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Acute effects of lactate administration prior to endurance exercise on intramuscular signaling

Kenya Takahashi[1],Yu Kitaoka[2],Yutaka Matsunaga[1],Hideo Hatta[1]
1.The Universiy of Tokyo
2.Kanagawa University

Objective High-intensity exercise, which increases blood lactate concentration, is known as an effective method to induce mitochondrial biogenesis compared to traditional endurance exercise. In addition, it has been reported that lactate acts as a signaling molecule inducing mitochondrial biogenesis. Therefore, we hypothesized that efficacy of high-intensity exercise is partly induced by lactate. The purpose of this study was to investigate the effects of lactate administration on signaling related to mitochondrial biogenesis.
Methods 8-week-old male ICR mice were used in this study. Mice were intraperitoneally administrated phosphate buffered saline (PBS) or 1 g/kg of body weight of sodium lactate. Immediately after the administration, mice were kept sedentary or performed treadmill exercise (20 m/min) for 60 min. Hence, there are the following four groups in this study: the PBS-sedentary, the Lactate-sedentary, the PBS-exercised and the Lactate-exercised. The blood, and the soleus and the plantaris muscles were harvested immediately after the rest or exercise. Nucleus and mitochondria were isolated to assess the localization of p53. Two-way ANOVA (Lactate x Exercise) was performed for statistical analysis.
Results We first measured blood substrates and muscle glycogen concentrations. Lactate administration significantly increased blood lactate and plasma free fatty acid concentrations. Exercise significantly decreased glycogen concentration both in the soleus and the plantaris muscles. Furthermore, lactate administration significantly decreased muscle glycogen concentration only in the soleus muscle. To clarify the effects of lactate administration on intramuscular signaling, we assessed kinases related to mitochondrial biogenesis. Main effect of exercise was observed in phosphorylation state of AMPK, ACC, p38 MAPK, and CaMKII in the soleus and the plantaris muscles. There was a trend of negative effect of lactate in CaMKII phosphorylation in the soleus muscle. However, there was no effect of lactate administration on the other kinases. We also investigated phosphorylation and localization of p53. As a result, lactate administration tended to increase p53 phosphorylation in the plantaris muscle. However, p53 was not translocated to nucleus or mitochondria.
Conclusions Lactate administration affected plasma FFA concentration and muscle glycogen concentration. However, acute lactate administration did not dramatically change intracellular signaling assessed in this study.