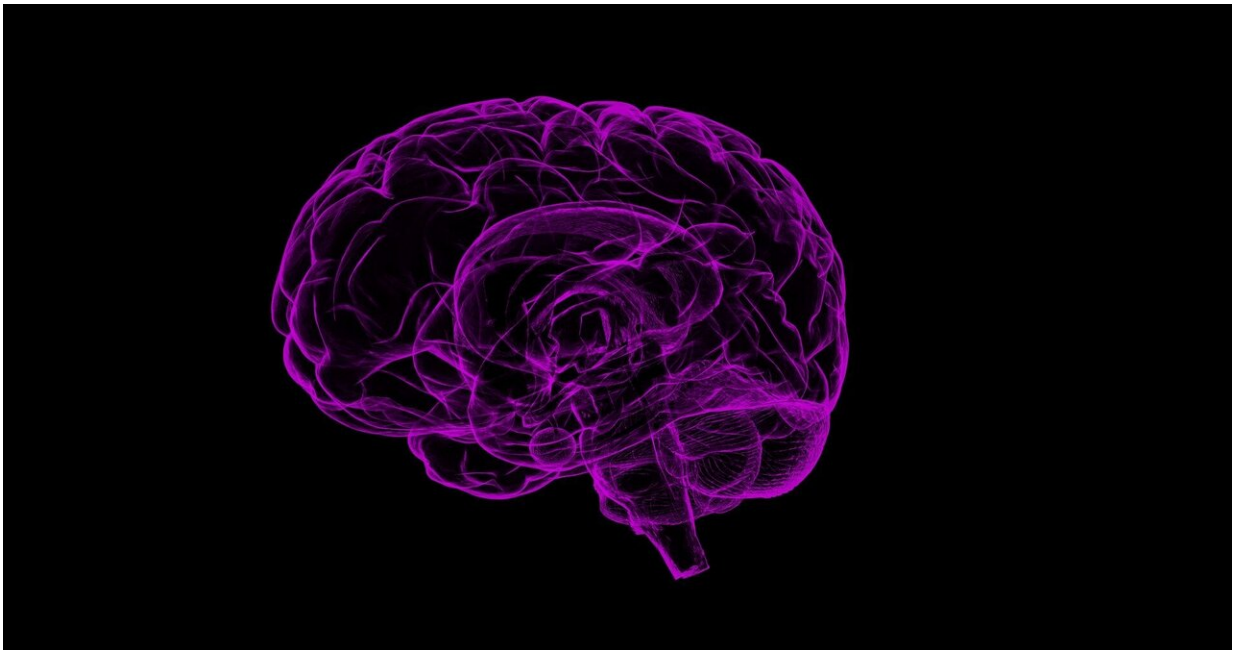


Study examines effects of low-level lead exposure and alcohol consumption

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A new IU study examining effects of low-level developmental lead exposure in mice could explain why some people dependent on alcohol return to using.

The study, published in *Neuropharmacology*, looked at whether developmental lead exposure can increase the propensity to relapse to [alcohol consumption](#) in mice. The researchers also looked at the effects

on the expression of synaptic and non-synaptic glutamate transporters—regulators of [brain](#) motivation and reinforcing circuits—in [brain regions](#) associated with drug addiction.

"Our data in mice suggests that early life, low-level lead exposure does not lead to the development of an alcohol use disorder in adults per se," said Stephen Boehm, professor in the Department of Psychology at IUPUI. "However, it does alter brain circuits in such a way that once a dependency is developed, it makes it harder to refrain from turning back to alcohol."

Although there have been efforts to reduce environmental exposure to lead and to prevent [lead poisoning](#), Boehm said exposure still exists and can cause serious harm and significant health problems, even at low levels. Studies of adults with a history of childhood lead exposure have consistently demonstrated cognitive impairments associated with sustained glutamate signaling.

Boehm and his team studied whether developmental lead exposure increased motivation to consume alcohol by testing mice in an alcohol self-administration paradigm.

The study suggests that low-level lead exposure for humans during childhood and adolescence—so low that kids growing up in old industrial cities might be exposed by merely kicking up dirt during normal play—may be sufficient to enhance relapse to alcohol use in adults struggling with alcohol use disorder.

Their study also suggests that reduced expression of proteins responsible for taking up the [neurotransmitter glutamate](#) in dorsolateral striatum—a brain region believed to be involved in the development of compulsive drug taking—may be associated with this effect. Glutamate is a key player in the executive control of dorsolateral striatal function. So,

dysfunction in [glutamate](#) signaling may well have implications for the development of compulsive drinking (characteristic of individuals with [alcohol](#) use disorder).

Boehm said future work is needed, but the current project demonstrates the need for additional policies around environmental lead exposure.

"This study not only gives us further insight into addiction and the brain, but it might also lead to a call for changes in policies around lead exposure that could help keep our children safe."

More information: Claudia Rangel-Barajas et al, Low-level developmental lead exposure does not predispose to adult alcohol self-administration, but does increase the risk of relapsing to alcohol seeking in mice: Contrasting role of GLT1 and xCT brain expression, *Neuropharmacology* (2020). DOI: [10.1016/j.neuropharm.2020.108339](https://doi.org/10.1016/j.neuropharm.2020.108339)

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