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High-intensity interval exercise impairs neuroelectric indices of reinforcement-learning

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ABSTRACT

A single bout of high-intensity interval exercise (HIIE) improves behavioural measures of cognitive function; however, investigations using event-related potentials (ERPs) to examine the systems that underlie these cognitive improvements are lacking. The reward positivity is a positive-going ERP component that indexes reward processing evoked by 'win' feedback and is a candidate marker of an underlying human reinforcement learning system. While HIIE improves behavioural measures of learning, it is unknown how HIIE affects the amplitude of the reward positivity. Therefore, the purpose of this study was to investigate how HIIE affects reward positivity amplitude in response to reward feedback in university students. Using a single-group randomly assigned counterbalance crossover design, 25 healthy university students performed HIIE and control visits on separate days. Electroencephalographic data was recorded before (pre-intervention) and 10 min after (post-intervention) the intervention period while participants played a novel gambling task. The HIIE intervention consisted of 4 separate body-weight exercises totaling 11 min in duration, including rest. The control visit intervention consisted of quietly watching a nature documentary for 11 min. Heart rate (HR) was measured at the same time intervals in both trials. Analysis revealed that HIIE significantly diminished the amplitude of the reward positivity whereas it remained unaffected in the control condition. HR was significantly higher following HIIE compared to control during post-intervention testing. These findings suggest that mechanisms of reinforcement learning are impaired shortly after HIIE cessation, possibly due to persistent, suboptimal arousal as evidenced by elevated HR post-HIIE.

1. Introduction

Participation in regular physical activity elicits beneficial structural and functional adaptations in the brain across the lifespan [1]. In addition to the benefits of habitual exercise, a single bout of aerobic exercise has been shown to be an effective stimulus for improving behavioural measures of cognitive function for up to an hour following cessation from exercise [2]. In particular, higher-order top-down processes that govern goal-directed behaviour in changing environments (executive functions) appear to be especially sensitive to acute exercise [3,4]. Mechanistically, exercise increases arousal through the reticular activating system, which has been shown to augment the amount of neural resources allocated to attentional tasks and may act to facilitate improvements in cognitive task performance [5–7].

These exercise-induced behavioural improvements are supported by underlying changes in neuroelectric activity, as evidenced by event-related potential (ERP) studies [3,8,9]. ERPs reflect electrophysiological underpinnings of cognitive function, and as a measurement tool, ERPs afford high temporal resolution in characterizing these neuroelectric events [10]. The majority of studies examining ERPs post-exercise have focused on the P300 [10–13], which is an endogenous component that is the neural substrate for processes of stimulus discrimination and information-processing governed by mechanisms of attention, working memory, and decision-making [14,15]. The general consensus in this literature is that continuous aerobic exercise positively modulates P300 amplitude [12,13] and decreases latency [11,12], suggesting that greater allocation of neural resources devoted to a cognitive task and faster task-related processing speed occur post-

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exercise, respectively. However, aside from the P300, only a few studies have examined the effect of exercise on other ERP components involved in executive-control [16,17].

This period of transiently improved cognitive function presents an opportunity to strategically prescribe tasks that require considerable cognitive control, such as learning a new skill (e.g., piano, free-throw shooting). In support of this, a single bout of HIIE performed on a cycle ergometer improves learning of a novel motor task in young males [18,19]. An important ERP component that has yet to be explored with respect to exercise, and is relevant in the context of learning, is the reward-positivity [20–22]. The reward positivity is a positive deflection that occurs approximately 250 ms following reward feedback and reflects neural activity associated with early reward evaluation by a reinforcement learning system within the medial frontal cortex [23,24]. Mechanistically, one prominent theory proposes that reward positivity reflects a phasic burst in dopamine firing within the mesencephalic reward system that projects to the anterior cingulate cortex (ACC) when the outcome of an action is better than predicted (i.e., a reward prediction error) [23,25,26]. Originally, processing of error feedback was associated with a negative deflection called the feedback related negativity (FRN) [27]; however, in recent years it has been proposed that this response reflects the modulation of a positive waveform, reward positivity (see Proudfit [21] for review). The reward positivity is thought to represent the addition of neural processes devoted to processing reward-related feedback and serves as biomarker for early learning, such that the magnitude of response following reward-related feedback predicts successfully learning on a variety of tasks [22,28–31]. As such, given the positive impact of exercise on cognitive function, it reasonable to suspect that the reward positivity amplitude would similarly benefit from a single bout of exercise.

Justification for the possibility of acute exercise modulating reward positivity amplitude is two-fold: first, dopamine signalling on the ACC is a proposed theoretical mechanism underlying the reward positivity [23]. Exercise increases dopamine release in the brain [32,33], which contributes to enhanced cognitive function following exercise through mechanisms of arousal [5,34,35]. Second, the ACC is a target structure for a myriad of higher ordered cognitive processes including reinforcement learning and attentional functions [36,37], the latter of which is positively modulated following a bout of exercise [1,17,38]. As such, given the upregulation of the dopaminergic pathway during exercise and the convergence of effect on the ACC, it stands to reason that a single bout of exercise may also enhance mechanisms of reward learning.

This leads us to propose that learning a new skill may benefit from performing a preceding bout of exercise that augments reinforcement learning systems in order to improve performance [22]. Accordingly, investigation into accessible and time-effective exercise modalities that boost learning is warranted. High intensity interval exercise (HIIE) is a time-effective exercise stimulus that elicits metabolic and fitness adaptations to the same magnitude as traditional continuous aerobic exercise but in a fraction of the time [39], and emerging evidence suggests that a bout of HIIE improves selective attention, inhibition, and learning [40–45]. Importantly, these functions are facilitated by the ACC [46,47], suggesting that HIIE modulates ACC activation in a manner that facilitates improved executive control. As such, we assert that a single bout of HIIE may have a modulatory effect of the amplitude of the reward positivity, which would have implications for learning; however, the effect of HIIE on reward positivity amplitude is currently unknown. Therefore, the purpose of this study was to examine the reward positivity amplitude response to a gambling task following an acute bout of whole-body HIIE in young adults, and given that HIIE results in behavioural improvements, we hypothesized that reward positivity amplitude would be enhanced following a bout of HIIE compared to rest.

2. Methods

2.1. Participants

Healthy, young male and female adults were invited to volunteer in this study via advertisements posted around the University of Victoria (n = 25, 16 females; age = 22.4 ± 3.5 years; BMI = 22.5 ± 2.4 kg/m^2). Prior to acceptance in this study participants were deemed suitable to perform high-intensity, whole body exercise as determined by an exercise screening questionnaire. Participants were excluded from this study if they indicated that they were diagnosed with ADHD or major depression on a medical history screening questionnaire. All participants read and signed consent forms that explicitly detailed study procedures and the associated risks. All experimental procedures were in accordance with the Declaration of Helsinki, and subsequent revisions, and this study was approved by the Health Sciences Research Ethics Boards at Queen's University and the University of Victoria.

2.1.1. Experimental *protocol*

This study consisted of two experimental visits (exercise and control) that were separated by a one week washout period. A counterbalanced crossover design was used, wherein participants were randomized to either exercise or control for their first visit, and subsequently performed the other experimental visit following the washout period (Fig. 1). Participants performed each experimental visit that the

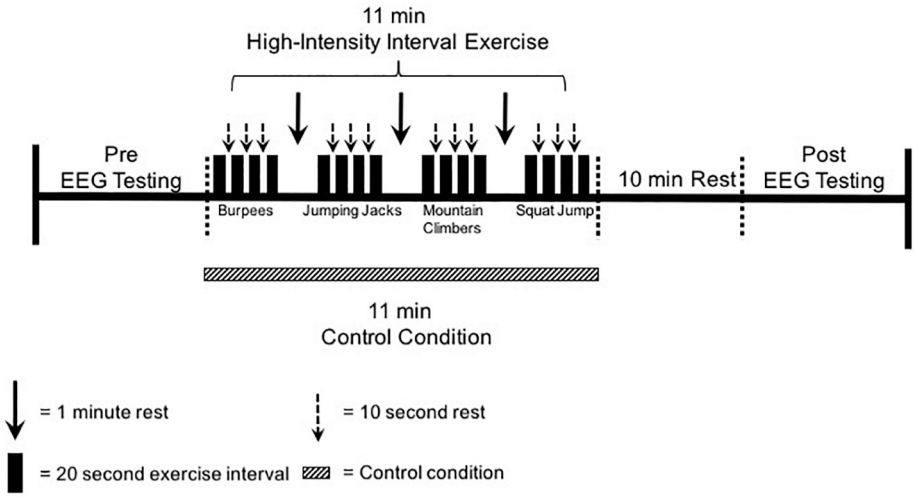

Fig. 1. Protocol schematic for the HIIE and control conditions.

same time of day to avoid potential differences in arousal due to time of day effects. For both visits, heart rate (HR) measures were obtained using HR monitors (Polar Electro, Kempele, Finland). Participants were instructed to abstain from exercise for 24 h and consumption of alcohol or caffeine for 12 h prior to the experimental visits. Otherwise, participants were instructed to maintain their regular activities in between visits to the lab. On the first visit to the lab, a researcher recorded measurements of height and weight for each participant.

2.1.2. Exercise ***visit***

For the exercise visit, participants were instrumented with an EEG headband (MUSE, InteraXon, Inc., Toronto, Ontario, Canada) and performed the pre-EEG testing session. Following completion of the EEG testing participants performed the HIIE protocol, which was a modified Tabata [48] protocol and was 11 min in duration including rest periods, with a total exercise time of 5.5 min. The protocol consisted of four exercise blocks, each consisting of four 20 s exercise intervals separated by 10 s rest intervals. Each exercise block consisted of a different whole-body exercise, which were: 1) burpees, 2) jumping jacks, 3) mountain climbers, and 4) squat-jumps. A one-minute rest period was provided following the completion of each exercise block (Fig. 1). Participants were instructed to perform each exercise as fast as possible while maintaining proper form. Researchers provided continual verbal encouragement and feedback regarding movement technique throughout the exercise session. Following completion of the HIIE bout, participants were given 10 min to recover prior to performing the post-exercise EEG testing. HR measures were obtained immediately before commencement of the pre- and post-EEG testing, and during the 1 min rest periods following an exercise block.

2.1.3. Control visit

For the control visit, participants performed the pre-EEG testing, after which they quietly watched a nature documentary in a dark room for 11 min to account for the elapsed time of the HIIE intervention, followed by a 10-min quiet recovery period prior to performing the second cognitive testing session (Fig. 1). Participants remained seated for the duration of the documentary, did not have access to electronic devices, and were not permitted to interact with other participants. HR measures were obtained at the same time points as the exercise visit.

2.1.4. Cognitive task

Participants were seated in a quiet room and performed a reward learning task on an 11" MacBook Air laptop (Apple, Inc., California, USA) that was connected via Bluetooth to the MUSE EEG headband. Participants made responses to the feedback with the "f" and "j" keys ("f" key for square appearing to the left and "j" key for square appearing to the right) on the laptop keyboard. On each trial, participants viewed a black fixation cross against a grey background (MATLAB RGB value = [108108108]). Participants were instructed to focus on the black fixation cross and to keep eye blinks to a minimum. This cross was presented for 300 to 500 ms and followed by a pair of squares on either side of the fixation cross. Participants were asked, on each trial, to select one of the squares and feedback was presented 300 to 500 ms after square selection and remained visible for 1000 ms ("WIN" for wins, "LOSE" for losses). The next trial began immediately after feedback offset. Each square was assigned a win probability such that one square would "win" more often than the other (60% vs. 10% win/loss ratio) in order to avoid contamination within the N200 time range. Specifically, previous work (e.g., Holroyd and Krigolson [49]) has shown that the reward positivity occurs coincidently with the N200 ERP component which is sensitive to stimulus frequency. As such, in order to avoid frequency effects impacting the amplitude of the reward positivity it is necessary to ensure equivalence between win and loss outcomes (Krigolson [76]). The location of each square (left, right) was randomly determined for each trial and win/loss ratio to colour mapping did not change throughout the experiment. New colours were randomly selected for every block. Participants completed 5 blocks of 20 trials. Behavioural measures of reaction time (i.e., speed of response) and accuracy (i.e., selection of the correct square) were recorded for each trial.

2.1.5. Data acquisition

The approach to acquiring data from the MUSE EEG system has been previously validated [50]. For data acquisition, all laptop batteries were fully charged and MacBook Air laptops were disconnected from power outlets as pilot work determined that 60 Hz noise could be introduced into EEG channels. Therefore, unplugging the laptops prevented electrical noise contamination. Data were recorded from a MUSE EEG headband with research preset AD (500 Hz sampling rate, no onboard data processing: InteraXon, Toronto, Ontario, Canada; see http://developer.choosemuse.com/hardware-firmware/hardware-specifications for full technical specifications). The MUSE EEG system has electrodes located analogous to Fpz, AF7, AF8, TP9, and TP10 with electrode Fpz used as a reference electrode. Data were streamed from the MUSE device via open sound control (OSC) protocol (see http://www.neuroeconlab.com/muse.html for all configuration, setup, methods and software). We sampled 1000 ms of data into MATLAB for every trial – from stimulus onset to 1000 ms after. Data were subject to a small, variable Bluetooth transmission delay measured elsewhere [50] (also see http://developer.choosemuse.com/protocols/data-streaming-protocol).

2.1.6. Muse data processing

The MUSE EEG was processed in the same manner as done previously [50]. The raw MUSE EEG data were converted to a format suitable for BrainVision Analyzer (available at http://neuroeconlab.com/muse-analysis.html) [50]. Following the analysis of the MUSE data, the ERP components were quantified. Data were re-referenced to the average of electrodes TP9 and TP10 and filtered with a dual pass Butterworth filter with a passband of 0.1 Hz to 15 Hz in addition to a 60 Hz notch filter [50]. Next, the data were segmented from stimulus onset to 600 ms after. A baseline correction was applied using a time window from stimulus onset (0 ms) to 50 ms after. This was done as data before stimulus onset were not collected. We shortened each trial epoch and then applied an artifact rejection algorithm in which voltage gradients larger than 10 μV/ms and/or an absolute voltage difference > 100 μV were removed. As a result of this analysis, four participants were removed from further analysis due to excessive (> 25%) EEG artifacts leaving a n of 21. Segmented data were separated according to condition (win or loss). Electrodes AF7 and AF8 were pooled and ERP averages were calculated for each condition for every participant. Finally, difference waveforms were constructed for each condition (i.e., Pre-HIIE, Post-HIIE, Pre-Control and Post-Control) by subtracting loss waveforms from the win waveforms (i.e., win – loss) for each participant. Finally, we calculated grant average conditional and difference waveforms by averaging across participants for each condition.

2.1.7. Data analysis

For the reward positivity, mean peak amplitudes were extracted from difference waves (i.e., Pre-HIIE – Post-HIIE; Pre-Control – Post-Control) using a peak window ± 20 ms from where the reward positivity was maximal. The peak times were identified from the grand average difference waveforms as 310 ± 20 ms and 260 ± 20 ms for Pre- and Post-Control, respectively, and 300 ± 20 ms and 300 ± 20 ms for Pre- and Post-HIIE, respectively. A 2 (condition: HIIE, Control) by 2 (time: Pre, Post) repeated measures ANOVA was used to examine the impact of HIIE on the reward positivity. The assumption of sphericity was examined and Greenhouse-Geisser corrections were applied as needed. The same ANOVA design was used to examine reaction time and performance accuracy. A 2 (condition: HIIE, Control) by 3 (time: Pre, Intervention, Post) repeated measures ANOVA was used to

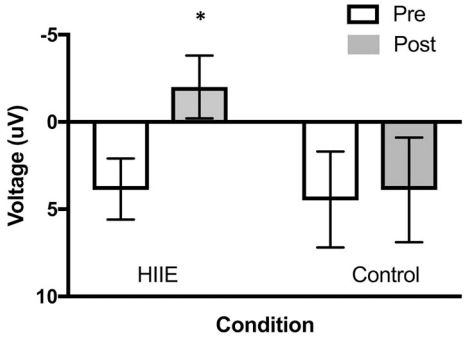

Fig. 2. Mean difference waveform peak amplitude (uV) on the gambling task for each condition. The voltage difference represents the reward positivity taken at 300 ± 20 ms, 300 ± 20 ms, 310 ± 20 ms and 260 ± 20 ms of the ERP waveform for pre- and post-HIIE, and pre- and post-control, respectively. * Significantly different compared to Pre, $p < .05$.

compare differences HR across and between conditions. Means are reported with 95% confidence intervals for all effects.

3. Results

3.1. Reward positivity responses

Analysis of each difference wave revealed a positivity consistent with the timing of the reward positivity for the Pre-HIIE, Pre-Control, and Post-Control conditions. A reduced reward positivity was observed in the Post-HIIE condition. This effect was verified by a repeated measures ANOVA ($F_{(1,20)} = 6.43$, $p < .05$) that demonstrated an interaction between experimental condition (HIIE versus control) and time (Pre versus Post) (see Figs. 2, 3, 4). In other words, the reward positivity amplitude was reduced Post-HIIE (−2.0 uV [−0.3–3.8]) relative to Pre-HIIE (3.9 uV [2.2 5.6]) ($p < .05$) but did not change between Pre (4.5 uV [1.7 7.2] and Post-Control (3.9 uV [0.9 6.9]) ($p > .5$) (Fig. 2).

3.2. Gambling task behavioural responses

There was a significant effect of time on reaction time performance on the gambling task, such that post-intervention reaction time was significantly faster compared to Pre for both HIIE and Control conditions, $F_{(1,20)} = 8.73$, $p < .05$ (Table 1). There was no difference in performance accuracy between time points or between conditions.

3.3. HR responses

There was a significant time x condition interaction on measures of HR ($F_{(2, 40)} = 75.47$, $p < .05$) (Fig. 5). Specifically, HR was significantly higher during HIIE compared to the Pre-EEG testing time point and remained significantly higher during the Post-EEG testing compared to Pre-EEG testing. HR in the HIIE condition was significantly higher than the Control condition during HIIE and the Post-EEG testing time point. There was no difference in HR over time in the Control condition.

4. Discussion

In the present study, we sought to investigate the effect of HIIE on the amplitude of the reward positivity, an ERP component associated with the evaluation of performance feedback. To the best of our knowledge, we are the first group to investigate the effect of exercise on the amplitude of the reward positivity. We instructed participants to make a choice between two alternatives that were linked to a particular probability to produce a win (0.6 compared to 0.1 win probabilities). Participants were tasked with learning the win probability associated with each alternative by trial and error. Behaviourally, participants responded significantly faster during the post-test compared to the pre-test, regardless of condition or order of randomization; however, task accuracy was unchanged throughout the trials (Table 1). This apparent uncoupling between behavioural responses and the reward-positivity has been reported previously when participants learn the task and come to expect reward [51]; however, given that our task switched rewarding stimuli every 20 trials and since the probability of winning was 60%, we do not believe that this was the case. Instead, this uncoupling may be related to repeated task performance within a short period of time. From a neuroelectric perspective, our results demonstrate that a preceding bout of HIIE abolishes the reward positivity during performance of a reward-learning task, suggesting that HIIE temporarily impairs reward learning mechanisms immediately after HIIE. This finding stands in contrast to reports that a bout of HIIE improves behavioural measures of executive function [40–42], and shed light on how HIIE affects mechanisms of reward-learning.

The present finding contradicts positive exercise effect on cognitive function observed elsewhere [2], as accumulating evidence supports the ability of acute exercise to transiently improve behavioural [2] and neuroelectric indices [10,44] of cognitive function for up to an hour post-exercise. Although acute exercise positively modulates the P300 [10,44], only one previous study has investigated ERP componentry related to reinforcement learning following acute exercise in young adults. From this work, it was found that 30 min of submaximal treadmill exercise did not affect the amplitude of a response-locked ERP component – the error related negativity (ERN) – upon error commission during performance of a flanker task [16]. However, it is important to note that the ERN reflects an immediate evaluation of the internal efference copy of a motor command – something that is potentially not susceptible to fatigue with exercise given the mechanism [23]. On the other hand, the reward positivity reflects the conscious processing of visual feedback and thus is associated with a different underlying cortical mechanism that, at least in terms of our data, is affected by acute exercise [20,21]. As such, the mechanisms underlying reward positivity might be differentially affected by acute exercise compared to ERN. Moreover, the resulting ERP from feedback-locked and response-locked tasks represent different aspects of the error-processing system [52] and therefore may have divergent responses to acute exercise. Most recently, we [45] and others demonstrated that brief, HIIE improves behavioural [40,41,53] and neuroelectric [44] measures of cognitive function, which led us to hypothesize that HIIE may also improve mechanisms of reward learning. However, the observed diminishment of the reward positivity following HIIE stands opposed to this hypothesis, especially given that acute exercise may increase the phasic dopamine firing [54,55].

4.1. What may account for the present observations?

Reward positivity amplitude is sensitive to a number of different factors including the perceived value of the reward [56], an individual's motivation [57], inter-individual differences in reward processing [57], acute mental [58] and physical stress [59], and task learning [22]. However, given that the reward positivity was unchanged in the control trials, we attribute the reduction in reward positivity observed following HIIE as being due to an effect of exercise per se (Fig. 2). This may be due to alterations in the dopamine reward pathway and/or competition for neural resources during recovery from exercise as explained by the reticular activating hypofrontality (RAH) model of acute exercise [5]. Acute exercise upregulates the midbrain dopamine system [55], increases cortical excitability [60], and enhances long-term potentiation in the motor cortex [61]. The neuromodulatory effect of the dopamine system on cortical pathways following acute exercise may have an additive effect over time, as long-term exercise training evokes plasticity of the mesocorticolimbic reward pathway [62] and reduces

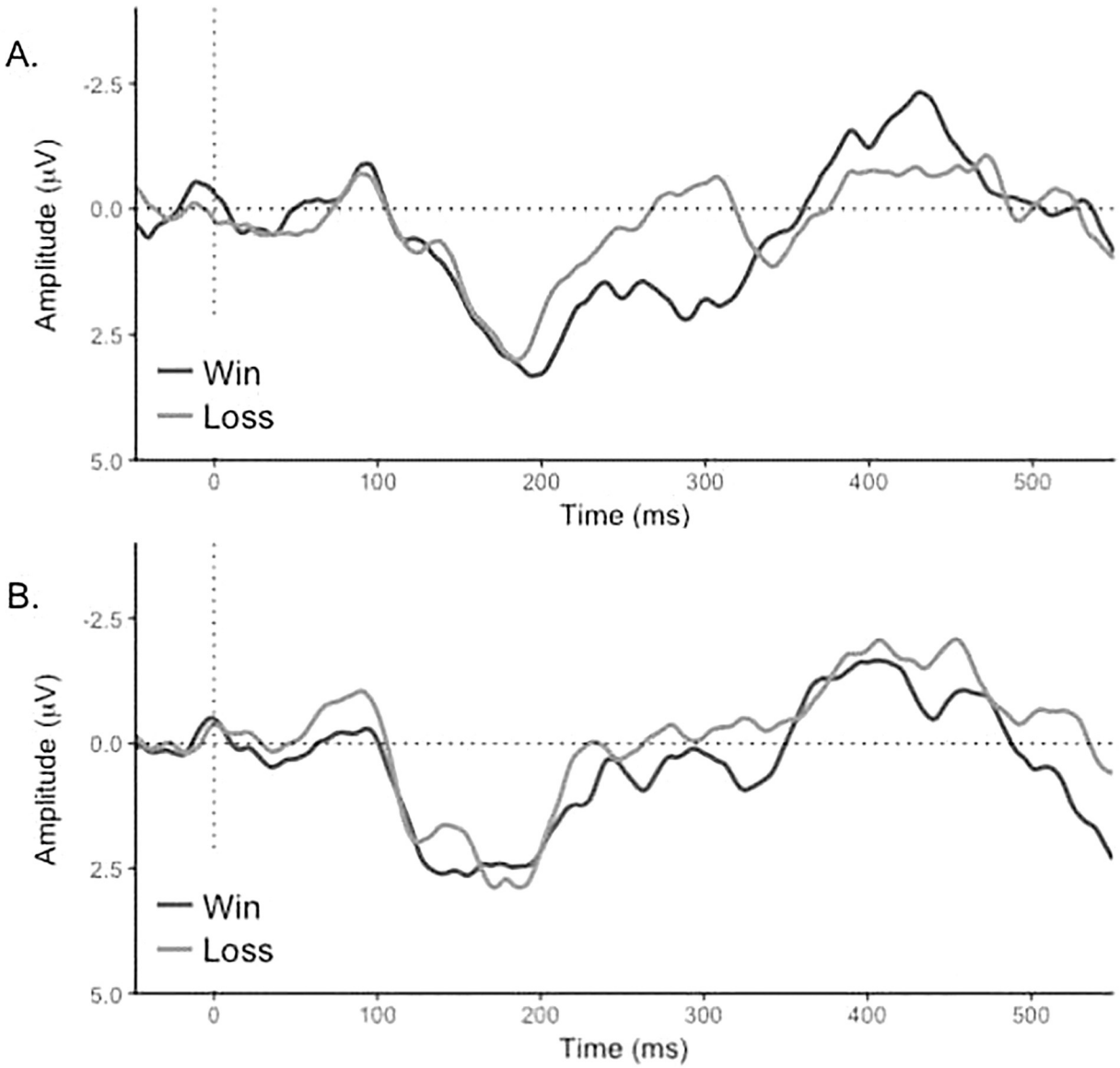

Fig. 3. Grand average conditional waveforms in response to the reward-learning task for A) Pre-HIIE, and B) Post-HIIE.

the positive reinforcing effects of cocaine in rats [63]. In humans, however, evidence suggests that reward-pathway activation is downregulated in response to reward stimuli following a bout of exercise, as evidenced by altered activation of the mesocorticolimbic reward pathway in response to smoking-related images in cigarette-addicted individuals [64]. However, acute exercise's effect on the midbrain dopamine system in humans is poorly understood given measurement limitations [54]. Peripherally, exercise significantly increases levels of plasma dopamine [53,54], which was found to predict aspects of learning in young adults [53]. However, the relationship between peripheral (circulating) and central (brain) levels of dopamine has yet to be established. Nonetheless, if the midbrain dopamine system is in fact upregulated by exercise in humans, as suggested by rodent studies, one might expect an enhanced reward positivity response; however, our findings suggest that HIIE may downregulate the reward pathway, which may be explained by competition for neural resources post-exercise.

A possible mechanism that may underlie the diminishment of the reward positivity is over-arousal via catecholamine signalling during HIIE. Initially proposed by Cooper in 1973 [65], the 'Arousal Theory' states that exercise engages the reticular activating system in the brainstem, which contains the primary nuclei for the serotonergic, dopaminergic, and noradrenergic systems and is a major hub for the integration of bidirectional, diffuse neural transmission during exercise [5,65]. Exercise increases arousal through the release of these monoamines, which in turn drives the potentiation of cognitive function following a bout of exercise [66]. However, there appears to be an inverted-U relationship between arousal and performance [67], such that over-arousal via acute exercise can impede task performance [43]. Cortisol levels may similarly impact reward systems, with higher levels of this stress hormone leading to reductions in reward sensitivity [58]. HIIE significantly increases cortisol levels compared to moderate intensity exercise [68], and this may have deleterious effects on reward-feedback processing in a similar manner to that observed in response to acute mental stress [58] and physical stressors like the cold pressor test [59]. Interestingly, higher levels of aerobic fitness may buffer the suboptimal effects of over-arousal and high cortisol levels with exercise, as Budde et al., [43] found that an acute bout of HIIE improved selective attention in participants with higher physical activity levels whereas no effect was found in those with lower activity levels. It is also possible that mechanisms of reward learning are differentially affected by increased arousal and cortisol compared to other cognitive

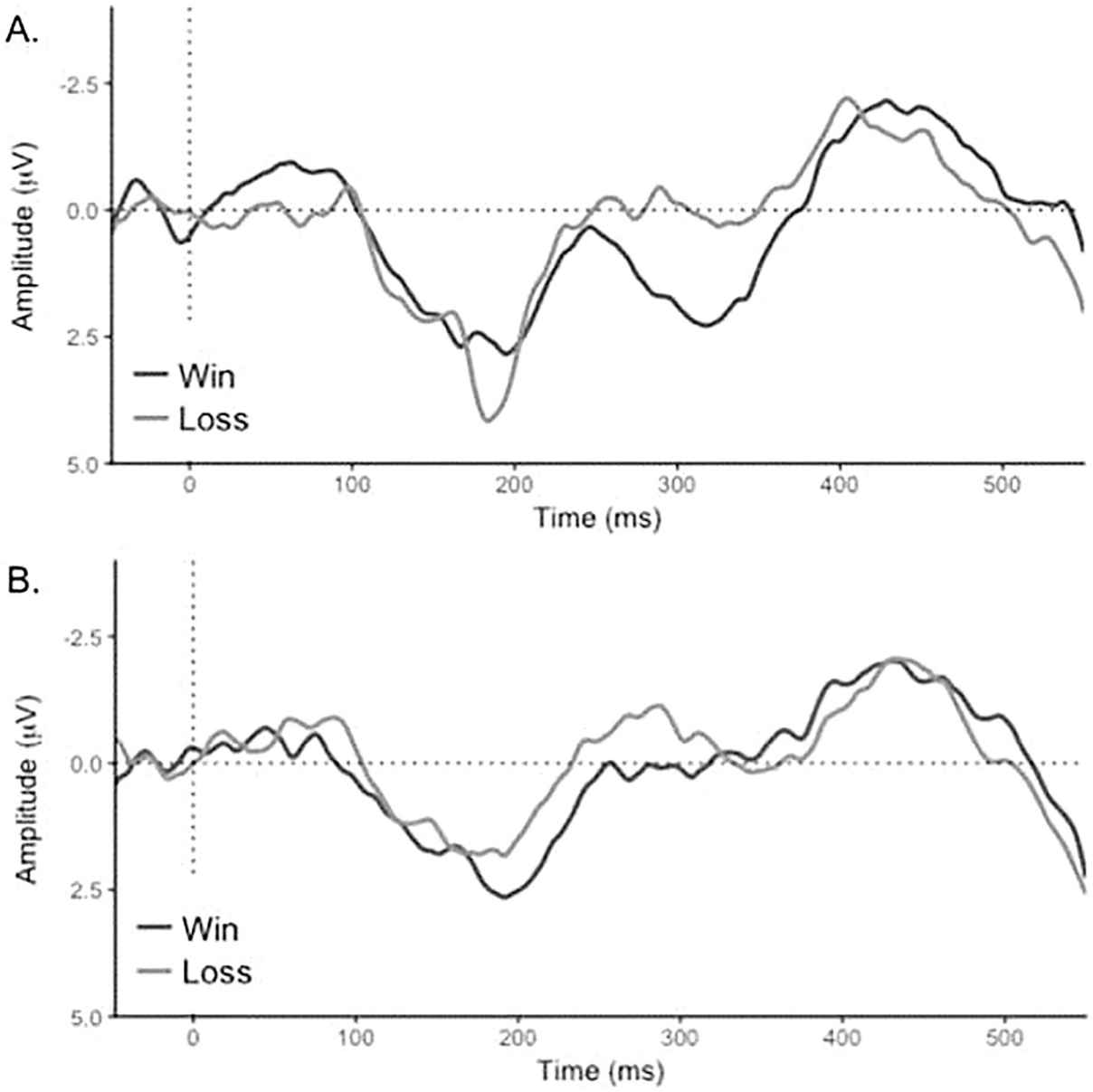

Fig. 4. Grand average conditional waveforms in response to the reward-learning task for A) Pre-Control, and B) Post-Control trials.

Table 1
Gambling task behavioural performance.

	Pre-HIIE	Post-HIIE	Pre-Control	Post-Control
Reaction time (ms)	467 [385548]	385 [332436]*	435 [333538]	358 [305410]*
Task accuracy (% correct)	72.6 [67.4 77.9]	74.5 [70.0 79.1]	75.3 [69.8 80.8]	79.7 [73.6 85.7]

* Significantly different compared to Pre in the same condition, $p < .05$.

functions. In support of this, van Rensburg et al. [64] observed reduced activation of the dorsolateral prefrontal cortex and increased activation of the ACC and frontal areas responsible for maintenance of homeostasis in response to rewarding stimuli following exercise. The apparent down-regulation of the reward-pathway by HIIE may be due to persistent over-arousal following cessation from exercise [69,70], as evidenced by HR being elevated by 21.0 ± 15.9% in post-HIIE testing compared to pre-HIIE in the present study (Fig. 2). Interestingly, this impairment appears to be short-lived following exercise cessation [2,5], which guided our rationale for prescribing a 10-minute recovery period following HIIE. We previously observed improved selective attention following the very same HIIE protocol [45] and deemed this a suitable duration for a recovery period. However, it is possible that 10 min was insufficient for adequate recovery period from HIIE, thereby impairing mechanisms of reward-learning. Accordingly, a relatively longer post-exercise recovery period and/or multiple post-exercise testing time points might reveal differential modulation of the reward positivity post-HIIE due to an optimization of arousal and cortisol levels within the brain [5,68].

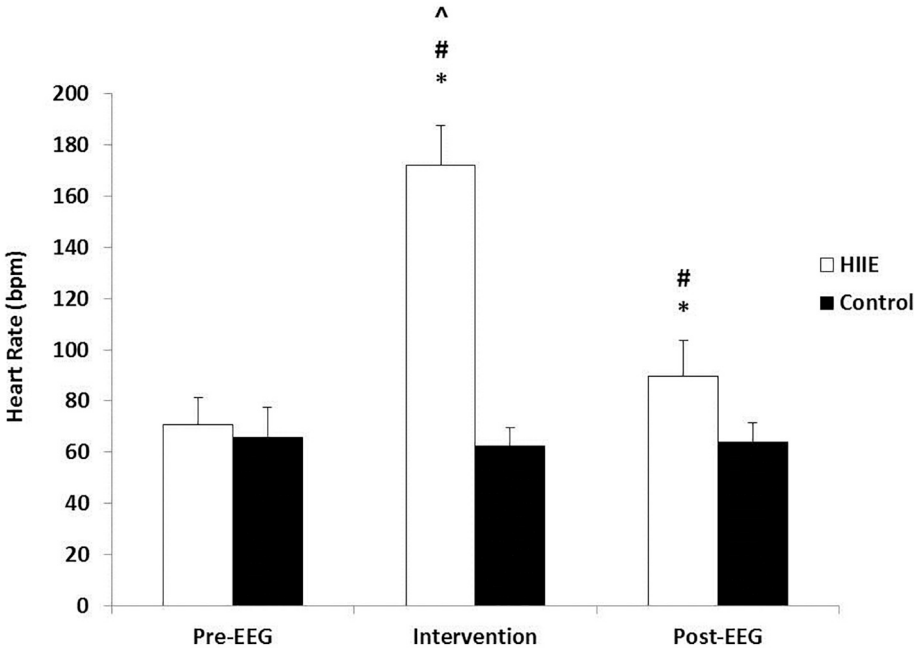

Fig. 5. Heart rate responses to HIIE and Control conditions. * Significantly different compared to Control condition at the same time point. # Significantly different compared to both Pre-EEG HIIE and Control conditions. ^ Significantly different compared to both Post-EEG HIIE and Control conditions.

4.2. Implications

In the earliest stages of learning, before the value of an action is known, external feedback regarding performance is required for successful learning. The reward positivity indexes the underlying mechanisms of reinforcement learning that accompany feedback regarding a better than expected outcome on task [23]. Modulation of reward positivity amplitude reflects the discrepancy between the predicted and actual outcome of an action, as well as adjustments in learning mechanisms as an individual moves from external to internal sources of information for error-processing [22,31,71]. Functionally, the reward positivity serves as a biomarker of the processes that underlie the earliest stages of learning. While our findings suggest that HIIE impairs mechanisms of learning, we emphasize that these results should not be extended to the entire post-exercise time period, as more testing time points beyond 10 min are required to clarify how HIIE affects reward positivity amplitude.

The reward positivity has implications beyond learning, as individuals with major depression [57], anxiety [72], and addiction [73,74] appear to have dysfunctional reward pathways. In individuals with depression, the reward positivity is consistently smaller relative to people without depression [57], whereas reward positivity amplitude appears to be hypersensitive to unexpected reward in individuals with problem gambling behaviours [73]. Both acute and chronic exercise reduce symptoms of depression and anxiety [75] and positively modulate responses to addictive stimuli [64]; however, whether reward-pathway function (i.e., reward positivity) is improved via exercise is currently unknown. Given the wealth of evidence supporting the therapeutic utility of exercise for mood disorders and addiction, and findings from animal studies showing positive brain plasticity in the mesocorticolimbic reward pathway [62], there is good evidence that the reward positivity may be positively implicated. As such, characterizing the effect of acute exercise on the reward positivity stands to advance our understanding of how exercise affects mechanisms of learning and could elucidate how exercise remedies dysfunctional neural pathways.

4.3. Future directions and conclusions

We are the first group to investigate the effect of exercise on the reward positivity. Our finding that reward positivity is abolished in response to reward feedback suggests that mechanisms of reinforcement learning are down-regulated during the initial post-exercise recovery period, possibly due to persistent over-arousal. This work represents a preliminary exploration into the effect of exercise on the reward positivity, and as such, there are a number of questions that remain to be answered. First, post-exercise measurements need to be extended beyond 10 min in order to fully characterize the reward positivity response. These measures should be coupled to indices of arousal, such as HR, in order to gain a greater understanding of potential mechanisms underlying the modulation of the reward positivity post-exercise. Second, future research should compare differences between HIIE and continuous aerobic exercise, and systematically investigate the potential of an intensity dose-response relationship. Finally, the implications of an altered reward positivity on learning should also be investigated in individuals with normal and dysfunctional reward pathways, in order to further our understanding of how exercise can be used for enhancing learning.

Declaration of interest

None.

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