THE CARLAT REPORT CHILD PSYCHIATRY

A CME Publication

Subscribe today!
Call 866-348-9279

UNBIASED INFORMATION FOR CHILD PSYCHIATRISTS

Glen Elliott, MD, PhD
Editor-in-Chief

Volume 8, Number 3
May 2017
www.thecarlatchildreport.com

IN THIS ISSUE

Marijuana in Children and Adolescents

Learning Objectives
After reading these articles, you should be able to:
1. Describe some of the current marijuana preparations and their effects on users. **2.** Identify ways clinicians can effectively discuss key information about marijuana use with their patients. **3.** Summarize some of the current findings in the literature regarding psychiatric treatment for children and adolescents.

Cannabis in 2017: Preparations and Modes of Delivery

Mark Elliott, MD, psychiatrist in San Francisco, CA; Timmen L. Cermak, MD, private practice in psychiatry and addiction psychiatry in San Francisco and Marin County, CA

Dr. Elliott and Dr. Cermak have disclosed that they have no relevant financial or other interests in any commercial companies pertaining to these educational activities.

While the age-old tradition of smoking cannabis remains very common, several other options for preparing the drug are becoming widely available and frequently used. In this article, we'll update you on the modern preparations and routes of administration.

The main psychoactive component of cannabis is Δ9-tetrahydrocannabinol

Continued on page 2

In Summary

- Cannabis preparations can be separated into two major categories: inhaled preparations, which include flowers and concentrates, and ingested preparations, which include edibles and medicinal tinctures.
- Current methods of using inhaled preparations include butane hash oil, dabbing, and CO_2 oil.
- Patients can experience intense negative effects from consuming edibles due to their delayed onset and greater difficulty in determining dose amount.

Q&A With the Expert

Adolescent Marijuana Use and Clinical Practice

Timmen L. Cermak, MD

Private practice in psychiatry and addiction psychiatry in San Francisco and Marin County, CA; former president of the California Society of Addiction Medicine

CCPR: Clinicians working with adolescents have seen that kids are frequently using marijuana now—and stronger strains of it. What does this exposure mean for them? Is there any suggestion that marijuana use is affecting development?

Dr. Cermak: There is conclusive evidence that marijuana use, particularly in early adolescence, can affect brain development. The best research on this is probably the Dunedin Study done in New Zealand (Meier MH, *Proc Natl Acad Sci* 2012;109(40):E2657–E2664). Researchers did full neurocognitive testing on 13-year-olds and then followed them at various intervals up to age 38. They demonstrated that certain subpopulations had as much as an 8 point decrease in IQ from age 13 to age 38 with marijuana use. Now, it's important not to over-interpret these results: Importantly, the only ones who demonstrated that much of an IQ change were those who began using at the very earliest ages of adolescence. Those who didn't smoke until they were age 18, say, but then smoked continuously and heavily from then until age 38, did not show a decline in IQ. So, that's the best evidence that youth who begin smoking quite early on are at the highest risk of adverse effects.

Continued on page 3

Cannabis in 2017: Preparations and Modes of Delivery

Continued from page 1

(THC), which is present to some extent in all preparations and is responsible for most of the "high" from the drug. Cannabis plants produce more than 100 other identified cannabinoids, most of which we know little about. One of these, cannabidiol (CBD), has received a lot of attention recently. While it's not as markedly psychoactive as THC, CBD is increasingly believed to be a major, if not *the* major contributor to the therapeutic effects of cannabis. It has been shown to have anxiolytic, antipsychotic, anti-inflammatory, anticonvulsant, and antineoplastic effects. CBD-enriched preparations have become increasingly popular in recent years, and as long as the THC concentrations are less than 0.3%, they are not currently restricted by the DEA (Mead A, *Epilepsy Behav* 2017 doi:10.1016/j.yebeh.2016.11.021).

Cannabis preparations have exploded in diversity. They can be separated into two categories: inhaled preparations (including both flowers and concentrates) and ingested preparations.

Inhaled preparations

Flowers (or buds)

Flowers are what most people call "pot," "weed," "trees," "herb," "bud," etc. They are harvested primarily from unpollinated female cannabis plants (pollination creates unwanted seeds) and trimmed away to leave only the buds, which are high in cannabinoid content. The concentration of THC in these preparations can vary widely—from less than 0.3% to 30% or so, with most modern samples in the 4%–20% range. They are generally smoked, either in cigarettes by themselves as a "joint," with tobacco as a "spliff," in a cigar as a "blunt," or as "bowls" smoked with pipes of various sorts, including "bongs" (water pipes). When people smoke flowers, the effect begins within seconds, with peak concentrations around 10 minutes and subjective effects lasting 3–5 hours (Huestis MA, *Chem & Biodiversity* 2007;4(8):1770–1804). Smoking flowers likely remains the most common route of administration in the US today, especially when used recreationally and abused.

Concentrates

Concentrates are more diverse. These include kief, hash, butane hash oil (BHO), CO_2 oil, and many more, all of which are derived from cannabis plant material. The cannabinoids are concentrated before consumption by various means; their cannabinoid content is largely dependent on the source material and the means of extraction.

Kief (pronounced "keef"), the simplest of the concentrates, is merely the separated and collected dried resin, or trichomes, of the plant. Its THC concentration is generally around 20%–60%, many times higher than the concentration in flowers. Kief is usually added to flowers or tobacco to enhance cannabinoid content; it can also be smoked by itself in a pipe.

Hash (also known as "hashish" or "water hash"), one of the oldest and best-known concentrates, is comprised of compressed and purified kief. It has a long history in the Middle East and in much of Europe as the primary mode of cannabis use. In modern preparations, hash has been estimated to contain 50%–80% THC. It is generally added to tobacco and smoked as a cigarette (a spliff), added to cannabis flowers, or used in a pipe or bong. This preparation is relatively unusual in the US.

BHO has undergone a dramatic popularity increase in recent years. It can be prepared in several forms that go by different names—"oil," "wax," "budder," and "shatter," for instance. These are all created by running butane over whole plant material or hash, then collecting and evaporating the excess butane. This leaves a thick substance rich in cannabinoids, with THC concentrations ranging from 60%–90%. Depending on the process, the product may be hard and clear (shatter), sticky and soft (wax), or somewhere in between (budder). The concentrates can be consumed in many ways: through addition to tobacco or other cannabis products, with electronic vaporizers, and through increasingly notorious "dabs."

Dabbing has gained significant popularity since around 2011 (https://trends.google.com/trends/explore?date=all&q=dab%20weed). It involves using "dab rigs," which are elaborate bongs created specifically to vaporize concentrates. In a standard bong, the cannabis is packed into a bowl, and the smoke is drawn through a column of water. In dab rigs, in place of the bowl there is a "nail," a piece of metal that can be heated with a blowtorch or, in the higher-tech versions, charged up and heated electronically with lithium ion batteries. The concentrate is picked up with the dab tool (looks like a dental pick) and then laid directly onto the heated nail. The vapor is then inhaled through the water column.

Formal research has been sparse, but dabs have achieved notoriety for their reportedly intense, short high and elaborate ritual, as well as the

Continued on page 3

EDITORIAL INFORMATION

Publisher: **Daniel Carlat, MD**

Editor-in-Chief: **Glen Elliott, MD, PhD,** is the former director of child and adolescent psychiatry and emeritus professor of clinical psychiatry at the University of California San Francisco, CA.

Deputy Editor: **Talia Puzantian, PharmD, BCPP,** is associate professor at the Keck Graduate Institute School of Pharmacy in Claremont, CA.

Executive Editor: **Janice Jutras**

Founding Editor: **Caroline Fisher, MD, PhD,** is training director and chief of child psychiatry at Samaritan Health Systems in Corvallis, OR.

Editorial Board:

Jonathan C. Gamze, MD, is a psychiatrist in private practice in Arlington Heights, IL.

Georgia Gaveras, DO, is the director of training and education in child and adolescent psychiatry and the director of the child and adolescent psychiatry consultation liaison service at Mount Sinai St. Luke's Hospital in New York, NY.

Peter Parry, MBBS, is consultant child & adolescent psychiatrist and senior lecturer at Flinders University in Adelaide, Australia.

John Preston, PsyD, is a professor emeritus at Alliant International University in Sacramento, CA.

Jess Shatkin, MD, MPH, is vice chair for education and professor of child & adolescent psychiatry and pediatrics at NYU School of Medicine in New York, NY.

All editorial content is peer reviewed by the editorial board. Dr. Fisher, Dr. Gamze, Dr. Gaveras, Dr. Parry, Dr. Preston, Dr. Puzantian, and Dr. Shatkin have disclosed that they have no relevant financial or other interests in any commercial companies pertaining to this educational activity.

This CME/CE activity is intended for psychiatrists, psychiatric nurses, psychologists, and other health care professionals, with an interest in the diagnosis and treatment of psychiatric disorders.

Cannabis in 2017: Preparations and Modes of Delivery

Continued from page 2

potential danger of working with volatile, flammable liquids in the preparation of BHO. Many suspect that dabs have more addictive potential than other routes of administration, and one survey found that users reported more tolerance and withdrawal with dabs than with other means (Loflin M and Earleywine M, *Addictive Behav* 2014;39(10):1430–1433).

CO_2 oil is produced by pressurizing CO_2 into its liquid form. A newer process, it requires elaborate machinery and produces a purer concentrate that can be used in different applications compared to BHO. These are currently primarily being distributed as premade cartridges for "vape pens," which have become very popular in cannabis dispensaries.

Ingested preparations

Edibles have been around for a very long time and are relatively simple to prepare. In general, they are created by extracting cannabinoids from plant material with an edible fat, such as butter or cooking oil. This potent fat is then used to prepare a myriad of products, from brownies to lollipops to Rice Krispies® treats. Compared with smoking, oral administration produces a delayed and less intense but much longer effect, with peak blood concentrations at 2 to 6 hours after ingestion and subjective effects lasting as long as 24 hours.

It is important to note that, while peak blood concentrations are lower in an edible preparation compared to smoking the same dose of cannabinoid, it is much more difficult to determine appropriate edible dosing due to the delayed onset, and people are much more liable to take a larger dose than they intend. This may lead to overwhelming experiences that can land a person in the emergency room, including severe anxiety, paranoia, perceptual disturbances, and tachycardia. Edibles can also be accidentally ingested by toddlers or children.

Medicinal tinctures were the primary mode of preparation prior to the criminalization of cannabis in the US in the early 20th century. These days, they are generally prepared as a source of CBD for therapeutic purposes.

CCPR VERDICT: The medicalization of the cannabis plant has accompanied a rapid increase in the variety of available cannabis preparations. Particularly in states with medical cannabis laws, these preparations are now being used and abused by many patients. It is important for clinicians to be familiar with some of these trends to be able to competently talk with patients about their drug use. Only time will tell how these preparations transform trends in use and abuse, and how they will affect cannabis addiction in our patients.

Expert Interview

Continued from page 1

CCPR: Any other possible effects on cognitive functioning with early marijuana use?

Dr. Cermak: Most of the effect is on memory, attention, and executive function. With respect to this last domain, adolescents who have been abstinent for 28 days still can show some executive function impairment.

CCPR: What points would you make for clinicians working with children and adolescents?

Dr. Cermak: Delay, delay, delay. The more success we have getting kids to delay onset of use, the more protection we provide.

CCPR: How are adolescents who start smoking a bit later, say at age 16, likely to be affected?

Dr. Cermak: The primary effect for those who begin a little bit later in adolescence is on their education, which can be life-changing. Youth who smoke regularly during adolescence, even if they don't start at age 13, are likely to be earning about two-thirds as much as the average person at age 30. That economic impact predominantly comes from interfering with education right at the point where the adolescent's educational trajectory is being determined. If you spend a couple of years stoned and not paying attention to your education, then stop smoking and clear your mind, you're still apt to go to a less rigorous college—or maybe not get into college at all—because of how poorly you did in the 10th and 11th grade.

CCPR: How do you think marijuana affects the ability of adolescents to mature normally?

Dr. Cermak: Everyone moving through adolescence must master numerous psychological developmental tasks in order to navigate their way from dependence on others to a place of independence early in adult life. Unfortunately, marijuana use often substitutes for the psychological work needed to achieve those goals.

CCPR: Can you give us an example?

Dr. Cermak: We all know that development of autonomy is essential. With marijuana, adolescents need only to light up to become a marijuana smoker, which, in many people's minds, is a step toward autonomy. They may think, "I'm told not to do this; however, I do it, so I'm an adult. I'm making my own decisions." And they join a subculture that is attractive in many ways, creating a sense of affiliation outside of the family. But it's an affiliation based on a narrow characteristic: drug users who share the same habit. It doesn't come from developing the capacity to have intimate relationships outside the family. So, kids think they're achieving movement toward adulthood when, in fact, using marijuana is a substitute for the real psychological task.

Continued on page 4

Expert Interview
Continued from page 3

CCPR: What are your thoughts about "amotivational syndrome"?

Dr. Cermak: I actually doubted the existence of this concept until recently. While it's clear that a lot of daily users prefer their couch over anything else, I wasn't sure one could measure motivation scientifically. However, recent work using brain imaging looked at the brain's response to anticipatory reward (Martz ME et al, *JAMA Psychiatry* 2016;73(8):838–844). For regular marijuana smokers who are not stoned at the time they are being tested, the brain reward mechanisms respond less to the anticipation of monetary reward when compared to the response seen in non-marijuana smokers. This suggests that the reward mechanisms develop a narrow salience for marijuana-related experiences that can dominate other kinds of rewards necessary for a broader range of development.

CCPR: What else should clinicians know?

Dr. Cermak: There are two other pieces of research I think everyone treating adolescents should know about. One is work by Staci Gruber that offers good evidence that marijuana users are processing affective cues differently than non-users (Gruber SA et al, *Drug Alcohol Depend* 2009;105(1–2):139–153). That fits with what I hear from many family members of regular smokers, which is that they lack a certain emotional presence. The other result is from research using the Iowa gambling task, which assesses real-life decision-making and risk-taking. Regular marijuana smokers are less deterred by the large losses and attracted to the large gains. This research shows that users are apt to stick with a strategy that trades lower overall payoff for higher short-term gain. It's a perfect example of people not learning from their mistakes (Wesley MJ et al, *Psychiatry Res* 2011;191(1):51–59).

"Kids think they're achieving movement toward adulthood when, in fact, using marijuana is a substitute for the real psychological task."

Timmen L. Cermak, MD

CCPR: Are there other potential adverse effects for adolescents?

Dr. Cermak: THC decreases the ability to respond to negative reinforcement. Our endocannabinoid system is a natural balm that helps prevent aversive experiences from overwhelming us; however, constant overstimulation of that system deprives us of an important source of learning.

CCPR: Let's go back to the clinician. What stance do you recommend when an adolescent insists on smoking?

Dr. Cermak: Motivational interviewing is important here. If you get into an argument with an adolescent who is smoking, you simply intensify the person's defenses. The most important thing in the beginning is to listen with as much curiosity as possible. You have to be nonjudgmental, putting your fears and opinions on the back burner. The goal is to develop a therapeutic relationship with a kid by demonstrating you genuinely want to know what makes using marijuana appealing. The more you can get someone to describe in detail the perceived benefits of using marijuana, the more you're giving that person an opportunity to be understood and find out what the problem is. Suppose your patient starts by saying, "When I smoke, I can chill. I can relax for the first time. It's the only thing that really gets me away from school and family stress." Okay, now you know you're dealing with a kid who may have excessive anxiety and stress—something you can address later on.

CCPR: How do you follow up from there?

Dr. Cermak: One thing almost everyone will endorse when we explore the benefits of marijuana is the kind of novelty it brings—an antidote to boredom, if you will. Essentially, all marijuana users like the freshness that makes walking down the hallway to the bathroom an adventure. I acknowledge that we all like that kind of novelty, then point out that THC is actually just mimicking our natural cannabinoids, which act in those same brain areas to increase the sense of novelty.

CCPR: So, you're drawing on what we've learned about the endocannabinoids.

Dr. Cermak: Right. I ask, "Do you know how marijuana works to produce the effects you like?" Most kids have scant knowledge of the subject and generally are intrigued to learn how marijuana works. Once we've started to talk science, I explain what we've learned regarding the effects of THC. For example, I mention that when marijuana hits the cannabinoid receptors, their number reduces by as much as 20%–60% in different parts of the brain. Hearing that, most kids easily guess what the effect is: It's the opposite of being stoned. If being stoned makes things novel, then if you're not stoned and don't have as many receptor sites, you're even more bored than before. If marijuana tends to relax you, and you reduce the receptors and you don't have any marijuana on board, then you're more likely to be tense and anxious or irritable. If it helps you go to sleep, not using is apt to lead to a little bit of insomnia.

CCPR: This lets you talk about what's not so good about a particular patient's marijuana use.

Dr. Cermak: Yes. Fairly frequently, people latch onto, "Yeah, I don't like …" This is where I give it a name: They don't like "a cannabinoid deficiency state." This is a much more palatable term than the word "withdrawal," which no one wants to hear. Kids get that using THC helps the deficiency but begins generating a roller coaster.

Continued on page 6

The Neuroscience of Marijuana

Daniel J. Carlat, publisher, The Carlat Child Psychiatry Report

Dr. Carlat has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

The neuroscience of cannabis has advanced at a remarkable pace. While it's a complicated story, the basic effects of marijuana on our brains are pretty well established and fairly easy to grasp. Some knowledge of these effects can be clinically useful, particularly when it comes to predicting the potential effects of excessive THC that your patients may ingest.

To set the scene, recall the big picture: Our brains are composed of billions of neurons that communicate signals to one another via neurotransmitters (NTs) and whose activity is further mediated by numerous neuroregulators (NRs). There are dozens of types of NTs—some are widely distributed throughout the brain, such as glutamate and GABA, while others work in smaller and more specific brain areas. These include serotonin, dopamine, and norepinephrine—NTs that we manipulate with many of our psychiatric drugs.

What you probably didn't learn during your training is that one of the most extensive neuroregulatory systems in the human brain (or in any animal brain, for that matter) is the endocannabinoid system (ECS). The ECS is an ancient NR system, and contrary to the strong belief of many adolescents and adults, its primary function is not to allow people to get high from smoking joints. Rather, it serves as one of the major neural modulators of the nervous system.

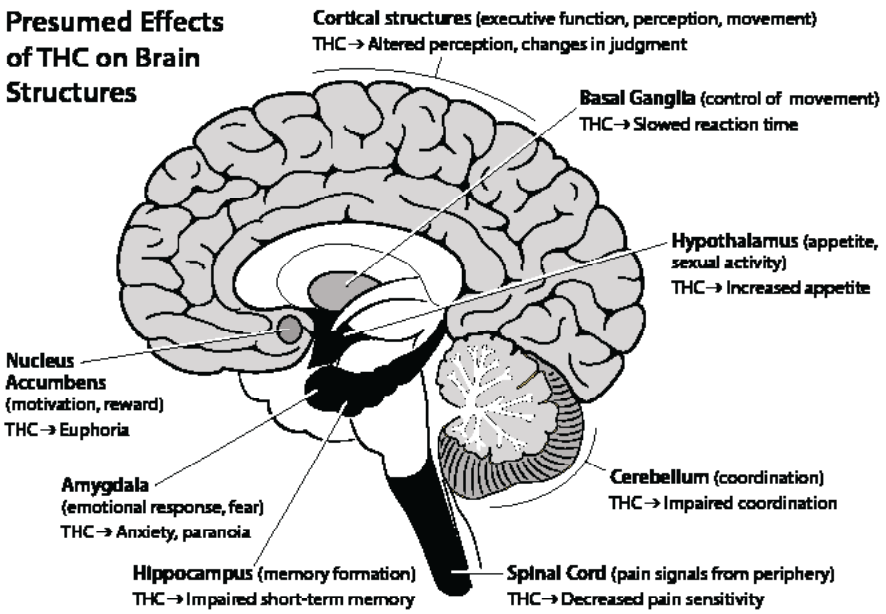
Here's how the ECS works. When a typical neuron is activated, it releases NTs into the synaptic cleft. The NTs travel across this tiny gap to bind to a specific receptor on the other side of the synapse. The binding then causes a chemical and electrical process that depolarizes the next neuron, creating an action potential that then activates the next neuron, and so on in a domino effect. This is how garden-variety NTs like glutamate and dopamine work.

But neurons need a modulating mechanism—something to put the brakes on neurotransmission so that our brain machinery can be finely tuned. That's the job of our endogenous cannabinoids, known as endocannabinoids. There are two of them: anandamide (named from the Sanskrit word for bliss) and 2-arachidonoyl glycerol.

The building blocks of the endocannabinoids are stored within the postsynaptic neurons. When an NT activates the postsynaptic neuron, it starts a process that synthesizes endocannabinoids and spits them out into the synaptic space. These endocannabinoids then travel backwards, or upstream, to the presynaptic neuron, where specialized cannabinoid receptors are located. (There are two cannabinoid receptors, called CB1 and CB2. The CB1 receptors live mainly in the brain, while the CB2 receptors are in the immune system.) Once the endocannabinoids bind to the cannabinoid receptors, they act to inhibit the neuron from firing. The process is known as "retrograde transmission" and causes "presynaptic inhibition"—ie, reduced NT release.

In other words, a primary function of the ECS is to buffer the brain's NT activity. This buffering process affects both excitatory (mainly glutamatergic) and inhibitory (mainly GABAergic) circuits. Putting the brakes on a glutamate neuron slows things down. But inhibiting a GABA neuron means reducing inhibition, so it speeds things up. Some researchers think that this dual effect helps to explain the various paradoxical psychoactive effects of cannabis: For example, the drug causes drowsiness on the one hand but enhances sensory experience on the other; it decreases anxiety at low doses but worsens it at higher doses.

This brings us right into the next topic—how does THC affect the ECS?



Research Updates IN PSYCHIATRY

ANXIETY

Can Guanfacine XR Be Used to Treat Anxiety in Kids?

REVIEW OF: Strawn J et al, *J Child Adolesc Psychopharmacol* 2017; 27(1):29–37

STUDY TYPE: Double-blind, placebo-controlled, exploratory phase-2 study

We use guanfacine XR (GXR), an alpha-adrenergic agonist, for various psychiatric issues in children, though its only FDA indication in psychiatry is for ADHD. Surprisingly, no studies have yet been conducted on the efficacy and safety of GXR for pediatric anxiety disorders—until now.

Researchers recruited 83 patients age 6–17 years from 32 clinical sites in the US. The average participant was 12 years old (SD = 3.38), white (81.9%), and female (57.8%) with a normal BMI (63.9%). Participants all met DSM-IV-TR criteria for generalized anxiety disorder, separation anxiety disorder, or social anxiety disorder. Patients were randomly assigned (3:1) to GXR or placebo, and treatment continued for 12 weeks, which included 6 weeks of dosage titration followed by 6 weeks of maintenance treatment. Participants received 1 mg–6 mg of GXR daily, with a maximum dose of 0.12 mg/kg/day. The mean dose was 2.7 mg/day.

The primary objective of this study was to assess the safety and tolerability of GXR, as measured by vital signs, EKGs, and side effects. Secondarily, the researchers assessed efficacy by administering standard symptom rating scales to patients.

RESULTS

GXR was as safe as placebo, with no clinically significant differences in vital sign parameters and no reported suicidal behaviors. Patients on GXR had slightly more side effects than those on placebo, and the most frequent were headache (35.5%), somnolence (27.4%), and fatigue (21.0%).

In terms of efficacy, patients taking GXR improved a bit more than those on placebo on various measures, though no statistical tests were done, presumably because the sample size was too small to trust the results.

CCPR'S TAKE

This small phase-2 study found GXR to be safe and well-tolerated but not clearly beneficial with respect to anxiety. If your patient's anxiety does not budge in response to an antidepressant, trying GXR is reasonable—it is safe, pretty tolerable, and may be effective—though we won't know for sure about its efficacy until we see larger studies confirming the efficacy signals present in this one.

—Kirsten Pickard, BA. Ms. Pickard has disclosed that she has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

Expert Interview

Continued from page 4

CCPR: What about the literature on marijuana's possible association with schizophrenia?

Dr. Cermak: I think it's conclusive at this point that there's a doubling in psychotic disorders that are either schizophrenia or schizophrenia-like; marijuana also can worsen psychosis regardless of the underlying process causing it, such as mania or depression as well as schizophrenia. The research gets complex, but over and over it shows the epidemiology is pretty clear: the earlier someone starts smoking, the more likely that person's risk of psychosis roughly doubles (McGrath J et al, *Arch Gen Psychiatry* 2010;67(5):440–447). The problem is, the prevalence of schizophrenia is so low that a doubling doesn't cause much alarm—unless you have one of these tragedies going on in your family or with your next-door neighbors. It's not going to tip public policy.

CCPR: Some clinicians won't prescribe any other medication to someone using marijuana. What's your stance?

Dr. Cermak: We simply don't know how someone bouncing between excessive and diminished endocannabinoid activity is going to affect other medications. If a kid is prescribed a psychoactive medication, I emphasize that I don't know what using marijuana will do, but that its effects are unlikely to be positive: Science tells us marijuana alters the activity of all the other neurotransmitters. So, if I'm trying to help mood or anxiety with a medication that affects a particular neurotransmitter, and the patient is using something that can dominate and modulate that neurotransmitter, we're working at cross purposes.

CCPR: Do you prescribe such medications if you know a kid is using marijuana?

Dr. Cermak: Sometimes I will, because I don't think any serious medical damage is likely, but I make it clear that using marijuana is likely undoing the effect of what I'm prescribing.

CCPR: This sounds like you're back to your earlier intervention.

Continued on page 8

CME Post-Test

To earn CME or CE credit, you must read the articles and log on to www.TheCarlatChildReport.com to take the post-test. You must answer 75% of the questions correctly to earn credit. You will be given two attempts to pass the test. Tests must be completed within a year of each issue's publication date. As a subscriber to *CCPR,* you already have a username and password to log onto www.TheCarlatChildReport.com. To obtain your username and password, please email info@thecarlatreport.com or call 978-499-0583.

The Carlat CME Institute is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. Carlat CME Institute is also approved by the American Psychological Association to sponsor continuing education for psychologists. Carlat CME Institute maintains responsibility for this program and its content. Carlat CME Institute designates this enduring material educational activity for a maximum of one (1) *AMA PRA Category 1 Credit™* or 1 CE credit for psychologists. Physicians or psychologists should claim credit commensurate only with the extent of their participation in the activity.

Below are the questions for this month's CME/CE post-test. This page is intended as a study guide. Please complete the test online at www.TheCarlatChildReport.com. Note: Learning Objectives are listed on page 1.

1. Which marijuana preparation has an average THC concentration in the 60%–90% range? (LO #1)
   [ ] a. Kief [ ] b. Hash [ ] c. Flowers [ ] d. Butane hash oil

2. Amotivational syndrome can result from short- or long-term marijuana use during adolescence. (LO #2)
   [ ] a. True [ ] b. False

3. Your adolescent patient tells you that many of the kids at his middle school have started smoking weed and eating pot brownies after school. He asks you about the difference between ingested and inhaled marijuana. Which of these facts is accurate to share? (LO #1)
   [ ] a. The effects of ingested marijuana begin within a few minutes and last about 3 hours
   [ ] b. Adolescents that use ingested marijuana have more withdrawal effects than those who use inhaled preparations
   [ ] c. The effects of ingested marijuana can last as long as 24 hours
   [ ] d. Ingested marijuana preparations cause fewer long-term memory challenges than inhaled preparations

4. According to studies, the risk of developing _____ doubles for individuals who begin using marijuana during early adolescence compared to later adolescence. (LO #2)
   [ ] a. Bipolar disorder [ ] b. Psychosis [ ] c. Disruptive mood dysregulation [ ] d. ADHD

5. A recent study of guanfacine XR compared to placebo found no difference in reported suicidal behaviors over the course of 12 weeks. (LO #3)
   [ ] a. True [ ] b. False

PLEASE NOTE: WE CAN AWARD CME CREDIT ONLY TO PAID SUBSCRIBERS

THC's effect on the ECS

When someone uses marijuana, THC insinuates itself throughout the user's ECS, latching onto cannabinoid receptors all over the brain and crowding out endocannabinoids. What does this mean for perception, emotion, and behavior? That depends on which parts of the brain we're talking about.

The figure on page 5 shows brain structures that are loaded with cannabinoid receptors and therefore especially vulnerable to the effects of THC. The psychoactive effects of THC match up pretty neatly with specific brain structures. For example, pot's impairment of short-term memory is likely due to THC slowing down neurotransmission in the hippocampus, where we normally create memories. Its usefulness for chronic pain may be an effect of influencing transmission in the spinal cord.

An interesting side note (one that should make all stoners happy) is that there are no cannabinoid receptors in the brain stem, which is responsible for respiration. This means high doses of pot do not cause respiratory depression and death—unlike opioid overdoses.

The bottom line for your patients

How can you use your knowledge of the ECS in your interactions with teenage pot smokers? Tell patients that we now know a lot about how pot works in specific areas of the brain. They will be fascinated that the brain makes its own cannabinoids to keep itself functioning smoothly. However, non-endogenous cannabinoids, such as THC, throw this system off kilter. If this happens occasionally, little if any harm is done. However, constant use—or perhaps use at critical periods of brain development, such as early adolescence—is apt to spark long-term effects such as poorer motivation and difficulty learning and remembering information.

Will sharing this simplified version of marijuana neuroscience have an impact on your patients? You won't know unless you try. (For more information, see *Journal of Psychoactive Drugs* January–March 2016;48(1).)

THE CARLAT REPORT: CHILD PSYCHIATRY

P.O. Box 626
Newburyport, MA 01950

PRSRT STD
US Postage
PAID
Nashville, TN
Permit 989

This Issue's Focus:
Marijuana in Children and Adolescents

Next Time in *The Carlat Child Psychiatry Report:* PANDAS, PANS, and Related Disorders

Expert Interview
Continued from page 6

Dr. Cermak: Yes. The primary rationalization we get for marijuana use is that "this is my medicine, and that's why I'm taking it." So, using motivational interviewing, I get very intrigued: "Okay, what is it you're taking the medicine for? What are the symptoms? Has that really been well evaluated?" Anyone describing marijuana as medicine is saying there is some sort of problem. I'm a doctor, so that's what I want to be able to help the person understand.

CCPR: I understand you're working on a book to try to help the dissemination of marijuana research within the field.

Dr. Cermak: Right. The working title is Taking Marijuana Seriously. My primary goal is to turn the research into a narrative, so that people will begin understanding how each piece of research led to the next piece and then the next. I hope it will give both clinicians and the general public much greater access to what we know about marijuana.

CCPR: Thank you for sharing your extensive experience, Dr. Cermak.

❖ ❖ ❖