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Changes in Cognitive Function, Response Preparation, and Arousal Level Following Moderate Exercise

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Abstract

HIGASHIURA, T., NISHIHIRA, Y., KIM, S.-R., HAYASHI, K., HAYASHI, Y., HATTA, A. and KUROIWA, K., Changes in Cognitive Function, Response Preparation, and Arousal Level Following Moderate Exercise. Adv. Exerc. Sports Physiol., Vol.15, No.1 pp. 9-15, 2009. The aim of this study was to investigate the patterns of changes in cognitive function, response preparation, and arousal level following moderate exercise using the event-related brain potentials (ERPs). In the exercise condition, 14 participants (24.2 ± 1.3 yrs) performed a Go/NoGo reaction time task before cycling (pre-exercise), immediately after (post-exercise 1), and after their esophageal temperature (Tes) and heart rate (HR) had returned to pre-exercise values (post-exercise 2). Exercise was moderate intensity (65% maximal HR) for 30 min. In the control condition, participants performed this task at equal intervals, as in the exercise condition. Go P3 amplitude at post-exercise 1 was significantly larger than that of pre-exercise. NoGo P3 amplitude was increased at post-exercise 1 compared to pre-exercise and post-exercise 2. The pattern of changes in early contingent negative variation (CNV) amplitude was similar to NoGo P3. These results indicate that the facilitation of cognitive function following moderate exercise is caused by the exercise-induced arousal level, and does not last long in the exercise protocol used in this study. In addition, the increases in late CNV amplitude were observed at post-exercise 1 and post-exercise 2 compared to pre-exercise. This finding suggests that response preparation facilitate after moderate exercise, and may represent sensitive to acute exercise.

Keywords: cognitive function, response preparation, arousal level, moderate exercise, event-related brain potentials (ERPs)

1. Introduction

To date, many investigators have investigated the effects of acute exercise on brain function using behavioral and/or neuroelectric indices. Recently, majority of studies have employed P3 component of an event-related brain potential (ERP) to assess cognitive function. P3 is generally considered to be a cognitive neuroelectric phenomenon (31). The theoretical interpretation of P3 is that its latency is related to the stimulus classification speed or stimulus evaluation time (22). P3 amplitude reflects the amount of attentional resources devoted to a given task (37) and context updating of working memory (7).

Several studies suggested that the decreases in P3 latency and/or the increases in P3 amplitude were observed immediately after acute exercise (18, 19, 20, 26), and after heart rate (HR) had returned to pre-exercise values (15, 24). It was considered that the changes in P3 after moderate exercise were caused by exercise-induced arousal level (17). On the other hand, Yagi et al. (38) observed the decreases in P3 latency and amplitude during moderate exercise but no changes in P3 latency and amplitude at post exercise. Grego et al. (12) also showed no changes in P3 latency and amplitude at post exercise (0 and 15 min after exercise) whereas the increases in P3 latency (108 and 144 min from the beginning of the acute exercise) and amplitude (72 and 108 min) during moderate exercise. The results of previous studies probably were inconsistent because of the differences in methodological factors (e.g. the intensity and duration of physical exercise, the nature of the cognitive tasks, and the time at which the psychological task was administered to the subjects). Thus, it is unclear about the patterns of changes in cognitive function caused by exercise-induced arousal level after acute exercise.

It is possible for a Go/NoGo reaction time (RT) task to measure cognitive function and arousal level together. Moreover, response preparation can be examined by this task. This task consists of a warning stimulus (S1) and an imperative stimulus (S2), and elicits NoGo P3 and contingent negative variation (CNV) in addition to Go P3 (abovementioned P3). NoGo P3 is generated in NoGo trials, and is associated with inhibitory response and/or cognitive function (2, 3). CNV appears between S1 and S2, and is separated into early and late components. It is known that the early component is related to attention and/or arousal level (32, 33), and the late component reflects response

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preparation (34, 35).

In RT studies, some investigators reported that facilitation of response preparation was observed during or immediately after moderate exercise (1, 9, 10). In ERPs study, Kamijo et al. (17) showed that late CNV amplitude increased immediately after moderate exercise for 20 min. In regard to the effects of acute moderate exercise on response preparation, the findings are consistent, but the patterns of changes in response preparation after exercise are unknown.

Thus, the aim of this study was to investigate the patterns of changes in cognitive function, response preparation, and arousal level following moderate exercise. We hypothesized that the facilitation of cognitive function and response preparation following moderate exercise were observed immediately after and after their esophageal temperature (T_{es}) and heart rate (HR) had returned to pre-exercise values exercise because of exercise-induced arousal level.

2. Materials and methods

2-1. Participants

14 right-handed healthy males participated in this experiment. Participant characteristics are presented in Table 1. All participants provided an informed consent to the study's procedures and possible risks associated with this experiment. The appropriate committee in the University of Tsukuba approved the experimental protocols.

Table 1. Participant characteristics (n = 14)

Age (yrs)	Age range (yrs)	Height (cm)	Body weight (kg)	Maximal oxygen uptake (ml/min/kg)	Maximal heart rate (bpm)
24.2 ± 1.3	19-36	170.9 ± 1.1	67.7 ± 2.6	48.4 ± 1.6	186.6 ± 1.1

Values are the means ± S.E.

2-2. Procedures

This experiment was conducted in a room at a constant ambient temperature (24-25 °C). Prior to this experiment, participants performed a graded exercise test (GXT) using a cycle ergometer to measure their maximal oxygen uptake ($\dot{V}O_{2max}$) and HR (HR_{max}). During the GXT, the work rate (WR) was increased by 15 W/min. More than 4 days after the GXT, each participant carried out the exercise condition and control condition (no exercise). The two conditions were randomly conducted on a different day, with an interval of at least 4 days. All measurements were conducted at the same time of day in each participant. In the exercise condition, ERPs were collected before cycling (pre-exercise), immediately after exercise (post-exercise 1), and after both their esophageal temperature (T_{es}) and HR had returned to pre-exercise values (post-exercise 2). Participants were seated comfortably except when cycling, and did reading and/or talking between post-exercise 1 and 2. They cycled at load corresponding to 65% HR_{max} for 30 min. The WR was adjusted mechanically to maintain the target HR of each participant. The pedaling rate was kept at 60 rpm. Physiological parameters (T_{es} and HR) were recorded every minute during the experiment. Rating of perceived exhaustion (RPE) and WR were captured every minute during exercise. RPE values were recorded using the Borg scale 4). In the control condition, participants performed this task at equal intervals, as in the exercise condition. Physiological parameters were recorded every minute through the experiment. In regard to the seven participants who performed the control condition first, time to T_{es} and HR recovery after exercise was previously measured by the exercise protocol used in the exercise condition.

2-3. Go/NoGo RT task

The Go/NoGo RT task, which consisted of a warning stimulus (S1) followed 2 s later by an imperative stimulus (S2), was used to elicit ERPs. A binaural 2,000 Hz tone (5 ms rise/fall, 50 ms plateau, 60 dB SPL) was used for S1. For S2, green and red LEDs (duration: 200 ms) at the fixation point 1 m in front of participants appeared randomly, with both colors appearing with the same probability. They were instructed to press a button with their thumb as fast as possible in response to the green LED, but were told not to respond to the red LED. The inter-trial interval was 10 s, and the task duration was about 13 min.

2-4. Recordings

EEG activity was recorded with Ag/AgCl electrodes from five electrodes positioned at Fz, Cz, Pz, C3, C4 of the International 10-20 System, which were referenced to linked earlobes. The electrooculogram (EOG) was recorded using a pair of surface electrodes above and below the right eye. The EEG and EOG activities were amplified with a time constant of 5 and 0.3 s, respectively, and a high-cut filter of 120 Hz. An electromyogram (EMG) was recorded using a pair of surface electrodes on the right-hand flexor pollicis brevis muscle. EMG activity was amplified with a time constant of 0.03 s and high-cut filter of 120 Hz. The sampling rate of digitalization was 500 Hz. The analysis period of P3 was 700 ms including 100 ms pre-S2. EEG samples were averaged for Go (target) stimulus and NoGo (non-target) stimulus, respectively. Go and NoGo P3 were designated as the largest positive-peak between 250 and 500 ms post-S2. Their amplitudes were measured relative to the 100 ms pre-S2 baseline, and peak latencies were defined as the time point of maximum positive amplitude. The analysis period of CNV was 3000 ms including 500 ms pre-S1. EEG samples were averaged for Go and NoGo stimulus, respectively. CNV amplitude was measured relative to the 500 ms pre-S1 baseline. We used the waveform of summed Go and NoGo trials after being

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averaged. The mean amplitudes during the 500-1000 ms after S1 (early CNV) and during the 500 ms until onset S2 (late CNV) were calculated with an averaged CNV. The electromyographic reaction time (EMG-RT) was measured as the time from S2 onset to a sharp increase in EMG bursts. Trials with eye blinks, movement (rejection levels: ± 50 μV), and response errors were excluded from these analysis.

2-5. Statistical analysis

Two-factor (Phase × Condition) analysis of variance (ANOVA) with repeated-measures was applied to EMG-RT. Four-factor (Phase × Condition × Electrode Site × Stimulus) ANOVA with repeated-measures was applied to P3. CNV was analyzed with three-factor (Phase × Condition × Electrode Site) ANOVA with repeated-measures. The Greenhouse-Geisser epsilon was used to adjust the degrees of freedom when sphericity was violated. Post hoc analyses were conducted using repeated t-test or Tukey's HSD. The significance level was set at $p<0.05$. Results are given as means ± S.E.

3. Results

3-1. Physiological data, RPE, and WR

Table 2 shows the mean RPE and WR during exercise in addition to mean T_{es} and HR in both conditions. It was mean 27.4 ± 2.1 min until the T_{es} and HR had returned to the baseline level after exercise.

3-2. EMG-RT

No significant main effects or interactions were observed for EMG-RT (Table 3).

Table 2. Physiological data (T_{es} and HR), RPE, and WR in both conditions (n = 14).

	Control 1	Control 2	Control 3
T_{es} (℃)	36.70 ± 0.03	36.71 ± 0.03	36.73 ± 0.03
HR (bpm)	61.4 ± 2.2	61.1 ± 2.0	61.9 ± 1.8
	Pre-exercise	Post-exercise 1	Post-exercise 2
T_{es} (℃)	36.74 ± 0.03	37.11 ± 0.07	36.82 ± 0.03
HR (bpm)	60.5 ± 1.9	121.7 ± 0.8	62.1 ± 1.9
		during exercise	
RPE		12.8 ± 0.8	
WR(W)		98.9 ± 5.2	

Values are the means ± S.E.

Table 3. EMG-RT in both conditions (n = 14).

	Control 1	Control 2	Control 3
EMG-RT (ms)	223.39 ± 8.04	223.59 ± 8.65	221.59 ± 9.10
	Pre-exercise	Post-exercise 1	Post-exercise 2
EMG-RT (ms)	221.6 ± 8.04	218.03 ± 9.11	220.94 ± 8.34

Values are the means ± S.E.

3-3. P3 latency and amplitude

Fig. 1 shows grand-averaged waveforms of P3 elicited by the Go and NoGo stimuli in the exercise condition.

A significant effect for Stimulus was found for P3 latency {F (1, 13) = 11.42, p = 0.005}. Post hoc analysis of Stimulus indicated that NoGo P3 latency was longer than that of Go P3 at all electrode sites in both conditions.

Significant effects of Phase, Electrode Site, and Stimulus were found for P3 amplitude {F (2, 26) = 3.65, p = 0.040, F (4, 52) = 25.37, p < 0.001, F (1, 13) = 8.78, p = 0.011, respectively}. Furthermore, the Phase × Condition interaction was observed {F (2, 26) = 7.71, p = 0.020}. Follow-up analysis for Phase × Condition indicated that Go P3 amplitude at post-exercise 1 increased compared to that of pre-exercise only in the exercise condition (Fig. 2 A). Also, NoGo P3 amplitude at post-exercise 1 was larger than that of pre-exercise and post-exercise 2 (Fig. 2B). Moreover, an Electrode Site × Stimulus interaction was also observed {F (4, 52) = 23.872, p < 0.001}. Follow-up analyses for Electrode Site × Stimulus indicated that NoGo P3 showed a more anterior distribution relative to Go P3 in

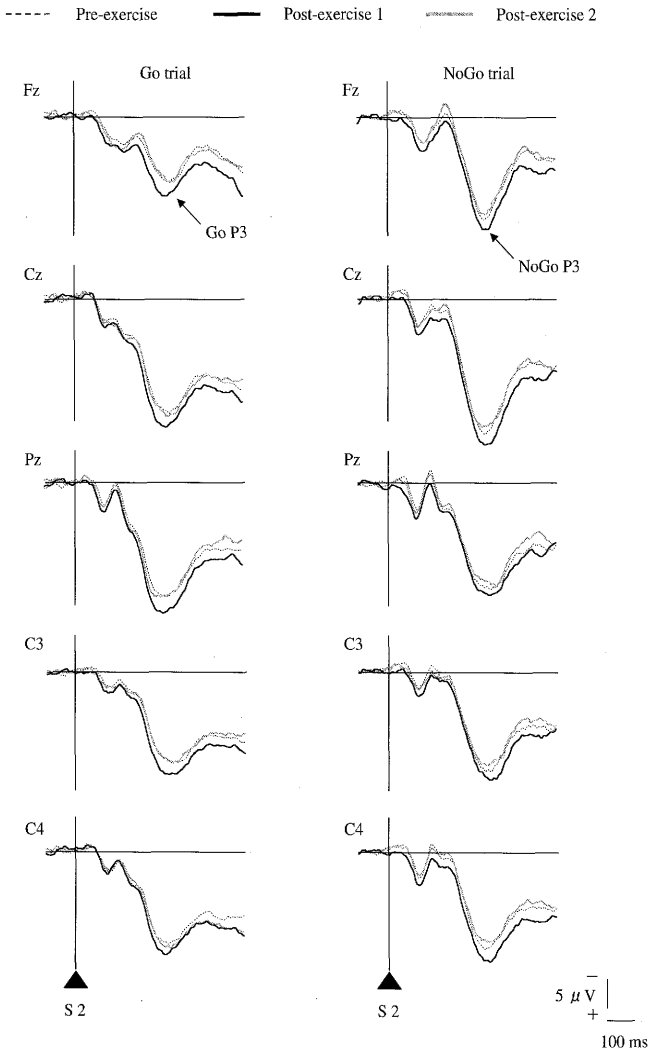

Fig. 1 Grand-average waveforms of P3 elicited by Go and NoGo trials from all electrode sites for exercise condition (n = 14).

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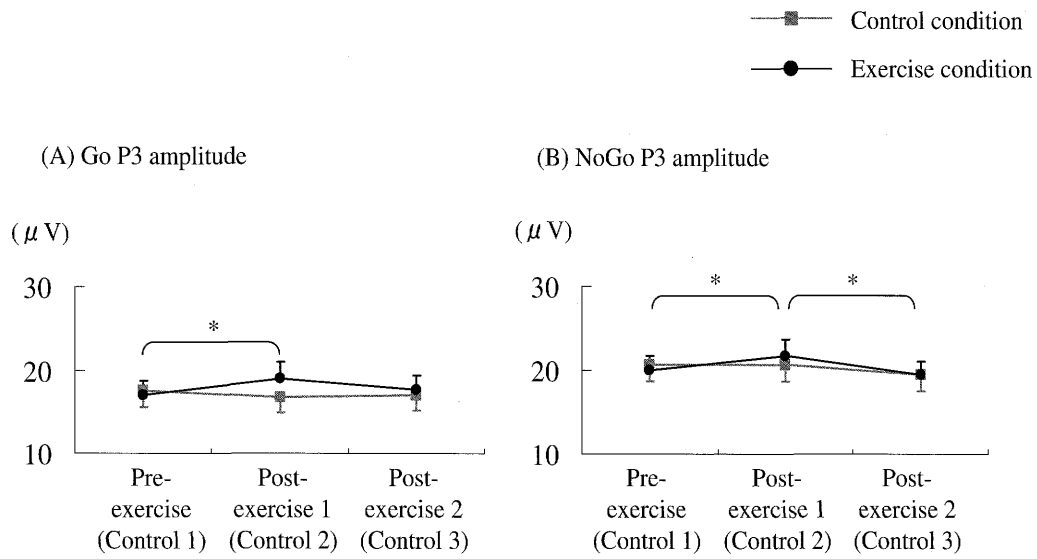

Fig. 2 The Phase × Condition interaction was observed for P3 amplitude {F (2, 26) = 7.71, p = 0.020}.
(A) Mean Go P3 amplitude for control and exercise conditions (n = 14). Go P3 amplitude at post-exercise 1 increased compared to that of pre-exercise only in the exercise condition.
(B) Mean NoGo P3 amplitude for control and exercise conditions (n = 14). NoGo P3 amplitude at post-exercise 1 was larger than that of pre-exercise and post-exercise 2.
Bars represent means (SE). *p < 0.05: exercise condition.

both conditions.

3-4. Early and late CNV amplitude

Fig. 3 shows grand-averaged waveforms of CNV in the exercise condition.

The main effect for Phase and Electrode Site on early CNV amplitude was significant {F (2, 26) = 5.29, p = 0.012, F (4, 52) = 7.82, p < 0.001, respectively}. A Phase × Condition interaction was also observed {F (2, 26) = 3.85, p = 0.034}, with follow-up analysis revealing a larger early CNV amplitude at post-exercise 1 relative to that of pre-exercise and post-exercise 2 only in the exercise condition (Fig. 4 A). Early CNV amplitude was maximum at Cz in both conditions.

The main effect for Phase and Electrode Site on late CNV amplitude was significant {F (2, 26) = 6.12, p = 0.007, F (4, 52) = 14.22, p < 0.001, respectively}. A Phase × Condition interaction was also observed {F (2, 26) = 4.29, p = 0.025}, with follow-up analysis indicating larger late CNV amplitude at post-exercise 1 and 2 compared to that of pre-exercise only in the exercise condition (Fig. 4 B). Late CNV amplitude was the largest for Cz.

4. Discussion

Recently, many researchers have attempted to explain the effects of acute exercise on brain function using behavioral and/or neuroelectric indices. However, inconsistent findings were induced by several methodological factors. This study focused on the patterns of changes in cognitive function, response preparation, and arousal level following acute moderate exercise.

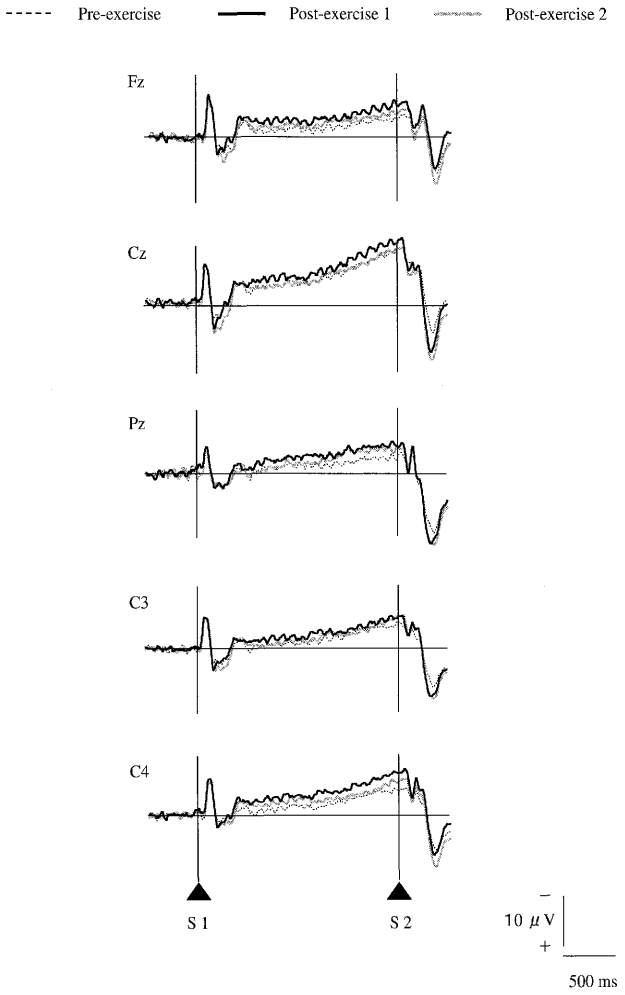

Fig. 3 Grand-average waveforms of CNV from all electrode sites for exercise condition (n = 14).

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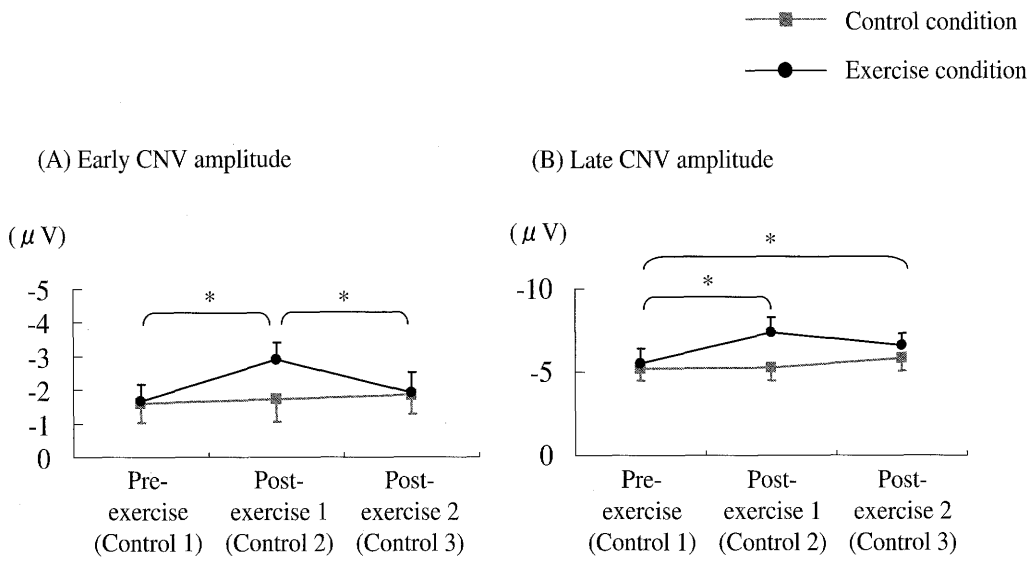

Fig. 4 (A) Mean early CNV amplitude for control and exercise conditions (n = 14). A Phase × Condition interaction was also observed {F (2, 26) = 3.85, p = 0.034}, with follow-up analysis revealing a larger early CNV amplitude at post-exercise 1 relative to that of pre-exercise and post-exercise 2 only in the exercise condition.
(B) Mean late CNV amplitude for control and exercise conditions (n = 14). A Phase × Condition interaction was also observed {F (2, 26) = 4.29, p = 0.025}, with follow-up analysis indicating larger late CNV amplitude at post-exercise 1 and 2 compared to that of pre-exercise only in the exercise condition.
Bars represent means (SE). *p < 0.05: exercise condition.

EMG-RT, Go and NoGo P3 latency were not significantly different among the three recording phases in this study. RT can be decomposed to include components such as stimulus evaluation, response selection, and response execution (8). Go and NoGo P3 latency reflects temporal aspects of cognition, such as stimulus classification speed or stimulus evaluation time (22). Thus, these results may imply that the exercise protocol used in this study did not affect temporal aspects of information processing such as stimulus evaluation time, response selection, or response execution. However, it was difficult to clarify the effects of acute exercise on RT, Go and NoGo P3 latency from the results of this study alone. Chodzko-Zajko (5) suggested that cognitive tasks which require effortful processing should be more sensitive to the effects of exercise than tasks which can be performed without or with minimal attention. Hillman et al. (15) and Kamijo et al. (20) also showed that shorter P3 latency after exercise was observed for more difficult tasks. Therefore, to gain a better understanding of the effects of acute exercise on temporal aspects of information processing in the CNS, it is necessary to examine them further using psychological tasks of varying difficulty.

Go P3 amplitude at post-exercise 1 increased compared to that of pre-exercise. When compared with the pre-exercise and post-exercise 2, NoGo P3 amplitude was significantly increased at the post-exercise 1. Go P3 amplitude is related to the amount of attentional resources devoted to a given task (37) and context updating of working memory (7). NoGo P3 amplitude reflects neural activity associated with inhibitory response and/or cognitive function in the frontal lobe (2, 3). Thus, the results of this study suggest that acute moderate exercise for 30 min facilitates cognitive function in the CNS; however, the facilitative effects do not last in the exercise protocol used in this study. In this study, the pattern of changes in early CNV amplitude was similar to NoGo P3 amplitude. In particular, early CNV amplitude in the frontal lobe is related to arousal level (32, 33). Therefore, our results suggest that arousal level is variable immediately after acute exercise, and it may be that Go and NoGo P3 amplitude is affected by these changes.

Although the mechanism of changes in Go and NoGo P3 amplitude caused by exercise-induced arousal level is uncertain, one possibility is brain neurotransmitter effects (28). The noradrenergic (NA) nucleus locus coeruleus (LC) is phasically activated by a variety of arousing or alerting stimuli, and projects into many brain regions involved in Go and NoGo P3 generators, e.g., the frontal cortex (27), hippocampus, amygdala (13), and thalamus (39). Some monkey studies confirmed that Go P3-like amplitude increased after administration of noradrenergic agonist (29), but decreased when using antagonist (30). Therefore, it is speculated that the NA-LC system is concerned with phasic changes in Go and NoGo P3 amplitude immediately

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after acute moderate exercise.

On the other hand, Hillman et al. (15) and Magnié et al. (24) observed that P3 amplitude increased after participant's oral temperature or HR returned to baseline (about 48 and 60 min post-exercise, respectively). We thought that this discrepancy may be induced by differences in exercise intensity. Hillman et al. (15) and Magnié et al. (24) used high intensity exercise (83.5% HR_{max} and maximal GXT, respectively), but exercise intensity was 65% HR_{max} in this study. Exercise intensity is one of the key factors modulating cerebral blood flow, brain metabolism (16) and brain neurotransmitter levels (11), which affect cognitive function and arousal level. Thus, further studies to investigate the relationships between exercise intensity and time-course of cognitive function and arousal level will be necessary to resolve this discrepancy.

The increases in late CNV amplitude were observed not only at post-exercise 1 compared to pre-exercise but also post-exercise 2. In the case of response to stimulus, late CNV is overlapped by readiness potential, a slow wave brain potential recorded prior to self-paced movement (21). So late CNV amplitude is related to response preparation (34, 35). The results of this study suggest that acute exercise affects response preparation in addition to cognitive function. The increases in late CNV amplitude at post-exercise 1 can be explained by exercise-induced arousal level as well as Go and NoGo P3 amplitude because they are affected by changes in arousal level (25). However, the larger late CNV amplitude at post-exercise 2 can not be explained by changes in arousal level. The generators of late CNV are different from those of Go and NoGo P3, and involve motor-related areas such as the primary sensorimortor cortex, supplementary motor area, and premotor area (14). These areas are directly associated with exercise, and activate during cycling (6). Thus, late CNV may be more sensitive to exercise although the time-course of regional brain activation after exercise is unclear from this study. However, it is necessary to investigate the effects of exercise on response preparation further using indices reflecting motor processing, e.g. movement-related cortical potential, in the CNS because late CNV is also associated with the anticipation of the response stimulus (23, 36).

In conclusion, it is indicated that the increases in Go and NoGo P3 amplitude are concomitant with the exercise-induced arousal level immediately after acute exercise, and may not persist in the exercise protocol used in this study. Moreover, the increases in late CNV amplitude were observed not only at post-exercise 1 compared to pre-exercise but also post-exercise 2. This finding suggests that response preparation facilitate after acute moderate exercise, and may represent sensitive to acute exercise.

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