New role for glial energy metabolism in addiction

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Addiction may be viewed as a disorder of reward learning. To date, addiction research has focused on the molecular adaptations through which memories of exposure to abused substances are encoded and maintained by nerve cells.

However, glia, the non-neuronal supporting cells of the brain, have now been implicated in the consolidation of cocaine-related memories. Researchers, led by senior author Dr. Jie Shi, Professor of Clinical Pharmacology at Peking University in China, report these new findings in the current issue of *Biological Psychiatry*.

Glia play a number of roles that support nerve cells. For example, they take up energy substrates, like glucose and acetate, and metabolize these chemicals into lactate, which they then release. Nerve cells absorb this lactate and use it to fuel many cellular functions.

In their new paper, Shi and colleagues report that reactivation of cocaine memories in rats alters the expression of a protein that releases lactate from glia and enables nerve cells to take up lactate. The authors found that if they blocked lactate release by glia or uptake by nerve cells, they produced a long-lasting prevention of cocaine relapse in rodents.

These data indicate that cellular metabolic communication plays a critical role in addiction-related behaviors, which may have implications for new strategies to prevent cocaine addiction.

"This study paves the way toward regional and time-specific therapeutics for drug craving and relapse," said Shi.

"This study suggests that glia provide a critical source of fuel that is necessary for addiction-related memories," added Dr. John Krystal, Editor of Biological Psychiatry. "It broadens our view of the neurobiology of addiction to appreciate a role for the often-neglected supporting cells of the brain."

More information: Yan Zhang et al. Inhibition of Lactate Transport Erases Drug Memory and Prevents Drug Relapse, *Biological Psychiatry* (2016). DOI: 10.1016/j.biopsych.2015.07.007

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