MedicalXpress

Marijuana increases alcohol toxicity in young rats

April 8 2008

Marijuana is among the most frequently used illicit drugs by women during their childbearing years and there is growing concern that marijuana abuse during pregnancy, either alone or in combination with other drugs, may have serious effects on fetal brain development.

There is strong evidence that THC, the main psychoactive component of marijuana, crosses the placenta, that maternal marijuana abuse results in intrauterine growth retardation and that infants exposed to marijuana exhibit a temporary syndrome that includes lethargy and decreased muscle tone. Fetal exposure to THC can also result in attention deficits, learning disabilities and behavioral problems.

A new study using rats found that THC combined with mildly intoxicating doses of alcohol induced widespread nerve cell death in the brain. The study is published in the *Annals of Neurology*, the official journal of the American Neurological Association.

Led by Henrik Hansen and Chrysanthy Ikonomidou, at the Neuroscience Research Center of the Humboldt University in Berlin and the Department of Pediatric Neurology, University of Technology Dresden, Germany, researchers administered THC, a synthetic form of THC, ethanol, MK-801 (an anticonvulsant) and phenobarbital by injection to rats between 1 and 14 days old. A previous study by the same group had shown that ethanol and drugs such as sedatives, anesthetics and anticonvulsants triggered widespread nerve cell death in the developing brain of immature rodents; the current study was conducted to determine

if cannabinoids had the same effect.

The results showed that THC and its synthetic form did not cause neurodegeneration when administered alone but did cause cell death when given with lower than toxic amounts of ethanol. This combined effect increased according to the dose of THC that was administered and was strongest when the rats were 7 days old. THC also enhanced the neurotoxic effect of phenobarbital and MK-801 (ethanol combines the mechanism of action used by these two drugs). In addition, marijuana activates CB1 receptor levels, which causes the psychomotor, memory, cognition and pain perception changes seen with this substance in adult humans and animals. In the current study, THC combined with ethanol increased these levels, and the CB1 receptor blocker Rimonabant, an anti-obesity drug that may be beneficial in treating addiction, reduced these levels. Mice that did not have functioning CB1 receptors (knockout mice) were less susceptible to the neurotoxic effects of ethanol.

“Neuronal degeneration became disseminated and very severe when THC was combined with a mildly toxic ethanol dose,” the authors state, adding that since this effect was completely counteracted with the CB1 receptor blocker Rimonabant, the activation of these receptors is responsible for ethanol’s increased toxicity. They note that experimental evidence suggests that endocannabinoid (compounds similar to cannabinoids that are naturally produced in the body) signaling may be involved in developmental processes such as cell proliferation and survival during the formation of the central nervous system, which would account for the age-dependent effect of the THC/ethanol combination seen in this study.

The authors acknowledge that the effect of cannabinoids on the neurotoxicity of ethanol on the developing brain requires further studies with longer survival periods. “With the use of behavioral and stereological techniques such studies would explore whether acute

changes reflect permanent neuronal loss and lead to behavioural deficits," they conclude. "The results of the acute studies have interesting potential therapeutic implications including the use of CB1 receptor antagonists for preventing brain damage in fetuses and neonates exposed to ethanol, sedative and/or anticonvulsant drugs."

Source: Wiley

Citation: Marijuana increases alcohol toxicity in young rats (2008, April 8) retrieved 24 April 2024 from https://medicalxpress.com/news/2008-04-marijuana-alcohol-toxicity-young-rats.html

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