

## WHAT THE SCIENCE SAYS ABOUT ADOLESCENT USE OF CANNABIS

By Linda Gertson, Ph.D.

Although scientific evidence exists that some cannabis strains may be beneficial for specific medical disorders, there is an increasing body of empirical evidence demonstrating harmful effects of cannabis use during adolescence. The current trend to increase the number of medical marijuana dispensaries and to legalize recreational use has brought attention to the risks of cannabis, particularly among youth. This paper focuses on the effects of cannabis on adolescents and young adults.

“Marijuana” refers to the dried flowers, leaves, stems and seeds of the cannabis plant. Delta-9-tetrahydrocannabinol (THC) is one of approximately 70 cannabinoids contained in this plant. THC is the primary psychoactive component of marijuana and activates the brain’s reward system causing the “high” that can include a sense of euphoria, relaxation and calm. The “high” can also stimulate appetite; cause distortions of time, color and sound; slow learning; disrupt concentration; and impair short-term memory (National Institute on Drug Abuse (NIDA), 2015). Another cannabinoid, cannabidiol (CBD), appears to be non-psychoactive and is believed to have a neuroprotective role (Iuvone, et al., 2004) and an anticonvulsant effect (Porter & Jacobson, 2013).

There are hundreds of varieties of cannabis. The different varieties contain different blends of cannabinoids which can cause a range of effects (Canadian AIDS Society, 2007). Consequently, results of research into the consequences of cannabis use may be confounded if the cannabinoid blend is not consistent across studies. Scientific studies of “medical marijuana” will likely include different blends of cannabinoids. For example, there are reports of compounds with low THC and high CBD (“cannabidiol-enriched cannabis”) having a positive effect in reduction of seizures in some children (Porter & Jacobson, 2013). Following a study on the heterogeneity in cannabis composition, Burgdorf, Kilmer and Pacula (2011) concluded that research on the THC to CBD ratio should continue in order to evaluate the health effects of both recreational and medical cannabis.

### CANNABIS USE AMONG ADOLESCENTS AND YOUNG ADULTS – CONSEQUENCES OF EARLY USE

According to the Substance Abuse and Mental Health Services Administration’s National Survey of Drug Use and Health (NSDUH), cannabis is the most frequent first specific drug associated with initiation of illicit drug use among 2.6 million persons aged 12 or older. In 2011, an estimated 1.5 million past year cannabis users started before the age of 18. The NSDUH shows more than 47% of Americans 12 years of age and older have used cannabis at some point

**EDITOR’S NOTE:** The field of substance abuse treatment and prevention is approaching a critical time of change. As more states move toward increased legal or decriminalized access to marijuana, we need reliable information on how substances like marijuana affect youth, a group which is disproportionately impacted. And while a drug like marijuana may well become legal, that doesn’t make it *safe*. Unfortunately, the data tell us that there will likely be an increase in the percentage of youth who abuse marijuana. Ventura County, recognized as a leader for its alcohol and drug policy and prevention work, is in a key position to equip local behavioral health treatment providers with a factual presentation of the best evidence on marijuana and its impact on the developing adolescent brain. I am pleased to add this new white paper on *What the Science Says About Adolescent Use of Cannabis* to our series on this critically important subject.

PATRICK ZARATE, DIVISION MANAGER, VENTURA COUNTY BEHAVIORAL HEALTH, ALCOHOL & DRUG PROGRAMS • APRIL 2016

during their life; more than any other illicit drug use. The survey also shows an estimated 16.7% of past year cannabis users ages 12 or older used cannabis on 300 or more days within the previous 12 months. This translates into daily or near-daily use of cannabis by nearly 5.0 million persons. An estimated 39.1% of current cannabis users age 12 or older (7.1 million people) used cannabis 20 or more times during a 30-day period (SAMHSA, 2012).

The Monitoring the Future (MTF) survey conducted by the University of Michigan has for decades been a trusted source for national trends in adolescent drug use. The 2015 MTF survey found that cannabis use remained steady among 8th graders at 6.5%, 10th graders at 14.8%, and 12th graders at 21.3%. 6% of 12 graders reported daily use of cannabis. For the first time, daily cigarette use (5.5%) was lower than daily cannabis use (6%) among high school seniors. In addition, only 31.9% of 12th graders reported a perception that regular use of cannabis puts the user at great risk compared to 78.6% in 1991. This indicates a “softening of perceived risks” (Johnston & Miech, 2015). The full report for the 2015 Monitoring the Future survey is available at [www.drugabuse.gov/related-topics/trends-statistics/monitoring-future](http://www.drugabuse.gov/related-topics/trends-statistics/monitoring-future).

The NSDUH report collects data on perceived risk of harm as an important factor influencing whether young persons will use particular substances. The report found that, among youths aged 12 to 17, the percentage reporting a perceived great risk of harm in smoking cannabis once or twice a week declined from 54.6% in 2007 to 44.8% in 2011. Combined data for 8th and 10th graders showed a similar decline in perceived risk over this time period from 69.4 to 61.8% (SAMHSA, 2012).

...only 31.9 percent of 12th graders reported a perception that regular use of cannabis puts the user at great risk compared to 78.6 percent in 1991. This indicates a “softening of perceived risks”...

## LOCAL TRENDS

A sample of 132 youths participating in treatment at three Ventura County Behavioral Health, Alcohol and Drug Programs sites (Simi, Ventura and Oxnard) over the course of 12 months (2014/2015) was examined as an indicator of local cannabis use among adolescents ages 12 to 17 years old.

<b>TOTAL CANNABIS USING CLIENTS ( n=129)</b>	98 percent
<b>TOTAL AVERAGE AGE OF ONSET</b>	13 years of age
<b>TOTAL CANNABIS FIRST DRUG (n=63)</b>	49 percent
<b>TOTAL CANNABIS DAILY USERS (n=53)</b>	41 percent
<b>TOTAL ALCOHOL FIRST DRUG (n=23)</b>	17 percent
<b>TOTAL STARTED WITH CANNABIS &amp; ALCOHOL (n=34)</b>	26 percent
<b>TOTAL STARTED W/ CANNABIS ALONE OR CANNABIS AND ALCOHOL (n=97)</b>	75 percent

As shown in the above table, 98% of the youth in a local treatment program used cannabis and the majority began use at age 13. As discussed below, “early onset” of cannabis use is one of the risk factors for the development of cognitive deficits and psychotic disorders. For nearly half of these youths, cannabis was the first substance of abuse. It should be noted that 75% of this treatment population began drug use with either cannabis alone or cannabis and alcohol in combination at the same age. This is of particular concern because there is evidence indicating the presence of alcohol in the bloodstream causes a faster absorption of THC. This can lead to the cannabis having much stronger effects than it would normally have (Alcohol and Drug Abuse Institute, 2013).

For the local youth who reported use of drugs in addition to cannabis and/or alcohol, use typically began 1 to 3 years after onset of cannabis use. (This survey was conducted by Dr. Gertson from the Screening Forms used at the sites).

Studies have shown that early age of onset is a strong predictor of rapid progression to a substance use disorder (Winters & Arria, 2011). Individuals who begin use of cannabis in adolescence are between 2 to 4 times more likely to have symptoms of cannabis dependence within 2 years of first use (Chen, Storr & Anthony, 2009).

## THE ADOLESCENT BRAIN

Neuroscientific research indicates that adolescents are particularly vulnerable to the effects of cannabis because of the unique nature of the adolescent brain.

At birth, the brain is only about 40% the size it will be in adulthood (Jensen, 2015). During adolescence the neural systems that control emotion and reward-seeking (the limbic system) are fully developed while the brain regions associated with executive functioning (for example

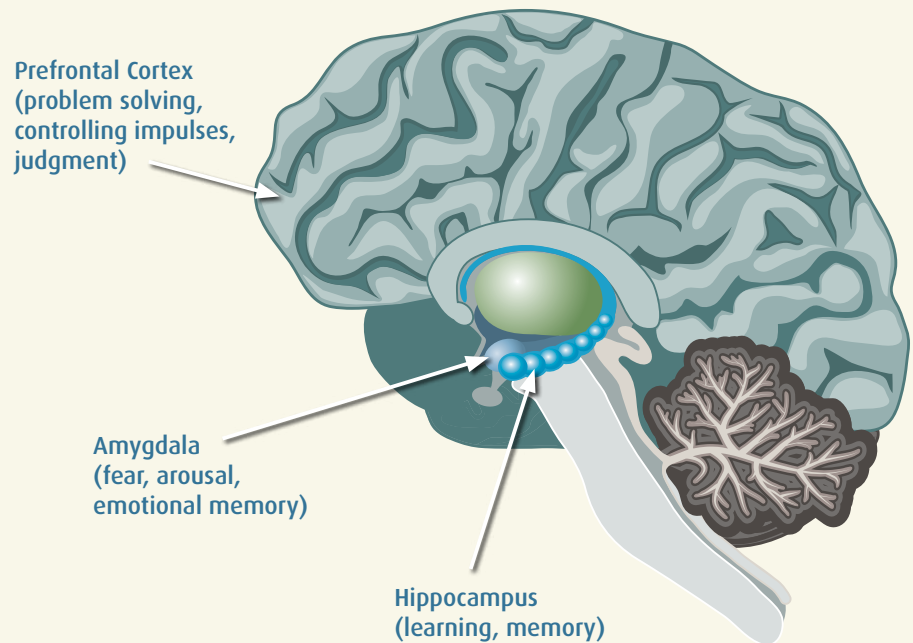
problem solving, planning, working memory, and emotional regulation) which includes the prefrontal cortex are still maturing (Lisdahl, et al., 2013). This non-uniform maturation pattern may contribute to an increase in risk taking and novelty seeking during adolescence (Dahl, 2004). The “high” produced by drugs floods the reward system which may create a strong drive to repeat the experience. The immature brain is more likely to repeat the experience without adequately considering the consequences (Andersen & Teicher, 2009). As the experience of drug taking is repeated, the brain reinforces the neural links between pleasure and drug-taking, making the association stronger and stronger, altering key brain areas necessary for judgment and self-control (NIDA, 2014).

Additionally, long-term heavy cannabis use has been shown to decrease the size of the hippocampus, a brain structure important for learning and memory, by 12%; and the amygdala by 7% (Yucel, Solowij et al., 2008).

A study by Winters and Lee (2008) found that only 4.4% of individuals who began smoking cannabis after age 21 became addicted within the first two years of onset **while 17.4%** of 13-year olds became addicted within the same time period.

In summary, the relevant brain research indicates that adolescents are in a critical stage of development which makes them prone to lasting adverse behavioral, emotional and cognitive effects of cannabis. The California Society of Addiction Medicine (CSAM) therefore concludes that the preponderance of scientific evidence provides adequate rationale for public policies that “deter, delay and detect” child and adolescent cannabis use (CSAM, 2009).

During adolescence, the prefrontal cortex and other areas associated with “executive functions” – including problem solving, controlling impulses and emotional regulation – are still maturing.



Long-term heavy cannabis use has been shown to:

- Decrease the size of the hippocampus by 12%
- Decrease the size of the amygdala by 7%

## IS CANNABIS ADDICTIVE?

In addition to other harmful effects of adolescent cannabis use, the question of addictive potential for cannabis should be addressed.

Although, as with all substances of abuse, not everyone who uses cannabis develops an addiction, the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) found that approximately 9% of cannabis users will become addicted as defined by the criteria in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (Lopez-Quintero, et al., 2011).

For those who start using cannabis during adolescence, the number increases to 17% and to between 25 and 50% among daily cannabis users (Hall & Degenhardt, 2009). In addition, Hall and Degenhardt (2007) found that early and heavy use of cannabis increases the risk of use and addiction to other drugs.

A drug is considered to be potentially addictive if it meets criteria such as the existence of a specific withdrawal syndrome related to cessation of the drug. There is scientific recognition of a cannabis withdrawal syndrome which includes: irritability, sleep difficulty, depression, anxiety and craving (Volkow, et al., 2014). These symptoms may undermine attempts to stop use of cannabis and contribute to relapse.

It is important to recognize that the potency of cannabis, as measured by concentration of THC, has increased by about 300% (ElSohy, 2014). The Office of National Drug Control Policy ([www.whitehouse.gov/ondcp](http://www.whitehouse.gov/ondcp)) advises that cannabis growers have been genetically altering marijuana plants to increase the percentage of THC which increases the potency of this substance. Inaba and Cohen (2011) report that high-potency cannabis is also due to increased indoor cultivation and a multitude of growing tips and techniques available on the Internet. The Potency Monitoring Project at the University of Mississippi reported that the average cannabis THC concentration increased from 3.7% in 1988 to 12.55% in 2013 (ElSohy, 2014).

DiForti et al., (2015) showed that frequent use of high potency cannabis demonstrated the highest risk for psychotic disorders (up to 24% of first episode cases of psychosis). **“Our findings show the importance of raising awareness among young people of the risks associated with the use of high-potency cannabis. The need for such public education is emphasized by the worldwide trend of liberalization of the legal constraints on cannabis and the fact that high potency varieties are becoming much more widely available” (DiForti, et al., 2015, p.6).**

**SYNTHETIC CANNABINOIDS** (such as “spice” or “K2”) can be even more powerful than THC. The acute effects of synthetic cannabis include trouble breathing, heart palpitations, panic attacks, hallucinations, vomiting and seizures. “Spiceophrenia” is a term used to describe the agitation, anxiety, paranoia and psychosis that can result from synthetic cannabinoids (Murray, 2015). The June 12, 2015 *Morbidity and Mortality Weekly Report* produced by the Centers for Disease Control and Prevention reported a 229 percent increase in calls to poison centers between January and May 2015 as compared to the same period in 2014. A major adverse effect, i.e., “symptoms that are life-threatening or result in substantial residual disability or disfigurement,” was experienced by 11.3% of the callers. Fifteen deaths were reported in 2015 which is a three-fold increase over the five deaths reported in 2014. Most of the people who call poison centers for bad reactions to synthetic marijuana are between 20 and 29 years old. The New York City Department of Health and Mental Hygiene in an Advisory dated April 17, 2015 described a sharp increase of visits to Emergency Departments beginning on April 8 from individuals who were experiencing “severe adverse events after suspected ingestion of synthetic cannabinoids.” The 120 cases identified through April 15 was more than 6 times the number of average weekly visits reported throughout the year.

## RISKS OF ADOLESCENT CANNABIS USE

Multiple scientific studies, including longitudinal research designs, have shown adverse effects on cognition, educational achievement, as well as psychiatric conditions, particularly schizophrenia, related to early onset of cannabis use.

### Cognition and Educational Achievement

As discussed above, the prefrontal cortex does not reach full maturity until the early twenties, and many complex executive processes of the brain are still developing during adolescence. Therefore, considerable research has explored how early onset of cannabis use affects cognition. Given the common finding of a relationship between early onset of cannabis and cognitive deficits, the authors of several of the studies described below emphasize the need for prevention or delay of cannabis use in adolescence and recommend efforts to ensure that cannabis legislation consider the potentially adverse developmental effects of this drug.

Data from three Australasian cohort studies involving over 6000 participants were analyzed to examine the associations between age of onset of cannabis use and educational achievement (Horwood et al., 2010). The authors compared data on age of onset use across three groups (below age 15, between ages 15 and 17, and never before age 18) and three educational outcomes (high school completion, university enrollment, and degree attainment). The study found that there were statistically significant associations between age of onset and all achievement outcomes such that rates of attainment were highest for those who had not used cannabis prior to age 18 and lowest for those whose first use occurred before age 15. The authors suggest that early use of cannabis may contribute up to 17% of the rate of failure to obtain high school completion, university enrollment and degree attainment. Below is a table reflecting some of the results of this study regarding academic achievement and onset of cannabis use. (Horwood, et al., 2010).

ACADEMIC ACHIEVEMENT	CANNABIS USE DURING AGES 15-17	NO USE BEFORE AGE 18
High school degree	1.9 times more likely to get degree than adolescent who uses before age 15	3.6 times more likely to get degree
College enrollment	1.5 times more likely to enroll than adolescent who uses before age 15	2.3 times more likely to enroll
College degree	1.9 times more likely to get degree than teen who uses before age 15	3.7 times more likely to get degree

In a study assessing the effects of cannabis use on verbal learning and memory, Solowij et al., (2011) found that, despite relatively brief exposure, adolescent cannabis users demonstrated similar memory deficits to those reported in adult long-term heavy users. There was strong evidence of greater impairment the earlier cannabis use commenced. The authors report that the vast majority of memory performance outcome measures worsened as a function of quantity, frequency, duration and early age of onset of cannabis use.

**“The fact that the young cannabis users within the current study, with far less exposure to cannabis over an average of 2.4 years, showed similar significantly impaired performance relative to their age-matched counterparts as adult users with 24 years use, suggests indeed greater adverse effects of cannabis use on the developing brain.” (Solowij et al., 2011, p.12)**

Meier et al., (2012) tested the association between persistent cannabis use and neuropsychological decline in an attempt to determine whether decline is concentrated among adolescent-onset cannabis users. The subjects evaluated

in this study were members of the Dunedin, New Zealand cohort which is a longitudinal prospective study of a birth cohort of 1,037 individuals who were followed from birth to age 38. The use of cannabis was ascertained in interviews at ages 18, 21, 26, 32 and 38 years. Neuropsychological testing was conducted at age 13, before initiation of cannabis use, and again at age 38 following a pattern of persistent cannabis use. The results of the study indicated that persistent cannabis use was associated with neuropsychological decline across five domains of functioning (executive function, memory, processing speed, perceptual reasoning and verbal comprehension), even after controlling for years of education. These deficits continued even after one or more years of abstinence from cannabis use.

Silins et al., (2014) integrated data from three large, longitudinal studies conducted in Australia and New Zealand, including the Dunedin study discussed above. They found that individuals who were daily cannabis users prior to age 17 had reductions in the odds of high school completion and degree attainment compared with those who never used cannabis. They also found that adolescent daily cannabis users were 18 times more likely to develop a cannabis use disorder, 8 times more likely to use other illicit drugs and 7 times more likely to attempt suicide in young adulthood.

Results of multiple studies comparing subjects who began cannabis use prior to age 16 (early onset use) with those whose use followed age 16 were reported by Gruber et al., (2012). The results of these studies found that early onset users demonstrated deficits in tests measuring neurocognitive performance including tasks that require executive control, decision-making, and abstract thinking. The cannabis users in this study had more difficulty than controls in ability to inhibit inappropriate responses and could not adequately utilize feedback to improve their performance. Similarly, Lisdahl et al., (2013) conducted a meta-analysis of studies examining the impact of early onset cannabis use on neurocognition. They found that, consistent with age of onset data, chronic cannabis use during adolescence is associated with cognitive deficits such as complex attention, verbal memory, sequencing ability, psychomotor speed and reduced inhibitory control.

***“It is important to not only reduce symptoms of drug abuse and dependence, but *delay the onset of regular use* from early teen years to early adult years in order to prevent long-term neuronal damage and ensure optimal brain health and cognitive functioning in youth.” (Lisdahl et al., 2013, p.13)***

## **Psychotic Disorders**

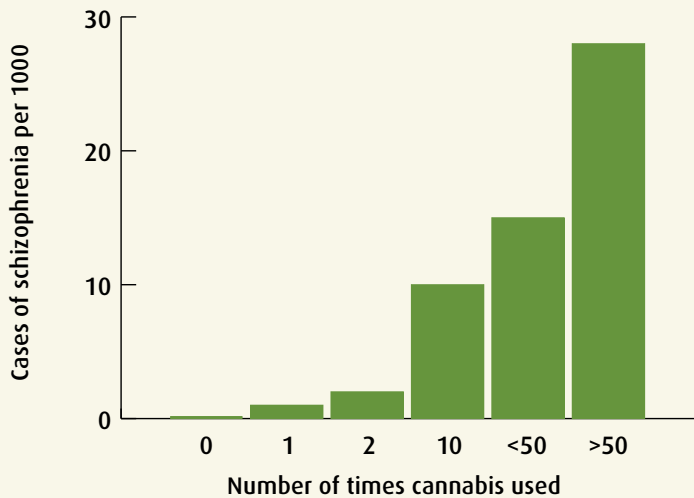
There is extensive scientific literature regarding the risks of cannabis use in the development of psychotic disorders, including schizophrenia. The studies included in this section are those most well-recognized and the most frequently cited in this body of literature.

***“COULD IT BE ALL THE MARIJUANA? Beginning in the mid-1980s, psychiatrists like me who specialize in schizophrenia started seeing an increasing number of previously well-functioning teenagers whose school performance had gradually dropped off, who had become increasingly alienated from their friends, and who then started having paranoid delusions and hallucinations. These patients puzzled us because most had been bright and sociable and showed no evidence of the usual risk factors for schizophrenia (e.g., family history, developmental insult to the brain). Family and friends would often ask, “Could it be all the marijuana they have been smoking?” We would confidently reassure them that they were mistaken and tell them that cannabis was known to be a safe drug. How wrong we were! Twenty-five years later, there is convincing evidence that heavy use of cannabis, especially the high-potency types, increases the risk of schizophrenia up to 5-fold.”***

From ***“Marijuana and Madness: Clinical Implications of Increased Availability and Potency” (Murray, 2015)***

One of the first longitudinal studies conducted on the long-term effects of cannabis was the Swedish conscription study (Andreasson et al., 1988). The original study consisted of 50,087 Swedish men who were examined for compulsory military training during autumn 1969 to spring 1970. The conscripts were ages 18-19 at the time of the initial data collection. Data from this cohort was followed for 15 years. The results of the study indicated a statistically significant risk for schizophrenia among high consumers of cannabis (use on more than fifty occasions) compared with non-users. Multiple additional follow-up studies have been conducted with this cohort over the past 26 years.

**Figure 1. Risk of schizophrenia in conscripts followed up to 15 years**



*Adapted from Andreasson et al., (1988)*

Using additional analysis with the same cohort described above, Zammit et al., (2002) showed that “heavy cannabis users” by the age of 18 were 6.7 times more likely than non-users to be diagnosed with schizophrenia 27 years later. The risk was reduced but remained significant after controlling for confounding variables such as disturbed behavior, low IQ score, urban background, and poor social integration.

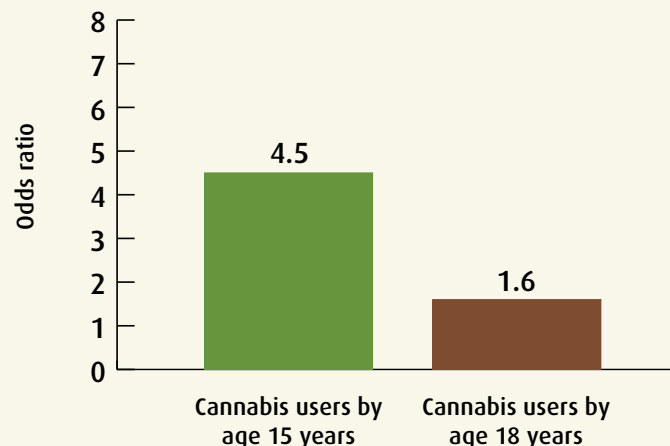
A composite of follow-up studies to the Swedish conscription study was published by Manrique-Garcia (2014) through the Department of Public Health

Sciences at the Karolinska Institute. Schizophrenia patients with a history of cannabis use showed a higher median duration of first hospital episode than those without (59 days v. 30 days). Patients with a history of cannabis use also had a higher median rate of readmission (10 times v. 4 times). The total number of hospital days was higher in patients with a history of cannabis use compared with those without (547 days v. 184 days).

Arseneault et al., (2002) analyzed data from a representative group of the Dunedin, New Zealand study (759 subjects) to investigate the risk for adult psychosis among adolescent cannabis users. In this cohort, cannabis use by age 15 and age 18 was found to be associated with more schizophrenia symptoms at age 26. The association was found to be stronger with earlier onset.

In a follow up study, Arseneault et al., (2004) concluded that cannabis use is not a sufficient cause for later psychosis because the majority of adolescent cannabis users do not develop schizophrenia in adulthood. However, it is likely to be part of a “causal constellation” that leads to adult schizophrenia. The authors conclude, “Cannabis use among psychologically vulnerable adolescents should be strongly discouraged by parents, teachers, and health practitioners . . . policy and law makers should concentrate on delaying onset of cannabis use” (Arseneault, et al., 2004, p. 115).

**Figure 2. Risk of schizophrenia-like psychosis by age 26 years**



*Adapted from Arseneault et al., (2002)*

The Christchurch, New Zealand study is a general population birth cohort that has examined the development of the participants for more than 20 years. Fergusson et al., (2003) found concurrent associations between cannabis dependence disorders and risk of psychotic symptoms at ages 18 and 21. Individuals who met the diagnostic criteria for cannabis dependence disorder at age 18 had a 3.7 fold increased risk of psychotic symptoms than those without cannabis dependence. This risk was 2.3 times higher at age 21 years. Leeson et al., (2012) found that cannabis use brings forward the onset of psychosis in people who otherwise would have good prognostic features, as indicated by premorbid cognition and social function.

**“Thus, the earlier age at onset in cannabis users could be due to the toxic action of cannabis rather than an intrinsically more severe illness. Public health policies aimed at preventing cannabis use, if successful, might therefore delay the onset of psychosis in vulnerable young people and improve outcomes further.” (Leeson et al., 2012, p. 879).**

## **Conclusions and Cautions in Interpreting the Data Related to “Cannabis-Induced” Psychotic Disorders**

Data indicating a correlation between or among disorders should always be interpreted with caution. Correlation does not necessarily signify causation. Not all adults with schizophrenia used cannabis in adolescence, and not all adolescent users develop schizophrenia or other psychotic disorders. Although there may be incidents of cannabis as a direct cause of psychosis, the majority of experts concur that cannabis use is likely part of a constellation of risk factors that contribute to the onset of a psychotic disorder like schizophrenia. Most studies conclude that the risk is greater with early onset of cannabis use, longer duration of use, and a positive family history of psychosis (e.g., genetic risk factors). Radhakrishnan, Wilkinson and D’Souza (2014) suggest that cannabis-induced psychotic disorder may be a distinct sub-type of a schizophrenia disorder spectrum. These authors conclude, “It is likely that cannabis is an important component cause in the development of psychotic disorders. This causal role is likely magnified when cannabis exposure occurs at an earlier age, in greater quantities, and over a longer time-course” (p. 15). In a review of the evidence linking cannabis use to psychosis, Burns (2013) suggests that individuals who present with early initiation and long-term cannabis use may present with psychotic symptoms which would not have occurred in the absence of cannabis use.

## **Summary of Risks of Adolescent Cannabis Use**

Following a review of the research over the past 20 years regarding adolescent use of cannabis, Hall (2014) concludes that 1 in 6 individuals who begin cannabis use in adolescence are at risk of developing a dependence disorder. In addition, adolescent use increases the likelihood of other illicit drug use, appears to produce cognitive impairment, impairs educational attainment as compared to non-users, and approximately doubles the risk of experiencing a psychotic disorder, including schizophrenia, especially if there is a family history of psychosis.

**The question, “Does cannabis use cause schizophrenia?” might better be phrased as, “What are the risk factors that link cannabis use to psychotic disorders, particularly schizophrenia?”**

**These risk factors include:**

- **Early onset of use**
- **High frequency of use**
- **Long duration of use**
- **Genetic predisposition**
- **Family history of psychotic disorders**



*“Repeated marijuana use during adolescence may result in long-lasting changes in brain function that can jeopardize educational, professional, and social achievements. However, the effects of a drug (legal or illegal) on individual health are determined not only by its pharmacologic properties but also by its availability and social acceptability. In this respect, legal drugs (alcohol and tobacco) offer a sobering perspective, accounting for the greatest burden of disease associated with drugs not because they are more dangerous than illegal drugs, but because their legal status allows for more widespread exposure.”*

– Volkow, N.D. et al. (2014)

Nora D. Volkow, M.D., is Director of the National Institute on Drug Abuse at the National Institutes of Health.

## **INTERVENTIONS FOR ADOLESCENT CANNABIS ABUSE**

Adolescent cannabis use should be identified and addressed as soon as possible to prevent or minimize the negative consequences described in the previous sections of this paper. Intervention can be beneficial even if the young person is using the drug but not demonstrating evidence of abuse or dependence. Treatment can begin at any stage of substance use including “experimentation” since this could lead to continued and increased use. Routine medical visits are an ideal time for the pediatrician or family practitioner to inquire about drug use, and standardized screening tools are available for use in primary care settings and can indicate when referral for specialized treatment is recommended. It should also be noted that treatment can work even if mandated and not voluntary (NIDA, 2014).

Treatment should be tailored to the unique needs of the individual adolescent and should focus on the whole person not just on his or her drug use, and approaches may include Cognitive-Behavioral Therapy (CBT), Contingency Management (CM) and Motivational Enhancement Therapy (MET). Appropriate treatment considers an adolescent’s level of psychological development, gender, relations with family and peers, how well he or she is doing in school, the larger community, cultural and ethnic factors, and any special physical or behavioral issues. It is important to identify any other mental health conditions and/or academic problems the adolescent may be experiencing (NIDA, 2014).

Treatment can help the adolescent stop drug use by increasing the motivation to change. This is accomplished by providing incentives, building skills to resist urges to use and/or peer pressure, and replacing drug use with more constructive activities. Monitoring for abstinence and relapse is also an important part of an effective approach. Length of treatment and titration to aftercare sessions should be individualized depending on the needs of the participant (NIDA, 2014).

## **CONCLUSIONS**

Following a review of the research over the past 20 years regarding adolescent use of cannabis, Hall (2014) concludes that 1 in 6 individuals who begin cannabis use in adolescence are at risk of developing a dependence disorder. In addition, adolescent use increases the likelihood of other illicit drug use, appears to produce cognitive impairment, impairs educational attainment as compared to non-users, and approximately doubles the risk of experiencing a psychotic disorder, including schizophrenia, especially if there is a family history of psychosis (Hall, 2014).

As more states, including California, move forward with the legalization of both medical and recreational use of cannabis, it is important to educate the public about the risks of adolescent use of this drug and to ensure that policies are included in the legislation which will provide for the prevention and treatment of early onset use. The majority of experts referred to in this report emphasize the need to “deter, delay and detect” use of cannabis during adolescence due to evidence from multiple scientific studies demonstrating that regular use during adolescence is associated with cognitive deficits, educational under-achievement and increased risk of psychotic disorders, especially in those with genetic vulnerability.

**LINDA GERTSON, PH.D.** is a Clinical Psychologist currently serving as Clinical Supervisor for Ventura County Behavioral Health in the Alcohol and Drug Programs Division. She has been specializing in addictions and co-occurring disorders for over 25 years. Dr. Gertson was responsible for the development of the VCBH Integrated Dual Diagnosis Treatment Program which was awarded the SAMHSA Science to Service Award for high-fidelity implementation of an evidence-based co-occurring disorders program. She is the recipient of the 2011 Ventura County Medical Resource Foundation's Fainer Award as "Psychologist of the Year." Dr. Gertson is currently responsible for the integration of mental health services in the VCBH "A New Start for Moms" perinatal treatment program.

## REFERENCES

- Alcohol and Drug Abuse Institute, University of Washington (2013). Learn about marijuana: Science-based information for the public. [LearnAboutMarijuanaWA.org/factsheets/adolescents.htm](http://LearnAboutMarijuanaWA.org/factsheets/adolescents.htm)
- Andersen, S.L. & Teicher, M. (2009). Desperately driven and no brakes: Developmental stress exposure and subsequent risk for substance abuse. *Neuroscience & Biobehavioral Reviews* 33(4), 516-524.
- Andreasson, S., Allebeck, P. & Engstrom, A. (1988). Cannabis and schizophrenia: A longitudinal study of Swedish conscripts. *Lancet*, ii, 1483-1485.
- Arseneault, L., Cannon, M., Poulton, R., Murray, R., Caspi, A. & Moffitt, T. (2002). Cannabis use in adolescence and risk for adult psychosis: Longitudinal prospective study. *British Medical Journal*, 325, 1212-1213.
- Arseneault, L., Cannon, M., Witton, J. & Murray, R.M. (2004). Causal association between cannabis and psychosis: Examination of the evidence. (2004). *British Journal of Psychiatry*, 184, 110-117.
- Burgdorf, J.R., Kilmer, B. & Pacula, R. L. (2011). Heterogeneity in the composition of marijuana seized in California. *Drug and Alcohol Dependence*, 117(1), 59-61.
- Burns, J.K. (2013) Pathways from cannabis to psychosis: a review of the evidence. *Frontiers in Psychiatry*, 4, 1-12.
- California Society of Addiction Medicine (CSAM). (2009). Impact of marijuana on children and adolescents: Evidence-based information on cannabis/marijuana. [www.csam-asam.org](http://www.csam-asam.org)
- Canadian AIDS Society (2007). Cannabis and HIV-AIDS. Canadian Public Health Society website. February 15, 2007. [www.cpha.ca](http://www.cpha.ca)
- Centers for Disease Control and Prevention, Morbidity and Mortality Weekly Report, June 12, 2015, 64(22), 618-619.
- Chen, C.Y., Storr, C.L. & Anthony, J.C. (2009). Early-onset drug use and risk for drug dependence problems. *Addictive Behavior*, 34, 319-322.
- Dahl, R.E. Adolescent brain development: A period of vulnerabilities and opportunities. In: Dahl, R.E., Spear, L.P., editors. (2004) *Adolescent Brain Development: Vulnerabilities and Opportunities*. Vol. 1021. New York: *Annals of the New York Academy of Sciences*, 1-22.
- DiForti, M., Marconi, A., Carra, E., Fraitetta, S., Trotta, A., Bonomo, M., Bianconi, F., Gardner-Sood, P., O'Connor, J., Russo, M., Stilo, S. A., Marques, T. R., Mondelli, V., Dazzan, P., Pariane, C., David, A. S., Gaughran, F., Atakan, Z., Iyegbe, C., Powell, J., Morgan, C., Lunskey, M. & Murray, R. M. (2015). Proportion of patients in south London with first-episode psychosis attributable to use of high potency cannabis: A case-control study. *Lancet Psychiatry*, 1-6.
- ElSohly, M.A. (2014). Potency Monitoring Program quarterly report number 124. Reporting period: 12/16/2013-03/15/2014. Bethesda, MD: National Institute on Drug Abuse.
- Fergusson, D. M., Horwood, L. I. & Swain-Campbell, N.E. (2003). Cannabis dependence and psychotic symptoms in young people. *Psychological Medicine*, 33, 15-21.
- Gruber, S.A., Sagar, K.A., Dahlgren, M.K., Racine, M. & Lukas, S. E. (2012). Age of onset of marijuana use and executive function. *Psychology of Addictive Behavior*, 2012 26(3), 496-605.
- Hall, W. (2014). What has research over the past two decades revealed about the adverse health effects of recreational cannabis use? *Addiction Monograph*, 1-17.
- Hall, W. & Degenhardt, L. (2007) Prevalence and correlates of cannabis use in developed and developing countries. *Current Opinion in Psychiatry*, 20, 393-397.
- Hall, W. & Degenhardt, L. (2009). Adverse health effects of non-medical cannabis use. *Lancet*, 374, 1383-1391.
- Horwood, L.J., Fergusson, D.M., Hayatbakhsh, M. R., Najman, J. M., Coffey, C., Patton, G. C., Silins, E. & Hutchinson, D. M. (2010). Cannabis use and educational achievement: findings from three Australasian cohort studies. *Drug and Alcohol Dependence*, 110, 247-253.

- Inaba, D.S. & Cohen, W.E. (2011). *Uppers, Downers, All Arounder: Physical and Mental Effects of Psychoactive Drugs (7th Edition)*. Medford, Oregon: CNS Productions, Inc.
- Iuvone, T., Esposito, G., Esposito, R., Santamaria, R., DiRosa, M. & Izzo, A.A. (2004). Neuroprotective Effect of Canabidiol, a Non-psychoactive Component from Cannabis Sativa, on B-amyloid-induced Toxicity on PC12 Cells. *Journal of Neurochemistry*, 89(1), 134-141.
- Jensen, F.E. (2015). *The teenage brain: A neuroscientist's survival guide to raising adolescents and young adults*. New York, New York: Harper Collins Publishers.
- Johnston, L.D. & Miech, R. (2015) Monitoring the Future National Results on Adolescent Drug Use: Overview of Key Findings, 2015. Ann Arbor: Institute for Social Research, The University of Michigan. Additional information on the MTF Survey, as well as comments from Dr. Volkow, can be found at [www.drugabuse.gov/drugpages/MTF.html](http://www.drugabuse.gov/drugpages/MTF.html). The University of Michigan press release can be found at [monitoringthefuture.org](http://monitoringthefuture.org).
- Leeson, V.C., Harrison, I., Ron, M.A., Barnes, T.R.E. & Joyce, E.M. (2012). The effect of cannabis use and cognitive reserve on age at onset and psychosis outcomes in first-episode schizophrenia. *Schizophrenia Bulletin*, 38(4), 873-880.
- Lisdahl, K.M., Gilbert, E.R., Wright, N.E. & Shollenbarger, S. (2013). Dare to delay: The impacts of adolescent alcohol and marijuana use onset on cognition, brain structure, and function. *Frontiers in Psychiatry*, 4, 1-19.
- Lopez-Quintero, C., Hasin, D.S., Perez de los Cobos, J., Pines, A., Wang, S., Grant, B.F. & Blanco, C. (2011). Probability and predictors of transition from first use to dependence on nicotine, alcohol, cannabis and cocaine: results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *Drug and Alcohol Dependence*, 115, 120-130.
- Manrique-Garcia, E. (2014). Cannabis, schizophrenia and other psychosis: Longitudinal studies on Swedish conscripts. Department of Public Health Services, Karolinska Institutet.
- Meier, M.H., Caspi, A., Ambler, A., Harrington, H., Houts, R., Keefe, R.S., McDonald, K., Ward, A., Poulton, R. & Moffitt, T. E. (2012). Persistent cannabis users show neuropsychological decline from childhood to midlife. *National Academy of Science*, E2657-E2664.
- Monitoring the Future National Survey Results on Drug Use: Key Findings on Adolescent Drug Use* (Institute for Social Research, University of Michigan, 2015).
- Murray, R. (2015) Marijuana and madness: Clinical implications of increased availability and potency. *Psychiatric Times*, 1-5.
- National Institute on Drug Abuse. *Principles of adolescent substance use disorder treatment: A research-based guide*. National Institutes of Health Publication Number 14-7953, January 2014.
- National Institute on Drug Abuse. *The Science of Drug Abuse & Addiction*, Revised September 2015. [www.drugabuse.gov/publications/drugfacts/marijuana](http://www.drugabuse.gov/publications/drugfacts/marijuana)
- New York City Department of Health and Mental Hygiene (2015). Increase in synthetic cannabinoid (marijuana)-related adverse events and emergency department visits, New York City, 2015 Advisory #6.
- Porter, B.E. & Jacobson, C (2013), Report of a parent survey of cannabidiol-enriched cannabis use in pediatric treatment-resistant epilepsy. *Epilepsy Behavior*, 29(3), 574-577.
- Radhakrishnan, R, Wilkinson, S.T. & D'Souza, D.C. (2014). Gone to pot: A review of the association between cannabis and psychosis. *Frontiers in Psychiatry*, 5, 1-24.
- Silins, E., Horwood, J., Patton, G.C., Fergusson, D. M., Olson, C. A., Hutchinson, D. M., Spry, E., Toumbourou, J. W., Degenhardt, L., Swift, W., Coffey, C., Tait, R. J., Letcher, P., Copeland, J. & Mattick, R. P. (2014). Young adult sequelae of adolescent cannabis use: An integrative analysis. *The Lancet*, 1, 286-293.
- Solowij, N., Jones, K.A., Rozman, M. E., Davis, S. M., Ciarrochi, J., Heaven, P. C. L., Lubman, D. I. & Yucel, M. (2011). Verbal learning and memory in adolescent cannabis users, alcohol users and non-users. *Psychopharmacology*, 1-14.
- Substance Abuse and Mental Services Administration, "Results from the 2011 National Survey on Drug Use and Health: Summary of National Findings." NSDUH series H-44, HHS publication no. (SMA) 12-4713 (Rockville, MD: Substance Abuse and Mental Health Services Administration, 2012).
- Volkow, N.D., Baler, R.D., Compton, W.M. & Weiss, S.R.B. (2014). Adverse health effects of marijuana use. *The New England Journal of Medicine*, 370(23), 2219-2227.
- Winters, K.D. & Arria, A. (2011). Adolescent Brain Development and Drugs. *Prevention Researcher*, 18, 21-24.
- Winters K.C. & Lee S. (2008). Likelihood of developing an alcohol and cannabis use disorder during youth: Association with recent use and age. *Drug and Alcohol Dependence*, 92, 239-247.
- Yucel, M., Solowij N., Respondek, C., Whittle, S., Fornito, A., Pantelis, C. & Lubman, D. I. (2008). Regional brain abnormalities associated with long-term heavy cannabis use. *Archives of General Psychiatry*, 65, 694-701.
- Zammit, S., Allebeck, P., Andreasson, S., Lundberg, I. & Lewis, G. (2002). Self-reported cannabis use as a risk factor for schizophrenia: Further analysis of the 1969 Swedish conscript cohort. *British Medical Journal*, 325(7374), 1199-1201.

# PREVENTING TEEN CANNABIS USE

*Adapted from "Protecting our Youth: Options for Marijuana Regulation in California"*

The prevention field has moved far beyond the confines of education and persuasion in understanding and developing effective youth prevention strategies in the context of drugs that are legally available to adults but illegal for youth (i.e., alcohol and tobacco). The science is well-developed and is particularly relevant to the implications for youth if marijuana is legalized in California.

First and foremost, the science has found that the most effective strategies for reducing and preventing youth problems include raising the price, reducing the availability, and restricting youth exposure to commercial advertising for the drugs involved. The National Academy of Sciences, National Institute on Alcohol Abuse and Alcoholism, U.S. Centers for Disease Control and Prevention, the World Health Organization, and the Surgeon General of the United States have documented the scientific evidence regarding these prevention strategies as they apply to alcoholic beverages and have made specific recommendations for their implementation at federal, state and local levels.

Understanding this new approach to prevention involves a paradigm shift from a focus on individual youth (seeking to educate and persuade them to be abstinent and to intervene if they ignore the abstinence message) to a focus on the social, cultural, political and economic environment that youth encounter in their daily lives. The messages youth receive in their everyday community in terms of how a legal drug is made available and marketed are far more

powerful than any countervailing messages they hear from parents, teachers, and health officials.

Environments are shaped by policy, which can be legislative (e.g., laws and regulations), institutional (e.g., company and school) and informal (neighborhood watch programs). Policies are therefore an integral part of any comprehensive drug prevention strategy.

Some examples include:

- Strict controls on commercial sales and furnishing to youth, focusing primarily on adult providers through well-funded compliance check programs;
- Limitations on the sale of products that are attractive to young people or put them at heightened risk of harm;
- Price controls through fees and taxes to maintain relatively high prices over time, although not too high to foster illegal production and sale, with revenues dedicated to compliance, implementation, enforcement, and prevention;
- Restrictions on the number, type, location and sales practices of marijuana retail outlets; and particular attention to product quality, environmental protection, and the prevention of public nuisance activities associated with marijuana cultivation, distribution and sales.

**Learn more: [venturacountylimits.org/mj](http://venturacountylimits.org/mj)**

PREPARED FOR:



VENTURA COUNTY  
**BEHAVIORAL HEALTH**  
ALCOHOL & DRUG PROGRAMS

For more information:

[www.venturacountylimits.org/mj](http://www.venturacountylimits.org/mj)

805-981-6831