In-person only, conducted in English.対面開催のみ.
Pre-registration is not necessary. 予約不要.

Feeding the hungry brain: Metabolic adaptations of synaptic transmission

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The brain requires a constant, ready source of energy to function properly. When the metabolic processes supporting energy generation go awry, for example secondary to ischemic stroke, or uncontrolled diabetes, loss of cognitive function follows rapidly. Paradoxically, even in a healthy brain, access to glucose is relatively unreliable. Baseline glucose concentration in the brain is substantially lower than blood serum which is further depleted by bouts of high neuronal activity. Emerging evidence suggests that neurons are metabolically plastic and can utilize alternative fuels like pyruvate as local glucose concentration declines in active circuits. However, the molecular mechanisms of pyruvate utilization in mammalian synapses and their impact on synaptic communication remain poorly understood. Using an unbiased RNA sequencing approach, we have uncovered a signaling pathway involving PGC1α and Sirtuin 3 that is essential for synaptic metabolic plasticity. Furthermore, we have applied quantitative optical and nanoscale imaging of individual synapses to demonstrate that mitochondrial pyruvate uptake modulates distinct steps in the synaptic vesicle cycle and is essential for synaptic function under oxidative conditions. In summary, our studies have uncovered some of the metabolic adaptations of neurons to glucose deprivation which ultimately ensure the fidelity of synaptic transmission.

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