NSSS ADVOCACY BULLETIN

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Marijuana's Pro-Psychotic Effects May Put Teens At Risk

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As a psychiatrist, the more I know, the more I realize how little I know. This drives my continuing education, as does what I witness every day in my office: the painful struggle of living with a mental illness and the liberation of recovery.

Through this blog, I hope to provide accurate, scientific information about mental illnesses, but also to reflect the experience of living with mental illness, because many voices of personal experience are silenced by shame, fear or lack of insight.

There were many disconcerting comments about my April 8 blog regarding the dangers of street pot. Some who commented hadn't actually read the blog, attributing beliefs to me I hadn't espoused. Others launched ad hominem attacks focusing on my profession and my character rather than the substance of the blog.

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Cannabis use has enormous physical, psychological and social consequences. Disregarding evidence that doesn't align with personal convictions reflects a closed mind. I am writing to the nearly 5,000 people who "liked" the blog, but also to those who read it with an open mind, whether they liked it or not.

Only a small percentage of pot smokers develop schizophrenia, but mountains of scientific evidence underpin the THC-psychosis relationship. In my April 8 blog, I explained that due to selective breeding the potency of THC in street pot has risen exponentially. This has inversely mirrored the reduction/elimination of cannabidiol, the purported antipsychotic, anticonvulsant, neuroprotective component of cannabis (the evidence for its benefits is mixed but promising). I have argued that THC is pro-psychotic. To appreciate the gravity of that assertion, it's important to understand the devastating consequences of psychotic disorders (see my April 19 blog).

If THC is associated with psychosis, some question why the incidence of schizophrenia is unchanged despite an increase in cannabis use and potency; however, the literature yields little recent data regarding incidence. What has been demonstrated is increased rates of schizophrenia in countries where the use of high-potency pot has become the norm (e.g. U.K.), compared to countries using lower-potency pot (e.g. Italy).

Some have posited teens with schizophrenia start to smoke pot to deal with their symptoms of psychosis or to manage drug side effects. That hasn't been born out in research, which shows that the most common reason teens smoke pot is to get high, whether or not they're psychotic. Smoking to manage psychotic symptoms is rarely reported.

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There is no single cause for schizophrenia. THC alone is not responsible, but there is an abundance of evidence that THC can provoke an earlier onset of schizophrenia by up to six years. There is also solid evidence to suggest a causal link between THC

and schizophrenia. Similar associations have not been established with other drugs or tobacco.

The age of onset of psychosis is most powerfully influenced by

gender (male) and the frequent use of high-potency pot before age 15. Critically important brain development occurs in the teen years and continues until about age 25. It's also a time when nearly all mental illnesses first present.

A later onset of psychosis is associated with higher educational attainment, stronger support networks and more mature social skills, resulting in greater independence, insight and engagement in treatment. Thus, the age of onset of schizophrenia may impact illness progression and life-long functioning.

For many patients, treatment, especially soon after the onset of illness, results in improvement or resolution of psychotic symptoms. Unfortunately, many discontinue treatment, whether due to side effects, negative personal/family/peer beliefs about medication, or lack of insight regarding the seriousness of their illness.

This usually results in an acute psychotic episode (the return or worsening of symptoms). Acute episodes of psychosis cause progressive brain changes associated with the disorder, resulting in a more chronic, treatment-resistant illness. Continuing to use pot once psychosis has been established is associated with higher relapse rates, wreaking havoc on an already vulnerable brain.

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There are several proposed mechanisms for the THC-psychosis relationship. One is that THC is a neurotoxin that provokes the onset of schizophrenia by damaging neurons needed to develop vital brain circuits.

THC affects the body's endocannabinoid system. This system is critically important for organ and brain development. Studies have demonstrated that long-term use of cannabis results in abnormal brain structure and function in areas rich in endocannabinoid receptors.

There is now evidence that THC-induced changes in brain structure and function may be inherited by subsequent generations. Known as epigenetic modification, the chronic use of high-potency THC is postulated to result in altered gene expression. The modified gene, which causes brain changes in a pot-smoking parent, may be passed on to their child, resulting in the same brain changes even if the child never smokes pot.

There is evidence that pot-related psychosis can be linked to several specific genes. A variant of the AKT1 gene increases the risk of psychosis in pot smokers and also heightens the pro-psychotic effect of THC.

Dopamine is a key neurotransmitter associated with schizophrenia and a specific gene for the dopamine receptor might also heighten THC-related psychosis risk.

Schizophrenia is a devastating illness for patients and those who love them and it's more devastating when it happens early in life, before vital skills are developed and independence has been attained.

Even if schizophrenia were inevitable, there is a strong argument to be made for the benefits of remaining well for as long as possible and avoiding pot after a diagnosis is established. Since we don't know who is most vulnerable to the effects of THC, all teens should be considered at risk and receive education about the association between pot and psychosis.