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Acute aerobic exercise effects on event-related brain potentials

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The study of acute exercise effects on neurocognitive function has grown in interest over recent decades, largely due to an increasing focus on the relationship between health behaviours and brain and cognition. For example, recent evidence suggests that aerobic fitness is related to better academic performance on standardized tests in gradeschool children (Castelli *et al.*, 2007). Other research has indicated that adults are able to better engage and classify information in their stimulus environment and process this information more quickly following acute aerobic exercise (Hillman, Snook and Jerome, 2003). Such findings have generated interest in the amount, intensity and duration of exercise necessary to improve cognitive function, with the hope of determining a practical application for improvements in cognitive performance.

Examination of the neuroelectric system has also become increasingly popular in the investigation of acute exercise effects on cognition because the various measures provide a more sensitive means of determining which underlying processes are affected by acute exercise, beyond that of overt action. That is, the temporal sensitivity of neuroelectric measurement allows for the investigation into a sub-set of processes that occur between stimulus encoding and response production. As such, these measures enable researchers to more precisely gauge the effects of acute exercise on cognition relative to task performance measures. Accordingly, this approach allows for an increased understanding of the means by which acute exercise improves cognitive health and effective function.

The purpose of this chapter is to describe a small body of research focused on the effects of acute, aerobic exercise on an aspect of the neuroelectric system, known as event-related brain potentials (ERPs). The scope will be further limited

1 to endogenous aspects of the neuroelectric system; that is processes independent
2 of the physical properties of the stimulus environment that are driven by internal
3 cognitive states. We will not engage in lengthy discussion of the various
4 theoretical perspectives since these viewpoints are described earlier in this
5 volume (see Chapters 1 and 3). Rather, the purpose herein is descriptive with
6 the goal of forming a cohesive understanding of a body of literature that
7 investigates acute exercise effects on aspects of the neuroelectric system. We
8 will, however, engage in discussion of executive control because it provides a
9 guiding framework for what is, at times, a seemingly disparate body of literature.

10 For example, at first glance, two similar studies yield contrasting findings
11 (Pontifex and Hillman, 2007; Yagi *et al.*, 1999); however, upon deeper inspection
12 the disparate findings are understandable and may even be predicted because
13 they require different cognitive processes that involve different neural networks.
14 Unfortunately, as discussed at the end of this chapter, often studies investigating
15 acute exercise effects on the neuroelectric system do not consider how the
16 relationship will be affected by the cognitive task employed. That is, considerable
17 thought is given to the exercise intervention, while considerably less thought is
18 given toward the aspect of cognitive function and the supporting brain tissue
19 involved in the sub-set of processes studied. Several well-written reviews de-
20 scribing how the nature of the exercise stimulus affects cognition have been
21 published previously (Kamijo, in press; Tomporowski, 2003b), but arguably
22 these reviews do not engage in an in-depth discussion of the cognitive neurosci-
23 ence involved in the various tasks employed. The goal of this chapter is to turn
24 our attention toward the neurocognitive processes affected by acute exercise.
25 Thus, one limitation of this chapter is that the aerobic exercise intervention will
26 not be considered in great detail, despite the fact that the interventions differ
27 considerably across studies.

28 Accordingly, research examining neuroelectric correlates of exercise and cogni-
29 tion have proceeded in two directions. The majority of studies have employed
30 simple stimulus discrimination tasks, which are described below in detail. Although
31 these tasks are well-grounded in previous neuroelectric literature and have pro-
32 vided insight into exercise effects on cognition, they have provided a fairly limited
33 understanding of the underlying neural networks that support cognitive change.
34 Alternatively, empirical investigations in our laboratory examined a selective
35 aspect of cognition referred to as 'executive control' or 'cognitive control'. These
36 investigations have been further aimed at a selective aspect of executive control,
37 known as 'interference control'. Thus, through the standardization of cognitive
38 tasks and measurement techniques, we are able to draw reasonable inferences that
39 are linked to a specific neural network that supports the sub-set of processes
40 involved in interference control. Through our study of the neuroelectric system, we
41 are able to better elucidate that those processes are influenced by acute exercise and
42 make inferences based on neuroimaging research regarding the brain tissue
43 underlying these processes.
44

8.1 Executive control

Executive control describes a sub-set of cognitive processes associated with the selection, scheduling and coordination of the computational processes that are responsible for perception, memory and action (Meyer and Kieras, 1997; Norman and Shallice, 1986). Executive processes are functionally distinct from the processes they organize and require conscious awareness (Rogers and Monsell, 1995), which prevents those tasks requiring executive control processes from becoming automatic or habituating over time.

Within the framework of executive control is a sub-set of executive processes responsible for adjustments in perceptual selection, biasing of responses and on-line maintenance of contextual information often referred to as cognitive control (Botvinick *et al.*, 2001). These cognitive control processes are often associated with a 'central executive' in many theoretical models (Baddeley, 1996a; Norman and Shallice, 1986) and this executive is responsible for the adaptability of the cognitive system. The cognitive control processes underlying the adaptability of the cognitive system are not unitary and need to be understood, not only in terms of what increased control does to an individual's interaction with the environment, but also how increased control is recruited and implemented under the appropriate circumstances. To address these issues, researchers have suggested that there are at least two dissociable systems of cognitive control termed 'regulative' and 'evaluative' (see Botvinick *et al.*, 2001 for review).

The regulative system exerts top-down control during ongoing information processing. That is, flexible adjustments in strategic support are provided for task-relevant interactions with the stimulus, allowing for improved attentional maintenance of task demands and representations. Thus, this is the aspect of cognitive control related to how alterations in top-down support alter an individual's behaviour and the quality of their environmental interaction. Available neuroimaging research indicates that this regulative support is likely provided, at least in part, by the dorsolateral prefrontal cortex (DLPFC) (MacDonald *et al.*, 2000).

The evaluative system of cognitive control monitors for instances of behavioural conflict during information processing. More specifically, the evaluative system detects the occurrence of conflict during cognitive processing of environmental information and sends signals to the processing centres responsible for compensatory adjustments of top-down control necessary to successfully adapt to specific task demands (Botvinick *et al.*, 2001). Neuroimaging research has suggested that the anterior cingulate cortex (ACC) is involved in the evaluative system of cognitive control and the signalling/detection of conflict. Most notably, results indicate that ACC activation is largest when cognitive control is weak and conflict is at its greatest due to specific task demands or constraints (Botvinick *et al.*, 1999; Carter *et al.*, 2000; Yeung, Botvinick and Cohen, 2004). Further, increased ACC activation in conjunction with weak cognitive control may indicate the increased signalling for adjustments in control to more strongly engage the strategic processes necessary

to improve subsequent behavioural interactions with the environment (MacDonald *et al.*, 2000).

Thus, the two components of cognitive control interact as part of a feedback loop to optimize task performance (MacDonald *et al.*, 2000). In this loop, the DLPFC implements cognitive control for novel or complex task-related behaviours and the ACC monitors for conflict during task execution. If conflict is present, the ACC signals for an up-regulation of cognitive control. The DLPFC responds to the conflict signal from the ACC with an adaptive adjustment to increase the engagement of control during task execution in an attempt to improve subsequent behavioural interactions with the environment.

8.2 Neuroelectric measurement

Although behavioural measures of performance have been useful in evaluating aspects of cognition, the evaluation of ERPs has provided additional insight into underlying mechanisms that occur during cognitive operations. ERPs refer to a class of electroencephalographic activity that occurs in response to, or in preparation for, a stimulus or response (Coles, Gratton and Fabiani, 1990). This neuroelectric activity is reflective of the synchronous activity of large populations of neurons (Hugdahl, 1995). ERPs can be obligatory responses (exogenous) or reflect higher-order cognitive processing that often requires active participation from the subject (endogenous) (Hugdahl, 1995). The stimulus-locked ERP is characterized by a succession of positive (P) and negative (N) components, which are constructed according to their direction and the relative time that they occur (i.e. N1-P2-N2-P3) (Hruby and Marsalek, 2003). Among the various stimulus-locked ERPs, the P3 has captured considerable attention because it is easily identifiable and has been linked to various cognitive processes during a variety of cognitive tasks. For more extensive descriptions of the stimulus-locked ERPs, the interested reader is referred to reviews by Coles and Rugg (1995) and Coles, *et al.* (1990).

P300

Originally discovered over four decades ago by Sutton *et al.* (1965), the P399 (also referred to as the P3) is a positive-going waveform that peaks approximately 300–800 ms after stimulus onset. This endogenous component reflects neuronal activity underlying basic cognitive functions such as attentional resource allocation (Donchin, 1981; Polich and Kok, 1995). The P300 is considered a cognitive neuroelectric phenomenon as it is elicited in tasks that require the participant to attend to and discriminate between different stimuli (Polich and Kok, 1995). The P300 (and other ERP components) may be decomposed according to amplitude and latency.

P300 amplitude, which is usually measured as the change in voltage from pre-stimulus baseline activity to the largest positive peak after the N1-P2-N2 complex, generally shows a spatial scalp distribution with increasing amplitude from frontal

to parietal electrode sites and lateral to midline electrode sites in young adults (Polich and Kok, 1995). P300 amplitude is reflective of the amount of attentional resources allocated towards a stimulus or task, with increased amplitude reflecting increased attention (Kok, 2001; Polich, 1987; Polich and Heine, 1996). P300 latency, which is defined as the time from stimulus onset to the maximum positive amplitude within a specified latency window (Polich and Kok, 1995), is considered to be a measure of stimulus identification and classification speed (Kutas, McCarthy and Donchin, 1977; Magliero *et al.*, 1984) that is independent of response selection processes (Duncan-Johnson, 1981; McCarthy and Donchin, 1981). It should be noted that shorter latencies are related to superior cognitive performance as P300 latency is negatively correlated with mental function (Emmerson *et al.*, 1989; Howard and Polich, 1985; Johnson, Pfefferbaum and Kopell, 1985; Polich and Martin, 1992; Polich, Howard and Starr, 1983).

Much of the literature on exercise-induced changes in cognitive function has focused on task performance measures (i.e. response speed and accuracy) to gain an understanding of the effects of exercise on behaviour. However, a growing number of researchers are turning towards neuroelectric measures, and particularly the P300 component, to measure underlying neurocognitive function as a means of elucidating why exercise influences behaviour. This approach has become particularly popular with regards to examining chronic physical activity behaviours and cardiorespiratory fitness (Hillman, Buck and Themanson, in press).

8.3 Event-related brain potentials during exercise

Relatively few studies have attempted to examine ERPs to assess underlying changes in cognitive function during acute exercise (i.e. Yagi *et al.*, 1999; Grego *et al.*, 2004; Pontifex and Hillman, 2007), with only a single study examining other ERP components in addition to the P300-ERP (Pontifex and Hillman, 2007). Table 8.1 provides a brief description of the existent literature on the changes in ERP function during acute exercise. Two of these studies have examined the P300 using various versions of the oddball task (i.e. Yagi *et al.*, 1999; Grego *et al.*, 2004). This task requires participants to discriminate between two stimuli with differing probabilities and to respond selectively to the infrequent stimulus, while ignoring the more frequent stimulus. That is, through instruction, a behavioural response is mapped to the infrequent (target) stimulus, while no response is mapped to the frequent (nontarget) stimulus. Finally, only a single study has attempted to assess changes in executive control function during acute exercise (Pontifex and Hillman, 2007).

Despite the fact that both Yagi *et al.* (1999) and Grego *et al.* (2004) have used similar tasks to assess stimulus discrimination during acute aerobic exercise, they have provided conflicting results, since one study reported a reduction in P300 amplitude and shorter P300 latency during exercise (Yagi *et al.*, 1999), while the other reported an increase in P300 amplitude and longer P300 latency during exercise (Grego *et al.*, 2004). Specifically, Yagi *et al.* (1999) examined changes in stimulus discrimination

Table 8.1 Summary of studies performed to assess the effects of acute in-task exercise on neuroelectric indices of cognition.

Author(s)	n	Time of test	Exercise intervention	Cognitive task	Result
Grego <i>et al.</i> (2004)	12 trained cyclists	Pretest, 3 min, 36 min, 72 min, 108 min, 144 min, immediately post, 15 min post.	180 min cycling at approximately 66% of $\dot{V}O_{2max}$.	Auditory oddball task	Increase in P3 amplitude at minutes 72 and 108, which remained elevated through the 144th minute. Steady increase in P3 latency during exercise, with significantly longer latency at 108 and 144 min following the start of the acute bout.
Pontifex and Hillman (2007)	41 college students	During and rest	6.5 min cycling at 60% HRmax	Modified flanker task	Decrease in N1 amplitude at parietal sites, increased P2 amplitude at frontal and central sites, reduction in N2 amplitude at all sites, and increased P3 amplitude at frontal and bilateral sites during exercise relative to rest. Longer N2 and P3 latencies during exercise relative to rest.
Yagi <i>et al.</i> (1999)	24 college students	Pre-test, during, and post-test	10 min cycling at an HR between 130 and 150 bpm.	Auditory and visual oddball tasks	Decrease in P3 amplitude and shorter P3 latency during exercise relative to a pre-test baseline.

1 during acute, aerobic exercise using both auditory (1000 Hz nontarget tone, 2000 Hz
2 target tone) and visual (white 'X' nontarget stimulus, white 'O' target stimulus)
3 oddball tasks presenting nontarget and target stimuli at a ratio of 80 : 20, respectively.
4 As mentioned above, they observed a reduction in P300 amplitude and shorter P300
5 latency during both oddball tasks while participants exercised on a cycle ergometer at
6 a heart rate between 130 and 150 bpm relative to a resting, baseline session. Given that
7 P300 amplitude to an oddball task is an index of resource capacity (Kok, 2001) and is
8 related to attentional allocation during working memory operations involved in
9 contextual updating of the stimulus environment, and P300 latency is related to
10 stimulus evaluation and classification speed, their results suggest that, regardless of
11 stimulus modality, individuals exhibit a reduced capacity to allocate attentional
12 resources, but a facilitation of cognitive processing speed during exercise (Yagi *et al.*,
13 1999). Stated another way, Yagi *et al.* (1999) data indicate that individuals have a
14 smaller capacity to allocate attentional resources, but process environmental infor-
15 mation more quickly while they are engaged in aerobic exercise. Taken together, we
16 might speculate that these measures suggest a cognitive strategy aimed at empha-
17 sizing the speed of stimulus acquisition at the cost of the quality of stimulus
18 engagement. Given that task performance did not change, it is not possible to know
19 whether these P300 changes were facilitative, reflecting neuroelectric efficiency, or
20 whether exercise was detrimental to individuals' cognitive capacity to engage
21 changes in the stimulus environment.

22 Alternatively, Grego *et al.* (2004) attempted to examine changes in stimulus
23 discrimination during long duration, acute, aerobic exercise at approximately 66%
24 of their maximum volume of oxygen uptake ($\text{VO}_{2\text{max}}$) using an auditory oddball task
25 (1000 Hz nontarget tone, 2000 Hz target tone), which presented nontarget to target
26 stimuli at a ratio of 80:20, respectively. During acute exercise Grego *et al.* (2004)
27 observed an increase in P300 amplitude after an hour of exercise (at minutes 72 and
28 108), which remained elevated through to the 144th minute. P300 latency increased
29 steadily during the exercise session, and became significantly longer at minutes 108
30 and 144. However, it should be noted that the authors chose to assess changes across
31 time relative to the third minute of exercise, rather than relative to the nonexercise pre-
32 test. It is not readily apparent why a time point that occurred after the initiation of
33 exercise was used for comparison; particularly as inspection of the means indicate that
34 P300 latency increased approximately 51 ms between the pre-test and the third minute
35 of the exercise intervention, 111 ms from pre-test to minute 36, and 132 ms from pre-test
36 to minute 72. Regardless, the data suggest that alterations in P300 occurred throughout
37 the course of the exercise intervention, although statistical significance was not
38 achieved at all time points. These data are in stark contrast to those of Yagi *et al.*
39 (1999) as an increase in P300 amplitude occurred after 36 min of exercise and longer
40 P300 latency occurred after only 3 min of exercise. If we were to draw conclusions
41 based solely on the Grego *et al.* (2004) findings, we would suggest, quite logically, that
42 cognitive processing speed was delayed due to the fatiguing influence of prolonged
43 exercise. We might then speculate that inefficient increases in attentional resources
44 were allocated to successfully execute task demands.

As such, two seemingly similar studies yielded disparate findings, which is understandable given the differences in the timing of the cognitive testing relative to the time of the intervention. Thus, the task now is to determine what logical generalizations can be made from these two studies. Given that both Yagi *et al.* (1999) and Grego *et al.* (2004) used similar stimulus discrimination tasks, the lack of consensus across studies is not readily apparent. It is possible that the P300 component fluctuates during exercise similar to that of other physiological measures and that the frequency of data collection (Grego *et al.*, 2004) was not sufficient to capture these changes. Examination of the early measurement period (i.e. minute 3) of Grego *et al.* (2004) provides minimal support for this speculation and supports the findings of Yagi *et al.* (1999). Specifically, P300 amplitude decreases by approximately 0.5 μ V when measured 3 min following the initiation of the exercise intervention. The later measurement periods indicate increases in amplitude. Accordingly, it is possible that brief exercise interventions may decrease the P300 component amplitude, while longer interventions increase amplitude. Future research should follow up on this possibility, which would be necessary to draw strong inferences regarding the underlying meaning of such a finding. Further, the implementation of a task that is challenging enough to yield behavioural changes during exercise would further allow for reasonable inferences to be drawn regarding the influence of in-task exercise on cognition and the resulting effect on task performance.

To date only a single study has examined the relationship between acute in-task exercise and ERPs using a task that requires variable amounts of executive control. The Eriksen flanker task (Eriksen and Eriksen, 1974) has frequently been used to test an individual's ability to manage interference from task-irrelevant information in the stimulus environment (Miyake *et al.*, 2000). This task requires participants to discriminate between two letters that are flanked by an array of other letters that have different action schemas associated with them. Variable amounts of interference control are required based on the compatibility of the letters that flank the target stimulus. Congruent stimuli (e.g. HHHHH) elicit faster and more accurate responses, whereas incongruent stimuli (e.g. HHSHH) elicit increased error rate and decreased response speed (Eriksen and Schultz, 1979) because the latter condition results in greater response competition (Kramer *et al.*, 1994; Spencer and Coles, 1999). This task has been associated through neuroimaging research with a neural network involved in the executive control of attention that includes the ACC and lateral prefrontal cortex, and parietal cortex (Bush, Luu and Posner, 2000; Fan *et al.*, 2005).

Pontifex and Hillman (2007) used a modified flanker task (Hillman *et al.*, 2006a; Posthuma *et al.*, 2002) in which participants were required to attend to a centrally located arrow flanked by other arrows oriented either in the same (i.e. congruent: <<<< or >>>>) or in the opposite directions (i.e. incongruent: <<>< or >><>). Pontifex and (2007) collected ERPs from 41 college-aged adults in counterbalanced conditions of rest and 6.5 min of steady-state exercise on a cycle ergometer at 60% of their maximum heart rate (HR_{max}) as determined during a

maximal exercise test. An increase in P300 amplitude was observed during exercise over frontal and bilateral electrode sites across both conditions of the flanker task relative to rest. Such a pattern of findings suggests an inefficiency of neuroelectric resources during stimulus engagement. Additionally, Pontifex and Hillman (2007) observed a global increase in P300 latency during exercise relative to rest, suggesting delays in stimulus evaluation and classification speed. However, Pontifex and Hillman (2007) also assessed changes in earlier ERP components that occur during acute exercise through the analysis of the N1, P2 and N2 ERP components. The N1 and P2 components relate to aspects of visual discrimination (Luck, 1995; Vogel and Luck, 2000) and selective attention (Talsma and Kok, 2001), respectively. The N2 component relates to response inhibition during tasks that elicit conflict (Ridderinkhof *et al.*, 2002), such as the incongruent condition of a flanker task (i.e. an increase in conflict is generated by the two response mappings as opposed to the single response mapping in the congruent condition).

With regard to the earlier ERP components, Pontifex and Hillman (2007) found a parietal reduction in N1 amplitude and increased P2 amplitude at frontal and central electrode sites, suggesting a decreased capability to visually discriminate stimuli, but an increase in selective attention during exercise. These results may seem contradictory, however Pontifex and Hillman (2007) suggested that the increase in P2 amplitude is indicative of the activation of an on-line, top-down cognitive control mechanism (as described above) to correct for deficits in stimulus engagement. Carter *et al.* (2000) further suggest that increases in top-down cognitive control would relate to reductions in response conflict leading to a reduced activation of the ACC (Colcombe *et al.*, 2004), which is thought to be the neural generator of the N2 component (van Veen and Carter, 2002). As such, additional support for this interpretation is provided through global reductions in N2 amplitude along with longer N2 latency, suggesting a reduced ability to inhibit inappropriate responses and delayed processing speed during exercise.

Taken in consideration with the P300 findings, Pontifex and Hillman (2007) suggested that the earlier ERP components index stimulus encoding deficits that occur during acute aerobic exercise leading to reduced quality of information processing and delays in cognitive processing speed; thus requiring an increased need to allocate attentional resources towards the task and upregulation of top-down cognitive control. Additionally, no ERP differences were observed between task conditions requiring variable amounts of executive control (i.e. congruent, incongruent), suggesting a more general shift in the underlying neuroelectric system that supports cognitive function during acute exercise.

Further support is garnered from the behavioural data, since participants exhibited reductions in response accuracy during exercise only in response to the incongruent condition, which requires increased interference control relative to the congruent condition. Taken together with the neuroelectric data, the findings suggest that in spite of deficits in stimulus acquisition and delays in cognitive processing, participants are still capable of responding quickly and accurately during exercise relative to rest during the congruent condition (low interference control requirement). That is, the

relative neuroelectric inefficiency observed on congruent trials during exercise did not influence task performance. However, the incongruent condition (high interference control requirement), which also exhibited neuroelectric inefficiency, did result in reductions in task performance during exercise, likely due to the inability to inhibit neural resources, resulting in a decrement in the ability to accurately discriminate between target and flanker stimuli or an inability to inhibit responses mapped to interfering stimuli, or both.

As the neuroelectric literature on the effects of acute, in-task, aerobic exercise is relatively sparse and conflicting, it is difficult to make broad generalizations regarding the nature of changes that occur during acute, aerobic exercise. However, it is clear that the P300 component exhibits changes across tasks that require relatively simple stimulus discrimination as well as tasks that require variable amounts of executive control. Although the assessment of ERPs during exercise was previously difficult (as even the slightest movements produced interference to the EEG signal), the advancement of various data collection techniques and sophisticated digital filters has led to a growing number of laboratories attempting to assess neuroelectric indices of cognition during exercise. As it is likely that this area will continue to grow, future research should attend more carefully to the design of the cognitive tasks employed in an effort to embrace a cognitive neuroscience perspective, utilizing tasks that target specific neural networks and structures to better make inferences regarding exercise effects on brain and cognition.

8.4 Event-related brain potentials following exercise

In contrast to the small literature base assessing neuroelectric changes in cognitive function during exercise, a relatively larger literature has investigated neuroelectric changes in cognitive function following acute exercise, with the majority of studies examining the P300 component. Further, many studies have opted to examine the P300 using versions of the oddball task (i.e. auditory and visual), which is described above. Other research has employed different (simple) stimulus discrimination tasks in which the participant receives an auditory warning stimulus (S1) followed by a second auditory (imperative) stimulus (S2) and they are instructed to respond to the latter stimulus (Kamijo *et al.*, 2004a). Finally, few studies have employed tasks requiring aspects of executive control (Hillman, Snook and Jerome, 2003; Kamijo *et al.*, 2004b; Themanson and Hillman, 2006). Although not all studies will be described in detail in this section, the interested reader may refer to Table 8.2 for a brief description of each.

Available evidence examining acute exercise effects on the P300 potential using an oddball task have again provided discrepant findings. That is, to the best of our knowledge, four published studies have examined acute aerobic exercise effects on the oddball-P300. Of these studies, two have observed no change in the P300 component as a function of an acute bout of exercise, while one has observed an increase in P300 amplitude and the other observed an increase in amplitude and

Table 8.2 Summary of findings of studies performed to assess the changes in neuroelectric indices of cognition following an acute bout of exercise.

Author(s)/n	n	Time of test	Exercise intervention	Cognitive task	Result
Duzova <i>et al.</i> (2005)	31 college students (separated into high, moderate, and low physical activity levels)	Pre-test and post-test	Maximal exercise test	Auditory oddball task	No effects for P ₃ . Increase in N2 amplitude following exercise at frontal sites for the high physically active group No effect
Grego <i>et al.</i> (2004)	12 trained cyclists	Pre-test, 3 min, 36 min, 72 min, 108 min, 144 min, post, 15 min post	180 min cycling at approximately 66% of VO ₂ max	Auditory oddball task	Increase in P ₃ amplitude following exercise relative to baseline Shorter P ₃ latency for incongruent trials following exercise relative to baseline
Hillman, Snook, and Jerome (2003)	20 young adults	Baseline and post-test	30 min running on a treadmill at approximately 83.5% of HRmax	Modified flanker task	Decrease in early and late CNV amplitudes following high intensity exercise.
Kamijo <i>et al.</i> (2004a)	12 adults (22–33 yr)	Baseline and post-test	Approximately 18 min of cycling until volitional exhaustion (high intensity), at an RPE between 12 and 14 (medium intensity), and at an RPE between 7 and 9 (low intensity).	St-S2 Reaction time task	Increase in CNV amplitude following moderate intensity exercise.

(Continued)

Table 8.2 (Continued)

Author(s) <i>n</i>	Time of test	Exercise intervention	Cognitive task	Result
Kamijo <i>et al.</i> (2004b)	12 adults (22–33 yr) Baseline and post-test	Approximately 18 min of cycling until volitional exhaustion (high intensity), at an RPE between 12 and 14 (medium intensity), and at an RPE between 7 and 9 (low intensity).	Go/no-go task	Go: Increase in P3 amplitude at Frontal and Central regions following medium intensity Global reduction in P3 amplitude following high intensity exercise No-go: Global increase in P3 amplitude following medium intensity exercise Reduction in P3 amplitude at Central sites following high intensity exercise No effect for N1, P2, or N2. Global increase in P3 amplitude after exercise. Global reduction in P3 latency after exercise.
Magnié <i>et al.</i> (2000)	20 college students (separated into high-fit and low-fit groups) Pre-test and post-test	Cycling to exhaustion	Auditory oddball task	Increase in P2 amplitude at frontal and central sites Increase in P3 amplitude at central and parietal sites No effect for N1 or N2 amplitude or for latency at any component
Nakamura <i>et al.</i> (1999)	7 adults (29–44 yr) Pre-test and post-test	30 min of jogging at a comfortable and self selected pace	Auditory oddball task	

Themanson and Hillman (2006)	28 young adults (separated into higher-fit and lower-fit groups)	Baseline and post-test	30 min running on a treadmill at approximately 82.8% of HRmax.	Modified flanker task	No effect for ERN or Pe
Yagi <i>et al.</i> (1999)	24 college students	Pre-test, during, post	10 min cycling at a HR between 130 and 150 bpm.	Auditory and visual oddball tasks	No effect

1 decrease in P300 latency. Specifically, both Grego *et al.* (2004) and Yagi *et al.* (1999)
2 observed that exercise was unrelated to modulation of the P300 component following
3 the cessation of exercise. As described in the previous section, both observed changes
4 during exercise, with conflicting results reported across the two studies. Despite the
5 differences reported during exercise, both Grego *et al.* (2004) and Yagi *et al.* (1999)
6 showed that P300 recovered from the acute bout of exercise such that the amplitude
7 and latency did not differ from the pre-exercise baseline. Further, both data sets did
8 not imply that repeated exposure to the task resulted in the nonsignificant findings
9 from pre- to post-exercise, since P300 was modulated in such a manner that the
10 latency increased during exercise (Yagi *et al.*, 1999) and then decreased following
11 exercise to the point where it did not differ from the pre-exercise measurement. In
12 other words, the pattern of findings indicated that cognitive processing speed was
13 decayed during exercise, but recovered to the baseline level upon cessation of the
14 exercise.

15 In the case of Grego *et al.* (2004), a nonsignificant increase in latency was
16 observed following the acute bout, suggesting that although participants engaged in
17 3 hours of cycling, the P300 recovered rather quickly, since latency differences were
18 not observed immediately and 15 min following the cessation of strenuous exercise.
19 However, it should be noted that P300 latency was 85 ms slower following exercise
20 relative to the pre-test measure, indicating that although significance was not
21 achieved between test sessions, delays in cognitive processing speed were observed.
22 This delay in P300 latency is not surprising given the nature of the exercise
23 intervention. What is surprising is that the large difference from pre- to post-
24 intervention (i.e. mean of 85 ms) did not achieve significance. Thus, the possibility
25 remains that this nonsignificant finding may have been the result of the relatively
26 small sample size employed. As such, future research should attempt to replicate
27 these findings with a larger sample to better determine the exact nature of long-term
28 exercise on cognitive processing speed. Nakamura *et al.* (1999) corroborated the
29 above mentioned findings as P300 latency was unchanged in a small sample ($N = 7$)
30 of individuals, who had completed a 30 min self-paced jog. Taken together, these
31 data indicate that cognitive processing speed, as measured via P300 latency, is
32 unrelated to acute aerobic exercise when assessed via an oddball task. However,
33 limitations in the generalization of these findings remain due to the high variability
34 observed in the P300 response and the small sample sizes across all three studies.

35 Given that the exercise interventions differed considerably across the three studies,
36 one may begin to believe that no relation exists between acute exercise and P300
37 latency. However, Magniè *et al.* (2000) also examined the P300 component to an
38 oddball task following a maximal exercise test and observed a decrease in P300
39 latency and an increase in P300 amplitude following the acute bout, suggesting
40 benefits to both cognitive processing speed and the allocation of attentional
41 resources during stimulus encoding. These findings are in direct opposition to the
42 earlier works and suggest that acute (and exhaustive) exercise is related to improve-
43 ments in cognitive function through an increase in attentional resource allocation
44 during the updating of working memory processes and faster cognitive processing

1 speed. Accordingly, the field has been unable to achieve consensus regarding whether
2 the oddball-P300 is affected by an acute bout of aerobic exercise.

3 Several obvious reasons may account for these differences, including properties of
4 the exercise intervention, properties of the oddball task used to elicit the ERP, time
5 of testing relative to the cessation of exercise, small sample sizes and individual
6 differences across the samples tested. The interested reader is referred to Kamijo (in
7 press) for a discussion of these factors. Relevant to the direction of this chapter is the
8 fact that the lack of consensus across studies may be due to the fact that this relatively
9 simple oddball task engages a large neural network that is involved in the allocation
10 and focusing of attention, the updating of working memory to changes in the
11 stimulus environment and stimulus maintenance. The neural networks and struc-
12 tural generators that have been linked to these processes (and ultimately result in
13 generation of the P300) include the frontal lobe, ACC, infero-temporal lobe,
14 hippocampal formation and parietal cortex (Polich, 2004). That is, it is possible
15 that differences in the various experimental protocols have led to differential findings
16 because several specific brain regions are involved and it is not clear how exercise
17 influences each of these specified regions. As such, the above mentioned findings
18 have brought a certain degree of confusion regarding the relation of acute exercise to
19 the P300 when elicited by the oddball task, and not much progress has been made in
20 linking the changes in cognition to the supporting brain tissue. One issue that would
21 bring clarity to these discrepant findings would be the use of a different task that was
22 sufficiently difficult to modulate task performance. Thus, this additional information
23 would provide a basis in which to better interpret the P300 findings, allowing for
24 reasonable inferences to be drawn.

25 Accordingly, other research has examined the relationship between acute exercise
26 and ERPs using tasks that elicit variable amounts of executive control, with the goal of
27 linking neuroelectric changes with frontal lobe function, since executive processes
28 have repeatedly been shown to be supported, in large part, by this region of the brain.
29 Although ERPs are not ideal for measuring specific sources of activation due to their
30 low spatial sensitivity, reasonable inferences can be drawn when neuroimaging
31 findings (i.e. functional magnetic resonance imaging, dipole modelling) are consid-
32 ered. Hillman, Snook and Jerome (2003) used a flanker task (described above) to
33 examine the relationship between a 30 min acute bout of moderately hard exercise
34 and the P300 potential. Findings supported, in part, those of Magnié *et al.* (2000),
35 as increased amplitude and decreased latency were observed. The interpretation
36 of the findings differed in that Magnié *et al.* suggested that exercise increased central
37 nervous system arousal, which related to a global increase in neuroelectric activation.
38 Alternatively, Hillman, Snook and Jerome, 2003) suggested that the findings,
39 although general across conditions of the flanker task (i.e. increased P300 ampli-
40 tude), were also selective to task conditions requiring greater amounts of interference
41 control, since P300 latency decreased following acute exercise only during the
42 incongruent condition. That is, the findings suggested that cognitive processing
43 speed was increased only during task conditions requiring increased executive
44 control. It is argued herein that the flanker task may be a superior means for

determining the relation between acute exercise and ERPs, since task conditions that elicit variable amounts of executive control are incorporated, allowing for greater comparison of the acute effects of exercise on cognitive processes involved in each task condition. In other words, the conclusion of a global increase in P300 following acute exercise may have been premature since only the target condition was compared prior to and after acute exercise. Through the comparison of multiple task conditions elicited by the flanker task, it appears that the sensitivity of cognitive processes to acute exercise changes with the amount of interference control required. Based on prior neuroimaging research (Colcombe *et al.*, 2004), we can begin to make inferences regarding the disproportionately larger effects of acute exercise on processes subserved by the neural networks that involve the frontal lobe.

Other research employing a different executive control task supports the notion that acute exercise may differentially influence executive control processes. Kamijo *et al.* (2004b) examined the P300 during a go/no-go task. The go condition is synonymous with an oddball task in that participants discriminate between two stimuli with varying probabilities and respond only to the target stimulus, which occurs during a minority of the trials. The no-go condition utilizes the same study parameters, but the participant is instructed to respond on the majority of the trials and withhold their response to the minority of the trials (i.e. the target stimulus in the go condition). This latter condition requires a greater amount of response inhibition since a prepotent response is created due to the high probability of the presentation of a stimulus requiring a response. Results from Kamijo and his colleagues indicated that both the go and no-go P300 amplitudes were increased following moderate exercise (the exercise condition most closely related to the exercise intervention in Hillman, Snook and Jerome, 2003). Although they did not compare the magnitude of the change across conditions, the mean data indicate a greater increase in the no-go condition, supporting the idea that tasks requiring greater amounts of executive control may be disproportionately influenced by acute exercise. Clearly, future research needs to directly test this hypothesis, but it should be noted that considerable evidence for the disproportionate relation of chronic exercise to changes in executive control functions have been reported across several studies (Hillman *et al.*, 2006a; Hillman, Castelli and Buck, 2005; see Colcombe and Kramer, 2003 for review).

Finally, several studies have examined other ERP components following acute exercise. Kamijo *et al.* (2004a) examined the early and late components of the contingent negative variation (CNV) and found that high intensity exercise reduced the amplitude of both components. Given that the early CNV is related to attentional orienting (Weerts and Lang, 1973) and the late CNV is related to motor preparation (van Boxtel and Brunia, 1994), this observed reduction following highly intense exercise suggests decrements in these aspects of cognition. Interestingly, low and moderate intensity exercise was unrelated to the modulation of the early and late components of the CNV. In addition, Themanson and Hillman (2006) examined the relationship between an acute bout of aerobic exercise and response-locked action monitoring processes by asking participants to exercise for 30 min on a treadmill at approximately 80% HRmax and then examining indices of self-regulatory action

1 monitoring (i.e. the error-related negativity [ERN] and the error positivity [Pe])
2 following acute exercise. The ERN and Pe are thought to relate to neural correlates of
3 action monitoring and error awareness, respectively. Action monitoring processes
4 are related to one's ability to monitor behavioural task performance and adapt
5 performance in order to improve the quality of one's subsequent interactions with
6 the environment. These processes are indexed with both neuroelectric and beha-
7 vioural measures and have been found to be related to levels of cardiorespiratory
8 fitness (Themanson and Hillman, 2006) as well as physical activity involvement
9 (Themanson, Hillman and Curtin, 2006).

10 However, Themanson and Hillman (2006) observed no relationship between an
11 acute bout of aerobic treadmill exercise and neuroelectric and behavioural measures
12 of action monitoring (i.e. ERN, Pe, reaction time, response accuracy). Alternatively, an
13 association was observed between levels of cardiorespiratory fitness and indices
14 of action monitoring such that the higher-fit group exhibited a relative reduction
15 (i.e. smaller ERN) in the conflict-related neuroelectric index of action monitoring
16 associated with error responses and a relative increase in both neural and behavioural
17 post-error adjustments in top-down attentional control (i.e. longer reaction time
18 latency on the trial immediately following error commission). Thus, it was concluded
19 that exercise may not produce a powerful enough influence on these cognitive
20 processes when it is distributed in a single, acute bout, but repeated involvement
21 in aerobic exercise leading to increases in cardiorespiratory fitness may be related to
22 improvements in self-regulatory action monitoring processes.

23 24 25 **8.5 Future directions and conclusions**

26
27 There are several interesting and potentially valuable directions for future research
28 that stand to increase cognitive health and effective functioning across individuals.
29 Specifically, no research to date has employed neurocognitive measures to examine
30 acute exercise effects on cognitive performance using samples of children, older adults
31 or individuals with cognitive or physical diseases. This area of research clearly needs to
32 receive attention as these populations may be disproportionately responsive to acute
33 exercise interventions on cognitive performance. Studies of chronic exercise partici-
34 pation in both pre-adolescent children (Hillman, Castelli and Buck, 2005) and older
35 adults (Hillman, Castelli and Buck, 2005) have supported the notion that exercise
36 promotes better cognitive performance during the early and late stages of the human
37 lifespan. These studies have further provided a basis from which to understand the
38 specific cognitive processes that are influenced by exercise participation. Similarly,
39 future research investigating acute exercise effects on cognition should employ these
40 populations to determine whether they would be more amenable to intervention,
41 which cognitive processes are affected and the duration of cognitive change.

42 However, future research efforts must also embrace a cognitive neuroscience
43 perspective that utilizes specific tasks that are supported by specific neural networks.
44 Much of the research to date has employed little rationale for why the particular tasks

were chosen or the relation between these tasks and their underlying neural networks. That is, the extant literature has not established a link between changes in cognition and the brain tissue supporting these changes. Further research efforts need to draw reasonable inferences through the selection of tasks that have been linked to specific brain tissue through prior neuroimaging research. The extant cognitive neuroscience literature has established many relationships related to both executive and nonexecutive functions. With this in mind, future research efforts should branch into the various other executive functions (i.e. response inhibition, working memory, mental flexibility) as there is a wealth of neuroimaging data available that provides a sound rationale for their examination through an understanding of the neural networks involved. As such, future research would be better able to translate basic laboratory findings to cognitive processes involved in every day life.

In conclusion, available data indicate that neuroelectric concomitants underlying various cognitive processes are influenced by acute exercise, but the body of literature is not as convincing as one might hope at this time. This is largely due to the competing results that have been observed across seemingly similar studies. One obvious distinction that has been observed in the literature is the deficits that emerge in both the neuroelectric profile and task performance during exercise relative to the observed improvements in task performance and changes in the neuroelectric profile that support task performance following acute exercise. This distinction illuminates the need for future efforts aimed at the timing of the various influences upon cognitive performance and the need to tie these influences to the underlying neural networks that are influenced by exercise. Finally, research into the acute effects of exercise on neurocognition remains an exciting area of study as findings allow for the increased understanding of how health factors influence specific processes underlying cognitive performance.

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