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# Acute cardiovascular exercise and executive control function

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#### Abstract

Acute cardiovascular exercise effects on cognitive function were examined using an executive control task by comparing neuroelectric and behavioral performance at baseline with post-exercise in 20 undergraduates. A withinsubjects design was used to assess the P3 component of an event-related brain potential (ERP) and behavioral performance using a task that varied the amount of executive control required. The baseline session involved participation on the Eriksen flankers task followed by a graded maximal exercise test to measure cardiovascular fitness. The exercise session consisted of a 30-min acute bout of exercise on a treadmill followed by the Eriksen flankers task after heart rate returned to within 10% of pre-exercise levels. Across midline recordings sites, results indicated larger P3 amplitude following acute exercise compared to baseline. Shorter P3 latency was observed during the baseline Eriksen flankers task for the neutral compared to the incompatible condition; an effect not found following the acute bout of exercise. These findings suggest that acute bouts of cardiovascular exercise affect neuroelectric processes underlying executive control through the increased allocation of neuroelectric resources and through changes in cognitive processing and stimulus classification speed. © 2003 Elsevier Science B.V. All rights reserved.

Keywords: Acute exercise; Cardiovascular exercise; Executive control; Cognition; P3; ERPs; Eriksen flankers task

# 1. Introduction

It has been widely accepted for many years that physical activity, and in particular cardiovascular exercise, promotes health and effective functioning (Kesaniemi et al., 2001; US Department of Health and Human Services, 2000). More specifically, Chodzko-Zajko (1991) suggested that exercise increases the efficiency of effortful cognitive processes, despite the fact that it may not influence overall intellectual functioning. Accordingly, automatic processes, or processes that are less effortful, are unaffected by exercise, since there is presumably little need for increased efficiency in these environments. Alternatively, tasks that require conscious control and increased effort, such as executive control processes, would be most affected. Executive control processes are thought to be functionally distinct, resource limited, and associated with conscious awareness (Rogers and Monsell, 1995). Extending Chodzko-Zajko (1991) notions, it is these processes that may benefit the

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most from physical activity due to the increased processing demands.

Norman and Shallice (1986) proposed a theoretical model of executive control that assumes a top-down approach to cognitive processing and action execution such that multiple subsystems interact and are controlled by two distinct mechanisms: contention scheduling and the supervisory attentional system. Contention scheduling is the lower-order level of control that automatically selects action schemas to execute well-learned processes, while the supervisory attentional system arbitrates by providing increased inhibition or activation to competing schemas when contention scheduling does not result in the emergence of a dominant action schema (Norman and Shallice, 1986). Hence, only certain environmental situations require extensive amounts of executive control.

Stimulus evaluation tasks have long been employed to study executive control using numerous paradigms that require participants to make a decision and/or execute a response. One such paradigm, the Eriksen flankers task (Eriksen and Eriksen, 1974), requires participants to discriminate between two letters that are flanked by an array of other letters, which have different action schemas associated with them. Congruent stimuli (e.g. HHHHH) elicit faster and more accurate responses, and incongruent stimuli (e.g. HHSHH) cause decreased response speed and accuracy (Eriksen and Schultz, 1979). This latter condition requires greater amounts of executive control since incongruent arrays result in response delay due to activation of the incorrect response (elicited by the flanker stimuli) before evaluation is completed, and this response competes with the correct response that is elicited by the target stimulus (Kramer et al., 1994: Kramer and Jacobson, 1991: Spencer and Coles, 1999).

To date, only two studies have examined the influence of an acute bout of exercise on cognitive processing using event-related potentials (ERP), neither of which employed tasks requiring extensive amounts of executive control. Nakamura et al. (1999) examined variations in P3 using an acoustic oddball paradigm after an acute bout of self-paced jogging and found increased amplitude at the Cz and Pz electrode sites, and no differences at Fz, when compared to baseline. Further, using the same paradigm, Magnié et al. (2000) measured changes in the P3 and N4 components before and after a maximal graded exercise test. Relevant to the present report were increased amplitude and decreased latency for P3 during the postexercise compared to the pre-exercise testing sessions. Magnié et al. (2000) suggested that the exerciseinduced differences in P3 were indicative of an overall increase in central nervous system arousal since differences were observed across all midline (i.e. Fz, Cz, Pz) recording sites. Polich and Lardon (1997) provided a theoretical rationale suggesting that physical activity affects neuroelectric performance through a general increase in biological arousal, and draw from empirical evidence indicating that P3 amplitude increases in a global fashion following physical activity.

The present study was designed to extend previous research by examining the effects of an acute bout of cardiovascular exercise on the P3 potential using an executive control task. It was hypothesized that increased P3 amplitude and decreased latency would be observed following exercise relative to baseline. Based on research by Polich and Lardon (1997) and Nakamura et al. (1999) it was predicted that the most robust differences would be observed at the Cz and Pz sites, indicating a more specific, rather than global, effect of exercise on P3 distribution.

# 2. Method

# 2.1. Participants

Twenty right-handed participants (10 males, 10 females) were recruited from undergraduate kinesiology courses at the University of Illinois at Urbana-Champaign. All participants were nonsmoking, reported no adverse health conditions, and participated in regular physical activity. Although an attempt was made to obtain individuals that varied in their physical activity participation, the majority of the sample was moderate to highly active. Thus, the data presented herein may not generalize to other populations such as sedentary individuals. Data from one female par-

Variable	Total $(n=19)$	Male $(n=10)$	Female $(n=9)$
Age	20.5 (0.5)	20.9 (0.5)	20.2 (1.0)
Height (cm)	172.3 (2.6)	181.2 (2.6)	163.8 (2.2)
Weight (kg)	67.5 (3.7)	73.5 (4.3)	61.3 (6.2)
$VO_2 \max (ml/kg/min)$	48.4 (3.0)	54.8 (3.9)	42.8 (3.9)

ticipant were discarded due to technical problems. Table 1 provides participants' descriptive data and cardiovascular exercise history.

# 2.2. Procedure

Participants visited the lab at the same time of day on two occasions with a maximum of seven days between sessions. The baseline session consisted of measuring participants' ERPs and behavioral responses during the Eriksen flankers task, followed by a graded exercise stress test (GXT) to measure participants' VO2 max, which is considered to be the criterion measure for cardiorespiratory fitness (American College of Sports Medicine, 2000). The exercise session began with a 30-min bout of self-paced treadmill exercise, followed by completion of the flankers task after participants' heart rate (HR) returned to within 10% of baseline (pre-exercise) level (M=48 min post exercise, S.E. = 1.9). Participants were instructed to exercise at a pace that was somewhat hard to hard on the ratings of perceived exertion scale (Borg, 1970). Mean HR for this intensity was 162.4 bpm (S.E. = 3.6 bpm), or 83.5% of maximal HR achieved on the GXT, confirming that participants were exercising at a hard, yet submaximal, level. Counterbalancing of the baseline and exercise sessions occurred across participants to minimize potential practice effects, and participants were asked not to exercise on the days they visited the laboratory.

# 2.3. Task

Incompatible and neutral conditions of the Eriksen flankers task (Eriksen and Eriksen, 1974) required participants to respond as quickly as possible to a centrally presented target letter. When 'F' was the target stimulus, participants responded with their left index finger. When 'X' was the target stimulus, a right index finger response was required. The incompatible condition had the target response flanked by the opposing target stimulus (i.e. FXF or XFX). The neutral target response was flanked by letters with no response assignment (e.g. LFL, LXL). The two conditions were equiprobable, and stimuli consisted of black letters on a white background, with each letter measuring 2.5 cm in height and 1.8 cm in width. Five blocks of 144 trials were presented for 500 ms with a 1500 ms inter-stimulus interval.

## 2.4. Data collection and reduction

The GXT was conducted on a treadmill using a modified Bruce protocol (Dengel et al., 1994). A computerized indirect calorimetry system collected 30-s averages for oxygen uptake ( $VO_2$ ) and respiratory exchange ratio (RER). All participants achieved  $VO_2$  max as defined by meeting two out of the three following criteria: RER>1.1, agepredicted maximum HR obtained, or an observed plateau in  $VO_2$  despite an increase in workload.

Linked mastoid-referenced electroencephalograms were measured from the Fz, Cz, Pz and Oz sites. AFz served as the ground electrode and electro-oculographic activity was collected from electrodes placed above and below the right orbit and on the outer canthus of each eye to assess bipolar eye movement artifacts. Impedance values for all electrodes were  $\leq 10$  k ohms.

The Semlitsch et al. (1986) algorithm was used to correct for vertical and horizontal eye movement artifacts. Epochs were created from continuous data and baseline corrected using the 100-ms prestimulus period. Data were then filtered using a 30-Hz low pass cutoff frequency (24 dB/octave). Artifact detection excluded trials containing amplitude excursions of  $\pm 100 \ \mu$ V and artifact-free data that were accompanied by correct responses were averaged. P3 was defined as the largest positive-going peak within a 250–500 ms latency window. Amplitude was measured as a change score from the pre-stimulus baseline and peak latency was defined as the time point of the maximum amplitude.

### 3. Statistical analysis

Separate  $2 \times 2 \times 4$  (Exercise  $\times$  Compatibility  $\times$  Site) ANOVAs with repeated measures were conducted on P3 amplitude and latency data. Behavioral data were analyzed using  $2 \times 2$  (Exercise  $\times$  Compatibility) ANOVAs with repeated measures, and all tests utilized the Greenhouse–Geisser correction. When appropriate, follow up analyses were conducted using univariate ANO-VAs and paired samples *t* tests with Bonferroni correction.

# 4. Results

#### 4.1. Behavioral data

Reaction time (RT) analyses revealed a main effect for compatibility, F(1, 18) = 34.5, P < 0.001,  $\Phi = 1.0$ , with the incompatible condition (M = 470.8, S.E. = 14.4) yielding longer RT compared to the neutral condition (M = 455.8, S.E. = 15.7). Response accuracy analyses indicated a significant main effect for compatibility, F(1, 18) = 23.8, P < 0.001,  $\Phi = 0.97$ , with more accurate responses for the neutral (M = 96.0, S.E. = 0.8) compared to the incompatible (M = 92.3, S.E. = 1.3) condition.

# 5. P3 Amplitude

P3 waveforms are depicted in Fig. 1. The omnibus analysis revealed a main effect for Exercise, F(1,18)=5.3, P<0.05,  $\Phi=0.58$ , with increased amplitude for the exercise compared to the baseline session (Exercise: M=11.5, S.E.=0.92; Baseline: M=9.7, S.E.=0.69). A Compatibility×Site interaction was also observed, F(2.5, 45.8)=6.4, P<0.002,  $\Phi=0.93$ , with follow up analyses indicating that only the Pz and Oz sites

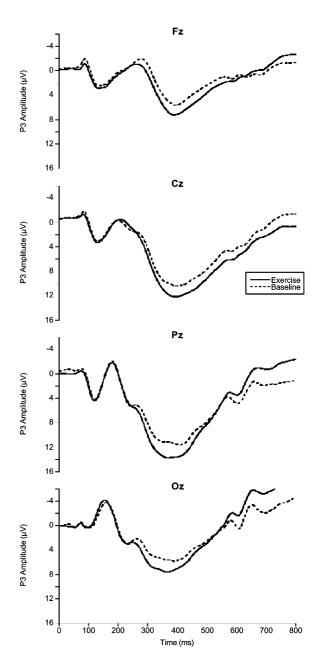


Fig. 1. P3 waveforms for the exercise and baseline sessions at each of the midline electrode sites averaged across incompatible and neutral conditions of the Eriksen flankers task.

yielded significant compatibility effects, t's(1, 18)=3.2,  $P \le 0.005$ , with increased amplitude for the neutral compared to the incompatible condition

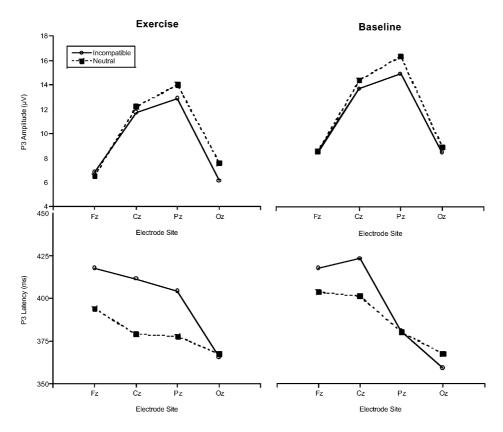


Fig. 2. Mean P3 amplitude and latency for incompatible and neutral conditions of the Eriksen flankers task during exercise and baseline sessions across electrode sites.

(see Fig. 2). Additionally, main effects for Compatibility, F(1, 18) = 6.4, P < 0.025,  $\Phi = 0.67$ , and Site, F(3, 35.1) = 24.0, P < 0.001,  $\Phi = 1.0$ , revealed increased amplitude for the neutral compared to the incompatible condition of the flankers task, and reduced amplitude at Fz and Oz relative to the Cz and Pz sites, t's(1, 19) > 3.9,  $P \le 0.001$  (see Fig. 2).

In addition to the primary analysis, a second Exercise×Compatibility×Site analysis was conducted that included the McCarthy and Wood (1985) normalization procedure, which standardizes differences in the topographic distribution. Topographic results were similar to the primary analysis, such that the Compatibility×Site interaction, F(2.3, 41)=4.6, P=0.01,  $\Phi=0.79$ , and Site main effect, F(1.8, 33.7)=26.8, P<0.001,  $\Phi=1.0$ , were observed. Post hoc analyses for the

Compatibility × Site interaction revealed a significant Compatibility effect only at the Fz site, t(1, 18) = 3.4, P = 0.003, with increased amplitude for the incompatible compared to the neutral condition. In addition, after Bonferroni correction the Compatibility effect approached significance at the Pz site (P = 0.025) with increased amplitude for the neutral compared to the incompatible condition. Follow up analyses for the Site main effect yielded similar findings to the primary analysis with increased amplitude at Cz and Pz relative to the Fz and Oz sites, t(1, 18) > 4.6, P < 0.001.

# 6. P3 Latency

Fig. 3 shows the Exercise × Compatibility interaction, F(1, 18) = 4.4, P = 0.05,  $\Phi = 0.51$ , with post hoc analyses indicating longer latency for the

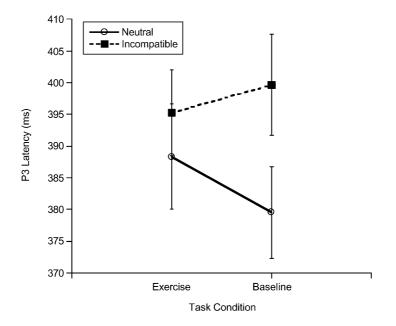


Fig. 3. Mean P3 latency for the neutral and incompatible conditions of the Eriksen flankers task during the exercise and baseline sessions.

incompatible relative to the neutral condition during the baseline session, t(1, 18) = 4.8, P < 0.001; an effect not found for the exercise session (P = 0.213). Additionally, main effects for Compatibility, F(1, 18) = 13.6, P = 0.002,  $\Phi = 0.94$ , and Site, F(1.9, 35.0) = 12.0, P < 0.001,  $\Phi = 0.99$ , were found with shorter latency for the neutral compared to the incompatible condition, and at Pz and Oz relative to the Fz and Cz, t's(1, 18) > 2.1,  $P \le 0.05$ .

# 7. Discussion

A consistent picture is beginning to emerge when the current findings are considered along with the two previous neuroelectric studies (Magnié et al., 2000; Nakamura et al., 1999) of acute exercise effects on information processing. Specifically, exercise was found to increase the amplitude of P3, suggesting that acute bouts of cardiovascular activity may be beneficial to cognitive functioning, and in particular, executive control processes. Contemporary theories of P3 suggest that the amplitude of this component reflects allocation of attention and context updating of working memory resources (Donchin and Coles, 1988), and has also been shown to be proportional to the amount of resources allocated to a particular task or stimulus (Wickens et al., 1983). As such, the current findings imply that acute bouts of cardiovascular exercise may facilitate the allocation of attentional and memory resources, and hence, benefit executive control function.

Magnié et al. (2000) provided an interpretation for the observed increases in P3 amplitude by suggesting that acute exercise facilitates cognitive processing via a general arousal effect. Similarly, Polich and Kok (1995) have suggested that P3exercise effects occur in a global fashion related to increases in general arousal, since variations in scalp topography were not observed following 'fluctuations in biological state'. The current data provide support for this notion since significant increases in P3 amplitude were observed across all sites.

P3 latency, which has been shown to reflect cognitive processing and classification speed (Duncan-Johnson, 1981), was also affected by acute exercise. However, these findings appear to be at odds with the interpretation of a general

arousal effect of exercise, because shorter P3 latencies were only observed for the incompatible condition. In fact, P3 latency for the neutral condition showed the opposite effect, as longer latencies were observed following exercise. Thus, as initially suggested by Chodzko-Zajko (1991), effortful or attentionally demanding tasks may be more sensitive to the beneficial effects of exercise compared to tasks that require minimal effort. Alternatively, given the increase in P3 latency for the neutral condition, it appears that tasks requiring minimal effort may be adversely affected by the rigors of an acute bout of cardiovascular exercise. Clearly, further examination is warranted using tasks that vary in difficulty to better determine the specificity of the relationship of acute exercise and cognitive processing.

Polich and Lardon (1997) have theorized that the effects of exercise on ERPs may be based in the fundamental changes observed in resting EEG that result from aerobic activity. As mentioned earlier, Polich and Kok (1995) reviewed previous P3 literature and convincingly established a relationship between fluctuations in biological state and the P3 component. In support of this notion, Kubitz and colleagues (Kubitz and Mott, 1996; Kubitz and Pothakos, 1997) found increased alpha activation following acute bouts of cardiovascular exercise and suggested that exercise may serve to increase neuronal synchrony. Further, Polich (1997) has shown that inter-participant variations in alpha power are related to individual variability in the P3 potential, indicating that changes in resting EEG activity may directly influence ERPs. Accordingly, exercise may help to increase alpha activity, which in turn, increases P3 amplitude and decreases P3 latency. Support for the relationship between the P3 component and resting EEG alpha has been reported (Bashore, 1989; Lardon and Polich, 1996). Less clear though, is the relationship between exercise and changes in alpha activity. Dustman et al. (1990) surmised that aerobic exercise promotes increased cerebral blood flow and draw from animal models, which have indicated that exercise improved neurotransmitter function and cerebral vascularization among other neurobiological changes. Additionally, Isaacs et al. (1992) observed significantly shorter diffusion distances from blood vessels in the cerebellum of rats in response to exercise training. Evidence from these studies suggests that despite the global arousal effects associated with exercise (i.e. increased blood flow), specific changes at the neurobiological level may also occur. Although the underlying mechanisms involved in exercise effects on neuroelectric performance still remain elusive, the above research pointing to increased vascularization and cerebral blood flow remain a promising direction for future research.

Lastly, exercise effects on behavioral measures were not as apparent as the neuroelectric results. Specifically, no relationship was observed between exercise and RT or accuracy. Although Spirduso (1980) reported improvements in simple and choice RT tasks as a function of physical activity participation in older adults, other research has failed to establish this relationship (e.g. Hillman et al., 2002; Kramer et al., 1999). Thus, additional research into the effects of physical activity participation on behavioral responses to cognitive tasks is necessary to elucidate this relationship.

# 8. Conclusions

Extending previous research, acute bouts of cardiovascular exercise were found to effect executive control function on the neuroelectric level. Mixed support was found for the general arousal interpretation of exercise-induced effects on P3, since amplitude findings supported this notion, but latency findings were inconclusive. The current results exemplify the need for a more sophisticated approach to understanding the relationship between exercise and cognitive function and provide a basis in which to further explore the use of exercise as a potential tool for maintaining mental health.

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