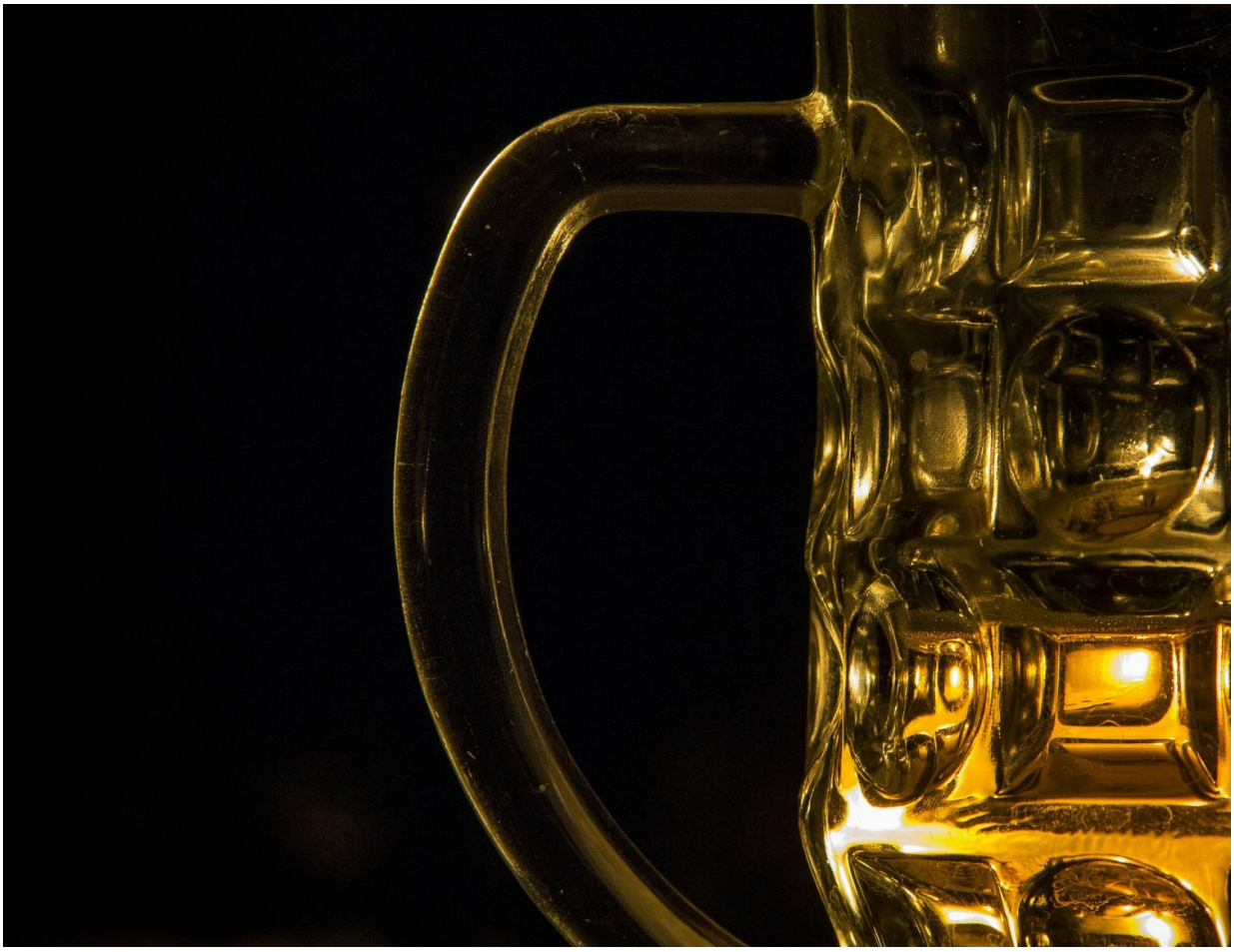
Why does prenatal alcohol exposure increase the likelihood of addiction?

July 7 2017, by Cathy Wilde



Credit: CC0 Public Domain

One of the many negative consequences when fetuses are exposed to

alcohol in the womb is an increased risk for drug addiction later in life. Neuroscientists in the University at Buffalo Research Institute on Addictions are discovering why.

Through a research grant from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) of the National Institutes of Health (NIH), Senior Research Scientist Roh-Yu Shen, PhD, is studying how prenatal alcohol exposure alters the reward system in the brain and how this change continues through adulthood.

The key appears to lie with endocannibinoids, cannabis-like chemicals that are produced by the brain itself.

"By understanding the role endocannibinoids play in increasing the brain's susceptibility to addiction, we can start developing drug therapies or other interventions to combat that effect and, perhaps, other negative consequences of prenatal alcohol exposure," Shen says.

Prenatal alcohol exposure is the leading preventable cause of birth defects and neurodevelopmental abnormalities in the United States. Fetal Alcohol Spectrum Disorders (FASD) cause cognitive and behavioral problems. In addition to increased vulnerability of alcohol and other substance use disorders, FASD can lead to other mental health issues including Attention Deficit Hyperactivity Disorder (ADHD), depression, anxiety and problems with impulse control.

"After the prenatal brain is exposed to alcohol, the endocannibinoids have a different effect on certain dopamine neurons which are involved in addicted behaviors than when brain is not exposed to alcohol," Shen says. "The end result is that the dopamine neurons in the brain become more sensitive to a drug of abuse's effect. So, later in life, a person needs much less drug use to become addicted."

Specifically, in the ventral tegmental area (VTA) of the brain, endocannibinoids play a significant role in weakening the excitatory synapses onto dopamine neurons. The VTA is the part of the brain implicated in addiction, attention and reward processes. However, in a brain prenatally exposed to alcohol, the effect of the endocannabinoids is reduced due to a decreased function of endocannabinoid receptors. As a result, the excitatory synapses lose the ability to be weakened and continue to strengthen, which Shen believes is a critical brain mechanism for increased addiction risk.

Shen's research appears in the latest issue of The *Journal of Neuroscience*.

Provided by University at Buffalo