2-B-03 Brain glycogen decreases with high-intensity intermittent exercise without hypoglycemia: possible role of brain monoamines

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Background: Brain glycogen, localizes in the astrocytes, is an important energy source for neurons during hypoglycemia. We currently found that prolonged exercise with hypoglycemia elicits increased noradrenalin and serotonin metabolism in the brain, and elicits brain glycogen reduction (Matsui *et al.*, J Physiol, 2011). However, it is unknown brain glycogen metabolism during short-duration high-intensity exercise (HIE) without hypoglycemia. We assumed that even under normoglycemic condition, astrocytic glycogen degrades with HIE supplying lactate to neurons via monoaminergic pathways.

Methods: We swam adult male rats fourteen 20-second bouts carrying a weight equal to 8% of their body weight, with a 10-second rest between bouts, and quantified brain glycogen, glucose, lactate, and monoamines using a high-power (10 kW) microwave irradiation method as a golden standard.

Results: At the end of HIE, glucose and lactate levels in blood and the brain, respectively, increased, while muscle glycogen levels decreased. Glycogen levels decreased significantly in the hippocampus, cerebellum, brainstem, and cortex, but not in the hypothalamus. The decreased brain glycogen did not positively correlate with blood and brain glucose, but did negatively correlate with lactate in the hippocampus, cerebellum and brainstem. In those brains, monoamine metabolites of noradrenalin and serotonin increased with swimming, and negatively correlated with brain glycogen.

Conclusion: HIE without hypoglycemia resulted in a decreased brain glycogen which was related to increased brain monoamines, suggesting a possible role of brain monoamines (noradrenalin and serotonin) in promoting astrocytic glycogenolysis during HIE without hypoglycemia.

Keywords: High-intensity intermittent exercise, brain glycogen, glycogen, brain monoamines

2-B-04 Brain glycogen decreases during prolonged exercise: possible role of brain monoamines

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Background: Brain glycogen, localizes in the astrocytes, is an important energy source for neurons during hypoglycemia. We currently found that the brain glycogen decreases in five brain loci during prolonged exercise induces hypoglycaemia. However, it remains unknown about the mechanism of decreasing brain glycogen. Previous study showed that brain glycogen is broken down into lactate via increased monoamines (Benington & Heller, 1995). We assumed that under prolonged exercise induces hypoglycaemia and muscle glycogen depletion, brain glycogen decreases to supply lactate to neurons via monoaminergic pathways.

Methods: We exercised male Wister rats on a treadmill for different durations (30-120 min) at moderate intensity (20 m/min) and measured their brain glycogen levels using high-power microwave irradiation (10kW). Also, we measured brain monoamines in cortex using High Performance Liquid Chromatography system.

Results: At the end of 30 and 60 min of running, the brain glycogen levels remained unchanged from resting levels, but liver and muscle glycogen decreased. After 120 min of running, the glycogen levels decreased significantly by 37 ~ 60% in five discrete brain loci compared to those of the sedentary control. The brain glycogen levels in all five regions after running were positively correlated with the respective blood and brain glucose levels. Further, in the cortex, the levels of MHPG and 5-HIAA, potential involved in degradation of the brain glycogen, increased during prolonged exercise and negatively correlated with the glycogen levels. These results support the hypothesis that brain glycogen could decrease with prolonged exhaustive exercise via increasing brain monoamines.

Conclusion: Prolonged exercise results in a decrease in brain glycogen to hypoglycaemia and to an increase of NA and 5-HT metabolism in the cortex.

Keywords: Prolonged exercise, brain glycogen, glycogen, brain monoamines