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Abstract

Approximately 30 million people are affected by diabetes in the United States alone. Insulin used to treat diabetics can lead to a variety of deleterious effects, including memory loss, during episodes of low blood glucose, or hypoglycemia. The branched polymer of glucose, known as glycogen, is a proposed neuroprotectant against the neurodegenerate affects of hypoglycemia, possibly serving as an alternate energy source to neurons during times of low blood glucose levels. Due to neurons inability to store glycogen as a glucose reservoir, glycogen within astrocytes undergoes glycogenolosis to form the transportable substrate lactate, which will then be shuttled to the neurons. Lactate is then converted back to pyruvate within the neuron and undergoes the TCA *cycle,* generating energy for the neuron. To test the hypothesis of glycogen acting as a neuroprotectant during times of hypoglycemia, mice with and without brain glycogen were exercised using a motor memory instrument, rotarod, while in various glycemic states. Brain tissue was then harvested and s'ubjected to a western blot in order to analyze the expression of memory proteins. When compared to wildtype, mice lacking brain glycogen have impaired memory formation during periods of hypoglycemia. Therefore, it is expected that mice lacking brain glycogen will have lower expression of memory proteins. Mice lacking brain glycogen also have higher levels of lactate within the blood following rotarod exercise, suggesting lactate's role as a compensatory mechanism for memory formation when blood glucose is unavailable.

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