

Positive Effects of Acute and Moderate Physical Exercise on Cognitive Function

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Abstract Some researchers have reported that moderate physical exercise improves cognitive function, and that exercise at high intensity beyond the optimal point attenuates performance, in an inverted U-relationship. The optimal intensity of physical exercise for cognitive function might be related closely to the anaerobic threshold. It has been regarded as an extremely useful index for effective training intensity in cardiorespiratory fitness. This review specifically addresses acute physical exercise around the anaerobic threshold in healthy subjects and its effects on task performance during or after exercise. We discuss physiological factors for the facilitating effect of moderate exercise on cognitive function, which indicates the initial basis in complicated mechanisms of the benefits of physical exercise on cognitive performance. *J Physiol Anthropol* 28(4): 155–164, 2009 <http://www.jstage.jst.go.jp/browse/jpa2>
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Introduction

Moderate physical exercise has the potential to improve cognitive performance (Davey, 1973; Chmura et al., 1994; McMorris and Keen, 1994; McMorris et al., 1999; Kashihara et al., 2000); however, additional high-intensity exercise above the optimal point might decrease that performance (Chmura et al., 1994). The relationship between the activation level of the central nervous system (CNS) (Sternberg, 1969) and the workload of exercise is known as the inverted U-relationship (Duffy, 1972; Schmidt, 1988). The optimal intensity of physical exercise for cognitive functions seems to be close to the anaerobic threshold (AT), the lactate threshold (LT) (Chmura et al., 1994). The LT has been regarded as a useful index for effective training intensity in cardiorespiratory fitness (Ivy et al., 1980; Jacobs, 1986), reflecting the efficiency of

aerobic metabolism in peripheral skeletal muscles.

This review is intended to investigate reports of previous studies, which have described the facilitating effects of physical exercise on cognitive function, and to explore its unclear mechanisms from a physiological viewpoint. Various kinds of physical exercise (e.g., type, intensity, or duration) and cognitive tasks have been used. We specifically examine the relationship between acute physical exercise around LT in healthy subjects and cognitive performance such as reaction times and working memory tasks during or following exercise; results indicate an initial and simple basis, which might be related to complicated physiological mechanisms underlying the benefit of physical exercise on cognitive performance.

In the first half of this review, the possibility of the optimal point around LT was explored; in the last half (from the section titled “Psychophysiological mechanism”), the psychophysiological mechanism related to LT and several hypotheses concerning how to produce the inverted U-relationship between physical exercise and cognitive function were discussed. Finally, debatable factors were reviewed.

Estimated $\text{VO}_{2\text{max}}$ and Heart Rate showing LT

Instead of a direct lactate measurement, the maximal oxygen uptake ($\text{VO}_{2\text{max}}$) is often used to determine the workload of physical exercise (Wasserman and Whipp, 1975). We firstly show the estimated level or range of $\text{VO}_{2\text{max}}$ indicating the LT. In fact, $\text{VO}_{2\text{max}}$ is used as an indicator of the functional capacity of respiratory, circulatory, and metabolic systems. The workload is generally normalized in a manner to accommodate individual differences of ability in cardiovascular response: $\%\text{VO}_{2\text{max}} = (\text{VO}_2 / \text{VO}_{2\text{max}}) \times 100$ (Mozrall and Drury, 1996). The LT in nonathletes is typically a value between 50–60% $\text{VO}_{2\text{max}}$ (Wasserman et al., 1973; Wasserman and Whipp, 1975; Şekir et al., 2002), although it tends to be higher (e.g. 65–75% $\text{VO}_{2\text{max}}$, Mazzeo and Marshall, 1989) in endurance athletes (Chmura et al., 1994; Chmura et al., 1998) with enhanced cardiopulmonary function.

Heart rate is usually available as a simple alternative index of exercise intensity to direct measurement of lactate or VO_2 ; it is useful for estimating $\% \text{VO}_{2\text{max}}$ during physical exercise (Yamaji et al., 1978). The VO_2 during physical exercise generally increases with cardiac output ($\text{l} \cdot \text{min}^{-1}$), as defined by the production of stroke volume ($\text{l} \cdot \text{stroke}^{-1}$) and heart rate ($\text{beats} \cdot \text{min}^{-1}$) (Yamaji et al., 1978; Mozrall and Drury, 1996). Increases in cardiac output are primarily attributable to the heart rate because the contribution of stroke volume to increased cardiac output reaches a plateau at almost $40\% \text{VO}_{2\text{max}}$ (Mozrall and Drury, 1996); the relationship between the heart rate and VO_2 is generally positive and linear. Indirect methods have been developed for predicting $\% \text{VO}_{2\text{max}}$ using the heart rate during submaximal exercise (Åstrand and Ryhming, 1954; Yamaji et al., 1978; Miyashita et al., 1985). Based on a previous report (Yamaji et al., 1978), the heart rate in young subjects can be estimated using the linear relationship between $\% \text{VO}_{2\text{max}}$ and heart rate showing the LT: a range between 115 and 135, which might be slightly higher in athletes.

The Possibility of an Optimal Point between Exercise and Cognitive Function

The positive effects of physical exercise on cognitive performance, such as reaction time, increase to an optimal point. Further increase in the activation of CNS attenuates task performance (Levitt and Gutin, 1971; Bender and McGlynn, 1976; Chmura et al., 1994). Previous studies have assumed that the intensity of physical exercise at the optimal point might have been related to the estimated LT (see above, 'Estimated $\text{VO}_{2\text{max}}$ and heart rate showing LT').

Levitt and Gutin (1971) assessed a choice-reaction task performance during exercise on a treadmill at graded speeds in male undergraduate students (115, 145, and $175 \text{ beats} \cdot \text{min}^{-1}$ in heart rate). The reaction time, which was improved at $115 \text{ beats} \cdot \text{min}^{-1}$, returned to baseline at $145 \text{ beats} \cdot \text{min}^{-1}$ and decreased below the baseline at $175 \text{ beats} \cdot \text{min}^{-1}$. Sjöberg (1975) compared performance in a choice-reaction task at five different workloads on a bicycle ergometer; task performance at a medium level (around $120 \text{ beats} \cdot \text{min}^{-1}$ in heart rate) was the most efficient. Reilly and Smith (1986) evaluated the influence of pedaling on a cycle ergometer at rates of $\% \text{VO}_{2\text{max}}$ (control, 25, 40, 55, 70, and 85% at a constant speed of 65 rpm) on a pursuit rotor task in subjects actively involved in sport. The estimated optimal workload showed $38\% \text{VO}_{2\text{max}}$ in the pursuit rotor task ($44\% \text{VO}_{2\text{max}}$ in mental arithmetic). This result seems to be smaller than LT, perhaps because of the dual task in the pursuit rotor task with exercise and/or the simple fitting curve using a second-order polynomial equation. Furthermore, Salmela and Ndoye (1986) evaluated the effects of pedaling a bicycle (rest, 115, 145, 160, and $180 \text{ beats} \cdot \text{min}^{-1}$ in heart rate at a constant speed of 50 rpm) on a visual five-choice reaction task in healthy physical education students. The choice reaction time and the number of missed trials were

greatly decreased at a heart rate of $115 \text{ beats} \cdot \text{min}^{-1}$. Consequently, the results suggest that the optimal point might be related to the estimated LT in healthy subjects.

Actually, some researchers have indicated that physical exercise around the AT improved cognitive task performance (Cooper, 1973; Lehmann et al., 1981; Chmura et al., 1994; Chmura et al., 1998). Chmura et al. (1994) tested an audio-visual five-choice reaction task during ergometer exercise incremented by 50 W until volitional exhaustion in male soccer players. Reaction time decreased gradually until exercise of almost $75\% \text{VO}_{2\text{max}}$ ($160 \text{ beats} \cdot \text{min}^{-1}$ in heart rate), but increased under the exercise of volitional exhaustion, suggesting a U-shaped relationship between reaction time and workload. The workload at the fastest reaction time was slightly greater than that showing LT or the threshold of adrenaline and noradrenaline.

Arcelin et al. (1997) evaluated the choice-reaction times during the beginning (3–5 min) and the end (8–10 min) of 10-min exercise using an ergometer at $60\% \text{VO}_{2\text{max}}$ in nonexpert subjects in decisional sports. The reaction times were significantly decreased during exercise. They were shorter at the end of exercise than at the beginning. McMorris et al. (1999) compared the decision-making performance of soccer players on a soccer decision-making test during exercise at the adrenaline threshold and at their maximum power output. The speed of decision was affected significantly by exercise, not the accuracy of the decision. The adrenaline threshold, which is related to the LT (McMorris et al., 2000b), might be indicative of increases in the allocatable resources of arousal and performance. Kashiwara et al. (2005) evaluated the effect of physical exercise at the estimated LT on a three-choice reaction task in healthy subjects. The physical exercise around LT attenuated the reaction time immediately after exercise, although its effect disappeared gradually as time progressed. Consequently, we infer that the exercise around the AT closely affects cognitive function.

The exercise workload at the optimal point might be slightly lower in non-athletes than in endurance athletes with enhanced cardiopulmonary function, reflecting the LT level. The workload at the optimal reaction time was higher in well-trained athletes than in nontrained subjects (Levitt and Gutin, 1971; Bender and McGlynn, 1976; Sothmann et al., 1991). The well-trained athletes also had shorter reaction times than sedentary ones (Spirduso et al., 1988; Rikli and Edwards, 1991). These results are supported by evidence that the workload at the LT is shifted toward a higher value through physical training (Henritze et al., 1985; Pierce et al., 1990). The positive effects of exercise in athletes seem to occur at workloads slightly above LT, which indicates the catecholamine threshold (Chmura et al., 1994; McMorris, 2000b).

Psychophysiological Mechanism

The improved effects of moderate physical exercise on cognitive performance appear to be attributable to various physiological factors related to the workload around the LT, rather than the direct action of the peripheral lactate, as in the following studies.

Direct effect on CNS

The cognitive facilitation by moderate exercise is presumably attributable to a direct improvement in cerebral circulation and the alteration of the action of neurotransmitters. The cerebral blood flow (CBF, e.g., in the middle cerebral artery) containing glucose, oxygen, and energetic substances is modulated by physical exercise (Ide and Secher, 2000b), including the intensity around LT (Ide et al., 2000a). The CBF at 60% $\text{VO}_{2\text{max}}$ was the fastest and showed an inverted U-relationship between the intensity of exercise and CBF (Moraine, 1993), which might correspond to that between the responses to exercise and cognitive task.

Furthermore, by exercise, the regional CBF (rCBF) might improve the brain area to control cognition because the cerebral oxygenation determined by near infrared spectroscopy (NIRS) is modulated in both mental (Hoshi and Tamura, 1993) and motor tasks (Obrig et al., 1996) in some brain areas. In addition, acute physical exercise might improve cognitive performance by changing the levels of neurotransmitters in CNS—acetylcholine, dopamine, norepinephrine, epinephrine, adrenocorticotrophic hormone (ACTH), and vasopressin (Spirduso, 1980; MacRae, 1989; Radosevich et al., 1989; Rikli and Edwards, 1991; Poehlman et al., 1992), which are known as specific neurotransmitters of the CNS (e.g., the hippocampus and brain cortex) to activate cognitive function (Gold and Zornetzer, 1983).

Previous animal studies have shown evidence that physical exercise modulates neurotransmitters in various brain regions. For instance, the hypothalamo-pituitary-adrenal (HPA) axis might be activated during running. Soya et al. (2007), examining 30-min running in rats, evaluated the relationship between exercise intensity and neural activities in the hypothalamus. The running above LT significantly increased blood glucose and lactate levels, and plasma ACTH and osmolality levels; it increased c-Fos induction in hypothalamic regions, including the paraventricular nucleus. Ohiwa et al. (2006a) showed that plasma concentrations of lactate and ACTH were significantly increased by running above the LT in rats, and increased c-Fos expression in the A1/A2 noradrenergic neurons projecting to the hypothalamus, correlating with exercise intensity. In the human brain, exercise can increase the oxidant capability, developing a trophic effect in cerebral centers involved in sensorial-motor function; the central executive function related to the frontal lobe and the hippocampus might also be improved in high-fitness humans (Spirduso, 1980; Churchill et al., 2002).

Brain neuronal activity

In the human brain, noninvasive methods to estimate the neuronal brain activities are effective for identification of mechanisms between physical exercise and cognition. Electroencephalographic (EEG) measurements quickly reflect neural activities, and spectral densities will become useful to quantify the neural activity (Kubitz and Mott, 1996) compared to functional MRI and positron emission tomography (PET). In particular, the latencies and the amplitudes of P300 are used as indices of cognitive speed, respectively unrelated to motor output and the attentional allocation (Nakamura et al., 1999; Yagi et al., 1999). Nakamura et al. (1999) studied the effect of 30-min jogging at a comfortable and self-paced cadence (no tiredness or exhaustion) on cognitive processes during the auditory oddball paradigm by measuring the P300 event-related potential before and after jogging in well-trained joggers. The amplitude of P300 increased significantly after jogging, suggesting a facilitatory effect of moderate jogging on cognitive processes. Yagi et al. (1999) reported that reaction times in both auditory and visual oddball tasks were significantly shortened during aerobic exercise for 10 min (heart rate of 130–150 beats·min⁻¹). The P300 latencies decreased during exercise, indicating the facilitated cognitive information processing in sensory modalities. Acute cardiovascular exercise including the intensity at AT should be evaluated in future studies.

Autonomic nervous system (ANS)

The ANS might be applicable as an effective index to elucidate the central mechanism in the positive effects of moderate exercise on cognition. Medullary centers in the brain reflect activation of autonomic nerves more directly and quickly than secretion of peripheral blood catecholamines; the activation of the ANS via peripheral feedback stimulates the secretion of noradrenaline in the adrenal cortex, hypothalamus, and brainstem (Genuth, 1998). Ohiwa et al. (2006b) identified the brain areas responding to different running speeds: the intensities reflect levels below and above LT (30 min). From c-Fos expression, it was shown that different running speeds caused exercise-intensity-dependent activation of each nucleus in the medulla; the caudal parts of the nucleus of the solitary tract and the caudal ventrolateral medulla were the sites most responsive to speed changes. Those results suggest the activation of the central ANS by exercise around LT.

The insular cortex is also considered a crucial region of cortical cardiovascular control. Using single-photon emission-computed tomography (SPECT) with magnetic resonance images (MRI), Williamson et al. (1997) demonstrated that the left insular cortex was identified as a site for cortical regulation of cardiac autonomic (parasympathetic) activity, despite the lack of change of heart rate during the volitional dynamic exercise. Williamson et al. (1999) also tested insular cortex activation in a cycling exercise. The rCBF data [62 (rest) to 82 (low) and 154 (high-intensity cycling) beats·min⁻¹ in heart rate] indicate that the magnitude of insular activation is related

to the level of perceived effort or central command.

In addition, peripheral autonomic arousal by physical exercise might influence emotion and cognition, although the cerebral function mediating peripheral ANS remains unclear in humans. Critchley et al. (2000) performed PET scanning during isometric exercise and mental arithmetic tasks. Both the exercise and mental tasks significantly increased rCBF in the cerebellar vermis, right anterior cingulate, and right insula with the mean arterial pressure; rCBF in the pons, cerebellum, and right insula was covaried with the increase of heart rate. Cardiovascular arousal was associated with decreased rCBF in prefrontal and medial temporal regions, the hippocampus, and amygdala as well as the left insula, cerebellum, and brainstem, suggesting increased parasympathetic activity. Exercise around the AT might also facilitate cognitive function by peripheral arousal of the ANS.

Heart rate variability (HRV) analysis is a useful method to evaluate the ANS noninvasively (Malliani et al., 1991) during cognitive performance (Vincent et al., 1996; Hansen et al., 2003) and physical exercise (Arai, 1989). Hansen et al. (2003) reported that HRV reflecting the vagal nerves was associated with better performance on simple reaction-time tasks that involve executive functions of the human brain. Arai et al. (1989) showed that HRV changed during the recovery period immediately after physical exercise. Even when cognitive performance is activated through the CNS after exercise around LT, the ANS might be modulated (Kashihara et al., 2005).

Peripheral to CNS

The LT shows the point at which blood lactate concentration is increased rapidly with activation of human growth hormone and catecholamines (Chmura et al., 1994; Chmura et al., 1998; Chwalbinska-Moneta et al., 1996; Chwalbinska-Moneta et al., 1998). The LT during exercise closely correlates with plasma catecholamine concentrations such as adrenaline and noradrenaline (Lehmann et al., 1981; Brooks and Gregg, 1988; Mazzeo and Marshall, 1989). The increase of plasma catecholamines by physical exercise stimulates lactate production in skeletal muscles via the action of β -adrenergic receptors, which determines the LT (Ivy et al., 1987; Chwalbinska-Moneta et al., 1989). Decreased pH in contracting muscles as a result of accelerated lactate production and metabolic changes might stimulate sympathetic outflow by neural afferent signals from muscle metabolic receptors, causing a quick release of catecholamines (McCloskey and Mitchell, 1972; Kjaer et al., 1989). The result suggests a contribution of the sympathoadrenal system to mechanisms of the LT.

It has been explained that moderate physical exercise activates cerebral catecholamine and facilitates arousal of the CNS (Chmura et al., 1998) because of the penetrated peripheral adrenaline against the blood-brain barrier (BBB) (Peyrin et al., 1987; Pagliari and Peyrin, 1995ab). Although the hypothesis that such neurotransmitters penetrate the BBB

remains debatable, the acute change of body temperature by physical exercise might reflect that it becomes easier for the catecholamine to penetrate the BBB. For example, Watson et al. (2005) examined changes in the serum concentration of S100 β , a peripheral marker of BBB permeability, following exercise in active subjects. With the elevation of core and skin temperatures, the heart rate, and the concentrations of blood glucose and lactate, the serum S100 β was shown to be increased after water immersion (39°C) and prolonged exercise (35°C) in a warm environment, suggesting altered permeability of the BBB.

McMorris et al. (2003) assessed whether increases in plasma concentrations of adrenaline and noradrenaline during exercise and power output acted as predictor variables of reaction and movement times during exercise; only power output was a significant predictor of the movement time. McMorris et al. (2008) also estimated the usage of norepinephrine and dopamine in the brain during exercise at 40% and 80% maximum power output (123 and 155 beats $\cdot$ min⁻¹ in heart rate). Although they were strong predictors of response time and movement time, the reaction time at 80% maximum power output increased significantly, and the movement time decreased. Because of the complicated results, they speculated that peripheral concentrations of catecholamines would not activate the CNS directly.

Brain-derived neurotrophic factor (BDNF) is known as the neurotrophic factor giving benefits in the animal brain after exercise, stimulating synaptic plasticity in the hippocampus (Cotman and Berchtold, 2002). In humans, Ferris et al. (2007) revealed that acute cycling on an ergometer at 20% below and at 10% above the ventilatory threshold increased serum BDNF related to the cognitive function (Vaynman et al., 2004). The cognitive function was improved significantly by acute exercise above the ventilatory threshold. The BDNF and Stroop task were correlated, indicating that the peripheral BDNF penetrates the BBB. The relationship between the LT and cognitive performance should be evaluated in future studies.

In summary, acute physical exercise around the AT might facilitate rCBF and neurotransmitters to regulate cognitive functions. Various neurotransmitters or hormones correlated with the lactate release from moderate exercise might produce positive effects on cognition.

Psychological factors

Physical exercise improves mood and psychological well-being, reducing psychological stress, anxiety, and depression (Fox, 1999). Acute exercise also improves mood states, which might indirectly facilitate cognitive function. McMorris et al. (2003) claimed that the effects of exercise on cognition might be attributable to an emotional response that introduces noradrenaline into the CNS directly. In particular, serotonin (Chaouloff, 1997; Weicker and Strüder, 2001) and β -endorphin (Anish, 2005) in the CNS may act as a physiological modulator for memory (Gold and Zornetzer, 1983) and impart positive effects on mood. Actually, 30-min exercise at 60%VO_{2max} on a

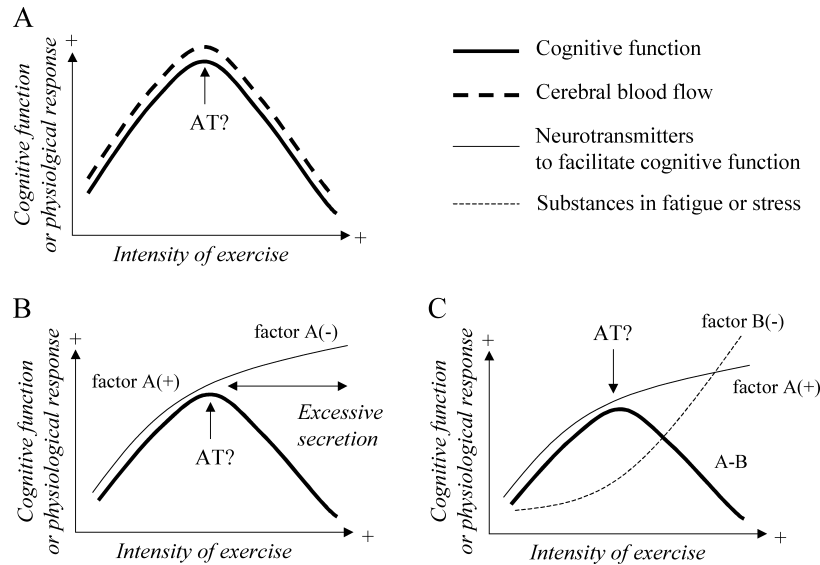


Fig. 1 Hypotheses to account for the inverted U-relationship between the intensity of exercise and cognitive function. A: physiological response corresponding to cognitive function. B: two-sidedness of a physiological factor. C: the interaction between positive and negative factors.

treadmill greatly increased β -endorphin in trained runners (Ferrel et al., 1982). Therefore, acute physical exercise around the AT might improve cognitive function and the mood state because of increased β -endorphin.

Acute running on a treadmill increased brain serotonin synthesis in trained rats (Chaouloff, 1997); exercise increased the ratio of circulating free tryptophan and penetrated the BBB. Furthermore, the beneficial effects of physical exercise on brain function might depend on the circulation of serum insulin-like growth factor I and might be associated with increased hippocampal neurogenesis as well as improved cognition and attenuated anxiety (Trejo et al., 2008). Therefore, moderate exercise might activate these factors, thereby resulting in a positive effect on brain function.

Resting frontal EEG asymmetry can predict affective responses to aerobic exercise at moderate intensities (Davidson, 2004). Greater relative left frontal activity predicts positive affect following exercise. Hall et al. (2007) tested whether resting frontal EEG asymmetry predicts affective responses following a maximal graded exercise for 20 min on a treadmill. Greater left frontal activity predicted tiredness and calmness during recovery from exercise, not tension or energy, thereby supporting the link between EEG asymmetry and affective responses to exercise. Therefore, acute physical exercise around AT might also modulate EEG asymmetry with improved cognition.

Hypotheses

The unknown psychophysiological mechanism still exists; however, some hypotheses will be considered. Figure 1 shows examples of the relationship between the intensity of exercise and cognition. If Fig. 1B or C explains the main reason for the psychophysiological mechanism, the physiological responses

depending on the intensities of exercise (e.g. Fig. 1A) might not directly correspond to the change in cognitive function. Figure 1B shows that the moderate secretion of neurotransmitters will positively affect human cognition; however, a large secretion might induce the rehexis of important neural networks (Weicker and Strüder, 2001). In Fig. 1C, the factors bringing positive effects are initially increased by exercise; however, those bringing negative effects might be dramatically increased after the LT. As a result, the higher intensities of exercise might induce negative or nonsignificant responses in cognition. For example, substances related to fatigue or stress might produce negative responses at higher intensities of exercise (see following section, “Blunted cognitive performance”). The possible factors inducing the positive and/or negative responses are summarized in Table 1. It should be evaluated whether the optimal point corresponds to the LT.

Debatable Factors

During vs. post exercise

Bender and McGlynn (1976) tested the relationship between reaction times and heart rates during walking and running of four stages (3 min) using a treadmill. The reaction time was increased with the increase of heart rate during exercise. Interestingly, despite having the same heart rate (80% of maximum), the reaction time in the post exercise (2 min) was less than that of the maximum workload at the fourth stage. The result suggests difficulty in performing cognitive tasks during exercise.

Positive effects on cognitive tasks during or after moderate exercise around the LT have been evaluated mainly within a few minutes (Chmura et al., 1994; McMorris and Keen, 1994).

Table 1 Possibility of positive and/or negative factors in psychophysiology

Inverted U-relationship (A)		Positive Factors (B, C)		Negative Factors (B, C)
1) Brain		2) Peripheral part	3) Peripheral part to Brain	
CBF	Neurotransmitters	Autonomic nervous system	Catecholamines	Stress hormone
glucose	acetylcholine	sympathetic	adrenaline etc.	cortisol
oxygen	dopamine	parasympathetic	BDNF	Fatigue
energetic substances	noradrenaline			blood lactate
	adrenaline			Excessive secretion
	ACTH			serotonin (dopamine)
	vasopressin			
	Psychological factors			
	β -endorphin			
	serotonin			

CBF; Cerebral blood flow, ACTH; adrenocorticotrophic hormone, BDNF; Brain-derived neurotrophic factor. Alphabets A, B, and C in () correspond to Fig. 1A–C.

Therefore, although continuous durations of cognitive effect are little known (Tomprowski, 2003), the positive effects of physical exercise around LT on the choice reaction time might occur mainly at the early stage of cognitive tasks (Kashihara et al., 2005). In addition, modulation in the vagal or sympathetic nerves by exercise might correlate with cognitive performance (Kashihara et al., 2005).

Simple vs. complicated cognitive tasks

The relationship between exercise-induced arousal and cognitive performance is complicated (Sibley and Beilock, 2007). The types and difficulties of cognitive or psychological tasks might engender different effects of the exercise on task performance (Sjöberg, 1977).

The activation of motor areas during or after exercise might contribute dominantly to cognitive facilitation, especially in a simple reaction task. Arcelin et al. (1998) reported that physical exercise improves performance directly by affecting motor functions and indirectly through other modes of information processing, such as attention and response preparation. Spirduso et al. (1988) reported that the number of taps and simple-reaction speed in young subjects were increased by exercise training, suggesting facilitation of motor function. Davranche et al. (2006) assessed the effects of physical exercise on simple reaction time performance, recording electromyographic signals from the thumb of the responding hand to the fraction reaction time in pre-motor and motor time (decision-making sports players). The results showed that exercise shortened the motor time, not the pre-motor time.

Sanders' model (Sanders, 1983) has been used to simulate the CNS function for cognitive tasks during or after exercise (Tomprowski, 2003). Sanders' model is a cognitive-energetic model of information processing in cognitive tasks; it consists of a computational level, an executive control level, and an energy pool level (Sanders, 1983; Jones and Hardy, 1989). The computational level is mainly affected by three energetic

generators in the energy pool level: an arousal system to change the effects of sensory inputs, an activation system to maintain the tonic readiness for motor action, and effort mechanisms related to decision-making and implicated in co-ordination between arousal and activation (Sanders, 1983; Arcelin et al., 1998; Tomprowski, 2003). The choice reaction task, as a popular and simple cognitive task, requires a perceptual-motor response, a discrete response following the perception, and processing of an invariant stimulus (Mozrall and Drury, 1996). Therefore, the positive effects of moderate exercise are attributable to the improvement of such cognitive processes (MacRae, 1989; Toole and Abourezk, 1989). Because the activated brain region will depend on the level of task difficulty, which might change the partial weight of cognitive processes in Sanders' model, the effects of moderate exercise might vary according to the difficulty and type of cognitive task.

For example, the P300 amplitudes decreased during exercise for 10 min (heart rate of 130–150 beats·min⁻¹), suggesting diminished attention level and error rates increased only in auditory, but not visual tasks (Yagi et al., 1999). Using a treadmill, Hillman et al. (2003) examined the effects of acute exercise for 30 min (162.4 beats·min⁻¹ in heart rate; 83.5% of maximal heart rate) on the response to a target letter as an executive control task, which was performed after the heart rate returned to within 10% of pre-exercise levels. Across midline recording sites, the P300 amplitude was greater in the cognitive task following exercise. The increase of attention related to accuracy might be required to use the larger intensities of exercise.

Examples of ineffective exercise

In some cases, physical exercise around LT does not significantly facilitate cognitive performance. For example, McMorris et al. (2000a) examined the effect of exercise at the epinephrine threshold and at maximum power output on the performance of a soccer skill requiring both decision-making

and motor performance. However, no significant effect of exercise was shown on the response speed by voice or whole body. McMorris et al. (2003) also evaluated the effect of exercise at 70% and 100% of their maximum power output on a four-choice response time task. A significant effect was not shown in the exercise at 70% of the maximum power output: only movement time during maximal intensity exercise was significantly faster. Furthermore, using a PET method, Wang et al. (2000) evaluated the effects of vigorous running on a treadmill for 30 min (healthy subjects with a history of regular exercise) on striatal dopamine release in the human brain, finding no significant changes in synaptic dopamine concentration.

Blunted cognitive performance

The facilitatory effect on cognition during or after moderate exercise would be attenuated by further exercise. Chmura et al. (1994) reported that the reaction time was increased by the physical exercise of a workload in excess of the LT level; they presumed that further increased blood lactate and physical fatigue caused by the choice reaction task after the exercise partly caused the increased reaction time. Furthermore, stress hormones such as cortisol might cause serious fatigue after vigorous physical exercise (Farrell et al., 1983).

The stress response by physical exercise results from a neuroendocrine mechanism. In fact, de Vries et al. (2000) clarified a pattern of activation in hormonal systems during an incremental cycling exercise at 40, 60, 80, and 100% $\text{VO}_{2\text{max}}$ graded every 10 min in healthy males. The increased heart rate (68 to 204 $\text{beats} \cdot \text{min}^{-1}$ at exhaustion and to 142 $\text{beats} \cdot \text{min}^{-1}$ at recovery), lactate, adrenalin, noradrenalin, and growth hormone during and after exercise depended on the workload. The quick response of the sympathoadrenal system (adrenocorticotrophic hormone, β -endorphin, and prolactin) and the delayed response of the HPA axis (cortisol as protective against tissue damage) to exercise might modulate effects on cognition, emotion, and psychological stress at different time points, indicating the reason for a complicated mechanism to produce the optimal point (Fig. 1C).

Perspective

Long-term or habitual physical training, as well as acute exercise, can improve the dysfunction of cognitive function. Recently, moderate physical training over a long term is anticipated to facilitate recovery of cognitive functions, even in elderly people (Kramer et al., 2006). Clarification of the physiological mechanism on facilitating cognitive function by acute physical exercise might suggest a method for more effective training and be applicable to a therapeutic exercise in the dysfunction of cognitive performance and to prevent aging.

The indices (e.g., duration and intensity) facilitating the positive effects of moderate exercise on cognition might be applied to various cases such as resting times in offices or schools. In various types of psychomotor tasks and moderate

physical exercise, further investigations will be required.

Conclusion

Acute physical exercise around the AT might positively affect cognitive task performance because of the modulated CNS. The reasons for improved cognitive function by the exercise around the AT seem to be not only the facilitated rCBF and neurotransmitters in the CNS but also psychological factors. However, the psycho-physiological mechanism causing the inverted U-relationship remains unclear. Advanced methodologies for brain functions (e.g., PET and functional MRI) as well as EEG or animal studies to observe the direct brain neuronal activity will clarify the complicated mechanism between physical exercise and cognitive processes in humans.

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