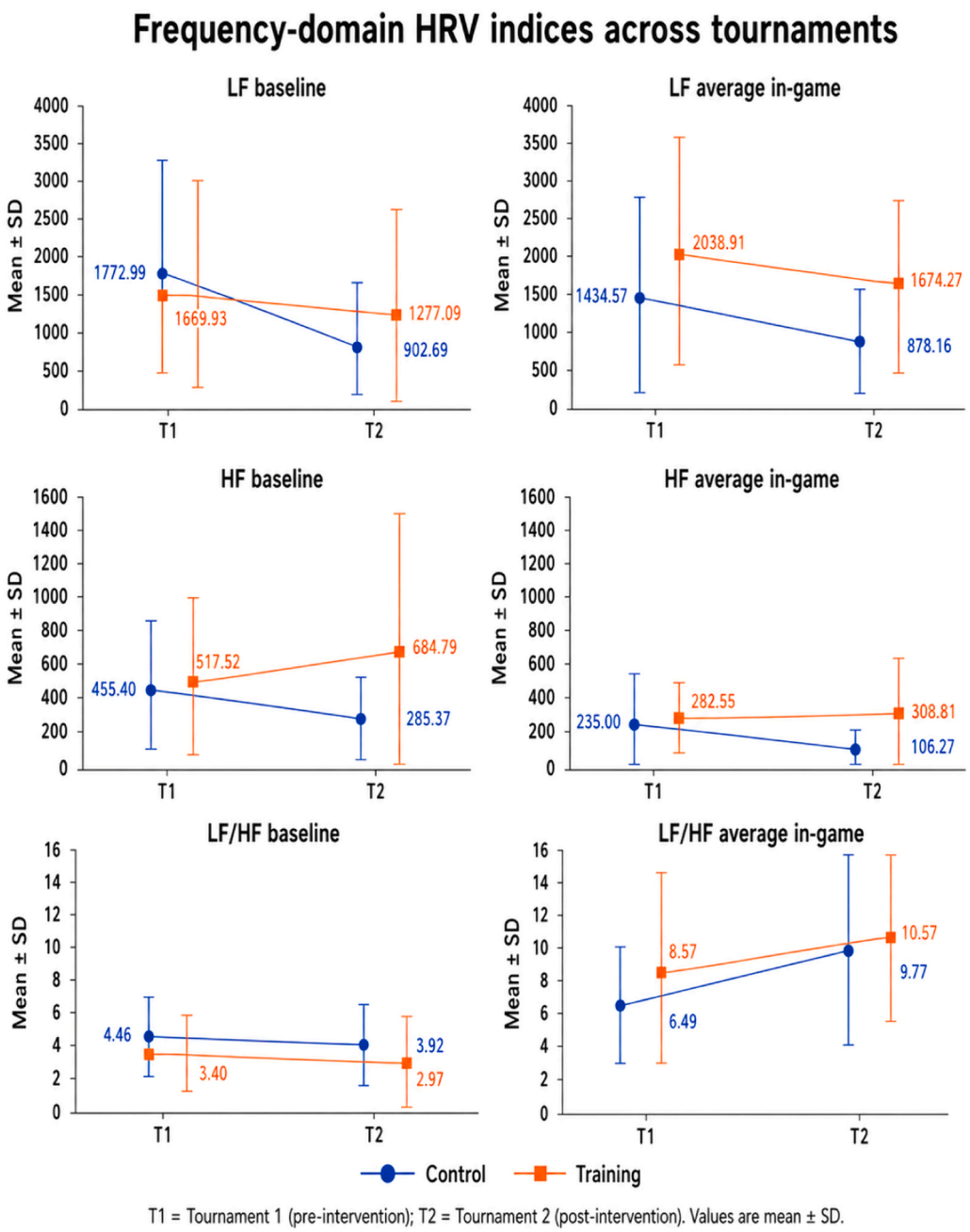

Fig. 4. Low-frequency (LF) power, high-frequency (HF) power, and LF/HF ratio are shown at baseline and during in-game performance for the control and training groups across both tournaments. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention. Data are presented as mean ± SD. Statistical significance refers to within-group changes from T1 to T2 and between-group differences at each tournament. n = 21 per group.

RBC = −1.00) (Fig. 3). Between-group comparisons at Tournament 1 and Tournament 2 showed no differences in cortisol at either time point (all Mann–Whitney U tests, $p > .05$) (Table 2). This tournament-related decrease is consistent with reduced endocrine reactivity upon re-exposure to the same competitive setting.

DHEA exhibited a trajectory opposite to cortisol, increasing from Tournament 1 to Tournament 2 at both sampling points in both groups (Fig. 3, Table 2). Pre-task DHEA increased significantly from Tournament 1 to Tournament 2 in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = 0.91) and in the control group (Wilcoxon signed-rank test, $p = .0029$; RBC = 0.83) (Fig. 3). Post-task DHEA also increased significantly in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = 0.68) and in the control group (Wilcoxon signed-rank test, $p < .001$; RBC = 0.99) (Fig. 3). Between-group comparisons did not show differences in DHEA at Tournament 1 or Tournament 2 for either time point (all Mann–Whitney U tests, $p > .05$).

Given the reciprocal cortisol and DHEA changes, the DHEA-to-cortisol ratio (DCR) was examined as an integrated index. DCR increased significantly from Tournament 1 to Tournament 2 in both groups: pre-task DCR increased in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = 1.00) and in the control group (Wilcoxon signed-rank test, $p < .001$; RBC = 1.00), and post-task DCR increased in the training group (Wilcoxon signed-rank test, $p < .001$; RBC = 0.96) and in the control group (Wilcoxon signed-rank test, $p < .001$; RBC = 0.99). Between groups, pre-task DCR did not differ at Tournament 1 or Tournament 2 (both Mann–Whitney U tests, $p > .05$). However, at Tournament 1, the training group showed higher post-task DCR than controls (Mann–Whitney U test, $p = .029$; RBC = 0.43), whereas no

Table 3
Low frequency across tournaments.

Variable	Tournament	Control	Training
LF baseline	T1	1772.99 ± 1286.54	1669.93 ± 1250.99
		1496.64 [2165.24]	1345.66 [1862.55]
	T2	902.69 ± 673.36	1277.09 ± 1179.66
		815.07 [1152.36]	772.55 [1878.83]
LF average in-game	T1	1434.57 ± 1267.24	2038.91 ± 1486.79
		922.07 [2122.31]	1818.14 [1831.68]
	T2	878.16 ± 724.55	1674.27 ± 1099.88
		627.13 [734.69]	1757.30 [1781.04]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; LF = low-frequency power; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

Table 4
High frequency across tournaments.

Variable	Tournament	Control	Training
HF baseline	T1	455.40 ± 365.38	517.52 ± 411.91
		425.91 [632.66]	365.19 [416.85]
	T2	285.37 ± 251.02	684.79 ± 779.62
		204.53 [406.31]	296.71 [824.46]
HF average in-game	T1	235.00 ± 240.20	282.55 ± 169.90
		147.62 [274.29]	258.27 [241.08]
	T2	106.27 ± 65.67	308.81 ± 304.08
		107.49 [102.12]	193.34 [328.71]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; HF = high-frequency power; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

between-group difference was observed at Tournament 2 (Mann–Whitney U test, $p = .76$; RBC = −0.06). Together, the increase in DHEA and the resulting rise in DCR from Tournament 1 to Tournament 2 are consistent with reduced endocrine stress reactivity during the second exposure.

Therefore, endocrine responses shifted across tournaments: cortisol decreased while DHEA increased from Tournament 1 to Tournament 2 in both groups, yielding a consistent rise in the DHEA-to-cortisol ratio (DCR). Taken together, this pattern suggests attenuated endocrine reactivity during the second exposure to the same competitive setting. Regarding SCP training, between-group contrasts for cortisol and DHEA did not show a systematic separation at the sampled time points, however, a discrete between-group effect emerged for post-task DCR at Tournament 1 in favor of the training group, which was not replicated at Tournament 2.

3.2. Heart Rate Variability (HRV) response

HRV-derived components were analyzed to determine whether SCP training influenced autonomic patterns across repeated exposure to the same competitive task.

Regarding frequency-domain indices (LF, HF, and LF/HF), baseline measures showed a re-exposure effect in the control group. Specifically, baseline LF decreased significantly from Tournament 1 to Tournament 2 in the control group (Wilcoxon signed-rank test, $p = .0014$; RBC = −0.77), indicating reduced low-frequency oscillatory modulation at rest (Fig. 4, Table 3). Similarly, baseline HF decreased significantly in the control group (Wilcoxon signed-rank test, $p = .0175$; RBC = −0.58) (Fig. 4, Table 4).

During gameplay (average in-game), a post-intervention between-group divergence emerged. The training group showed significantly higher LF than controls (Mann–Whitney U test, $p = .0257$; RBC = 0.44)

Table 5
LF/HF ratio across tournaments.

Variable	Tournament	Control	Training
LF/HF baseline	T1	4.46 ± 2.09	3.40 ± 1.79
		4.17 [2.41]	3.14 [1.87]
	T2	3.92 ± 2.14	2.97 ± 2.45
		3.84 [2.35]	2.56 [3.25]
LF/HF average in-game	T1	6.49 ± 3.03	8.57 ± 5.94
		6.71 [3.38]	7.67 [7.23]
	T2	9.77 ± 5.95	10.57 ± 5.18
		9.31 [6.79]	10.87 [5.21]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; LF/HF = low-frequency to high-frequency ratio; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

(Fig. 4, Table 3).

In parallel, the training group also showed significantly higher HF than controls (Mann–Whitney U test, $p = .0340$; RBC = 0.41) (Fig. 4, Table 4). Additionally, the LF/HF ratio during gameplay increased significantly from Tournament 1 to Tournament 2 in both groups (training: paired Student's t-test, $p = .0139$; $d = 0.36$; control: paired Student's t-test, $p = .0152$; $d = 0.70$) (Fig. 4, Table 5).

These results could indicate that, although the task elicited the expected physiological activation, trained participants displayed higher overall autonomic modulation during execution compared with controls.

For global/time-domain indices (HR, total power [TP], and RMSSD), several significant effects were observed (Fig. 5). Baseline HR did not differ significantly across tournaments within either group (Fig. 5, Table 6). During gameplay, HR did not show a significant pre–post change within the training group (paired Student's t-test, $p = .0901$; $d = -0.39$) or within the control group (paired Student's t-test, $p = .865$; $d = -0.04$). However, the magnitude of change differed between groups (Mann–Whitney U test, $p = .0201$; RBC = −0.45), indicating distinct tournament-related HR change patterns in the training group relative to controls (Fig. 5, Table 6).

For total power (TP), baseline values decreased significantly from Tournament 1 to Tournament 2 in the control group (Wilcoxon signed-rank test, $p < .001$; RBC = −0.80), whereas no significant baseline change was observed in the training group (Fig. 5, Table 7). During gameplay, TP also decreased significantly in the control group (Wilcoxon signed-rank test, $p = .0483$; RBC = −0.53), and post-intervention gameplay TP was significantly higher in the training group than in controls (Mann–Whitney U test, $p = .0157$; RBC = 0.47) (Fig. 5, Table 7).

For RMSSD, baseline values decreased significantly in the control group (paired Student's t-test, $p = .0183$; $d = -0.62$), while remaining stable in the training group (Fig. 5, Table 8. During gameplay, RMSSD decreased significantly from Tournament 1 to Tournament 2 in the control group (Wilcoxon signed-rank test, $p = .0385$; RBC = −0.56), whereas post-intervention gameplay RMSSD was significantly higher in the training group than in controls (Mann–Whitney U test, $p = .0312$; RBC = 0.41) (Fig. 5, Table 8). Overall, these uncorrected effects suggest a descriptive pattern of relatively higher HRV indices in the training group during task execution, but this pattern should not be interpreted as confirmatory evidence of training-related autonomic change.

Together, considering the exploratory nature of the study, these results suggest a descriptive pattern in which the control group showed reductions in overall HRV magnitude and short-term vagal-related indices at re-exposure, whereas the training group showed relatively stable HRV values across tournaments and higher post-intervention levels during gameplay. In the uncorrected analyses, the training group showed higher LF, HF, TP, and RMSSD during gameplay at Tournament 2 compared with controls. However, as these group-related

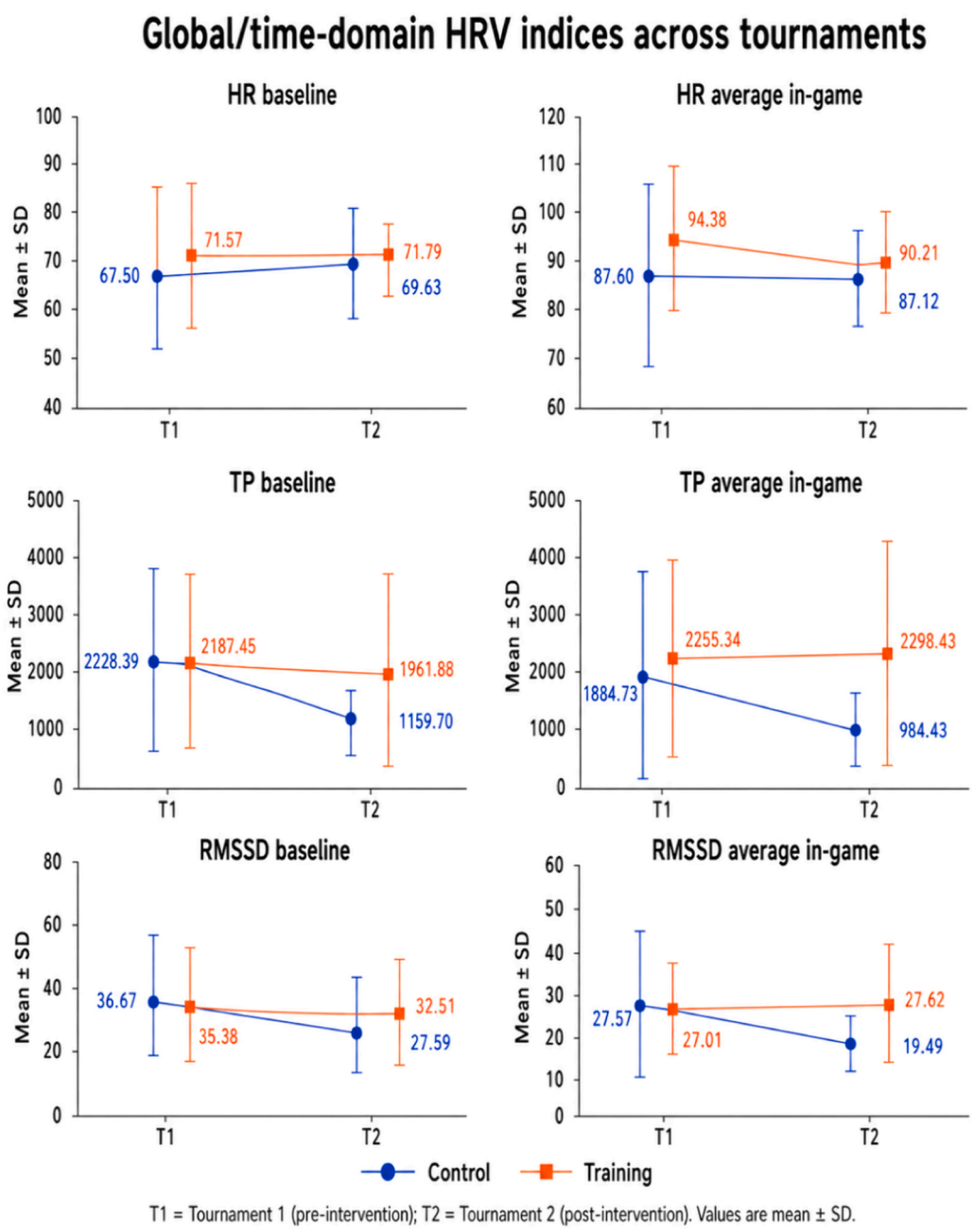

Fig. 5. Heart rate, total power and RMSSD before and during competitive performance. Heart rate (HR) before competition, Mean heart rate in-game, Total power of HRV before competition, Mean in-game total power, RMSSD before competition and Mean in-game RMSSD. Data are presented as mean ± SD. Statistical significance corresponds to pre- to post-tournament comparisons and between-group differences during the second tournament. $n = 21$ per group.

HRV findings did not remain significant after FDR correction (see appendix 1), they should be interpreted as exploratory descriptive patterns rather than as evidence of enhanced autonomic regulation.

3.3. *Performance during the tournament*

Performance outcomes were evaluated across both tournaments to determine whether SCP training influenced putting accuracy and overall game performance (Fig. 6, Table 9). For total score both groups showed small, non-significant decreases from Tournament 1 to Tournament 2 (training: paired Student's *t*-test, $p = .255$; $d = -0.29$; control: paired Student's *t*-test, $p = .206$; $d = -0.31$). Between-group differences were not significant at baseline (T1: independent Student's *t*-test, $p = .462$; $d = 0.23$) or at post-intervention (T2: independent Student's *t*-test, $p = .228$; $d = 0.38$). In addition, the pre–post change did not differ between groups ($p = .858$; $d = 0.06$).

For average in-game performance (Fig. 6, Table 9), the training group showed a small, non-significant change from Tournament 1 to Tournament 2 (paired Student's *t*-test, $p = .255$; $d = -0.29$), and the control group showed a similarly non-significant change (paired Student's *t*-test, $p = 0.182$; $d = -0.37$). Between-group comparisons were also non-significant at baseline (T1: independent Student's *t*-test, $p = 0.62$; $d = 0.15$) and at post-intervention (T2: independent Student's *t*-test, $p = 0.307$; $d = 0.32$), and the pre–post change did not differ between groups (independent Student's *t*-test, $p = 0.725$; $d = 0.11$).

Table 6
Heart Rate (HR) across tournaments.

Variable	Tournament	Control	Training
HR baseline	T1	67.50 ± 8.55	71.57 ± 9.58
		69.26 [10.51]	73.79 [13.75]
	T2	69.63 ± 9.84	71.79 ± 5.81
		72.30 [11.38]	72.52 [8.39]
HR average in-game	T1	87.60 ± 14.50	94.38 ± 12.21
		85.40 [17.27]	93.37 [14.66]
	T2	87.12 ± 9.79	90.21 ± 9.06
		86.32 [11.70]	91.86 [12.64]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; HR = heart rate; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

Table 7
Total power across tournaments.

Variable	Tournament	Control	Training
Total power baseline	T1	2228.39 ± 1616.06	2187.45 ± 1564.73
		1935.54 [2945.62]	1918.52 [2139.22]
	T2	1159.70 ± 849.22	1961.88 ± 1718.05
		1095.00 [1411.75]	1562.81 [2233.36]
Total power average in-game	T1	1884.73 ± 1757.58	2255.34 ± 1550.62
		1069.69 [3106.24]	2153.50 [2149.51]
	T2	984.43 ± 774.46	2298.43 ± 1856.74
		720.99 [821.46]	2317.98 [2746.09]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

Table 8
RMSSD across tournaments.

Variable	Tournament	Control	Training
RMSSD baseline	T1	36.67 ± 16.35	35.38 ± 14.80
		37.13 [27.63]	34.00 [16.59]
	T2	27.59 ± 12.54	32.51 ± 13.31
		23.40 [19.06]	30.92 [16.19]
RMSSD average in-game	T1	27.57 ± 15.55	27.01 ± 8.79
		24.59 [26.02]	27.46 [8.32]
	T2	19.49 ± 7.96	27.62 ± 12.10
		17.49 [8.90]	27.24 [19.05]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; RMSSD = root mean square of successive differences; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

3.4. Associations between HRV and hormonal responses

To examine whether endocrine responses covaried with autonomic regulation during competition, Spearman correlation matrices were computed between salivary hormones and HRV indices derived at baseline and during gameplay (Figs. 7 and 8).

In Tournament 1, hormone–HRV associations during gameplay were generally weak in the control group, with no consistent moderate correlations emerging across HRV markers. In contrast, the training group showed several moderate associations in Tournament 1, most notably between pre-task DCR and average in-game HR ($\rho = 0.69$), as well as negative correlations between post-task hormone levels and average in-game HR (post-task DHEA: $\rho = -0.43$; post-task cortisol: $\rho = -0.42$).

In Tournament 2, post-task cortisol correlated negatively with multiple gameplay HRV metrics, including average in-game HR ($\rho = -0.61$), average in-game RMSSD ($\rho = -0.43$), average in-game LF power ($\rho = -0.51$), average in-game HF power ($\rho = -0.42$), and average in-game TP ($\rho = -0.45$) (Fig. 7).

Conversely, in the training group at Tournament 2, post-task cortisol showed minimal associations with gameplay HRV, whereas DHEA and DCR displayed moderate positive correlations with HRV magnitude during gameplay. For example, pre-task DHEA correlated with average in-game HF power ($\rho = 0.52$) and average in-game TP ($\rho = 0.45$), and post-task DCR correlated with average in-game TP ($\rho = 0.54$) and average in-game HF power ($\rho = 0.50$) (Fig. 8).

The correlation matrices are therefore presented as exploratory descriptive analyses. Although some moderate uncorrected associations were observed between hormonal markers and gameplay HRV indices, none of the focal hormone–HRV gameplay correlations remained statistically significant after FDR correction, as reported in the Appendix 2. Accordingly, these results should be interpreted as hypothesis-generating patterns rather than as evidence of robust endocrine–autonomic covariation.

4. Discussion

The present pilot study examined the effects of a slow cortical potential (SCP) neurofeedback training protocol in amateur golfers and explored associated neurophysiological, endocrine, autonomic, and performance-related changes during a simulated competitive setting. SCP neurofeedback enables voluntary self-regulation of cortical excitability (Strehl et al., 2006), and the cortical results suggest that participants in the training group acquired the intended neural control. In line with previous evidence from our group using this SCP approach in competitive golfers (Lizama et al., 2025), these findings provide an initial validation of the training manipulation. Before interpreting the findings, it should be noted that this was an exploratory study with a modest sample size and multiple physiological endpoints. Although the main text reports the uncorrected analyses to describe the exploratory pattern of results, the principal group-related physiological and correlational findings did not remain statistically significant after FDR correction for multiple comparisons, as reported in the Appendices. Accordingly, the endocrine and autonomic findings should be interpreted cautiously as preliminary and hypothesis-generating rather than confirmatory.

Hormonal analyses revealed a tournament-dependent pattern rather than a specific training effect. Cortisol levels were higher during Tournament 1 at both pre- and post-task assessments in both groups, consistent with the novelty, uncertainty, and anticipatory demands of the first competitive exposure (Van Paridon et al., 2017). This profile is also coherent with previous endocrine observations in golf competition contexts (Doan et al., 2007; Wang et al., 2009). In Tournament 2, cortisol decreased markedly, whereas DHEA increased, leading to a strong increase in the DHEA/cortisol ratio (DCR) in both groups. This pattern could suggest a shift toward a less catabolic and potentially more adaptive endocrine profile with repeated exposure (Morgan et al., 2009), most likely reflecting habituation to the competitive context rather than a direct effect of SCP training (Kudielka and Wüst, 2010). The absence of consistent between-group differences supports this interpretation.

HRV analyses provided complementary information on autonomic regulation before and during competitive performance (Laborde et al., 2017). At baseline, several HRV indices decreased from Tournament 1

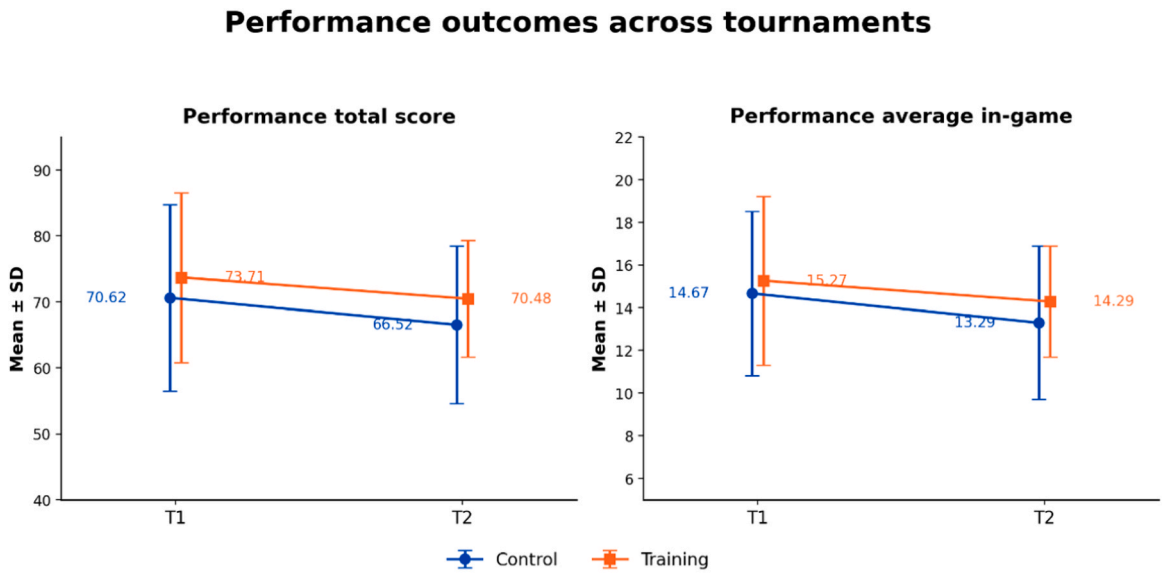

Fig. 6. Putting performance before and after the SCP intervention. **Left:** Total putting score for the control and training groups. **Right:** Mean in-game performance across all putting attempts. Data are presented as mean ± SD. No statistically significant differences were found between assessments. $n = 21$ per group.

Table 9
Performance across tournaments.

Variable	Tournament	Control	Training
Performance total score	T1	70.62 ± 14.12	73.71 ± 12.88
		71.00 [17.00]	69.00 [15.00]
	T2	66.52 ± 11.91	70.48 ± 8.81
		63.00 [12.00]	71.00 [9.00]
Performance average in-game	T1	14.67 ± 3.85	15.27 ± 3.96
		14.00 [6.67]	14.00 [5.00]
	T2	13.29 ± 3.59	14.29 ± 2.59
		12.67 [5.33]	14.33 [3.00]

Note. T1 = Tournament 1, pre-intervention; T2 = Tournament 2, post-intervention; Control = group with no intervention; Training = group with SCP intervention. Values are presented as M ± SD on the first line and Mdn [IQR] on the second line. M = mean; SD = standard deviation; Mdn = median; IQR = interquartile range.

to Tournament 2, particularly in the control group. One interpretation is that repeated exposure reduced anticipatory autonomic activation because the competitive situation became more familiar and less uncertain (Schommer et al., 2003). However, an alternative explanation is that the control group may have shown reduced engagement or motivational involvement at re-exposure, especially given the lack of intervention and the absence of blinding. Therefore, lower HRV in controls at Tournament 2 should not be interpreted solely as a maladaptive response; it may also reflect habituation, reduced challenge appraisal, or lower task salience.

During gameplay, the training group showed higher LF, HF, TP, and RMSSD than controls at Tournament 2, whereas several indices decreased in the control group. This pattern may suggest a relative preservation of autonomic variability during performance after SCP training. Nevertheless, this interpretation must remain cautious. Although HF and RMSSD are commonly considered vagal-related indices, respiration was not controlled during gameplay, and changes in breathing rate or depth may have influenced HF power and other HRV measures. Similarly, LF should not be interpreted as a pure marker of sympathetic activity because it includes both sympathetic and parasympathetic contributions and is strongly context-dependent (Billman, 2013; Shaffer and Ginsberg, 2017). For this reason, the LF/HF ratio was treated only as a complementary descriptive index, not as a direct measure of sympathovagal balance, given the substantial criticism of this interpretation in the HRV literature (Billman, 2013).

The observed preservation of TP and RMSSD in the training group may suggest a more sustained maintenance of autonomic variability during competitive execution, although this remains an inferential interpretation. Importantly, the study did not include direct measures of prefrontal activation during performance, functional connectivity, or mediation analyses linking SCP modulation to autonomic or endocrine outcomes. Therefore, any proposed cascade from SCP regulation to cortical control, autonomic modulation, and endocrine adaptation is speculative. The present findings are better understood as preliminary evidence that SCP training may be associated with altered psychophysiological regulation during re-exposure to competition, while the specific cortical–autonomic mechanisms remain untested.

The broad age range of the sample should also be considered when interpreting HRV and endocrine outcomes. Participants ranged from 22 to 64 years old, and both HRV and endocrine stress markers are known to vary with age. Age-related declines in HRV and changes in HPA-axis and adrenal hormone activity may have contributed to interindividual variability and may partly explain the wide dispersion observed in some HRV indices. In addition, several HRV variables showed substantial variability, especially frequency-domain measures such as LF, HF, and TP. These extreme or highly dispersed values are consistent with the known sensitivity of spectral HRV indices to individual differences, recording conditions, breathing patterns, and movement-related noise. Consequently, the group-level patterns should be interpreted with caution, particularly in the context of a small pilot sample.

The behavioral results are theoretically important. No statistically significant improvement was observed in putting performance, either for total score or average in-game performance. This null effect indicates that SCP training, despite producing evidence of learned neural regulation and being associated with some physiological differences, did not translate into measurable performance gains in this protocol. Several explanations are possible. First, physiological regulation may change before behavioral improvement becomes detectable, especially in complex motor tasks where performance depends on technical skill, practice history, attentional control, and contextual factors. Second, the putting task may not have been sensitive enough to capture subtle performance changes. Third, the intervention may have influenced psychophysiological state regulation without producing a direct

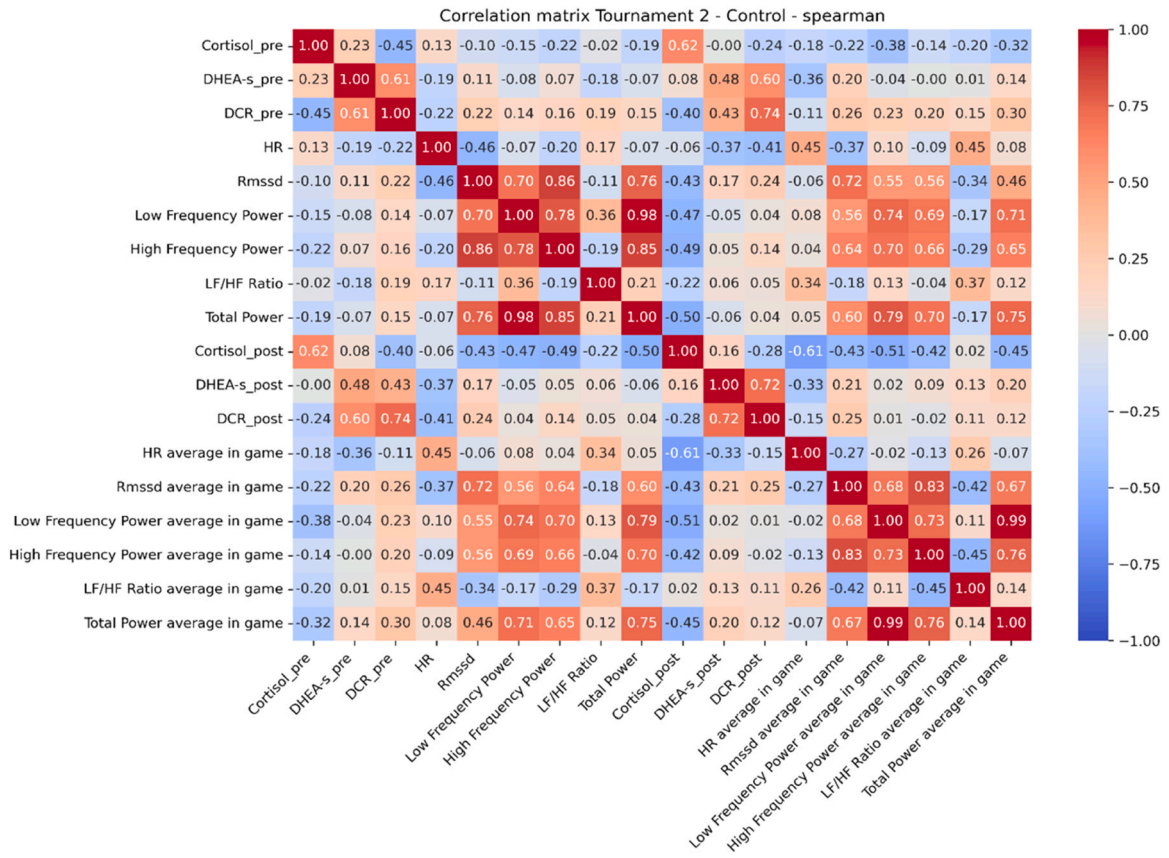

Fig. 7. Uncorrected Spearman correlation matrix (Tournament 2-Control group) between salivary hormones and HRV indices. Cells show Spearman's rho (ρ) for pairwise associations among cortisol and DHEA concentrations collected pre and post-task (Cortisol_pre, Cortisol_post, DHEA_pre, DHEA_post) and HRV metrics computed at baseline and during gameplay (average in game), including average HR, RMSSD, Low Frequency Power, High Frequency Power, LF/HF ratio, and Total Power. Warmer colors indicate positive associations and cooler colors indicate negative associations (scale −1–1). Correlations are presented without correction for multiple comparisons and should therefore be interpreted cautiously as exploratory associations.

improvement in motor accuracy. Therefore, the discrepancy between physiological changes and null performance findings is central to the interpretation of the study and indicates that SCP training cannot be considered performance-enhancing on the basis of the present data alone.

The exploratory correlation matrices could add preliminary information about endocrine–autonomic covariation across tournaments and groups. In Tournament 1, hormone–HRV associations during gameplay were generally weak in controls, whereas the training group showed some moderate associations between DCR, post-task hormones, and average in-game heart rate. In Tournament 2, controls showed negative associations between post-task cortisol and several gameplay HRV indices, whereas the training group showed weaker cortisol–HRV correlations and moderate positive associations between DHEA/DCR and HRV power. These patterns may suggest that endocrine and autonomic measures varied differently across groups at re-exposure. However, given the large number of pairwise correlations, the lack of correction for multiple comparisons, and the small sample size, the risk of Type I error is substantial. These associations should therefore be interpreted as exploratory, not confirmatory, and they require replication in larger, adequately powered samples.

5. Limitations

Several limitations should be acknowledged. First, this was a pilot study with a modest sample size, and the a priori power was set at 70%, meaning that the study was powered only to detect large effects. Consequently, statistical power was limited, confidence intervals are likely wide, and small-to-moderate intervention effects may have gone undetected. Second, despite stratified randomization, the sample included a broad age range and considerable heterogeneity in competitive skill/handicap, and data analysts were not formally blinded to group allocation during HRV and hormone processing; these factors may have increased variability, introduced potential assessment-related bias, and reduced sensitivity to detect performance-related effects. Third, the statistical approach was exploratory and relied primarily on multiple paired and between-group tests across several HRV indices, hormonal endpoints, performance outcomes, and correlation matrices. Although this approach was chosen because of the pilot nature of the study and the limited sample size, it does not fully model the factorial structure of the design — Group × Tournament × Time for variables with pre/post sampling — and increases the risk of Type I error. A mixed-effects model or repeated-measures ANOVA would provide a more integrated test of interaction effects in a larger confirmatory study. Moreover, no formal correction for multiple comparisons was applied to the primary exploratory analyses; therefore, the physiological and correlational findings should be interpreted cautiously as preliminary and hypothesis-generating rather than confirmatory. Fourth, the wait-list control group did not control for placebo or expectancy effects, and the absence of sham neurofeedback limits causal attribution of the observed psychophysiological differences to SCP training specifically. Fifth, the study did not include direct measures of prefrontal activation, functional connectivity, or mediation pathways during performance; therefore, any interpretation involving cortical–autonomic coupling or a cascade from SCP modulation to autonomic and endocrine adaptation remains speculative. Sixth, SCP modulation was assessed under standardized

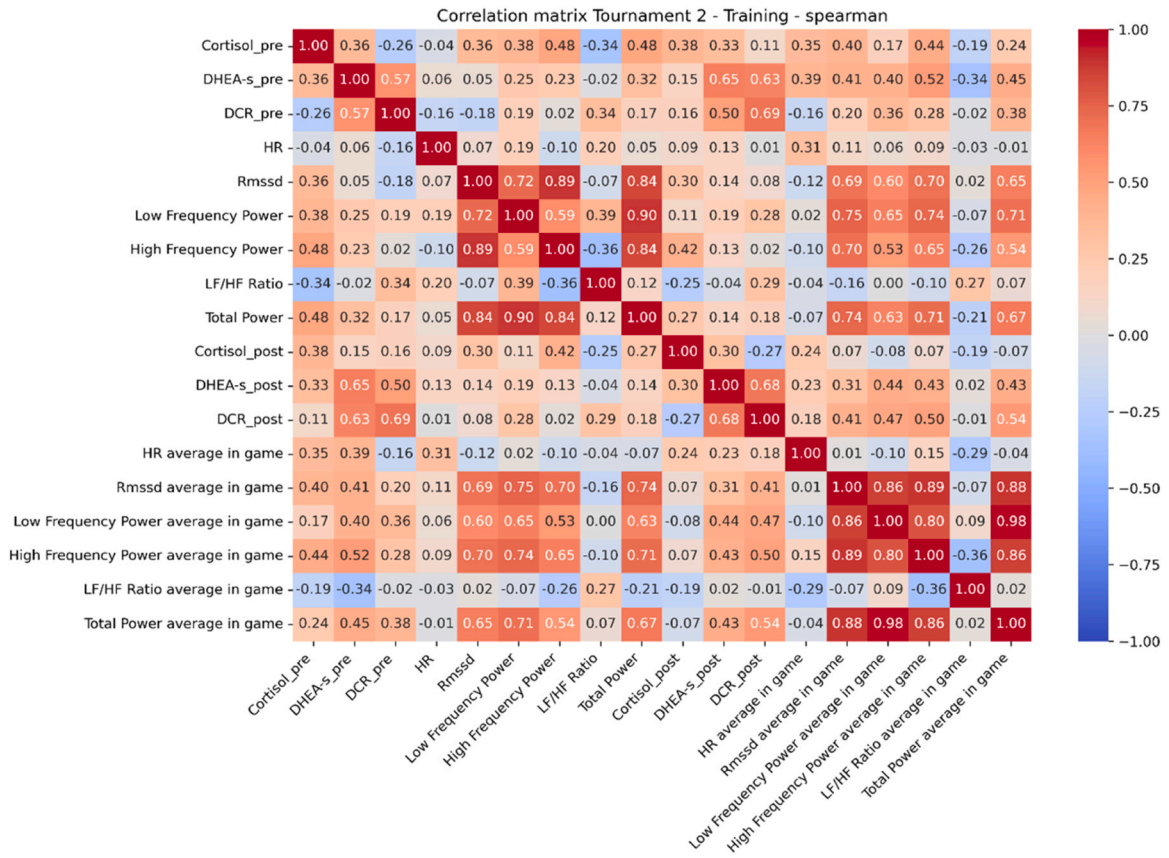

Fig. 8. Uncorrected Spearman correlation matrix (Tournament 2-Training group) between salivary hormones and HRV indices. Cells show Spearman's rho (ρ) for pairwise associations among cortisol and DHEA concentrations collected pre and post-task (Cortisol_pre, Cortisol_post, DHEA_pre, DHEA_post) and HRV metrics computed at baseline and during gameplay (average in game), including average HR, RMSSD, Low Frequency Power, High Frequency Power, LF/HF ratio, and Total Power. Warmer colors indicate positive associations and cooler colors indicate negative associations (scale −1–1). Correlations are presented without correction for multiple comparisons and should therefore be interpreted cautiously as exploratory associations.

evaluation conditions, and the extent to which these EEG changes generalize to real-time regulation during putting execution remains unknown. Seventh, HRV was recorded using a Polar H10 chest strap, which is acceptable for field-based HRV assessment but is not equivalent to gold-standard ECG systems. In addition, respiration was not controlled or monitored during gameplay; because HF power is strongly respiration-sensitive, breathing rate or depth may have influenced HF and related HRV indices, limiting interpretation of vagal modulation. Eighth, saliva samples were collected immediately after questionnaires and performance, whereas cortisol typically shows a delayed response of approximately 15–25 min after stress exposure; therefore, post-task cortisol may partly reflect anticipatory or early task-related stress rather than the full endocrine response to performance. Finally, psychological self-report data were not reported because the manuscript focused on physiological responses per se. This limits interpretation of whether the physiological patterns were accompanied by changes in appraisal, expectancy, perceived control, or intervention credibility.

6. Conclusions

In conclusion, SCP neurofeedback was associated with successful acquisition of cortical self-regulation, while endocrine responses appeared to be primarily driven by re-exposure or habituation to the competitive setting rather than by training-specific effects. The uncorrected HRV analyses suggested that, at Tournament 2, the training group showed higher gameplay LF, HF, TP, and RMSSD than controls, whereas several HRV indices decreased in the control group. However, these group-related physiological findings did not remain statistically significant after FDR correction for multiple comparisons and should therefore be interpreted as exploratory. Similarly, the correlation analyses suggested that endocrine and autonomic measures varied differently across groups at re-exposure, but these associations were preliminary and did not provide confirmatory evidence of training-related physiological integration. Importantly, the observed physiological responses and associations did not translate into significant improvements in putting performance. Given the pilot design, modest sample size, limited power, multiple endpoints, lack of sham control, absence of respiration monitoring, and no direct measures of cortical–autonomic mechanisms during performance, the present findings should be considered hypothesis-generating. Future studies should use larger samples, preregistered primary outcomes, active or sham-control conditions, correction for multiple testing, respiration monitoring, age- and skill-adjusted analyses, and direct measures of cortical activity or connectivity to determine whether SCP training is capable of producing reliable changes in physiological responses and sport performance.

CRediT authorship contribution statement

López Gonzalo: Visualization, Software, Data curation. **Carolina Pereira:** Investigation, Data curation. **Eugenio Lizama:** Writing – original draft, Resources, Project administration, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Bastian Carcamo-Herrera:** Writing – original draft, Visualization, Investigation. **Serrano Miguel-Angel:** Writing – review & editing, Validation, Project administration, Methodology, Conceptualization.

Ethical statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the I Ethics Committee of University of Valencia (protocol code 2024-PSILOG−3368845 and approved in 11/7/2024).” for studies involving humans.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used ChatGPT (Open AI, USA) in order to improve English language. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Funding

This research received no external funding.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors would like to thank all the volunteer golfers for their participation.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2026.107934.

References

Aydemir, M., Makaracı, Y., Avcı, B., Ürkmez, Y.C., Cintineo, H.P., 2025. The psychophysiologic stress and salivary cortisol and alpha-amylase awakening responses to cross-country running competitions in national-level female athletes. J. Strength. Cond. Res. 39 (5), e676–e683. https://doi.org/10.1519/JSC.0000000000005055.

Balthazar, C.H., Garcia, M.C., Spadari-Bratfisch, R.C., 2012. Salivary concentrations of cortisol and testosterone and prediction of performance in a professional triathlon competition. Stress 15 (5), 495–502. https://doi.org/10.3109/10253890.2011.642033.

Bangsbo, J., 2015. Performance in sports: With specific emphasis on the effect of intensified training. Scand. J. Med. Sci. Sports 25 (S4), 88–99. https://doi.org/10.1111/sms.12605.

Bertollo, M., Di Fronso, S., Filho, E., Conforto, S., Schmid, M., Bortoli, L., Comani, S., Robazza, C., 2016. Proficient brain for optimal performance: The MAP model perspective. PeerJ 4, e2082. https://doi.org/10.7717/peerj.2082.

Billman, G.E., 2013. The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. Front. Physiol. 4. https://doi.org/10.3389/fphys.2013.00026.

Bortoli, L., Bertollo, M., Hanin, Y., Robazza, C., 2012. Striving for excellence: A multi-action plan intervention model for shooters. Psychol. Sport. Exerc. 13 (5), 693–701. https://doi.org/10.1016/j.psychsport.2012.04.006.

Brito, M.A.D., Fernandes, J.R., Esteves, N.S., Müller, V.T., Alexandria, D.B., Pérez, D.I.V., Slimani, M., Brito, C.J., Bragazzi, N.L., Miarka, B., 2022. The effect of neurofeedback on the reaction time and cognitive performance of athletes: A systematic review and meta-analysis. Front. Human. Neurosci. 16, 868450. https://doi.org/10.3389/fnhum.2022.868450.

Carson, H.J., Robazza, C., Collins, D., Toner, J., Bertollo, M., 2020. Optimizing performance in sport. In: Bertollo, M., Filho, E., Terry, P.C. (Eds.), Advancements in mental skills training. Routledge, pp. 15–27. https://doi.org/10.4324/9780429025112-3.

Costa, Y., Domingos-Gomes, J., Lautenbach, F., Hayes, L., Nakamura, F., Lima, J., Castellano, L., Batista, G., 2022. Salivary hormone concentrations and technical-tactical performance indicators in beach volleyball: Preliminary evidence. Front. Sports Act. Living 4, 830185. https://doi.org/10.3389/fspor.2022.830185.

Damoun, N., Amekran, Y., Taiek, N., El Hangouche, A.J., 2024. Heart rate variability measurement and influencing factors: Towards the standardization of methodology. Glob. Cardiol. Sci. Pract. 2024. https://doi.org/10.21542/gcsp.2024.35.

Dias, G., Couceiro, M., Mendes, P., Gomes, R., Mendes, R., Vaz, V., Martins, F., Gama, J., Castro, M.A., 2021. Golf-putting performance in skilled golfers at different distances to the hole. Appl. Sci. 11 (24), 11785. https://doi.org/10.3390/app112411785.

Doan, B., Newton, R., Kraemer, W., Kwon, Y.-H., Scheet, T., 2007. Salivary cortisol, testosterone, and T/C ratio responses during a 36-hole golf competition. Int. J. Sports Med. 28 (6), 470–479. https://doi.org/10.1055/s-2006-924557.

Dutheil, F., De Saint Vincent, S., Pereira, B., Schmidt, J., Moustafa, F., Charkhabi, M., Bouillon-Minois, J.-B., Clinchamps, M., 2021. DHEA as a biomarker of stress: A systematic review and meta-analysis. Front. Psychiatry 12, 688367. https://doi.org/10.3389/fpsyt.2021.688367.

Elbert, T., 1993. Slow cortical potentials reflect the regulation of cortical excitability. In: McCallum, W.C., Curry, S.H. (Eds.), Slow potential changes in the human brain. Springer, pp. 235–251. https://doi.org/10.1007/978-1-4899-1597-9_15.

Filaire, E., Alix, D., Ferrand, C., Verger, M., 2009. Psychophysiological stress in tennis players during the first single match of a tournament. Psychoneuroendocrinology 34 (1), 150–157. https://doi.org/10.1016/j.psyneuen.2008.08.022.

Fronso, S.D., Robazza, C., Bortoli, L., Bertollo, M., 2017. Performance optimization in sport: A psychophysiological approach. Mot. Rev. De. Educ. ção F. íSci. 23 (4). https://doi.org/10.1590/s1980-6574201700040001.

Gamaiunova, L., Kreibig, S.D., Dan-Glauser, E., Pellerin, N., Brandt, P.-Y., Kliegel, M., 2022. Effects of two mindfulness-based interventions on the distinct phases of the stress response across different physiological systems. Biol. Psychol. 172, 108384. https://doi.org/10.1016/j.biopsycho.2022.108384.

Gerra, G., Zaimovic, A., Mascetti, G.G., Gardini, S., Zambelli, U., Timpano, M., Raggi, M. A., Brambilla, F., 2001. Neuroendocrine responses to experimentally induced psychological stress in healthy humans. Psychoneuroendocrinology 26 (1), 91–107. https://doi.org/10.1016/S0306-4530(00)00046-9.

Gevensleben, H., Albrecht, B., Lütcke, H., Auer, T., Dewiputri, W.I., Schweizer, R., Moll, G., Heinrich, H., Rothenberger, A., 2014. Neurofeedback of slow cortical potentials: Neural mechanisms and feasibility of a placebo-controlled design in healthy adults. Front. Human. Neurosci. 8. https://doi.org/10.3389/fnhum.2014.00990.

Gladdines, S., Von Gerhardt, A.L., Verhagen, E., Beumer, A., Eygendaal, D., GRIPP 9 Study Collaborative, 2022. The effectiveness of a golf injury prevention program (GRIPP intervention) compared to the usual warm-up in Dutch golfers: Protocol design of a randomized controlled trial. BMC Sports Sci. Med. Rehabil. 14, 144. https://doi.org/10.1186/s13102-022-00511-4.

Godoy, L.D., Rossignoli, M.T., Delfino-Pereira, P., Garcia-Cairasco, N., de Lima Umeoka, E.H., 2018. A comprehensive overview on stress neurobiology: Basic concepts and clinical implications. Front. Behav. Neurosci. 12, 127. https://doi.org/10.3389/fnbeh.2018.00127.

Hasslinger, J., Meregalli, M., Bölte, S., 2022. How standardized are “standard protocols”? Variations in protocol and performance evaluation for slow cortical potential neurofeedback: A systematic review. Front. Human. Neurosci. 16, 887504. https://doi.org/10.3389/fnhum.2022.887504.

Hertting, K., Wagnsson, S., Grahn, K., 2020. Perceptions of stress of Swedish volunteer youth soccer coaches. Sports 8 (11), 146. https://doi.org/10.3390/sports8110146.

James, K.A., Stromin, J.I., Steenkamp, N., Combrinck, M.I., 2023. Understanding the relationships between physiological and psychosocial stress, cortisol and cognition. Front. Endocrinol. 14, 1085950. https://doi.org/10.3389/fendo.2023.1085950.

Kimura, I., Noyama, H., Onagawa, R., Takemi, M., Osu, R., Kawahara, J., 2024. Efficacy of neurofeedback training for improving attentional performance in healthy adults: A systematic review and meta-analysis. Imaging Neurosci. 2. https://doi.org/10.1162/imag_a_00053.

Kudielka, B.M., Wüst, S., 2010. Human models in acute and chronic stress: Assessing determinants of individual hypothalamus-pituitary-adrenal axis activity and reactivity. Stress 13 (1), 1–14. https://doi.org/10.3109/10253890902874913.

Laborde, S., Mosley, E., Thayer, J.F., 2017. Heart rate variability and cardiac vagal tone in psychophysiological research: Recommendations for experiment planning, data analysis, and data reporting. Front. Psychol. 8, 213. https://doi.org/10.3389/fpsyg.2017.00213.

Lee, J.-K., Park, J.-K., Kim, H., Kang, J.-Y., Park, J.-Y., Do, S.-H., Ahn, R.-S., 2020. Association of the HPA axis response to upcoming competition and shooting outcomes in elite junior shooting players. Stress 23 (2), 153–161. https://doi.org/10.1080/10253890.2019.1660871.

Lennartsson, A.-K., Theorell, T., Rockwood, A.L., Kushnir, M.M., Jonsdottir, I.H., 2013. Perceived stress at work is associated with lower levels of DHEA-S. PLOS ONE 8 (8), e72460. https://doi.org/10.1371/journal.pone.0072460.

Lizama, E., Lorenzon, L., Pereira, C., Serrano, M.A., 2025. Enhancing neural efficiency in competitive golfers: Effects of slow cortical potential neurofeedback on modulation of beta activity—An exploratory randomized controlled trial. NeuroSci 6 (4), 104. https://doi.org/10.3390/neurosci6040104.

Lu, S., Wei, F., Li, G., 2021. The evolution of the concept of stress and the framework of the stress system. Cell. Stress. 5 (6), 76–85. https://doi.org/10.15698/cst2021.06.250.

MacDonald, D., Wetherell, M.A., 2019. Competition stress leads to a blunting of the cortisol awakening response in elite rowers. Front. Psychol. 10, 1684. https://doi.org/10.3389/fpsyg.2019.01684.

Makaracı, M., Makaracı, Y., Zorba, E., Lautenbach, F., 2024. Effects of ten biofeedback sessions on athletes’ physiological, psychological, and cognitive functioning: A randomized controlled trial with international tennis players. Percept. Mot. Skills 131 (5), 1664–1686. https://doi.org/10.1177/00315125241274834.

Matusik, P.S., Zhong, C., Matusik, P.T., Alomar, O., Stein, P.K., 2023. Neuroimaging studies of the neural correlates of heart rate variability: A systematic review. J. Clin. Med. 12 (3), 1016. https://doi.org/10.3390/jcm12031016.

Mayer, K., Blume, F., Wyckoff, S.N., Brokmeier, L.L., Strehl, U., 2016. Neurofeedback of slow cortical potentials as a treatment for adults with attention-deficit/hyperactivity disorder. Clin. Neurophysiol. 127 (2), 1374–1386. https://doi.org/10.1016/j.clinph.2015.11.013.

Meijen, C., Turner, M., Jones, M.V., Sheffield, D., McCarthy, P., 2020. A theory of challenge and threat states in athletes: A revised conceptualization. Front. Psychol. 11, 126. https://doi.org/10.3389/fpsyg.2020.00126.

Mirifar, A., Beckmann, J., Ehrlenspiel, F., 2017. Neurofeedback as supplementary training for optimizing athletes' performance: A systematic review with implications for future research. Neurosci. Biobehav. Rev. 75, 419–432. https://doi.org/10.1016/j.neubiorev.2017.02.005.

Morgan, C.A., Rasmusson, A., Pietrzak, R.H., Coric, V., Southwick, S.M., 2009. Relationships among plasma dehydroepiandrosterone and dehydroepiandrosterone sulfate, cortisol, symptoms of dissociation, and objective performance in humans exposed to underwater navigation stress. Biol. Psychiatry 66 (4), 334–340. https://doi.org/10.1016/j.biopsych.2009.04.004.

Panjeh, S., Nordahl-Hansen, A., Cogo-Moreira, H., 2023. Establishing new cutoffs for Cohen's *d*: An application using known effect sizes from trials for improving sleep quality on composite mental health. Int. J. Methods Psychiatr. Res. 32 (4), e1969. https://doi.org/10.1002/mpr.1969.

Pearce, A.J., Kidgell, D.J., 2009. Corticomotor excitability during precision motor tasks. J. Sci. Med. Sport. 12 (2), 280–283. https://doi.org/10.1016/j.jsams.2007.12.005.

Quigley, K.S., Gianaros, P.J., Norman, G.J., Jennings, J.R., Berntson, G.G., De Geus, E.J. C., 2024. Publication guidelines for human heart rate and heart rate variability studies in psychophysiology—Part 1: Physiological underpinnings and foundations of measurement. Psychophysiology 61 (9), e14604. https://doi.org/10.1111/psyp.14604.

Schaeuble, D., Myers, B., 2022. Cortical-hypothalamic integration of autonomic and endocrine stress responses. Front. Physiol. 13, 820398. https://doi.org/10.3389/fphys.2022.820398.

Schommer, N.C., Hellhammer, D.H., Kirschbaum, C., 2003. Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. Psychosom. Med. 65 (3), 450–460. https://doi.org/10.1097/01.PSY.0000035721.12441.17.

Shaffer, F., Ginsberg, J.P., 2017. An overview of heart rate variability metrics and norms. Front. Public. Health 5, 258. https://doi.org/10.3389/fpubh.2017.00258.

Smith, S.M., Vale, W.W., 2006. The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. Dialog.-. Clin. Neurosci. 8 (4), 383–395. https://doi.org/10.31887/DCNS.2006.8.4/ssmith.

Stöckl, M., Lamb, P.F., 2018. The variable and chaotic nature of professional golf performance. J. Sports Sci. 36 (9), 978–984. https://doi.org/10.1080/02640414.2017.1347269.

Strehl, U., 2009. Slow cortical potentials neurofeedback. J. Neurother. 13 (2), 117–126. https://doi.org/10.1080/10874200902885936.

Strehl, U., Leins, U., Goth, G., Klinger, C., Hinterberger, T., Birbaumer, N., 2006. Self-regulation of slow cortical potentials: A new treatment for children with attention-deficit/hyperactivity disorder. Pediatrics 118 (5), e1530–e1540. https://doi.org/10.1542/peds.2005-2478.

Strehl, U., Aggensteiner, P., Wachtlin, D., Brandeis, D., Albrecht, B., Arana, M., Bach, C., Banaschewski, T., Bogen, T., Flaig-Röhr, A., Freitag, C.M., Fuchsenberger, Y., Gest, S., Gevensleben, H., Herde, L., Hohmann, S., Legenbauer, T., Marx, A.-M., Millenet, S., Pniewski, B., Rothenberger, A., Ruckes, C., Wörz, S., Holtmann, M., 2017. Neurofeedback of slow cortical potentials in children with attention-deficit/hyperactivity disorder: A multicenter randomized trial controlling for unspecific effects. Front. Human. Neurosci. 11. https://doi.org/10.3389/fnhum.2017.00135.

Thayer, J.F., Åhs, F., Fredrikson, M., Sollers 3rd, J.J., Wager, T.D., 2012. A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. Neurosci. Biobehav. Rev. 36 (2), 747–756. https://doi.org/10.1016/j.neubiorev.2011.11.009.

Thielmann, B., Zavgorodnii, I., Zub, K., Böckelmann, I., 2022. The perception of stress, behavior in stressful situations and mental health of bank employees within a German-Ukrainian comparative study. Int. J. Occup. Med. Environ. Health 35 (1), 81–94. https://doi.org/10.13075/ijomeh.1896.01739.

Tsigos, C., Kyrou, I., Kassi, E., & Chrousos, G.P. (2000). Stress: Endocrine physiology and pathophysiology. In K. R. Feingold, R. A. Adler, S. F. Ahmed, M. R. Blackman, A. Boyce, G. Chrousos, E. Corpas, W. W. de Herder, K. Dhatariya, K. Dungan, A. Grossman, J. M. Hershman, J. Hofland, G. Kaltsas, C. Koch, P. Kopp, M. Korbonits, C. S. Kovacs, M. Kuohung, B. Laferrère, M. Levy, E. A. McGee, R. McLachlan, J. E. Morley, M. New, J. Purnell, R. Sahay, F. Singer, C. A. Stratakis, D. L. Trence, A. Vinik, & D. P. Wilson (Eds.), Endotext. MDText.com.

Van Paridon, K.N., Timmis, M.A., Nevison, C.M., Bristow, M., 2017. The anticipatory stress response to sport competition: A systematic review with meta-analysis of cortisol reactivity. BMJ Open. Sport. Exerc. Med. 3 (1), e000261. https://doi.org/10.1136/bmjsem-2017-000261.

Wang, H.-T., Chen, S.-M., Lee, S.-D., Hsu, M.-C., Chen, K.-N., Liou, Y.-F., Kuo, C.-H., 2009. The role of DHEA-S in the mood adjustment against negative competition outcome in golfers. J. Sports Sci. 27 (3), 291–297. https://doi.org/10.1080/02640410802520810.

Wang, K.-P., Cheng, M.-Y., Chen, T.-T., Huang, C.-J., Schack, T., Hung, T.-M., 2020. Elite golfers are characterized by psychomotor refinement in cognitive-motor processes. Psychol. Sport. Exerc. 50, 101739. https://doi.org/10.1016/j.psychsport.2020.101739.