

MULTIPLE STAGES OF INFORMATION PROCESSING ARE MODULATED DURING ACUTE BOUTS OF EXERCISE

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Abstract—Acute bouts of aerobic physical exercise can modulate subsequent cognitive task performance and oscillatory brain activity measured with electroencephalography (EEG). Here, we investigated the sequencing of these modulations of perceptual and cognitive processes using scalp recorded EEG acquired during exercise. Twelve participants viewed pseudo-random sequences of frequent non-target stimuli (cars), infrequent distractors (obliquely oriented faces) and infrequent targets that required a simple detection response (obliquely oriented faces, where the angle was different than the infrequent distractors). The sequences were presented while seated on a stationary bike under three conditions during which scalp recorded EEG was also acquired: rest, low-intensity exercise, and high-intensity exercise. Behavioral target detection was faster during high-intensity exercise compared to both rest and low-intensity exercise. An event-related potential (ERP) analysis of the EEG data revealed that the mean amplitude of the visual P1 component evoked by frequent non-targets measured at parietal–occipital electrodes was larger during low-intensity exercise compared to rest. The P1 component evoked by infrequent targets also peaked earlier during low-intensity exercise compared to rest and high-intensity exercise. The P3a ERP component evoked by infrequent distractors measured at parietal electrodes peaked significantly earlier during both low- and high-intensity exercise when compared to rest. The modulation of the visual P1 and the later P3a components is consistent with the conclusion that exercise modulates multiple stages of neural information processing, ranging from early stage sensory processing (P1) to post-perceptual target categorization (P3a). © 2015 IBRO. Published by Elsevier Ltd. All rights reserved.

Key words: attention, physical activity, aerobic exercise, cognition, EEG, ERP.

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Abbreviations: ANOVA, analysis of variance; BPM, beats per minute; EEG, electroencephalography; ERP, event-related potential; FDR, false discovery rate; NTVA, neural theory of visual attention; RPE, Rating of Perceived Exertion; RTs, reaction times; VO₂max, maximal oxygen consumption.

INTRODUCTION

Prolonged physical exercise can have important sustained and transient benefits across the human lifespan. Higher levels of aerobic fitness can enhance cognitive performance in children (Drollette et al., 2014; Hillman et al., 2014) and adults (Themanson and Hillman, 2006; Bullock and Giesbrecht, 2014), and consistent aerobic activity may help alleviate age-related cognitive decline (Colcombe et al., 2004, 2006; Hayes et al., 2013). Brief, acute bouts of exercise also have transient and generally positive effects on behavioral performance across numerous cognitive domains (for reviews see Lambourne and Tomporowski, 2010; Chang et al., 2012). Scalp recorded electroencephalography (EEG) measurements acquired after an acute bout of exercise have not only revealed subtle changes in patterns of brain activity post-exercise, but also that these changes correlate with human performance (e.g. Hillman et al., 2003; Drollette et al., 2014; Tsai et al., 2014).

While there are clear effects of exercise on brain activity measured subsequent to the bout of physical activity, it is also important to elucidate the nature of the changes that occur in the brain during exercise because these changes may be linked to cognitive performance in a way that is different than when the measurements are made after exercise. A handful of studies have acquired EEG data during an acute bout of exercise (Yagi et al., 1999; Grego et al., 2004; Pontifex and Hillman, 2007), and this approach has the potential to offer unique insight into how patterns of neural activity associated with human performance are influenced during exercise. Indeed, global patterns of neural activity in the alpha, beta and theta frequency bands can be modulated during a bout of exercise (e.g. Kubitz and Mott, 1996; Nybo and Nielsen, 2001; Bailey et al., 2008; Fumoto et al., 2010). These findings provide valuable insight into exercise-induced changes in global oscillatory activity; however, the analytical techniques used in these studies involve computing a Fourier transform over timespans of several minutes, thus precluding one from being able to observe the temporal dynamics of information processing.

What are the potential effects of exercise on brain responses measured during physical activity? Theories of attention suggest that cognitive performance can decline when there is competition for resources (Kahneman, 1973; Hockey, 1997). One suggestion is that activation of the neural circuits involved in the control of gross muscle movements during physical activity may

draw processing resources away from the frontal lobe networks, causing a decline in the performance of more complex cognitive tasks (Dietrich, 2003; Dietrich and Sparling, 2004). This hypothesis predicts effects on relatively late stages of cognitive processing, which is generally consistent with studies that have acquired EEG data during exercise (Yagi et al., 1999; Grego et al., 2004; Pontifex and Hillman, 2007). However, evidence from several recent intra-cranial recordings in mice during treadmill running suggests that behavioral state can have strong modulatory effects on early visual processing (Niell and Stryker, 2010; Ayaz et al., 2013; Saleem et al., 2013; Fu et al., 2014). Thus, acute bouts of physical activity may affect multiple stages of neural information processing.

The event-related potential (ERP) technique is well-suited to isolate the specific stages at which experimental manipulations influence information processing and several studies have used this technique to investigate fluctuations in post-perceptual stages of information processing during exercise. Two studies have focused on the P3 ERP component: a large, robust positive deflection typically centered around the parietal cortex which is considered to reflect brain activity associated with updating of the stimulus environment in memory (Donchin, 1981). P3 amplitude may reflect the amount of resources that are allocated to stimulus processing (e.g. Polich, 1987, 2007; Kok, 2001) and the latency of the P3 is considered to index stimulus classification speed (Kutas et al., 1977; Magliero et al., 1984). For example, Yagi et al. (1999) had participants perform visual and auditory “oddball” tasks, whereby they either monitored a stream of visual or auditory stimuli and responded to infrequent target stimuli (oddballs) while ignoring frequent stimuli (standards). Participants performed this task at rest, during a bout of cycling (at 65–75% of heart rate max), and during a post-exercise recovery session. When comparing the exercise condition to rest and recovery conditions, not only were reaction times (RTs) reduced and error rates increased, the peak latency and amplitude of the parietal P3 ERP component evoked by targets was also reduced. However, these results are difficult to interpret given that the changes in P3 amplitude and latency may just reflect the speed-accuracy trade-off rather than the effects of acute exercise per se. There is also evidence that extended bouts of exercise can have markedly different effects on the P3. Grego et al. (2004) had participants perform an auditory oddball task during a three-hour bout of cycling at ~66% of maximal oxygen consumption (VO_2max). They found a temporary increase in P3 amplitude between the first and second hour, followed by increased peak latency after 2 h, suggesting possible effects of both physiological arousal and fatigue on processing during an extended bout of exercise. The evidence from these studies suggests a complex effect of exercise on the neural indices of information processing, which may be influenced by several factors, such as task demands and the intensity and duration of the bout of exercise.

While the P3 is thought to measure post-perceptual recognition processes, one study has used the ERP technique to test whether earlier stages of information processing are modulated during an acute bout of exercise. Pontifex and Hillman (2007) had subjects perform a flanker task (Eriksen and Eriksen, 1974) during rest and exercise at 60% of maximum heart rate. Modulations of the parietal N1, fronto-central P2 and global N2 components during exercise were observed, which the authors suggested reflects modulation of earlier processes associated with visual discrimination (Vogel and Luck, 2000), selective attention (Talsma and Kok, 2001) and conflict monitoring (Yeung et al., 2004). Additionally, there were increased errors on incongruent trials during exercise compared to rest and increased P3 amplitude and latency at frontal and lateral sites. When considered together, these results indicate that exercise may modulate patterns of neural activity associated with both early and later stages of cognitive processing.

Present aims

While extant ERP studies have offered valuable insight into how dynamic patterns of brain activity are modulated during acute bouts of aerobic exercise, there are a number of questions that remain unanswered. First, although early visual processing associated with the flanker task can be modulated during a bout of exercise (Pontifex and Hillman, 2007), this task is not ideal for the investigation of the earliest stages of processing in extrastriate visual cortex, because the visually evoked response measured at the scalp is a combination of the response to the target and the flankers, so it is unclear when exercise is having its earliest effects on task-relevant information processing. Second, the mechanism by which arousal influences later stages of information processing is currently unclear. Polich (2007) suggests that arousal may increase resources and this might be an effective mechanism for suppressing the response to task-irrelevant stimuli. However, none of the previous ERP study designs allow for a measure of the unique neural response to rare task-irrelevant stimuli. Third, previous ERP investigations have all involved comparison of neural activity during rest and relatively high-intensity exercise, but there have been no attempts to look at how these effects interact with varying intensities of exercise.

The present study had three main aims. First, to test whether exercise can modulate early sensory processing we presented participants with stimuli known to evoke a robust parieto-occipital P1 component (Kasper et al., 2014). Second, to investigate the influence of exercise-induced arousal on both task-relevant and task-irrelevant stimuli, we presented a “three-stimulus” version of the oddball task. Participants viewed a stimulus sequence consisting of frequently appearing non-targets (standards), rare-non-targets (distractors) and targets (targets), and only responded to targets. Distractors and targets presented in a three-stimulus oddball task are known to evoke two subcomponents of the P3 complex which are commonly referred to as the P3a and P3b,

respectively (Polich and Criado, 2006; Polich, 2007). The P3a and P3b subcomponents typically have distinct spatiotemporal properties; with P3a considered to stem from frontal attentional mechanisms, whereas P3b originates from temporal/parietal activations and the hippocampal formation and is associated with memory processing (for a review, see Polich, 2007). Third, to investigate the interaction between exercise intensity and neural processing, participants performed the task at rest and during bouts of low- and high-intensity exercise.

Given that previous studies have shown modulation of both early processing (Pontifex and Hillman, 2007) and later processing (Yagi et al., 1999; Grego et al., 2004) during exercise, it is reasonable to predict that we will also see modulation of neural activity at multiple stages of processing during exercise. Due to the complex patterns of results it is difficult to predict the precise direction of these effects (i.e. reductions or enhancements). It is also difficult to make specific predictions regarding the effects of different exercise intensities on ERPs, but given that several studies demonstrate changes in cortical oscillatory activity as a function of different exercise intensity (Brümmner et al., 2011a,b), it is plausible that exercise intensity may also influence different stages of processing.

EXPERIMENTAL PROCEDURES

Participants

Twelve adult student volunteers from the University of California, Santa Barbara (UCSB) community took part in the study and received course credit or financial compensation (\$20/h). Demographic and physiological data from this sample are shown in Table 1. All participants completed the Physical Activity Readiness Questionnaire (PAR-Q; National Academy of Sports Medicine) to determine their eligibility to participate in aerobic activity. All participants reported having normal or corrected to normal vision and provided informed consent before the study began. All procedures were approved by the UCSB Human Subjects Committee and the US Army Human Research Protection Office.

Stimuli and stationary bike setup

Visual stimuli. Visual stimuli consisted of 8-bit grayscale images of faces and cars that were obtained from the Max Planck Institute for Biological Cybernetics face database (Troje and Bülthoff, 1996). Twelve images

were cars and twelve were faces (half were presented at 45° facing left, and half at 45° facing right). Images were filtered to match the average power spectrum calculated across all images. Independent Gaussian white noise fields were also filtered by the average power spectrum and added to the original images (Das et al., 2010). Stimuli were presented on an 19-inch CRT monitor with custom scripts that utilized the Psychophysics Toolbox for MATLAB (Brainard, 1997). The viewing distance was ~100 cm and images subtended ~7.4 × 7.4° of visual angle.

These stimuli were chosen because their lower level visual properties (i.e. overall size, shape, contrast, luminance) are equated between the different classes of stimuli, thus minimizing the contribution of physical stimulus differences to the visually evoked activity across trials. Standardization also means that participants are required to process the content of each stimulus, rather than relying on these lower level properties to perform the discrimination task. Furthermore, this set of images has been used in several previous studies (Das et al., 2010; Cecotti et al., 2011, 2014) and they are known to evoke robust, reliable ERP components (Kasper et al., 2014).

Stationary bike. The stationary bike was a CycleOps 400 Pro Indoor Cycle (Saris Cycling Group, Madison, WI, USA). T2 + Profile Design Aero Bars (Profile Design, Long Beach, CA, USA) were attached to the handlebars and a Logitech Trackball Mouse (Logitech, Newark, CA, USA) was fixed to the end of the bars (Fig. 1A, B). The addition of the aero bars served two important purposes. First, the participant could lean their elbows onto the bars leaving the hands free to respond to the task. Second, the bars stabilized the participant and helped reduce head and body movement, which is a critical factor for reducing noise during EEG recording. A CycleOps wireless heart rate monitor was used along with Trainer Road software (Trainer Road, Reno, NV, USA) to monitor heart rate.

Procedure

Stimulus presentation. An example of the oddball task is shown in Fig. 1C. Participants were required to monitor the sequence and respond to targets (left oriented faces, $p(\text{target}) = 0.10$) as quickly and as accurately as possible while ignoring distractors (right oriented faces, $p(\text{distractor}) = 0.10$) and standard non-targets (cars, $p(\text{standard}) = 0.80$). Participants responded by pressing the right mouse button with their thumb (Fig. 1B). Presentation order was pseudo-randomized to ensure a minimum of three standards appeared between each distractor/target. The orientation of the target and distractors was counterbalanced across participants.

The oddball task was presented in two separate sessions at two different presentation rates: ~1 Hz (SOA = 200 ms, ISI = 800 ms + jitter) and ~2 Hz (SOA = 200 ms, ISI = 300 + jitter). The jitter on each trial was a random number between 1 and 250 ms. For clarity, these two presentation rates are referred to as the 1-Hz and 2-Hz oddball tasks, respectively. Each

Table 1. Mean and standard deviation values for demographic and cardiovascular data

Measure	Mean participant information
<i>n</i>	12 (6 males)
Age (years)	20 (1.08)
Height (cm)	166.54 (8.60)
Weight (kg)	62.38 (9.81)
BMI (kg/m ²)	22.52 (3.21)
Resting heart rate (BPM)	69.54 (8.90)
VO ₂ max (ml/kg/min)	49.08 (9.96)

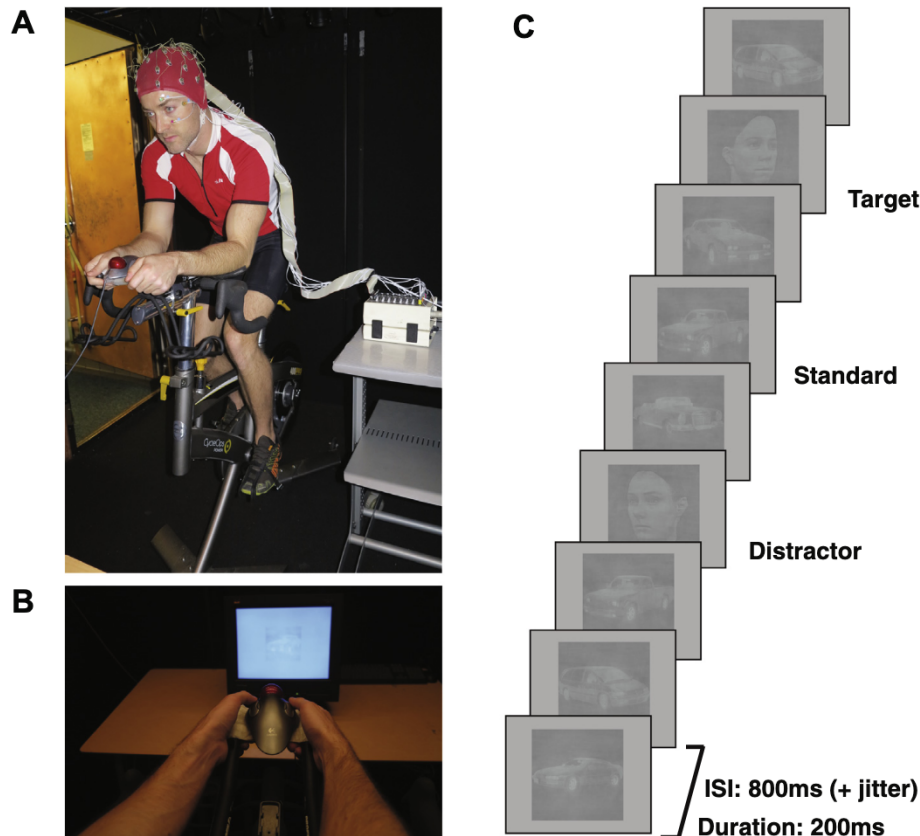


Fig. 1. Methods and stimuli. (A) The participant is shown fitted with an EEG cap and positioned on the stationary bike leaning with their elbows on the aero bars. (B) The participant used their right thumb to respond to target stimuli presented on the screen. (C) Example of the oddball task (1 Hz). Participants were required to detect targets (faces oriented right) in a stream of distractors (faces oriented left) and standards (cars). The individual shown here is the lead author, for demonstration purposes.

session consisted of 1000 images divided into five blocks of 200. The 1-Hz oddball task lasted ~20 min and the 2-Hz oddball task lasted ~10 min. Only the 1-Hz oddball data analyses are reported in this paper, for two main reasons. First, the 2-Hz data were collected for the purpose of another study, which involves testing the efficacy of a brain computer interface under conditions of physical stress. Second, the ERPs evoked by stimuli presented at this rate are subject to a high degree of temporal overlap from adjacent responses, resulting in increased noise and difficulties in isolating temporal dynamics of multiple stages of information processing with high precision (Woldorff, 1993).

Preparation for testing. Each participant was first familiarized with the Rating of Perceived Exertion (RPE) scale (RPE; Borg, 1970, 1982). RPE is a subjective rating of the intensity of physical sensations a person experiences during physical activity, including increased heart rate, respiration rate, muscle fatigue and physical discomfort. Participants were informed that when prompted by the experimenter during the experiment, they were to rate their feeling of exertion by viewing a scale and reporting a number between six (no exertion) and twenty (maximal exertion). Participants then mounted the exercise bike and were trained to maintain a smooth consistent

pedaling cadence of 50 rpm (RPM) in synchrony with a metronome set to 100 beats per minute (BPM) while minimizing head and body movement. BPM was kept constant between different exercise intensity conditions; only the resistance was changed. In combination with the training to keep the head and body still, this ensured that the motion artifact was held constant. Participants remained on the bike and then completed single practice blocks of both the 1-Hz and 2-Hz tasks. A measure of VO_2max was then obtained using the Åstrand–Ryhming Submaximal Bike Test (Åstrand and Ryhming, 1954). The procedure is fully documented in Bullock and Giesbrecht (2014).

Main experiment protocol. Each participant mounted the stationary bike and performed the tasks under three conditions of exercise intensity: at rest, during low-intensity cycling, and during high-intensity cycling. At rest, participants sat on the bike without pedaling. In the low-intensity exercise condition, participants warmed up by pedaling at a minimal resistance level (40 W) for 5 min. At the end of the warm-up, the participant reported their RPE level and then continued pedaling at 40 W resistance for the duration of the low testing session. All participants reported an “extremely light” to “very light” level of exertion (RPE 7–9) at end of the

warm-up and at the end of the low-intensity condition. In the high-intensity exercise condition, the participant also warmed up for 5 min at 40 W resistance, but then resistance was incrementally increased until workload intensity reached a level that the participant reported to be “somewhat hard” (RPE 12–14). This resistance level was set between 70 W and 120 W on an individual participant basis and was maintained for the duration of the testing session, followed by a cooling down session for 5 min at 40 W. Note that for ease of labeling we will refer to the three exercise conditions as rest, low and high intensity from here onwards. Two measures ensured that participants exercised at the desired intensity during the exercise conditions. First, participants were prompted by the experimenter to report their RPE at ~5 min intervals during each testing session. Second, an experimenter continuously monitored pedaling cadence and gave verbal instructions between blocks of the task to increase or decrease cadence if 50 RPM was not being maintained. Exercise conditions were completed in an order that was fully counterbalanced between participants and care was taken to ensure that heart rate returned to within 10 BPM of resting baseline between conditions.

During each of the three exercise intensity conditions, participants had their EEG recorded in four distinct sessions. In sessions 1 and 2, 5 min of continuous EEG were recorded with eyes open and eyes closed, respectively. The aim of these sessions was to record baseline brain activity during different exercise intensities while participants were not engaged in a cognitive task (these data are not discussed any further, as they are beyond the scope of the present paper). In sessions 3 and 4, participants performed the 1-Hz and 2-Hz oddball tasks. The order in which sessions 3 and 4 were completed was counterbalanced between participants. The total duration of the rest condition was ~45 min in total. The low- and high-intensity exercise conditions were longer in duration (~50 min total) because participants were required to warm up for 5 min before the first recording session.

EEG data acquisition

EEG data were recorded for each participant using a BioSemi Active Two system (BioSemi, Amsterdam, Netherlands) consisting of 32 Ag–AgCl sintered active electrodes arranged in an elastic cap (Electro-Cap, Eaton, OH, USA) and placed in accordance to the 10–20 system. Additional electrodes were placed at the right and left mastoids, as well as 1 cm lateral to the left and right canthi (horizontal) and above and below each eye (vertical) for the electrooculography (EOG). Data were sampled at 512 Hz and referenced offline to the average mastoid signal. At the beginning of each investigation all impedances were < 20 k Ω . All recording took place in an electrically shielded chamber to ensure minimal interference from external sources of electrical noise. The chamber was air-conditioned and the EEG cap was made of a breathable mesh, both of which helped minimize sweating.

Data analysis

EEG data pre-processing. MATLAB (version 2013b, Massachusetts, The MathWorks Inc.) was used for offline processing of the EEG data, along with the EEGLAB (Delorme and Makeig, 2004) and ERPLAB (Lopez-Calderon and Luck, 2014) toolboxes. The continuous data were low- and high-pass filtered at .01 Hz and 30 Hz, respectively (slope 6 dB octave⁻¹) and then ocular artifacts were removed using the Automatic Artifact Removal toolbox (Gomez-Herrero et al., 2006), available as an extension for EEGLAB. One participant's data were excluded from all further EEG analysis as their data contained excessive levels of noise.

The design characteristics of our 1-Hz oddball task allowed us to primarily focus on two main ERP components: the visual P1 and the P3 complex (P3a/P3b). We performed mean amplitude and peak latency analyses to determine whether the early and later stages of processing thought to be represented by these components were modulated during conditions of varying exercise intensity. The data were binned according to stimulus category and exercise condition and epoched from –100 ms pre-stimulus to 500 ms post-stimulus onset. Epoched data were then submitted to a threshold rejection routine, whereby any trial exceeding $\pm 125 \mu\text{V}$ measured at a priori scalp electrodes of interest (CP1, CP2, Pz, P3, P4, PO3, PO4, Oz, O1, and O2) was excluded. Rejection rates were 1.25%, 10.24% and 8.20% for rest, low- and high-intensity exercise conditions, respectively. Grand average ERP waveforms were created for each stimulus category by averaging across trials and participants. Only target trials with correct responses were included in the waveform.

P1 mean amplitude was calculated by averaging data from occipital and parieto-occipital channels (Oz, O1, O2, PO3 and PO4), finding the peak latency of the positive going component between 100 and 150 ms post stimulus onset and calculating mean amplitude ± 10 ms around this latency. Mean amplitude was calculated 127 ± 10 ms for standards, 126 ± 10 ms for distractors, and 125 ± 10 ms for targets. P3a and P3b peak latencies were calculated for distractor and target trials, respectively, by determining the peak latency of the positive going component between 300–500 ms post-stimulus onset and averaging across subjects and over channels CP1, CP2, Pz, P3, P4, PO3, and PO4. Mean amplitude was calculated from a window around the peak latency (between 391 ± 25 ms for P3a and 423 ± 25 ms for P3b). Mean amplitude and peak latency were not computed for standards, as these stimuli typically do not evoke a reliable P3 in this task.

Statistical analyses

All physiological, behavioral and EEG data were within three standard deviations of the condition means and the majority of data were normally distributed. Greenhouse–Geisser corrections were applied to all analyses of variance (ANOVAs) in order to account for

violations of the sphericity assumption. All instances of non-normality are reported in the text and any significant differences between conditions are supported with non-parametric tests. Post hoc analyses were computed using paired-samples *t*-tests correcting for multiple comparisons using the false discovery rate (FDR) method with a threshold of .05 (Benjamini and Hochberg, 1995). For completeness, both the uncorrected and FDR adjusted *p*-values (*q*) are reported for the post hoc tests.

RESULTS

The results are reported in three sections. First, we evaluate the physiological data to confirm the effectiveness of the exercise manipulation. Second, we examine the behavioral data, to determine whether exercise can influence target detection response time and accuracy. Third, we test whether event-related neural activity (i.e. measured with the ERP technique) is modulated by different conditions of exercise intensity.

Exercise physiology

Mean and standard deviation values for heart rate, RPE, cadence and power are presented in Table 2. A repeated-measures ANOVA was computed for the heart rate data, with exercise intensity [rest, low, high] as the within-participant factor. Paired samples *t*-tests were calculated to compare RPE, cadence and power data between low and high exercise intensity conditions. Heart rate significantly increased as a function of exercise, [$F(2,22) = 162.40$, $p < .001$, $\eta^2 = .93$]. Post hoc tests confirmed significantly increased heart rate from rest to low-intensity [$t(11) = 9.69$, $p < .001$, $q = .001$], low- to high-intensity [$t(11) = 9.42$, $p < .001$, $q = .001$] and rest to high-intensity conditions, [$t(11) = 16.28$, $p < .001$, $q = .001$]. RPE also significantly increased as a function of exercise intensity [$t(11) = 19.16$, $p < .001$]. Cadence did not differ between low- and high-intensity exercise [$t(11) = 1.72$, $p = .11$]. Power significantly increased from low to high intensity [$t(11) = 9.77$, $p < .001$].

Behavior

Mean accuracy and RT data are shown in Fig. 2. Repeated measures ANOVAs were computed with exercise intensity [rest, low, high] as the within-participant factor. RT significantly decreased as a function of exercise intensity [$F(2,22) = 7.48$, $p = .003$, $\eta^2 = .41$]. Post-hoc tests confirmed significantly faster

RTs during high-intensity exercise compared to rest [$t(11) = 3.25$, $p = .008$, $q = .012$] and during high-intensity compared to low-intensity exercise [$t(11) = 3.69$, $p = .004$, $q = .012$]. RTs did not significantly differ between low-intensity and rest conditions [$t(11) = 1.00$, $p = .34$, $q = .34$]. Target detection accuracy was not modulated by exercise [$F(2,22) = .29$, $p = .75$, $\eta^2 = .03$]. Overall false alarm rate was low (overall mean) and did not differ as a function of exercise intensity [$F(2,20) = 0.23$, $p = .98$, $\eta^2 = .002$].

Electrophysiology

P1 component. Inspection of scalp topographies from revealed a robust parieto-occipital P1 component in response to standards, distractors and targets presented during the task (Fig. 3). Repeated measures ANOVAs with exercise intensity [rest, low, high] and electrode position [PO3, PO4, O1, Oz, O2] as within-participant factors were computed for standards, distractors and targets for P1 mean amplitude and peak latency. P1 mean amplitude was modulated by exercise on standard trials [$F(2,20) = 8.92$, $p = .003$, $\eta^2 = .47$]. Post hoc tests confirmed significantly larger mean P1 amplitude during low-intensity exercise compared to rest [$t(10) = -5.38$, $p < .001$, $q = .003$]. There were trends for larger P1 amplitudes during low-intensity exercise compared to high-intensity exercise [$t(10) = 2.33$, $p = .042$, $q = .063$] and rest compared to high-intensity exercise [$t(10) = -1.82$, $p = .09$, $q = .09$]. There was no main effect of electrode location [$F(4,40) = 3.67$, $p = .07$, $\eta^2 = .27$] and exercise intensity and electrode location did not interact [$F(8,80) = .48$, $p = .72$, $\eta^2 = .05$]. P1 mean amplitude was not modulated by exercise intensity on distractor trials or target trials

Table 2. Mean and standard deviation values of physiological data recorded during 1 Hz oddball task

	Heart rate (BPM)	RPE	Cadence (RPM)	Power (W)
Rest	83.79 (12.12)	–	–	–
Low	114.36 (15.75)	7.58 (.67)	52.76 (2.99)	38.68 (3.15)
High	147.64 (15.83)	13.13 (.95)	51.43 (1.57)	93.75 (19.98)

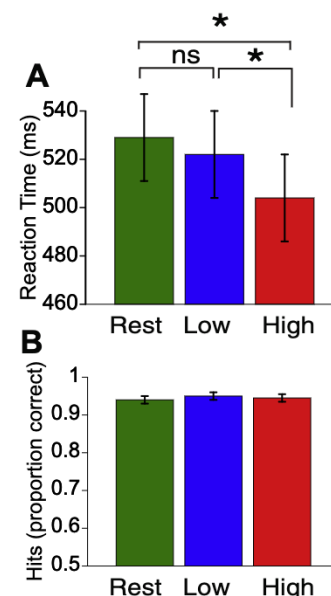


Fig. 2. Behavioral data. Plots show (A) reaction time and (B) accuracy data for the 1 Hz oddball task. Error bars = \pm SEM. $p < .05$.

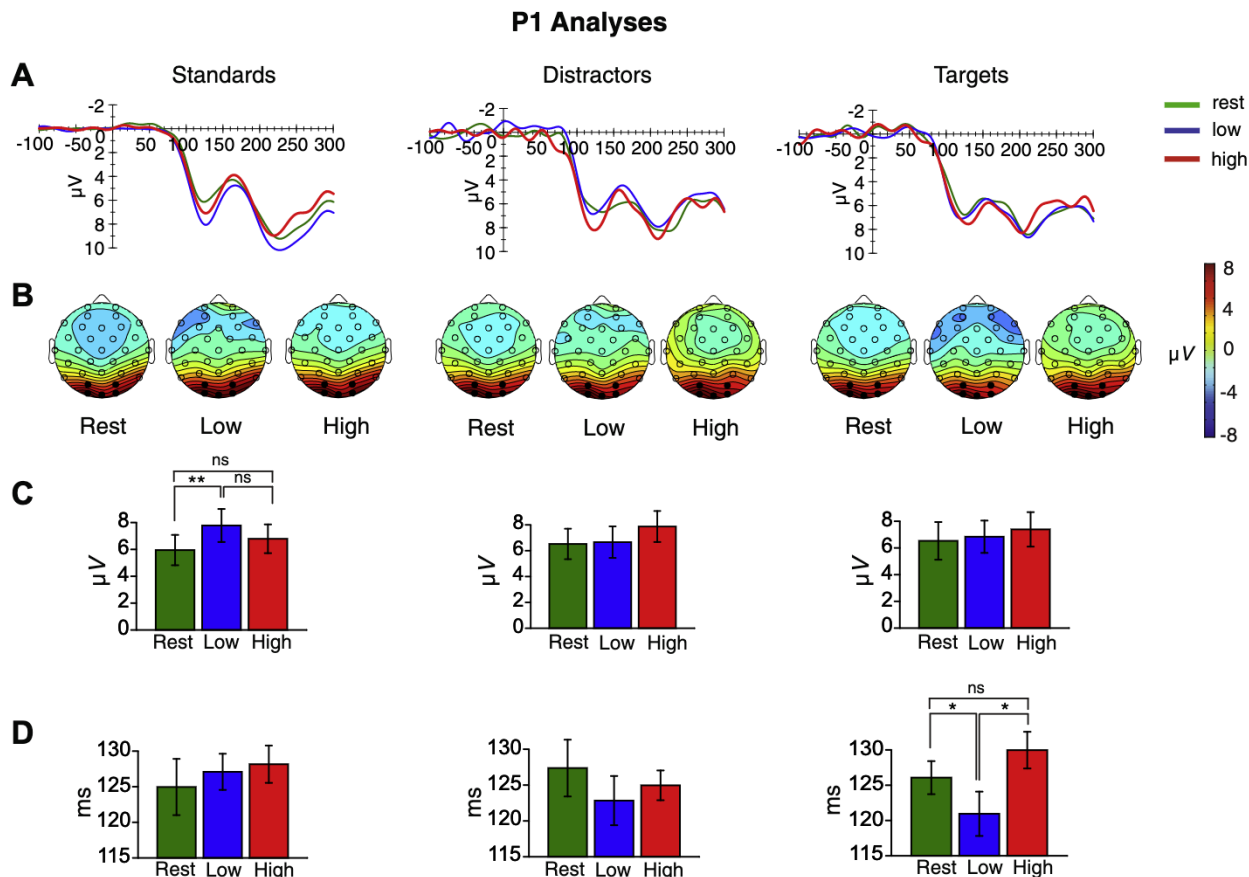


Fig. 3. Parieto-occipital P1 mean amplitude modulation by exercise in standard, distractor and target trials. (A) Grand averaged ERPs collapsed across electrodes PO3, PO4, O1, Oz, and O2. (B) Scalp topographies. Filled circles represent electrodes used in analyses. (C) Mean amplitude plots. (D) Peak latency plots. Error bars = \pm SEM. * $p < .05$, ** $p < .01$.

[$F(2,20) = 2.72$, $p = .08$, $\eta^2 = .25$; $F(2,20) = .35$, $p = .69$, $\eta^2 = .03$, respectively]. There was a main effect of electrode location on target trials [$F(4,40) = 4.09$, $p = .049$, $\eta^2 = .29$] such that the response at occipital locations was larger than at parieto-occipital locations. Electrode location was not significant on distractor trials [$F(4,40) = 3.51$, $p = .063$, $\eta^2 = .26$] and exercise intensity and electrode location did not interact [$F(8,80) = .41$, $p = .79$, $\eta^2 = .04$; $F(8,80) = .38$, $p = .76$, $\eta^2 = .04$, respectively].

P1 peak latency was modulated by exercise intensity on target trials [$F(2,20) = 5.62$, $p = .025$, $\eta^2 = .36$]. Post hoc tests confirmed that the P1 peaked significantly faster during low-intensity exercise compared to rest [$t(10) = 2.79$, $p = .019$, $q = .038$] and during low-intensity compared to high-intensity exercise [$t(10) = 2.63$, $p = .025$, $q = .038$]. Peak latency did not differ between rest and high-intensity exercise [$t(10) = 1.50$, $p = .16$, $q = .164$]. There was no main effect of electrode location [$F(4,40) = 2.44$, $p = .11$, $\eta^2 = .20$] and exercise intensity and electrode location did not interact [$F(8,80) = .78$, $p = .54$, $\eta^2 = .07$]. P1 peak latency was not modulated by exercise intensity on standard trials [$F(2,20) = 1.31$, $p = .29$, $\eta^2 = .12$] or distractor trials [$F(2,20) = .67$, $p = .50$, $\eta^2 = .06$]. Peak latency was earlier at right hemisphere locations when

compared to left hemisphere locations in both standard and distractor trials [$F(4,40) = 11.34$, $p < .001$, $\eta^2 = .53$; $F(4,40) = 4.02$, $p = .018$, $\eta^2 = .29$, respectively], but exercise intensity and electrode location did not interact on standard or distractor trials [$F(8,80) = .74$, $p = .54$, $\eta^2 = .07$; $F(8,80) = 1.04$, $p = .39$, $\eta^2 = .09$, respectively].

P3 components. P3a and P3b components were measured at centro-parietal, parietal and parieto-occipital channels in response to targets (P3a) and distractors (P3b) (Fig. 4). Repeated measures ANOVAs with exercise intensity [rest, low, high] and electrode position [CP1, CP2, P3, Pz, P4, PO3, PO4] as within-participant factors were computed for distractors and targets for P3a and P3b mean amplitude and peak latency. Peak latency data were non-normally distributed in both the high-intensity distractors and target conditions, so post hoc non-parametric Wilcoxon Signed Ranks tests were computed for these data. The pattern of results matched the post hoc t -tests, so only the parametric statistics are reported.

P3a peak latency was modulated by exercise intensity on distractor trials [$F(2,20) = 6.92$, $p = .008$, $\eta^2 = .41$]. Post hoc paired samples t -tests confirmed that P3a peaked earlier during high-intensity exercise compared

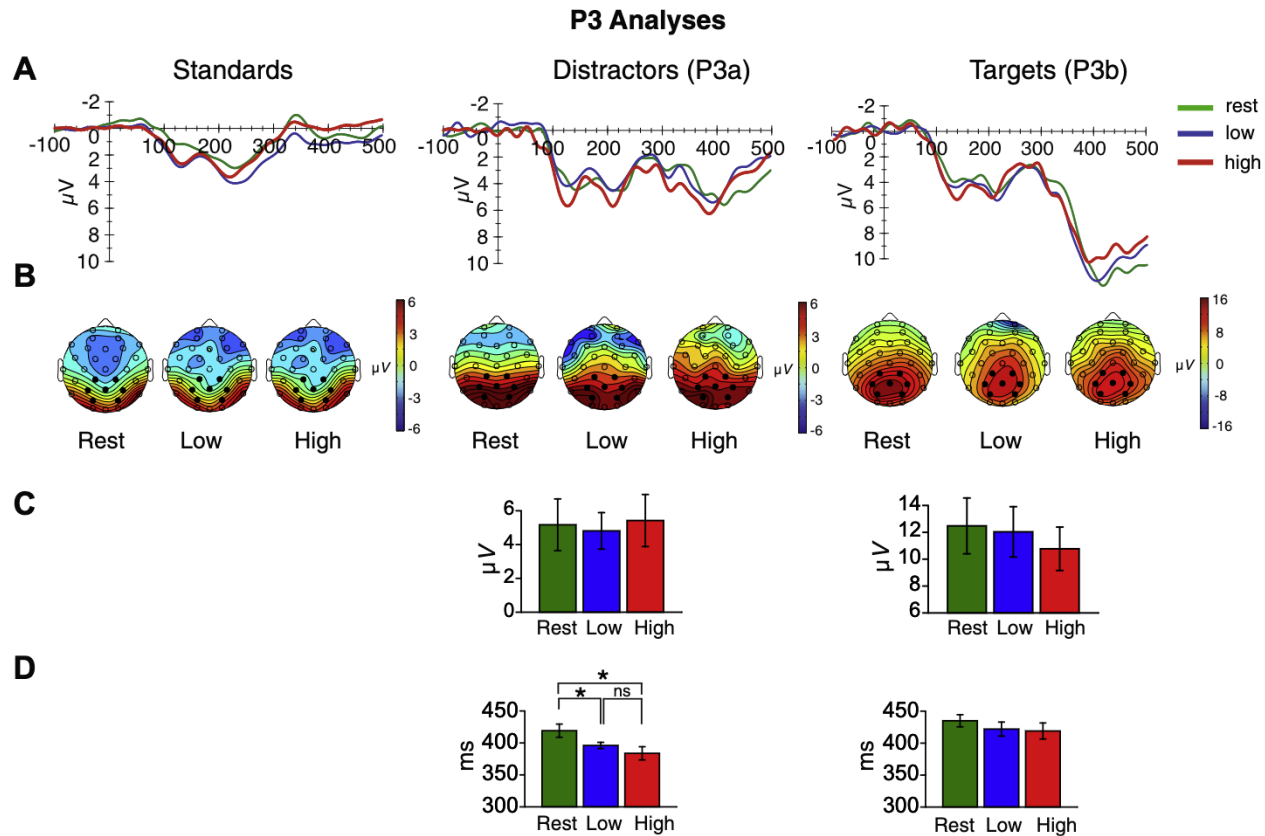


Fig. 4. Parietal-P3 peak latency modulation by exercise. (A) Grand averaged ERPs collapsed across electrodes CP1, CP2, P3, Pz, P4, PO3, and PO4. (B) Scalp topographies. Filled circles represent electrodes used in analyses. (C) Mean amplitude plots. (D) Peak latency plots. Error bars = \pm SEM. $p < .05$.

to rest [$t(10) = 3.04$, $p = .013$, $q = .0255$] and during low-intensity exercise compared to rest [$t(10) = 2.86$, $p = .017$, $q = .0255$]. There was no significant difference between low- and high-intensity exercise conditions [$t(10) = 1.41$, $p = .19$, $q = .19$]. P3b peak latency was not modulated by exercise intensity on target trials [$F(2,20) = 1.39$, $p = .27$, $\eta^2 = .12$]. There were main effects of electrode location for both distractor and target trials [$F(6,60) = 5.29$, $p = .008$, $\eta^2 = .35$; $F(6,60) = 8.28$, $p = .001$, $\eta^2 = .45$, respectively] which were largely driven by faster P3a and P3b responses at parieto-occipital locations when compared to centro-parietal and parietal locations. Exercise intensity and electrode location did not interact on either trial type [$F(12,120) = 1.03$, $p = .40$, $\eta^2 = .09$; $F(12,120) = .315$, $p = .99$, $\eta^2 = .03$].

P3a and P3b mean amplitude was not modulated by exercise intensity on distractor trials or target trials [$F(2,20) = .08$, $p = .85$, $\eta^2 = .01$; $F(2,20) = .83$, $p = .45$, $\eta^2 = .08$, respectively]. There were no main effects of electrode location in either distractor and target trials [$F(6, 60) = 3.13$, $p = .06$, $\eta^2 = .24$; $F(6, 60) = 2.66$, $p = .08$, $\eta^2 = .21$, respectively] and no interaction between exercise intensity and electrode location in either condition [$F(12, 120) = .72$, $p = .60$, $\eta^2 = .07$; $F(12, 120) = 1.27$, $p = .30$, $\eta^2 = .11$, respectively].

We also tested whether peak latency and mean amplitude were significantly different for P3a and P3b across the different exercise conditions. Repeated measures ANOVAs were computed with component [P3a, P3b], electrode location [CP1, CP2, P3, Pz, P4, PO3, PO4] and exercise intensity [rest, low, high] as within-participant factors. Results confirmed that the P3a peak was at significantly lower latency and reduced amplitude than the P3b peak during all three exercise conditions [$F(1,10) = 25.49$, $p = .001$, $\eta^2 = .72$; $F(1,10) = 51.88$, $p = .001$, $\eta^2 = .84$, respectively].

DISCUSSION

The goal of the present study was to examine the effects of an acute bout of physical exercise on multiple stages of information processing. Three key findings emerged from the behavioral and EEG data recorded during the task. First, response time to targets was faster during high-intensity exercise compared to both rest and low-intensity exercise. Second, mean amplitude of the parieto-occipital P1 component evoked by standards was larger during low-intensity exercise compared to rest and the P1 component peaked significantly earlier during low-intensity exercise when compared to rest and high-intensity exercise. Third, peak latency of the parietal P3a component evoked by distractors

decreased as a function of low- and high-intensity exercise compared to rest. In the following sections we discuss these findings in relation to the extant literature documenting exercise effects on behavior and brain activation

Behavioral performance

Target detection was more rapid during high-intensity exercise compared to low-intensity exercise and rest. There was no significant response-time benefit of low-intensity exercise compared to rest, but the trend was in the same direction, suggesting that the amount of response-time benefit depends on exercise intensity. These data are consistent with Yagi et al. (1999), who also demonstrated reduced response times to visual targets presented in a two-stimulus oddball task during a similar intensity level of exercise (130–150 BPM) when compared to rest. Unlike Yagi et al. (1999), however, we did not observe an increase in error rates during exercise compared to rest. In other words, where Yagi et al. (1999) observed modulations in neural activity in the presence of a behavioral speed-accuracy tradeoff, no such tradeoff was present in our data. Thus, any modulations in neural activity that we observe across exercise conditions are likely not due to the confounding effects of a behavioral speed accuracy tradeoff. We should acknowledge, however, that behavioral performance in the present manuscript was near ceiling across all conditions in our task, thereby precluding any negative effects of exercise on performance. Our data also concur with at least 20 other studies that demonstrate enhanced information processing during a bout of sustained cycling exercise performed at intensities ranging from 40% to 70% of VO_2max (Audiffren et al., 2008) and are consistent with recent meta-analyses that show a small but positive enhancement of cognitive performance during cycling when compared to rest (Lambourne et al., 2010; Chang et al., 2012). This pattern of results suggests that exercise effects on task performance are dependent on a number of factors, such as exercise intensity and the nature and difficulty of the cognitive task.

Early perceptual processing

The present data suggest that early visual information processing can be modulated during a bout of aerobic exercise. Specifically, we demonstrate that the amplitude of the visual P1 ERP component evoked on standard trials was significantly larger during low-intensity exercise compared to rest. Furthermore, on target trials the P1 peaked significantly earlier during low-intensity exercise when compared to both rest and high-intensity exercise. To our knowledge, these data are the first to show modulation of the P1 as a function of an acute bout of aerobic exercise.

The P1 component forms part of the visual evoked potential and is considered to have multiple sources across extrastriate cortex, near the border of Brodmann's Areas 18 and 19 (Clark et al., 1994; Heinze et al., 1994; Di Russo et al., 2001). In the ERP literature,

modulation of P1 amplitude has been shown in numerous studies to reflect the direction of selective attention, such that an attended item in a visual display will evoke a larger P1 than an unattended item (Van Voorhis and Hillyard, 1977). A sensory gain control mechanism has been proposed as one explanation for this effect, whereby stimulus-evoked neural activity is either increased or decreased depending on how much attention is allocated to the stimulus (for a review, see Hillyard et al., 1998). When interpreted within this framework, the present data suggest that exercise may influence the sensory gain mechanism. Specifically, under the current cognitive and exercise demands, low-intensity exercise may enhance the level of stimulus-evoked neural activity associated with processing of the visual stimuli in the present task. While exercise does not appear to alter the temporal dynamics of the P1 waveform evoked by standard trials, our data indicate that on target trials the wave peaks significantly earlier during low-intensity exercise when compared to rest and high-intensity exercise. While latency effects must be interpreted with some caution (Luck, 2014), this suggests that the stimulus-evoked neural activity associated with P1 occurs as a function of exercise intensity.

There is evidence that the P1 is modulated by the focus of spatial attention (Mangun and Hillyard, 1991; Hillyard et al., 1998); here we show that P1 can also be modulated by physical exercise and hence exercise may influence the perceptual selection process. Under the present task demands the most robust P1 mean amplitude and peak latency enhancement occurs during low-intensity exercise, which suggests that there may be an ideal level of exercise that is necessary for optimal sensory gain and more rapid processing. It is possible that enhanced early stage processing may contribute to the performance gains observed during cycling in the present study and perhaps in other previous studies that also demonstrate beneficial effects of cycling on performance (see Lambourne et al., 2010; Chang et al., 2012). There is evidence to support a relationship between P1 (and N1) amplitude and aspects of visual cognitive task performance, such as visual discrimination (Hanslmayr et al., 2005). However, one cannot make strong links between amplitude effects and cognitive task performance from the current data, as P1 amplitude was only modulated on standard trials where participants did not make a behavioral response. Making a direct link between peak latency and task performance is also complicated because P1 peak latency was shorter during low-intensity exercise compared to rest and high-intensity exercise on target trials, while the corresponding pattern was not observed in the behavioral data. Further work with paradigms better suited to specifically exploring early visually evoked potentials is necessary to determine precisely how P1 amplitude and peak latency interact with exercise intensity and selective attention.

Post-perceptual processing

In addition to modulation of early perceptual processing during exercise, our data also show modulation of

neural activity associated with later cognitive processing. The P3a component evoked by distractors peaked significantly earlier during both low- and high-intensity exercise when compared to rest. The P3b component evoked by targets also demonstrated a similar trend, but did not reach statistical significance. Our data are unique in that they demonstrate how different intensities of exercise can influence post perceptual processing when compared to rest.

The P3 ERP complex is thought to reflect brain activity associated with the updating of the representation of the stimulus environment in memory (Donchin, 1981). The P3a and P3b subcomponents are considered to stem from frontal and temporal/parietal activations from generators around the temporal/parietal junction (TPJ) and the hippocampal formation (for reviews see Polich and Criado, 2006; Polich, 2007). Typically P3a and P3b exhibit different scalp topographies, with P3a peaking earlier and showing a more anterior distribution across frontal and central sites, while P3b is distributed more around parietal areas (Polich, 2007). Our P3b data do show this more typical topography and the P3a does peak significantly earlier than the P3b; however the distribution of the P3a is more posterior than anterior. This may be accounted for by the relative ease of our oddball task. P3a topography can vary as a function of attentional and task demands, such that if discrimination between targets and non-targets in a three-stimulus oddball task is easy, then both the P3a and P3b show maximal amplitude at parietal locations and the P3a response is smaller than the P3b (Katayama and Polich, 1998; Comerchero and Polich, 1999). Performance in our task was near ceiling, so the topographic distribution and amplitude of P3a and P3b are consistent with these previous findings.

P3 peak latency is considered to be an index of stimulus classification speed (Kutas et al., 1977; Magliero et al., 1984), hence the present data suggest faster classification of distractor stimuli during conditions of both low- and high-intensity exercise compared to rest. Our data are unique in that they demonstrate exercise-induced speeding of P3a, which indexes the processing of task-irrelevant distractor stimuli without the contamination by neural activity relating to the production of an overt motor response. The data suggest that arousal can increase availability of cognitive resources for suppressing the response to task-irrelevant distractor stimuli. This finding aligns with numerous other studies that demonstrate how various aspects of biological state can influence cognitive operations associated with the P3 (Polich and Kok, 1995).

Both our P3 peak latency data and response time data largely corroborate previous findings from Yagi et al. (1999), who use a two-stimulus oddball task to demonstrate earlier P3 latency and faster target response times during a brief bout of cycling exercise compared to rest. However, whereas Yagi et al. observed reduced P3b amplitude during exercise – which they suggest indicates diminished attention resource allocation – we saw no significant effects of exercise on P3a or P3b amplitude. Differences in cognitive task demands and exercise duration (our participants were exercising for a longer duration

than their participants) may explain why Yagi et al. observed amplitude modulation and we did not. Furthermore, the P3 amplitude and latency changes observed by Yagi et al. during exercise may also just reflect the speed-accuracy trade off in their data. It is also difficult to draw comparisons between the present findings and Grego et al. (2004) as they only observed P3 modulation after 1 h of exercise. Furthermore, given the drastically different task demands, it is not surprising that our data are different than those reported by Pontifex and Hillman (2007), who observed increased P3 amplitude at frontal and lateral sites, and increased latency during exercise relative to rest. Importantly, Pontifex and Hillman (2007) used a flanker task, which is far more demanding than the task used here and requires greater levels of cognitive control.

Implications for theories of exercise induced arousal and attention

The relationship between exercise and cognition is complex. It is likely that exercise type, intensity, experience and fitness levels interact with different types of information processing tasks via different physiological mechanisms to either facilitate or degrade task performance (for a review see Lambourne and Tomporowski, 2010). One hypothesis is that the improvements in relatively simple information processing tasks during a bout of moderate, steady-state exercise, are driven by increased physiological arousal and its influence on neurotransmitter systems in the brain (McMorris and Graydon, 2000; Tomporowski, 2003; Davranche and Audiffren, 2004). McMorris et al. (2009) proposed a neuroendocrinological model whereby physical exercise causes the hypothalamus to trigger catecholamine synthesis in the sympathetic-adrenal-system-axis. Increased exercise intensity then triggers the release of adrenaline and noradrenaline in the adrenal medulla, which in turn triggers catecholamine release in the brain. Increased levels of dopamine and norepinephrine are thought to influence brain networks responsible for information processing and these neurotransmitters could influence prefrontal attentional systems by altering the signal-to-noise ratio of background neural noise relative to target saliency (Mesulam, 1990). This may facilitate information processing at stages ranging from early sensory processing to response selection and execution, thus explaining speeded RTs during steady-state exercise. This hypothesis is consistent with the suggestion that arousal may govern the availability of attentional resources and that increased arousal may be an effective mechanism for suppressing the response to task-relevant stimuli (Polich, 2007). However, few attempts have been made to characterize how specific neural mechanisms associated with information processing are modulated during bouts of acute exercise.

Previous attempts to elucidate the influence of exercise-induced arousal on the brain have used behavioral measures coupled with electromyography (EMG) to test the influence of arousal on the different neural and motor stages involved in making a speeded

response to a stimulus. Converging data from choice reaction time tasks have led to the suggestion that exercise-induced arousal facilitates activation of motor responses, but not the neural information leading up to the response (Davranche et al., 2005, 2006; Audiffren et al., 2008). However, several studies demonstrate modulation of the critical flicker fusion (CFF) threshold after a bout of aerobic exercise compared to before exercise (Davranche and Audiffren, 2004; Davranche and Pichon, 2005; Davranche et al., 2005), which provides indirect evidence for modulation of early sensory processing as a function of acute exercise. Critically, the present ERP data allow direct insight into patterns of neural activity associated with processing stimuli that do require a behavioral response (targets) and stimuli that do not require a response (standards and distractors). This not only demonstrates that exercise can modulate information processing at early sensory and later stimulus classification stages, but also that these effects can be independent of motor preparation and execution activity.

In a previous paper (Bullock and Giesbrecht, 2014) we speculate that the neural theory of visual attention (NTVA; Bundesen, 1990; Bundesen et al., 2005) may account for the effects of acute exercise and aerobic capacity on visual task performance. According to NTVA, the rapid and accurate categorization of a behaviorally relevant object is dependent on the size of the neural ensemble that is available to encode that object. A greater number of neurons may mean more rapid and accurate categorization. In the current context of acute exercise-induced arousal, enhanced metabolic energy supply (Secher et al., 2008) may increase neural recruitment during exercise, meaning larger populations of neurons are available to represent the visual stimuli. In conjunction with the proposed facilitatory effects of increased levels of dopamine and norepinephrine on target salience (Mesulam, 1990), these mechanisms may account for the modulation of multiple stages of information processing demonstrated in the present study, from sensory gain to faster stimulus categorization and RTs.

CONCLUSION

In this study we used the ERP technique to demonstrate that both early sensory and later cognitive processes can be selectively modulated during bouts of low- and higher intensity cycling exercise compared to rest. The present findings are important and novel for three main reasons. First, we show modulation of neural activity by exercise at early sensory stages of processing. Second, we provide neural evidence that exercise can facilitate neural activity associated with rapid stimulus classification, independent of a motor response. Third, we demonstrate that different levels of exercise intensity can selectively influence early and later stages of information processing. Together these results converge with the current literature on exercise and cognition and, more broadly, may be explained by current cognitive information processing frameworks.

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REFERENCES

- Åstrand P-O, Ryhming I (1954) A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. *J Appl Physiol* 7:218–221.
- Audiffren M, Tomporowski PD, Zagrodnik J (2008) Acute aerobic exercise and information processing: energizing motor processes during a choice reaction time task. *Acta Psychol (Amst)* 129:410–419.
- Ayaz A, Saleem AB, Schölvinck ML, Carandini M (2013) Locomotion controls spatial integration in mouse visual cortex. *Curr Biol* 23:890–894.
- Bailey SP, Hall EE, Folger SE, Miller PC (2008) Changes in EEG during graded exercise on a recumbent cycle ergometer. *J Sports Sci Med* 7:505–511.
- Benjamini Y, Hochberg Y (1995) Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Stat Soc* 57:289–300.
- Borg G (1970) Perceived exertion as an indicator of somatic stress. *Scand J Rehabil Med* 2:92–98.
- Borg G (1982) Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 14:377–381.
- Brainard DH (1997) The psychophysics toolbox. *Spat Vis* 10:433–436.
- Brümmner V, Schneider S, Abel T, Vogt T, Strüder HK (2011a) Brain cortical activity is influenced by exercise mode and intensity. *Med Sci Sports Exerc* 43:1863–1872.
- Brümmner V, Schneider S, Strüder HK, Askew CD (2011b) Primary motor cortex activity is elevated with incremental exercise intensity. *Neuroscience* 181:150–162.
- Bullock T, Giesbrecht B (2014) Acute exercise and aerobic fitness influence selective attention during visual search. *Front Psychol* 5:1–11.
- Bundesden C (1990) A theory of visual attention. *Psychol Rev* 97:523–547.
- Bundesden C, Habekost T, Kyllingsbæk S (2005) A neural theory of visual attention: bridging cognition and neurophysiology. *Psychol Rev* 112:291–328.
- Cecotti H, Eckstein MP, Giesbrecht B (2014) Single-trial classification of event-related potentials in rapid serial visual presentation tasks using supervised spatial filtering. *IEEE Trans Neural Netw Learn Syst* 25(11):2030–2042.
- Cecotti H, Sato-Reinhold J, Sy JL, Elliott JC, Eckstein MP, Giesbrecht B (2011) Impact of target probability on single-trial EEG target detection in a difficult rapid serial visual presentation task. *Proc Annu Int Conf IEEE Eng Med Biol Soc EMBS*:6381–6384.
- Chang YK, Labban JD, Gapin JI, Etnier JL (2012) The effects of acute exercise on cognitive performance: a meta-analysis. *Brain Res* 1453:87–101.
- Clark VP, Fan S, Hillyard SA (1994) Identification of early visual evoked potential generators by retinotopic and topographic analyses. *Hum Brain Mapp* 2:170–187.
- Colcombe S, Erickson KI, Scalf PE, Kim JS, Prakash R, McAuley E, Elavsky S, Marquez DX, Hu L, Kramer AF (2006) Aerobic exercise training increases brain volume in aging humans. *J Gerontol A Biol Sci Med Sci* 61:1166–1170.
- Colcombe S, Kramer AF, Erickson KI, Scalf P, McAuley E, Cohen NJ, Webb A, Jerome GJ, Marquez DX, Elavsky S (2004) Cardiovascular fitness, cortical plasticity, and aging. *Proc Natl Acad Sci U S A* 101:3316–3321.
- Comerchero MD, Polich J (1999) P3a and P3b from typical auditory and visual stimuli. *Clin Neurophysiol* 110:24–30.
- Das K, Giesbrecht B, Eckstein MP (2010) Predicting variations of perceptual performance across individuals from neural activity using pattern classifiers. *Neuroimage* 51:1425–1437.

- Davranche K, Audiffren M (2004) Facilitating effects of exercise on information processing. *J Sports Sci* 22:419–428.
- Davranche K, Burle B, Audiffren M, Hasbroucq T (2005) Information processing during physical exercise: a chronometric and electromyographic study. *Exp Brain Res* 165:532–540.
- Davranche K, Burle B, Audiffren M, Hasbroucq T (2006) Physical exercise facilitates motor processes in simple reaction time performance: an electromyographic analysis. *Neurosci Lett* 396:54–56.
- Davranche K, Pichon A (2005) Critical flicker frequency threshold increment after an exhausting exercise. *J Sport Exerc Psychol* 27:515–520.
- Delorme A, Makeig S (2004) EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods* 134:9–21.
- Di Russo F, Martinez A, Sereno MI, Pitzalis S, Hillyard SA (2001) Cortical sources of the early components of the visual evoked potential. *Hum Brain Mapp* 11:95–111.
- Dietrich A (2003) Functional neuroanatomy of altered states of consciousness: the transient hypofrontality hypothesis. *Conscious Cogn* 12:231–256.
- Dietrich A, Sparling PB (2004) Endurance exercise selectively impairs prefrontal-dependent cognition. *Brain Cogn* 55:516–524.
- Donchin E (1981) Presidential address, 1980. Surprise!... Surprise? *Psychophysiology* 18:493–513.
- Drollette ES, Scudder MR, Raine LB, Moore RD, Saliba BJ, Pontifex MB, Hillman CH (2014) Acute exercise facilitates brain function and cognition in children who need it most: an ERP study of individual differences in inhibitory control capacity. *Dev Cogn Neurosci* 7:53–64.
- Eriksen B, Eriksen C (1974) Effects of noise letters upon the identification of a target letter in a nonsearch task. *Percept Psychophys* 16:143–149.
- Fu Y, Tucciarone JM, Espinosa JS, Sheng N, Darcy DP, Nicoll RA, Huang ZJ, Stryker MP (2014) A cortical circuit for gain control by behavioral state. *Cell* 156:1139–1152.
- Fumoto M, Oshima T, Kamiya K, Kikuchi H, Seki Y, Nakatani Y, Yu X, Sekiyama T, Sato-Suzuki I, Arita H (2010) Ventral prefrontal cortex and serotonergic system activation during pedaling exercise induces negative mood improvement and increased alpha band in EEG. *Behav Brain Res* 213:1–9.
- Gomez-Herrero G, Clercq W, Anwar H, Kara O, Egiastian K, Huffel S, Paesschen W (2006) Automatic removal of ocular artifacts in the EEG without an EOG reference channel. *Proc 7th Nord Signal Process Symp NORSIG* 2006:130.
- Grego F, Vallier J-M, Collardeau M, Berson S, Ferrari P, Candito M, Bayer P, Magnié M-N, Brisswalter J (2004) Effects of long duration exercise on cognitive function, blood glucose, and counterregulatory hormones in male cyclists. *Neurosci Lett* 364:76–80.
- Hanslmayr S, Klimesch W, Sauseng P, Gruber W, Doppelmayr M, Freunberger R, Pecherstorfer T (2005) Visual discrimination performance is related to decreased alpha amplitude but increased phase locking. *Neurosci Lett* 375:64–68.
- Hayes SM, Hayes JP, Cadden M, Verfaellie M (2013) A review of cardiorespiratory fitness-related neuroplasticity in the aging brain. *Front Aging Neurosci* 5.
- Heinze HJ, Mangun GR, Burchert W, Hinrichs H, Scholz M, Münte TF, Gös A, Scherg M, Johannes S, Hundeshagen H (1994) Combined spatial and temporal imaging of brain activity during visual selective attention in humans. *Nature* 372:543–546.
- Hillman CH, Pontifex MB, Castelli DM, Khan NA, Raine LB, Scudder MR, Drollette ES, Moore RD, Wu C-T, Kamijo K (2014) Effects of the FITKids randomized controlled trial on executive control and brain function. *Pediatrics* 134:e1063–e1071.
- Hillman CH, Snook EM, Jerome GJ (2003) Acute cardiovascular exercise and executive control function. *Int J Psychophysiol* 48:307–314.
- Hillyard SA, Vogel EK, Luck SJ (1998) Sensory gain control (amplification) as a mechanism of selective attention: electrophysiological and neuroimaging evidence. *Philos Trans R Soc Lond B Biol Sci* 353:1257–1270.
- Hockey GR (1997) Compensatory control in the regulation of human performance under stress and high workload: a cognitive-energetical framework. *Biol Psychol* 45:73–93.
- Kahneman D (1973) *Attention and Effort*. Englewood Cliffs, NJ: Prentice-Hall, Inc..
- Kasper RW, Cecotti H, Touryan J, Eckstein MP, Giesbrecht B (2014) Isolating the neural mechanisms of interference during continuous multisensory dual-task performance. *J Cogn Neurosci* 26:476–489.
- Katayama J, Polich J (1998) Stimulus context determines P3a and P3b. *Psychophysiology* 35:23–33.
- Kok A (2001) On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology* 38:557–577.
- Kubitz K, Mott A (1996) EEG power spectral densities during and after cycle ergometer exercise. *Res Q Exerc Sport* 67:91–96.
- Kutas M, McCarthy G, Donchin E (1977) Augmenting mental chronometry: the P300 as a measure of stimulus evaluation time. *Science* (80-) 197:792–795.
- Lambourne K, Audiffren M, Tomporowski PD (2010) Effects of acute exercise on sensory and executive processing tasks. *Med Sci Sports Exerc* 42:1396–1402.
- Lambourne K, Tomporowski P (2010) The effect of exercise-induced arousal on cognitive task performance: a meta-regression analysis. *Brain Res* 1341:12–24.
- Lopez-Calderon J, Luck SJ (2014) ERPLAB: an open-source toolbox for the analysis of event-related potentials. *Front Hum Neurosci* 8:213.
- Luck SJ (2014) *An introduction to the event-related potential technique*. MIT Press.
- Magliero A, Bashore TR, Coles MGH, Donchin E (1984) On the dependence of P300 Latency on stimulus evaluation processes. *Psychophysiology* 21:171–186.
- Mangun GR, Hillyard SA (1991) Modulations of sensory-evoked brain potentials indicate changes in perceptual processing during visual-spatial priming. *J Exp Psychol Hum Percept Perform* 17:1057–1074.
- McMorris T, Graydon J (2000) The effect of incremental exercise on cognitive performance. *Int J Sport Psychol* 31:66–81.
- McMorris T, Tomporowski PD, Audiffren M (2009). In: McMorris T, Tomporowski PD, Audiffren M, editors. *Exercise and Cognitive Function*. Hoboken, NJ: John Wiley & Sons Ltd.. p. 41–68.
- Mesulam MM (1990) Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Ann Neurol* 28:597–613.
- Niell CM, Stryker MP (2010) Modulation of visual responses by behavioral state in mouse visual cortex. *Neuron* 65:472–479.
- Nybo L, Nielsen B (2001) Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *J Appl Physiol* 91:2017–2023.
- Polich J (1987) Task difficulty, probability, and inter-stimulus interval as determinants of P300 from auditory stimuli. *Electroencephalogr Clin Neurophysiol* 68:311–320.
- Polich J (2007) Updating P300: an integrative theory of P3a and P3b. *Clin Neurophysiol* 118:2128–2148.
- Polich J, Criado JR (2006) Neuropsychology and neuropharmacology of P3a and P3b. *Int J Psychophysiol* 60:172–185.
- Polich J, Kok A (1995) Cognitive and biological determinants of P300: an integrative review. *Biol Psychol* 41:103–146.
- Pontifex MB, Hillman CH (2007) Neuroelectric and behavioral indices of interference control during acute cycling. *Clin Neurophysiol* 118:570–580.
- Saleem AB, Ayaz A, Jeffery KJ, Harris KD, Carandini M (2013) Integration of visual motion and locomotion in mouse visual cortex. *Nat Neurosci* 16:1864–1869.
- Secher NH, Seifert T, Van Lieshout JJ (2008) Cerebral blood flow and metabolism during exercise: implications for fatigue. *J Appl Physiol* 104:306–314.
- Talsma D, Kok A (2001) Nonspatial intermodal selective attention is mediated by sensory brain areas: Evidence from event-related potentials. *Psychophysiology* 38:736–751.
- Themanson JR, Hillman CH (2006) Cardiorespiratory fitness and acute aerobic exercise effects on neuroelectric and behavioral measures of action monitoring. *Neuroscience* 141:757–767.

- Tomprowski PD (2003) Effects of acute bouts of exercise on cognition. *Acta Psychol (Amst)* 112:297–324.
- Troje NF, Bühlhoff HH (1996) Face recognition under varying poses: the role of texture and shape. *Vision Res* 36:1761–1771.
- Tsai C-L, Chen F-C, Pan C-Y, Wang C-H, Huang T-H, Chen T-C (2014) Impact of acute aerobic exercise and cardiorespiratory fitness on visuospatial attention performance and serum BDNF levels. *Psychoneuroendocrinology* 41:121–131.
- Van Voorhis S, Hillyard SA (1977) Visual evoked potentials and selective attention to points in space. *Percept Psychophys* 22:54–62.
- Vogel EK, Luck SJ (2000) The visual N1 component as an index of a discrimination process. *Psychophysiology* 37:190–203.
- Woldorff MG (1993) Distortion of ERP averages due to overlap from temporally adjacent ERPs: analysis and correction. *Psychophysiology* 30:98–119.
- Yagi Y, Coburn KL, Estes KM, Arruda JE (1999) Effects of aerobic exercise and gender on visual and auditory P300, reaction time, and accuracy. *Eur J Appl Physiol Occup Physiol* 80:402–408.
- Yeung N, Botvinick MM, Cohen JD (2004) The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychol Rev* 111:931–959.

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