REACTISPORT: Reducing the Impact of Competitive Stress on Decision-Making and Motor Precision Through Vagal Recalibration and EEG-Based Neuroregulation

Alina ROBU, Alina-Diana NEMES

Abstract

Elite athletic performance is frequently compromised in decisive competitive moments due to stress-induced neurocognitive destabilization rather than insufficient technical preparation. Acute stress alters prefrontal cortical functioning, disrupts executive control networks, and reduces autonomic flexibility, thereby impairing decision-making accuracy and fine motor execution. The present randomized controlled longitudinal study (REACTISPORT) evaluated the effectiveness of a 10-week integrated neurophysiological intervention designed to recalibrate autonomic balance and optimize cortical network coherence in professional athletes. Eighty elite athletes (football, handball, basketball) were randomly assigned to an experimental group (n = 40) receiving multimodal brain-based training or to a control group (n = 40) undergoing standardized assessments only. The intervention combined EEG-based neurofeedback, transcranial photobiomodulation (10 Hz alpha; 40 Hz gamma), heart rate variability (HRV) biofeedback, transcutaneous vagal nerve stimulation, and binaural auditory stimulation. Outcomes included quantitative EEG coherence (BA24/32), HRV parameters (RMSSD, LF/HF), perceived physiological stress (GP-8), and sport-specific performance under simulated competitive pressure. Compared to controls, the experimental group demonstrated significant increases in RMSSD (+12.5 ms), improved autonomic balance (LF/HF –1.3), enhanced anterior cingulate EEG coherence (+13%), substantial reductions in perceived stress (–16 points), and a 35% decrease in motor errors under pressure ($p < .001$ for all outcomes). These findings indicate that competitive resilience is a trainable neuroregulatory capacity. Integrated modulation of cortical and autonomic systems significantly reduces stress-related performance degradation, offering a scalable framework for embedding applied neuroscience within elite sport performance infrastructures.

Keywords: *Competitive stress, Neurofeedback, Heart rate variability (HRV), Vagal nerve stimulation, Executive control network, Anterior cingulate cortex, Elite sport performance.*

1. Introduction

In contemporary elite sport, marginal differences determine competitive outcomes. At the highest level, athletes are typically comparable in physical conditioning, tactical preparation, and technical proficiency. The decisive distinction between victory and defeat often emerges not from strength or endurance, but from the capacity to execute precise motor actions and make optimal decisions under acute psychological and physiological pressure. Penalty kicks in the final minutes, last-second shots, high-risk passes in densely contested zones, or uncharacteristic execution errors in overtime are rarely attributable to insufficient training. Rather, they frequently reflect transient neurocognitive destabilization induced by competitive stress.

A substantial body of neuroscientific and psychophysiological literature demonstrates that acute and chronic stress profoundly alter central and autonomic nervous system functioning (McEwen, 2007). Under high-pressure conditions, activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis leads to increased catecholamine and glucocorticoid release. These neurochemical changes exert

direct effects on prefrontal cortical regions responsible for executive control, working memory, behavioral inhibition, and flexible decision-making (Arnsten, 2009). Excessive noradrenergic and dopaminergic stimulation, particularly during acute stress, can impair dorsolateral prefrontal cortex (dlPFC) functioning, biasing behavior toward habitual or reflexive responses rather than contextually optimal strategies.

From a performance perspective, this neurobiological cascade manifests in several ways. First, inhibitory control may decrease, resulting in impulsive decisions such as premature shots or poorly timed passes. Second, rapid decision accuracy declines as cognitive flexibility is reduced. Third, fine motor coordination can become destabilized, even in highly trained athletes. Research in sport psychology has shown that pressure can disrupt automatized motor skills through mechanisms commonly described as "choking under pressure," whereby heightened self-focus and anxiety interfere with procedural execution (Beilock & Carr, 2001). Similarly, Nieuwenhuys and Oudejans (2012) demonstrated that anxiety alters attentional control and perceptual–motor performance in real sport environments, confirming that stress-induced attentional shifts contribute directly to performance errors.

Importantly, competitive stress should not be conceptualized solely as a subjective psychological phenomenon. While constructs such as performance anxiety or fear of failure are relevant, converging neuroimaging and autonomic research indicates that stress represents a systemic neurophysiological state involving large-scale brain networks and cardiovascular regulation (Thayer & Lane, 2000). Within the brain, the Salience Network (SN), anchored in the anterior insula (Brodmann area 13) and the anterior cingulate cortex (BA24/32), plays a critical role in detecting emotionally relevant stimuli and allocating attentional resources (Menon, 2011). Under competitive pressure, hyperactivation of the SN may amplify perceived threat value, increasing emotional reactivity and biasing cognitive resources toward vigilance rather than strategic planning.

Simultaneously, the Executive Control Network (ECN), centered in the dorsolateral and anterior prefrontal cortices (BA9, BA10, BA46), is responsible for rational decision-making, planning, and behavioral inhibition (Miller & Cohen, 2001; Cole & Schneider, 2007). Acute stress has been shown to disrupt functional connectivity within this network and weaken its regulatory influence over limbic structures (Arnsten, 2009). Dysfunction at the interface between the SN and ECN, particularly within the anterior cingulate cortex, may therefore explain why athletes sometimes fail to maintain composure in decisive moments. The anterior cingulate cortex is crucial for error monitoring, conflict detection, and adaptive response selection (Bush et al., 2000; Etkin et al., 2011). When this regulatory hub becomes dysregulated, impulsive or maladaptive motor responses are more likely to occur.

Beyond cortical networks, competitive stress is reflected in measurable autonomic markers. Heart rate variability (HRV), particularly time-domain indices such as the root mean square of successive differences (RMSSD), provides a robust indicator of parasympathetic (vagal) tone and adaptive flexibility (Shaffer & Ginsberg, 2017). Reduced HRV is associated with diminished emotional regulation capacity, impaired cognitive performance under pressure, and increased susceptibility to stress-related errors (Thayer et al., 2012). According to the neurovisceral integration model, efficient communication between the prefrontal cortex and cardiac vagal control systems underpins adaptive behavior (Thayer & Lane, 2000). In elite athletes, low HRV during competition may therefore reflect compromised top-down regulation, narrowing the window of optimal performance.

Traditional psychological interventions in sport, such as imagery training, cognitive restructuring, or motivational coaching, primarily operate through top-down cognitive mechanisms. While these approaches are valuable, they may not directly recalibrate the underlying neurophysiological imbalances induced by stress. Emerging evidence suggests that neurotechnological interventions, including EEG-based neurofeedback, can stabilize prefrontal activation patterns and reduce maladaptive high-beta activity associated with anxiety

(Gruzelier, 2014; Ros et al., 2014). Similarly, non-invasive vagal nerve stimulation has demonstrated efficacy in enhancing parasympathetic tone and reducing emotional hyperreactivity (Breit et al., 2018). Photobiomodulation and oscillatory entrainment techniques have also shown potential in modulating cortical excitability and network synchronization (Hamblin, 2016).

Despite these advances, a significant gap remains in applied sport neuroscience research. Few studies have examined integrated, multimodal neurophysiological interventions explicitly targeting competitive stress in elite athletes, while simultaneously correlating objective EEG and HRV markers with real performance errors in ecologically valid contexts. Most existing research isolates single modalities or relies predominantly on laboratory-based cognitive tasks rather than sport-specific execution measures.

The REACTISPORT study was designed to address this gap. Its scientific rationale rests on the premise that competitive resilience is not merely a psychological trait but a trainable neuroregulatory capacity grounded in network-level brain dynamics and autonomic flexibility. By targeting both cortical (SN–ECN connectivity, anterior cingulate coherence, prefrontal stability) and autonomic (vagal tone, HRV balance) systems, the study aims to reduce stress-induced performance degradation and enhance decision precision in high-stakes situations.

In doing so, REACTISPORT moves beyond performance enhancement in the abstract and toward a model of applied neurocognitive regulation. It conceptualizes elite performance under pressure as the product of adaptive salience detection, preserved executive control, efficient heart–brain communication, and stable motor automatization. Within this framework, errors in decisive moments are not random events but predictable consequences of neurophysiological dysregulation, processes that, in principle, can be measured, trained, and optimized.

2. Objectives of the Study

2.1 General Objective

The primary objective of the REACTISPORT study is to evaluate, within a controlled randomized clinical framework, the effectiveness of an integrated neurophysiological training protocol in reducing the impact of competitive stress on motor precision and decision-making in elite athletes. Specifically, the study seeks to determine whether multimodal brain-based interventions, targeting cortical network regulation and autonomic recalibration, can enhance performance stability in high-pressure competitive contexts.

This objective is grounded in evidence indicating that acute stress disrupts prefrontal cortical functioning, weakens executive control, and impairs motor execution (Arnsten, 2009; McEwen, 2007). By directly modulating neural activity in the anterior cingulate cortex and prefrontal regions, while simultaneously improving vagal tone and heart rate variability (HRV), the intervention aims to restore adaptive neurocognitive functioning during stress exposure. In practical terms, the overarching goal is not merely to improve baseline performance, but to reduce stress-induced performance degradation, particularly in decisive moments of competition.

2.2 Specific Objectives

1. **To reduce emotional and autonomic reactivity under competitive pressure.** This objective focuses on increasing parasympathetic activity, as indexed by HRV (particularly RMSSD), and decreasing sympathetic dominance (LF/HF ratio), thereby enhancing physiological adaptability and stress tolerance (Thayer et al., 2012; Shaffer & Ginsberg, 2017).

2. **To optimize functional activation and coherence within the anterior cingulate cortex (BA24/32).** Through EEG-based neurofeedback and complementary interventions, the study aims to improve regulatory control within the Salience Network, facilitating adaptive error monitoring, emotional regulation, and behavioral inhibition (Bush et al., 2000; Etkin et al., 2011).
3. **To maintain functional integrity of the Executive Control Network (BA9, BA10, BA46) under stress.** The intervention seeks to preserve prefrontal executive functioning during acute stress exposure, supporting rational tactical decision-making and impulse control (Miller & Cohen, 2001; Cole & Schneider, 2007).
4. **To decrease motor execution errors in simulated high-pressure conditions.** By stabilizing heart–brain dynamics and reducing maladaptive cortical hyperactivation, the study aims to reduce stress-related motor disruptions commonly described as performance choking (Beilock & Carr, 2001; Nieuwenhuys & Oudejans, 2012).
5. **To enhance recovery speed following stress exposure.** The protocol targets rapid autonomic and neurocognitive return to homeostasis after critical game situations, strengthening competitive resilience and decision consistency across successive high-intensity phases.
6. **To validate a standardized, scalable neurophysiological intervention model for elite sport.** Beyond individual-level outcomes, the study seeks to demonstrate the feasibility, reproducibility, and applied value of integrating objective EEG and HRV-guided training into professional sports performance systems.

Together, these objectives define a coherent investigative framework linking neurophysiological regulation to observable performance outcomes, with direct implications for competitive reliability in elite sport.

3. Study Design

The REACTISPORT study was structured to combine methodological rigor with applied relevance in elite sport. The design sought to ensure internal validity, statistical robustness, and ecological applicability within the constraints of an active competitive season. By integrating randomized allocation, longitudinal monitoring, and objective neurophysiological markers, the study was positioned to evaluate not only whether the intervention works, but also how it translates into measurable changes in stress-regulated performance.

REACTISPORT is a **randomized, controlled, longitudinal clinical trial** with two parallel arms:

- **Experimental group:** Athletes receiving the full integrated neurophysiological intervention.
- **Control group:** Athletes undergoing standardized assessments (EEG, HRV, psychophysiological scales, and performance simulations) without receiving the intervention.

This design allows for:

- Between-group (inter-group) comparisons of outcome evolution;
- Within-group (intra-group) longitudinal assessment;
- Control of seasonal adaptation effects and natural performance fluctuations.

Such a randomized controlled framework remains the most reliable method for evaluating intervention efficacy in applied human performance research (Schulz et al., 2010).

Randomization was performed using a computerized 1:1 allocation sequence. To ensure comparability between groups, stratification was conducted according to the following variables: Type of sport (football, handball, basketball); Playing position (e.g., goalkeeper, midfielder, attacker, playmaker); Competitive exposure (minutes played, national team participation); Baseline perceived stress level at T0. This stratified procedure reduced the likelihood of systematic imbalance in performance-critical characteristics and strengthened statistical interpretability.

Duration and Assessment Timeline

The intervention extended over **10 consecutive weeks**, a timeframe selected to allow sufficient neuroplastic adaptation while remaining compatible with the demands of a professional competitive calendar. Assessments were conducted at three predefined time points:

- **T0 – Baseline (pre-intervention)**
- **T1 – Midpoint evaluation (week 5)**
- **T2 – Post-intervention (week 10)**

This structure permitted the evaluation of early adaptive changes as well as sustained neurophysiological and behavioral effects.

Participants and Eligibility Criteria

The study included a minimum of 80 professional athletes, with 40 allocated to the experimental group and 40 to the control group. All participants were actively competing in professional football, handball, or basketball leagues.

Inclusion criteria were: age between 18 and 35 years; active participation in official competitions during the current season; consistent match involvement; absence of diagnosed neurological or psychiatric disorders; and availability for the full 10-week protocol.

Exclusion criteria were: history of concussion or cranial trauma within the previous six months; current use of psychotropic medication; participation in parallel structured mindfulness or neurostimulation programs; and cardiovascular conditions contraindicating vagal stimulation or HRV biofeedback.

These criteria ensured medical safety, minimized confounding influences, and preserved homogeneity of neurophysiological baseline conditions.

Intervention Structure

Participants in the experimental arm completed:

- **3 sessions per week**
- Over **10 weeks**
- Totaling **30 sessions**

Each session lasted approximately 60 minutes and consisted of:

1. EEG-based personalized neurofeedback – 20 minutes
2. Transcranial photobiomodulation (10 Hz Alpha + 40 Hz Gamma) – 10 minutes
3. HRV biofeedback and heart–brain coherence training – 15 minutes

4. Transcutaneous vagal nerve stimulation (tVNS) – 10 minutes
5. Binaural audio stimulation (theta–gamma entrainment) – 5 minutes

All procedures were conducted in specialized clinical settings under supervision of trained personnel. Intervention parameters were individualized according to baseline qEEG and HRV assessments.

Multicenter Implementation

The protocol was designed for implementation across multiple professional settings, including club training centers and affiliated clinical facilities. Standardization of procedures and centralized supervision ensured methodological consistency across sites. This multicenter capacity enhances external validity and supports the scalability of the intervention in diverse elite sport environments.

The study was conducted in accordance with the Declaration of Helsinki and approved by an independent ethics committee. All participants provided written informed consent prior to enrollment. Data were pseudonymized, securely stored, and processed in compliance with EU Regulation 2016/679 (GDPR). All neurostimulation components were non-invasive and applied within established medical safety parameters.

Beyond efficacy testing, the design was conceived to demonstrate feasibility and practical integration within professional sport systems. By combining objective neurophysiological measures (qEEG, HRV) with ecologically valid performance assessments, the study establishes a framework for embedding applied neuroscience into elite training infrastructures. The design therefore supports both scientific validation and real-world implementation.

4. Structure of the Intervention

The REACTISPORT intervention was developed as an integrated neurophysiological protocol designed to simultaneously target cortical regulation, autonomic balance, and stress-related motor stability. Rather than relying on a single modality, the intervention combines complementary techniques that act on interconnected neural and cardiovascular systems. Each session lasts approximately 60 minutes and is structured to sequentially modulate executive control networks, salience processing, and vagal tone, thereby reinforcing heart–brain coherence and adaptive decision-making under stress. The intervention was delivered exclusively to the experimental group and individualized based on baseline qEEG and HRV profiles.

4.1 EEG-Based Personalized Neurofeedback (20 minutes)

Neurofeedback represents the core regulatory component of the protocol. Using real-time EEG monitoring, athletes were trained to modulate their own cortical activity through visual and auditory feedback. Individualized protocols targeted dysregulated patterns identified at baseline, particularly within the anterior cingulate cortex (BA24/32) and dorsolateral prefrontal regions (BA9/46). The primary objective was to reduce excessive high-beta activity associated with hyperarousal and competitive anxiety while enhancing alpha and sensorimotor rhythm (SMR) activity linked to relaxed focus and motor stability. By reinforcing adaptive oscillatory patterns, neurofeedback aimed to restore functional balance between the Salience Network and the Executive Control Network, supporting inhibitory control and tactical clarity under pressure (Gruzelier, 2014; Ros et al., 2014).

4.2 Transcranial Photobiomodulation (10 minutes)

Photobiomodulation (PBM) was applied transcranially using low-intensity pulsed LED stimulation at 10 Hz (Alpha) and 40 Hz (Gamma). The stimulation targeted frontal and parietal regions associated with executive

processing and cognitive integration. PBM is known to enhance mitochondrial function, increase ATP production, and improve cortical metabolic efficiency (Hamblin, 2016). Gamma-frequency stimulation has been associated with improved attentional synchronization, while alpha-frequency entrainment promotes adaptive calmness without cognitive suppression. In the context of elite sport, this dual-frequency application was intended to optimize alertness while preventing hyperactivation.

4.3 HRV Biofeedback and Heart–Brain Coherence Training (15 minutes)

Autonomic recalibration was addressed through HRV biofeedback. Athletes practiced paced breathing protocols synchronized with real-time cardiac variability feedback, aiming to increase RMSSD and strengthen vagal tone. This component was grounded in the neurovisceral integration model, which posits that efficient prefrontal–cardiac communication underlies adaptive stress responses (Thayer & Lane, 2000). By enhancing parasympathetic flexibility, HRV biofeedback sought to widen the athlete's physiological window of tolerance, facilitating rapid recovery after critical game moments and stabilizing emotional reactivity.

4.4 Transcutaneous Vagal Nerve Stimulation (10 minutes)

Non-invasive vagal nerve stimulation (tVNS) was applied at the auricular branch of the vagus nerve (tragus region) using medically certified devices. Stimulation frequencies between 20–25 Hz were used to enhance parasympathetic activation. Vagal stimulation directly influences anterior insular and cingulate regions involved in interoception and emotional regulation (Breit et al., 2018). In competitive settings, increased vagal tone is associated with improved emotional resilience, reduced sympathetic overdrive, and enhanced clarity in decision-making under stress.

4.5 Binaural Audio Stimulation (5 minutes)

The session concluded with binaural auditory stimulation combining theta (approximately 4 Hz) and gamma (approximately 40 Hz) frequencies delivered via stereo headphones. This brief entrainment phase aimed to facilitate network synchronization and promote a state of focused readiness. Binaural beats have been shown to influence cortical oscillatory activity and modulate attentional and affective states (Orozco Perez et al., 2020). Within the REACTISPORT protocol, this component functioned as a consolidating phase, reinforcing neural integration achieved during earlier stages of the session.

In combination, these five components form a coherent and synergistic intervention model. Neurofeedback stabilizes cortical control networks; photobiomodulation enhances metabolic and oscillatory efficiency; HRV biofeedback and vagal stimulation recalibrate autonomic balance; and binaural stimulation supports integrative synchronization. Together, they target the core neurophysiological mechanisms underlying stress-induced performance errors, transforming competitive resilience from an abstract psychological trait into a measurable and trainable capacity.

6. Results

The outcomes of the REACTISPORT trial demonstrate statistically robust and clinically meaningful improvements in autonomic regulation, cortical coherence, perceived stress, and sport-specific execution under pressure. When compared to the control group, athletes who completed the 10-week integrated neurophysiological intervention exhibited consistent multidimensional adaptations that collectively support the central hypothesis of the study: competitive stress vulnerability can be reduced through targeted neuroregulatory training.

Table 1. Summary of Primary Outcomes (T0–T2 Changes)

Variable	Experimental Group	Control Group	p-value
RMSSD (ms)	+12.5 ± 3.0	+2.0 ± 2.0	< .001
LF/HF Ratio	–1.3 ± 0.5	–0.2 ± 0.4	< .001
GP-8 Score	–16.0 ± 3.0	–3.0 ± 2.0	< .001
EEG Coherence (BA24/32)	+13.0% ± 2.5	+3.0% ± 2.0	< .001
Motor Errors Under Stress	–35% ± 8.0	–8% ± 5.0	< .001

Autonomic Regulation: RMSSD and LF/HF Ratio

The most pronounced physiological adaptation was observed in heart rate variability. The experimental group demonstrated a mean increase of 12.5 ms in RMSSD, compared to a modest 2.0 ms increase in controls. RMSSD is widely recognized as a reliable index of cardiac vagal tone and parasympathetic flexibility (Shaffer & Ginsberg, 2017). In elite performance contexts, higher vagal tone is associated with superior emotional regulation, attentional stability, and adaptive behavioral responses (Thayer et al., 2012). This magnitude of change over a 10-week period is consistent with structured HRV biofeedback interventions reported in performance populations (Lehrer & Gevirtz, 2014). From a neurophysiological perspective, increased RMSSD suggests enhanced functional coupling between the prefrontal cortex and subcortical autonomic centers, supporting the neurovisceral integration framework (Thayer & Lane, 2000). In practical terms, athletes developed a broader physiological "window of tolerance," enabling faster recovery after stress exposure.

Complementing this finding, the LF/HF ratio decreased significantly (–1.3) in the experimental group, reflecting reduced sympathetic dominance and improved autonomic balance. Chronic sympathetic bias is known to impair executive functioning and increase impulsivity under pressure (Arnsten, 2009). The observed recalibration therefore represents not merely relaxation, but improved physiological adaptability during competitive stress.

Perceived Physiological Stress: GP-8 Reduction

The experimental group showed a substantial 16-point reduction in GP-8 scores, compared to a minor reduction in controls. This scale captures subjective markers of psychophysiological strain, including muscular tension, irritability, sleep disruption, and cognitive fatigue. Such reductions are consistent with studies showing that vagal stimulation and HRV training reduce perceived stress and improve emotional resilience (Breit et al., 2018). Importantly, subjective improvement paralleled objective autonomic enhancement, suggesting congruence between internal experience and physiological adaptation. In elite sport, where cumulative stress burden often undermines performance consistency, this reduction represents a meaningful protective effect.

EEG Coherence in BA24/32: Functional Network Reorganization

One of the most compelling findings was the 13% increase in EEG coherence within BA24/32 regions of the anterior cingulate cortex (ACC), compared to a 3% increase in the control group. The ACC plays a central role in conflict monitoring, error detection, and emotional regulation (Bush et al., 2000; Etkin et al., 2011). Enhanced coherence suggests improved synchronization and functional integration within the Salience Network.

Stress has been shown to disrupt prefrontal–cingulate connectivity, leading to impaired inhibitory control and heightened emotional reactivity (Arnsten, 2009). Neurofeedback-based modulation of cortical oscillations has previously been associated with increased frontal coherence and improved cognitive flexibility (Ros et al., 2014; Gruzelier, 2014). The present findings extend this literature by demonstrating that such network stabilization translates directly into sport-relevant performance gains. In performance terms, enhanced

ACC coherence likely supports more efficient filtering of emotionally salient stimuli (e.g., crowd noise, scoreboard pressure), preserving executive clarity in decisive moments.

Reduction of Motor Errors Under Stress

The most practically significant outcome was a 35% reduction in motor execution errors during standardized high-pressure simulations. The control group showed only an 8% reduction, likely attributable to repetition effects. This finding directly addresses the phenomenon of "choking under pressure," whereby stress disrupts automatic motor programs (Beilock & Carr, 2001). Anxiety-induced attentional shifts interfere with procedural memory retrieval, causing overcontrol or timing errors (Nieuwenhuys & Oudejans, 2012). By stabilizing both autonomic balance and prefrontal-cingulate regulation, the REACTISPORT intervention appears to protect motor automatization from stress-related disruption. The magnitude of error reduction indicates not merely improved psychological confidence, but genuine neurophysiological resilience under simulated competitive pressure.

The convergence of autonomic, cortical, subjective, and behavioral improvements supports a coherent mechanistic explanation. Increased vagal tone enhances physiological adaptability; improved ACC coherence strengthens emotional regulation and error monitoring; preserved executive network stability maintains tactical clarity; and together, these adaptations reduce performance-degrading interference during high-stakes execution.

All between-group differences reached strong statistical significance ($p < .001$), reinforcing both internal validity and effect robustness. Importantly, the multidimensional nature of improvement, spanning heart–brain dynamics and observable performance, distinguishes REACTISPORT from isolated single-modality interventions. These results suggest that competitive resilience can be conceptualized as a trainable neuroregulatory capacity grounded in measurable brain–body integration. Rather than viewing performance breakdown as a purely psychological phenomenon, the findings indicate that stress-induced errors reflect modifiable neurophysiological imbalances that can be systematically recalibrated through integrated intervention.

References

- Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10*(6), 410–422. https://doi.org/10.1038/nrn2648
- Beilock, S. L., & Carr, T. H. (2001). On the fragility of skilled performance: What governs choking under pressure? *Journal of Experimental Psychology: General, 130*(4), 701–725. https://doi.org/10.1037/0096-3445.130.4.701
- Breit, S., Kupferberg, A., Rogler, G., & Hasler, G. (2018). Vagus nerve as modulator of the brain–gut axis in psychiatric and inflammatory disorders. *Frontiers in Psychiatry, 9*, 44. https://doi.org/10.3389/fpsyt.2018.00044
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215–222. https://doi.org/10.1016/S1364-6613(00)01483-2
- Cole, M. W., & Schneider, W. (2007). The cognitive control network: Integrated cortical regions with dissociable functions. *NeuroImage, 37*(1), 343–360. https://doi.org/10.1016/j.neuroimage.2007.03.071

- Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2017). EEG-neurofeedback as a tool to modulate cognition and behavior: A review tutorial. *Frontiers in Human Neuroscience, 11*, 51. https://doi.org/10.3389/fnhum.2017.00051
- Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences, 15*(2), 85–93. https://doi.org/10.1016/j.tics.2010.11.004
- Friedman, L. M., Furberg, C. D., & DeMets, D. L. (2015). *Fundamentals of clinical trials* (5th ed.). Springer.
- Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. *Neuroscience & Biobehavioral Reviews, 44*, 124–141. https://doi.org/10.1016/j.neubiorev.2013.09.017
- Hamblin, M. R. (2016). Shining light on the head: Photobiomodulation for brain disorders. *BBA Clinical, 6*, 113–124. https://doi.org/10.1016/j.bbacli.2016.09.002
- Lehrer, P. M., & Gevirtz, R. (2014). Heart rate variability biofeedback: How and why does it work? *Frontiers in Psychology, 5*, 756. https://doi.org/10.3389/fpsyg.2014.00756
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87*(3), 873–904. https://doi.org/10.1152/physrev.00041.2006
- Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences, 15*(10), 483–506. https://doi.org/10.1016/j.tics.2011.08.003
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202. https://doi.org/10.1146/annurev.neuro.24.1.167
- Nieuwenhuys, A., & Oudejans, R. R. D. (2012). Anxiety and perceptual–motor performance: Toward an integrated model of concepts, mechanisms, and processes. *Psychological Research, 76*(6), 747–759. https://doi.org/10.1007/s00426-011-0384-x
- Orozco Perez, H. D., Dumas, G., & Lehmann, A. (2020). Binaural beats through the auditory pathway: From brainstem to connectivity patterns. *eNeuro, 7*(2), ENEURO.0232-19.2019. https://doi.org/10.1523/ENEURO.0232-19.2019
- Ros, T., Enriquez-Geppert, S., Zotev, V., Young, K. D., Wood, G., Whitfield-Gabrieli, S., Wan, F., Vuilleumier, P., Vialatte, F. B., & Gruzelier, J. H. (2014). Consensus on the reporting and experimental design of clinical and cognitive-behavioural neurofeedback studies (CRED-nf checklist). *Brain, 137*(10), 2677–2691. https://doi.org/10.1093/brain/awu138
- Schulz, K. F., Altman, D. G., & Moher, D. (2010). CONSORT 2010 statement: Updated guidelines for reporting parallel group randomised trials. *BMJ, 340*, c332. https://doi.org/10.1136/bmj.c332
- Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health, 5*, 258. https://doi.org/10.3389/fpubh.2017.00258
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders, 61*(3), 201–216. https://doi.org/10.1016/S0165-0327(00)00338-4
- Thayer, J. F., Åhs, F., Fredrikson, M., Sollers, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies. *Biological Psychology, 90*(2), 148–160. https://doi.org/10.1016/j.biopsycho.2011.11.009