The Cannabis-Psychosis Link: Mind Your Mind

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By Marie-josee Lynch, MD, Rachel A. Rabin, MSc, and Tony P. George, MD, FRCPC

Marijuana doesn't count, does it?" Clinicians are familiar with this common reply when screening for drug use. Cannabis—the most common illicit substance—has managed to exempt itself from the hazardous reputation held by other illicit drugs.[1]

As mental health practitioners, it is our duty to educate our patients about the potential harms and consequences of cannabis use. This important task is complicated by the disagreement and uncertainty surrounding the nature of the interaction between cannabis and psychotic disorders.

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While research suggests that cannabis use can induce an acute psychotic state, there is controversy about whether it may precipitate psychotic disorders, such as schizophrenia. In this article, we provide an update on the literature on this important issue, emphasize areas in need of research, and provide clinically useful recommendations.

More than 16 million Americans use cannabis on a regular basis, typically beginning in adolescence. Notably, it is estimated that approximately 4% of the population has a diagnosis of either cannabis abuse or dependence.[1] A history of cannabis misuse is even more common in patients who are schizophrenic than in the general population; 25% of patients with schizophrenia have a comorbid cannabis use disorder. Cannabis use disorders are especially common in younger and first-episode patient samples and in samples with high proportions of males.[2]

Neurobiology

Marijuana contains more than 400 chemical compounds, including over 60 cannabinoids that contribute to its

the second major psychoactive constituent of cannabis.[3] The ratios of these and other cannabinoids vary enormously in preparations of cannabis, and little information exists about the concentration of each of the particular cannabinoids in commonly used cannabis products. Concerns have been expressed regarding the large increase in the potency of cannabis and the surrounding health implications. In the 1960s, the THC content was thought to be in the range of 1% to 3%; today it can reach up to 20%.[4]

The endogenous cannabinoid system consists of 2 types of G-protein-coupled receptors: cannabinoid 1 (CB1) and cannabinoid 2 (CB2) receptors. CB1 receptors are the most abundant in the brain, while CB2 receptors predominate on immune cells. CB1 receptors are highly concentrated in brain regions implicated in the putative neural circuitry of psychosis and cognitive function. These include the hippocampus, prefrontal cortex, anterior cingulate, basal ganglia, cerebellum, and cortex, with lower levels present in the thalamus, hypothalamus, and amygdala. Activation of CB1 receptors mediates the behavioral and physiological effects of both endogenous and exogenous cannabinoids in the brain.[4]

An important role of the CB1 receptor is to modulate neurotransmitter release in a manner that maintains homeostasis by preventing excessive neuronal activity in the CNS.[5] CB1 receptors are localized on presynaptic neuron terminals on both inhibitory and excitatory neurons, yet they predominate on γ-aminobutyric acid interneurons.[6] It is the inhibitory neurons that are thought to mediate most of the effects of cannabinoids. In addition, the action of cannabinoids includes interactions, albeit indirectly, with the dopaminergic system.

THC is a partial agonist at the CB1 receptors, where it has modest affinity and low intrinsic activity. In contrast, CBD shows very little affinity for CB1 receptors. Moreover, the precise molecular mechanism of action of CBD remains unclear. The main endocannabinoids are anandamide and 2-arachidonylglycerol. In contrast to classic neurotransmitters, endocannabinoids can function as retrograde synaptic messengers—they are released from postsynaptic neurons and travel backward across synapses, activating CB1 on presynaptic axons and suppressing neurotransmitter release.

Cannabinoids produce an increase in the dopaminergic activity in the mesolimbic reward pathway, which plays a pivotal role in mediating the reinforcing effects of most drugs of abuse. The increased dopaminergic drive elicited

SECTION EDITOR: SCHIZOPHRENIA

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by the cannabinoids could underlie the abusive property of the drug and increases in positive psychotic symptoms induced by THC.[7] Recurrent cannabis use produces prolonged and excessive stimulation of the CB1 receptor, and this is thought to disrupt endocannabinoid system function.[8] Several lines of evidence exist to suggest a role for cannabinoids and their receptors in the pathophysiology of schizophrenia. It has also been proposed that this CB1 receptor overstimulation may be a contributing factor in triggering THC-induced psychosis.[9]

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C

L. Marjorie @ Sat, 2014-07-19 19:59

reply

B

L. Marjorie @ Sat, 2014-07-19 19:59

reply

I think cannabinol d's should be investigated for possible therapeutic aspects. Why do people with schizophrenia smoke pot and do spice? My son says it relaxes him. How can some ingredient in pot be incorporated into their meds?

Diane @ Fri, 2014-07-18 13:59

reply

As a relative expert in statistics I can verify for you that none of the evidence here presented is scientifically valid. These are merely observations with suppositions attached to them. It is clearly stated that cannabis users with psychosis are more likely to have the gene responsible for the disorder than other people. What the numbers actually say is that there are 3% of the general population who have psychosis and 4.2% of marijuana users. As marijuana users are a subset of society the numbers compared are much smaller for the cannabis users than the general population. This alone would introduce errors into the equations that render the results highly suspect. The standard deviation of the smaller subset would naturally be higher in the smaller group than the larger group. This would mean that the error margin in the data collection would exacerbate any findings and lead towards an exaggerated deviation from the larger group of the general population and nullify the results. In other words the smaller group has greater margin of error than the larger group. With such a small difference between the groups it is just as likely, if indeed the difference even exists, that the marijuana users were just slightly more inclined to seek help when compared to the general population. When we analyse by these statistics and even if we consider that there was 20% of the population of the United States to use marijuana this would represent then a subset of 0.2% of the population being affected by this supposed disorder. This is not even worth considering to be a real malady, it is just a figment of the imagination of the researchers. This is compared to the well established fact that 20% of people who have psychosis are also heavy drinkers.

Brian @ Tue, 2014-07-08 02:02

reply

Interesting article and the comments it generated. In my practice I have seen K2 induced psychosis is worse than regular THC induced mood/thought alterations. Hope this debate continues as we are legally accepting ganja more and more into our society.

reply

Substance Use Disorders
Adolescent Marijuana Use and Vulnerability for Neuropsychiatric Disorders
Effects of Adolescent Marijuana Use on Cognition

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shihab @ Tue, 2013-12-24 09:43

This is an excellent review of the correlations between cannabis use, symptoms associated with schizophrenia, the physiological, gender, and age factors that figure into the risk.
My question is why do so many "scientists" need to discredit good analyses...are personal and political views compromising our "science?"

reply

Donna @ Wed, 2013-10-30 18:43

[The comment by "TheClean" has been removed due to promotional material. -The Editors]

reply

TheClean @ Tue, 2013-10-29 17:56

Good point, Ken.

reply

Betty @ Tue, 2013-10-29 13:48

The author does not deal with the data that show psychosis rates are consistent cross-culturally, regardless of whether the culture has a high cannabis use rate (e.g., Canada) or a low cannabis use rate (e.g., Japan), or, why psychosis rates are consistent despite dramatic spikes in a culture's use of cannabis (e.g., U.S. 1960's).

reply

Ken @ Tue, 2013-10-29 11:29

The author does not account for the fact that most adolescents are going through a period of experimentation, and the fact that if a teen is using cannabis there is most likely concurrent use of other illicit or illegally obtained phamaceuticals which could very likely contribute to psychosis. Also by the time an individual reaches the age of 26 they have typically chosen their prefered vice, those that have chosen cannabis are not experiencing psychosis as they have determined what gives them pleasure and what causes distress including emotional and psychological distress.
Also if a child is experiencing signs of psychosis at an early age (11) then most likely they are trying to cope with unfamilar thoughts, emotions, and urges. These children who are dealing with mental illness during preteen years are still subject to peer pressure and are trying to fit in with their peers. The desire to numb these unusual thoughts, emotions, and urges can easily be a contributing factor to the early use of cannabis or any other "numbing" agent.
Why is it when medical studies are conducted the human variable is always ignored?
As members of the profession that studies and treats the human mind we must always consider the human variable, and remember when studies are conducted in a controlled environment, that which makes us human is removed, freedom of choice.
Jen

reply

Jeanette @ Tue, 2013-10-29 15:31