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Repeated high-intensity interval exercise shortens the positive effect on executive function during post-exercise recovery in healthy young males

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HIGHLIGHTS

- The effect of different lactate accumulation on executive function (EF) was examined.
- Lactate accumulation decreased with repeated high-intensity interval exercise (HIIE).
- Repeated HIIE was accompanied by a shorter positive effect on EF.
- A potential link between lactate accumulation and EF should be further elucidated.

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ABSTRACT

A single bout of aerobic exercise improves executive function (EF), but only for a short period. Compared with a single bout of aerobic exercise, we recently found that high-intensity interval exercise (HIIE) could maintain a longer improvement in EF. However, the mechanism underlying the effect of different exercise modes on the modifications of EF remains unclear. The purpose of the current investigation was to test our hypothesis that the amount of exercise-induced lactate production and its accumulation affects human brain function during and after exercise, thereby affecting post-exercise EF. Ten healthy male subjects performed cycle ergometer exercise. The HIIE protocol consisted of four 4-min bouts at 90% peak VO_2 with a 3-min active recovery period at 60% peak VO_2. The amount of lactate produced during exercise was manipulated by repeating the HIIE twice with a resting period of 60 min between the 1st HIIE and 2nd HIIE. To evaluate EF, a color-word Stroop task was performed, and reverse-Stroop interference scores were obtained. EF immediately after the 1st HIIE was significantly improved compared to that before exercise, and the improved EF was sustained during 40 min of the post-exercise recovery. However, for the 2nd HIIE, the improved EF was sustained for only 10 min of the post-exercise recovery period, despite the performance of the same exercise. In addition, during and following HIIE, the glucose and lactate accumulation induced by the 2nd HIIE was significantly lower than that induced by the 1st HIIE. Furthermore, there was an inverse relationship between lactate and EF by plotting the changes in lactate levels against changes in EF from pre-exercise during the late phase of post-exercise recovery. These findings suggested the possibility that repeated bouts of HIIE, which decreases lactate accumulation, may dampen the positive effect of exercise on EF during the post-exercise recovery.

1. Introduction

During exercise, lactate, a glycolytic product, is formed in contracting skeletal muscle and utilized continuously in diverse cells under fully aerobic conditions. Lactate exchange occurs not only between white glycolytic and red oxidative fibers within a working muscle bed but also between working skeletal muscle and the heart, liver, kidneys, and brain as oxidative and gluconeogenic substrates [1]. During prolonged exercise, muscle metabolism (e.g., lactate kinetics) and brain cognitive function change [1–5]. In light of the fact that systemic lactate is an energy source for the brain [6], there might be an unexplored association between muscle metabolism and the function of the brain. Given its reliance on lactate as a fuel, particularly as lactate concentrations rise, variations in lactate concentration in the blood likely impact cognitive function in the peri-exercise period.

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During prolonged dynamic exercise, cerebral blood flow gradually decreases towards resting values in association with hyperventilation [7,8]. Similar to the decrease in cerebral blood flow (CBF), the exercise-induced facilitation of cognitive function disappears during such prolonged exercise [9]. We previously hypothesized that cognitive function might be impaired during prolonged exercise and could be restored by an increase in CBF. However, this hypothesis was not validated by our finding that cognitive function was not impaired during prolonged exercise, despite a conflict between an increase in cerebral metabolism and a decrease in CBF [10]. In addition, hypercapnia-induced increases in CBF did not improve cognitive function [10]. Soya and colleagues reported that improved EF after acute aerobic exercise was associated with increased left-dorsolateral prefrontal cortex (L-DLPFC) activity in the brain [11,12]. Moreover, their recent study showed that improved EF after exercise correlated with enhanced psychological arousal levels [11]. These findings suggest that improved cognitive function during exercise may be due to the augmented cerebral neuronal activation and metabolism associated with exercise, rather than cerebral perfusion [10].

Previous studies have demonstrated that during heavy exercise, compensatory increases in the uptake (the difference between arterial and venous concentration (a–v difference)) of lactate, glucose and oxygen support elevated brain neuronal activity and metabolism [13]. When arterial lactate is elevated during exhaustive physical exercise, the brain takes up lactate in amounts that may supersede the uptake of glucose [14]. Furthermore, the arterial lactate concentration increased several-fold during exercise, and the reduced oxygen-to-carbohydrate ratio (cerebral metabolic ratio: CMR) during recovery was associated with relatively high lactate uptake by the brain [15,16]. In addition, increased lactate availability through intravenous lactate infusion increased lactate utilization by the brain [6]. These results suggest that lactate fuels the human brain during and after exercise to satisfy the augmented cerebral neuronal activation and metabolic demand, thereby affecting cognitive function. Indeed, lactate has emerged as a central player in the maintenance of neuronal function and long-term memory [17]. For example, albeit in animals, the administration of a glycogen phosphorylase inhibitor in rat hippocampus resulted in abolished extracellular lactate accumulation and long-term memory, whereas exogenous L-lactate administration rescued memory loss [18]. In addition, intracerebroventricular or intravenous injection of lactate has been shown to exert a neuroprotective effect during experimentally induced hypoglycemia or cerebral ischemia [19–21]. Furthermore, the intravenous infusion of 100 mM L-lactate improved cognitive recovery by preserving cerebral ATP generation following traumatic brain injury (TBI) in rats [22]. Recently, George Brooks' group has examined the advantages of using inorganic and organic lactate salts, esters and other compounds in TBI patients [23–25]. The authors compared dextrose + insulin treatment to exogenous lactate infusion in TBI patients with intact hepatic and renal functions, demonstrating that the latter results in normal glycemia and provides nutritive support to the injured brain [23–26]. However, no previous studies have investigated the effect of exercise-induced lactate production on cognitive function.

High-intensity interval exercise (HIIE) training is emerging as an effective alternative to current health-related exercise guidelines [27]. It has been reported that long-term HIIE was more effective at increasing exercise capacity and metabolic and cardiovascular health compared to long-term moderate-intensity continuous exercise in healthy individuals [3,4,28]. Importantly, the effectiveness of HIIE has been demonstrated in older individuals and patients with chronic diseases such as diabetes [29,30], chronic obstructive pulmonary disease [31], and heart failure [32,33]. In addition, Rognmo et al. [34] reported that the adaptation of HIIE for cardiac rehabilitation had a low risk of acute adverse cardiovascular events in a large population. Thus, in regard to ethical issues, it is well known that HIIE can be safely applied to various populations. However, few studies to date have examined the impact of HIIE on the cerebrovasculature and corresponding implications for cognitive function [27]. Recently, we found that HIIE-induced improvements in cognitive function (especially EF) after exercise were sustained for significantly longer periods than after moderate-intensity continuous exercise [35]. However, it is unclear whether the higher production of lactate induced by HIIE compared to that induced by moderate-intensity continuous exercise could sustain EF for a longer time period.

Lactate is produced continuously even under fully aerobic conditions, especially during exercise, when rates of glycogenolysis and glycolysis are elevated [36]; decreased muscle glycogen during and/or following prolonged exercise attenuates lactate production [3,4]. With this knowledge, we hypothesized that repeated high intensity exercise (e.g., first bout of HIIE) would reduce muscle glycogen and hence result in low lactate production and low lactate availability during and after second bout of HIIE and that in addition, this low lactate production/accumulation would affect EF. To test our hypothesis, we examined lactate production and EF in response to the first (1st HIIE) and second (2nd HIIE) rounds of HIIE. In this protocol, it was expected that lactate production and hence EF would be lower following the 2nd HIIE than following the 1st HIIE, although exercise workload (i.e., exercise volume: intensity × duration) between 1st and 2nd bout of HIIE is identical.

2. Methods

2.1. Subjects

Ten healthy, male subjects (mean ± SEM, age: 22.9 ± 0.6 yr, height: 171.4 ± 1.6 cm, weight: 67.5 ± 2.3 kg, peak oxygen uptake (peak VO_2): 46.8 ± 2.1 ml/min/kg) participated in this study. The subjects were informed of the experimental procedures and potential risks and provided written consent to participate in the study. All subjects were right-hand dominant and free of any known neurological, cardiovascular, and pulmonary disorders as well as color-blindness and abnormal vision. All procedures were approved by the Ethics Committee of Ritsumeikan University (BKC-IRB-2015-005). Subjects were instructed to avoid strenuous physical activity in the 24 h prior to each experimental session. Each subject also abstained from food (overnight fasting), caffeine, and alcohol for 12 h prior to each experiment.

2.2. Experimental procedure

Before the day of the experiment, subjects were familiarized with the EF test using the color-word Stroop task (CWST) [37] on their first visit to our laboratory. The CWST was practiced until the subject achieved consistent scores. Subsequently, peak VO_2 was measured to calculate the exercise intensity required for the exercise protocols.

On the day of the experiment, subjects ate a breakfast of approximately 580 kcal 2 h before the 1st exercise (8:00 a.m.). Thereafter, the subject rested until approximately 20 min before the 1st exercise and practiced the CWST for at least 10 min before pre-exercise data were recorded to prevent the learning effect. Next, the subjects rested for 5 min before undergoing measurements of cardiovascular and psychological parameters and the collection of fingertip blood samples for the pre-exercise data, which was concluded within 5 min. Ten min after the practice CWST was conducted, the subjects performed the pre-exercise CWST. Subsequently, the subjects performed the HIIE protocol (1st HIIE). The post-exercise recovery period was set to 60 min, during which the CWST was measured six times at 10-min intervals, including immediately after exercise (i.e., 0 min, 10 min, 20 min, 30 min, 40 min, and 50 min after exercise, Post 0, 10, 20, 30, 40, and 50, respectively). The subjects drank 50 ml of water while resting after the CWST. Shortly thereafter, the subjects performed the same HIIE (2nd HIIE) and post-exercise recovery protocol again.

2.3. Experimental conditions

The experimental protocol used in this study is presented in Fig. 1.