

Title: Chicken or the Egg: The Cannabis and Psychosis Debate

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Moderator: Hello, and welcome to today's webinar, Chicken or the Egg: The Cannabis and Psychosis Debate. This topical webinar is paid for by Newport Healthcare. Newport Healthcare is the national leader in teen and young adult behavioral health treatment, providing sustainable healing for mental health and co-occurring disorders.

Some important points before we get started. Michael Roeske, the presenter for today's webinar, is a senior director at Newport Healthcare Center for Research and Innovation. As such, he has both a financial and intellectual interest in the content. The purpose for this presentation is to provide a balanced view on the cannabis and psychosis debate. APA does not endorse any products or services. The content was created by Newport Healthcare and does not reflect the views of APA or its editorial staff. Emails advertising@apa.org to learn more.

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Now, let me introduce our presenter. Michael Roeske is a senior director at Newport Healthcare Center for Research and Innovation. As a licensed clinical psychologist, Roeske has worked and studied in a variety of treatment settings since 2003, including adult substance use disorder and adolescent mental health programs, community-based psychological services, and inpatient psychiatric hospitals. He is trained as a full battery assessor of children to older adults and has functioned as a supervisor, clinical director, educator, presenter, and operator. Welcome, Dr. Roeske.

Michael Roeske: Thank you. Let me see if I can advance the slide here. Anyway, hey, everybody. The topic of cannabis and psychosis is obviously a topical one that a lot of people have interest in, both in the community environment and in the clinical environment. Today, we'll talk about, why is this debate important? Why is causality discovery something that's of interest to clinicians and researchers?

We'll take a look at the history and trends of cannabis use, get into what we know about the cannabis and psychosis associations, try to give some context to where the argument lands right now. Obviously, at some point, we need to consider, how does this inform treatment, or how do we consider these associations with respect to presenting clients? We'll give some consideration to the societal impact on here, and then we'll conclude with a summary.

The chicken or the egg. I don't know if people use that reference anymore. I remember, as a kid, that was a metaphor that was-- given chickens lay eggs and eggs come from chicken, which came first. It's sort of a metaphor, maybe it is older people, maybe young people don't use it anymore, of any time you have a difficult time explaining what caused the other.

The question of why this is important, a lot of people think that mental healthcare advancements really are going to require causality discovery and also at a sufficient pace and scale, because we see it in medical treatments, the advancement that happened when we were able to really trace the pathway back in cancer treatment, heart disease, infectious disease.

When we started to hear, myself included, more anecdotal information about heavy cannabis users with psychotic symptoms coupled with the legalization movement, the greater acceptability, higher potency products out in the market, it obviously was going to spur the question, is the cannabis causing the psychotic symptoms? Are people that are predisposed to diseases such as schizophrenia just more vulnerable using cannabis? Maybe there's no relationship at all. We'll take a look at that.

I want to start with a case example. This is back in 2014. I was a clinical director at a primary substance use disorder treatment facility that was trying to open its aperture and accept more complicated patients. We were meeting with the clinical team with admissions, looking at the exclusion criteria. One of them was active psychosis.

One day, the clinical director whose office was directly below me called me down and said, "Hey, there's a young man. He's in an inpatient hospital environment. He has a diagnosis of cannabis-induced psychosis. Can you take a look at the--" I think it was facts. I don't know. I'm sort of dating myself here. I remember it was facts. I don't know if hospitals use facts. It's been a while since I've been in those environments. I read through it and I said, "Hey, let's get him on the phone." They set up about a 15-minute call with him. I got off the call and said, "Hey, let's bring him in."

He's a 22-year-old male. What happened was, one day, his mother, his biological mother, was suddenly looking out the window, and he pulls up in his car, and he has some of his things, and he was a college student. Comes in, doesn't say anything, goes up to his room. Shortly after, she gets a phone call from what she then found out was his ex-girlfriend saying that she was worried about him. He hadn't returned home. His roommate had called over to see if he was there.

She informed the mother that they had broken up a week earlier. She wasn't really sure what was going on. He had been really isolating, acting a little strange, so wasn't really sure what was going on. Mom said he's there. The mom thought that he had moved home largely because of the breakup in the relationship. She left him alone for a day, but she was worried. She didn't want him to lose out on a semester of college.

The next day, she went into his room and immediately was concerned. She saw him what appeared to be responding to internal stimuli. He was sort of talking to himself, and she said, "What's going on?" They had a pretty close relationship, so he told her, he said, "Hey, I have some concerns." What he told her was that he started to hear

his friends talk about him, even when they weren't around, and he could still hear them at that time, and that's what he was responding to. He was telling himself it wasn't real, even though he could hear the voices.

The mother was obviously concerned, and they drove to a local emergency department. There, he made the connection to having started smoking dabs eight weeks earlier, butane, hash oil, cannabis concentrates, about eight weeks earlier. He said shortly after starting smoking the dabs when he started to sense some decline in functioning, some increase in paranoia, and shortly thereafter, he had his first glimpse of fixed ideas and delusions and a little evidence of auditory hallucinations.

By the time he arrived at us, we calculated he was 15 days absent. He had stopped 10 days early. He was in the hospital for five days. He had stopped 10 days earlier, largely thinking that it was the dabs that were causing this, hoping that it would go away. When it didn't, he returned home.

A little bit of background on him. He started drinking at the age of 14, smoking leaf cannabis at 15. He had early life stress. His dad appeared to have a severe alcohol use disorder, created some chaos in the home, not physical violence, but yelling, unmanageable, had a difficult time holding a job. Around seven or eight years old, the mother left the father, took the only child, took this young man with her, and she never remarried, at least the last that I was in contact with her. She had never remarried or had any other children.

He had no prior mental health diagnosis, although he did say that he typically skewed anxious. That's why he liked cannabis. I want to say this, too. When we're talking about the intro here, this talk is not about whether or not, as a researcher and as a clinician, I try to be as dispassionate as possible about what we're talking about. This talk is not going to be about whether or not cannabis is good or bad, whether people should abstain or use. I'm really going to try to stick to the question about what the association is to psychotic symptoms, and how we might think about it.

Current labs were good. His physical is unremarkable. When I met with him, no suicidal or homicidal ideation. He had good ADLs. I've worked with individuals with schizophrenia diagnosis in the past. I didn't see a lot of the obvious things that sometimes come with that diagnosis, particularly around the keptness, he had good eye contact, good gait. He had poor judgment. He broke up with a girlfriend, even though he knew it wasn't quite right. He had insight into it all, but clearly, at base, he still had delusions and hallucinations when I met with him.

Going through a differential diagnosis, we took off the psychotic disorder relationship to MDD or to bipolar or to medical conditions and sort of left with two different options, schizophrenia, which he hadn't quite met the time requirement. DSM recommends you can start to consider something outside of CIP or cannabis-induced psychosis past the 30-day mark, and schizophrenia typically has that six-month deterioration component. You start to see a decline in prodromal symptoms prior. We're going to keep with the substance-induced psychosis diagnosis.

At the end there, I think it was day 47, he was in treatment for 32 days. He was still experiencing positive symptoms, hearing the voices. Very much, he had the fixed idea that they were talking about him. He had the negative symptoms as well. He

was socially withdrawn, difficulty emoting. He did well in treatment, got along fairly well with his peers, but had a lot of the alogia, the different elements that you typically see with someone with schizophrenia in particular. I did give, did recommend a provisional diagnosis there, and we referred him to a FEP program. First episode psychosis program, which was local. I'll talk about those programs later.

What are we really talking about here? Cannabis sativa, which is where the active ingredients of cannabis comes from, has 400 bioactive compounds, but it really narrows down to the singular one, delta-9-tetrahydrocannabinol. It's responsible for the psychoactive effects, the effects of getting high. It's associated with immune and stress response and appetite and mood and cognition. It attaches to the CB1 receptors, which are most dense in the brain. Then the CB2, which are peripheral immune cells.

Activity depends, around like CB1 density, just different elements on there. It also stimulates dopamine release. Those of you that have worked in substance use disorder treatment world, you know the argument, or there is an argument, that one of the explanations for the power of substances for people who have a severe substance use disorder is dopamine is essentially equated to evolutionary survival, neurotransmitter. Think of sex, eating, things that really depend upon your life, give dopamine release. It also does that with substances. That is some explanation as to why people end up making choices, in the most extreme cases.

The desire to care for your children, which is a profound impulse that people have, gets circumnavigated, gets sort of usurped by the compulsion to use substances. Here's what it looks like. You can see in the upper left-hand corner, now I'm 52 years old, so I guess friends told me that that's not what cannabis used to look like in the past. You can see there, it's very pungent. This is typically what you'll find often in cannabis shops. It's very sticky.

Then those are the concentrates in the upper right-hand, upper right is shatter, wax, crumble, butter, and oil. They're all similar in terms of the THC percentage, the potency on there. They're just made a little bit differently. Then you can see down in the lower left, that's an edible there. Obviously, Oreo, and then you see the chocolates. The one in the middle there is a vape, and the items on the bottom right are ways to use the cannabis concentrates.

I don't have a little pointer, I wish I had a little pointer I could show you. I don't know if you can see my cursor, but this right here, if you take a look, this is called a nail or a banger. That's where they put the concentrate, and then they use a blowtorch, takes a high heat in order to convert it into a usable substance for inhaling.

Trends. I'll start with the caveat. There are two large databases that people look to around substance use trends, mental health trends. You have the National Survey of Drug Use and Health, which is the SAMHSA study. Then there's Monitoring the Future studies. I tend to go with the Monitoring the Future studies, but they're not equivalent. They have different questionnaires, different patient populations. Just provide some caution.

For example, with the SAMHSA study that came out, I think, for 2023 has a little bit lower estimates in terms of percentage of use. Most people were surprised to learn that high schoolers, really, since the middle of the '90s, since what we call it a drug relapse period, or at least that's what the authors of Monitoring the Future call that period in the mid-90s where we saw just this escalation in substance use among teenagers really across all substances, really without explanation, but it's really been going down since then.

Then in 2018, 2019, largely coupled with the advent of the rise of vaping, it really took a spike up. Then the pandemic hit, and we saw our largest one-year drop in the time that we've been recording the data in terms of use. It really has maintained those levels since. Now, you can see here, though, I don't want to minimize it. It's still estimated that close to one in three 12th graders have used in the past year, and still one in eight use daily. It's still a substantial portion of the population. It just is down from what it was many years ago.

Again, they're different products. Somebody smoking cannabis in the '70s was smoking something sort of almost qualitatively different. Then when we think about young adults, this is different. We see this clinically where a lot of our teens that come into treatment don't have a lot of substance use history. Substance use tends to be initiated through social experience.

When I was a young person, you'd go to a party, and people would have alcohol or people would show you how to use drugs. Teens don't do that anymore. They're delayed getting driver's licenses. They're delayed in romantic relationships. They don't hang out with friends as much. Young adults, they go off to college or they move out of their parents' home, and then they do start to go to bars or congregate.

Then we start to see it really pick up. The highest levels reached since 1988. You can see almost half. The daily use is still about the same as teenagers. Then you can see that we really see an increase in number, 35 to 50-year-olds, still less than the peak years of young adults from emerging adulthood, but still a substantial portion of individuals.

The two common explanations that are often given or questions around the connections between these things are around increased availability and increased potency. We'll start with the first one. I was born in 1972, so I wasn't obviously alive in the '60s. I certainly remember seeing movies of it where it seemed like every time there was a clip of someone in the hippie movement, they were smoking cannabis. It really became a time where decriminalization became a conversation.

It wasn't until a couple of decades later when Surgeon General Elder suggested studying legalization. That really, if we look at it from a historical perspective, was the beginning of the legalization movement. Then California, which typically is often first when it comes to these type of initiatives, did the, what was called, the Medical Marijuana Initiative in 1996, which essentially gave immunity from prosecution to qualified individuals and their caretakers that were in possession of cannabis or were producing cannabis. Currently, there are 38 states that have legalized cannabis and 24 allow for recreational use.

Now, it is interesting that the Controlled Substances Act, which is the federal legislature on scheduling, and whether or not a substance is legal or illegal, still lists cannabis as illegal. It's still federally viewed that way. They just haven't allowed the states to do their thing. When we get into the societal implications, I will circle back around to that, whether or not that's a good thing.

When we think about our ability to collect data, to have similar data sets to really understand, you really do need a federal or a way to which, because a lot of the data that we have, it does come down to state-specific research. Then it is used in higher and legalized states, although we need more studies to understand whether or not those are just secular trends, in which case, there was a popular political movement to legalize it because more people were interested in doing it, or it has resulted in increased use just because it's more available.

Interestingly though, again, Coley and colleagues this year came out and took a look and said, "Hey, it actually is not as common. High schoolers in legalized states don't typically smoke as much or as frequently, but those that do use cannabis, there's more daily users in that population now." Then comes the potency, and that's 1995, that's my time period, or my friends, my friends were looking at cannabis at the time.

You can see if you purchased cannabis then, it was about 4% THC, give or take. By 2020, it's 20%, so five times the amount. That's similar to what you'll find if you go to a cannabis shop when it comes to leaf cannabis. You can see the concentrates go from 52% to 69%, but they can go as high as 90% to 95%. We're really talking about something quite different when it is 15 to 20 times greater potency than the cannabis you might have been-- again, depending on your age, you might have had friends that use it as well.

Again, you don't need to volunteer, but if we were in a live audience, you can share your own examples of it. There is evidence as well that the concentrates are increasingly popular. I can say anecdotally, I certainly heard it. I'm not a practicing clinician for a few years. I still supervise psych associates, so I have less sort of a finger on the pulse of it. Certainly, there's a lot of evidence that concentrates are increasingly popular. Lacks representative data though, so we're not entirely sure about that either.

Switching gears then to psychosis. Now, obviously, this is, I assume, largely psychologists or other behavioral health clinicians that are on this call. This is not new information, but I thought it would be useful to put into context here what we're talking about when somebody that's using cannabis arrives either in an emergency department, into their college healthcare program, into your private practice, or if you work at a clinic, especially if it's primary SUD.

If you remember from graduate school, the two sort of pillars are delusions and hallucinations. Then you start to see other things, inappropriate behavior, decline in hygiene, the negative symptoms, lack of motivation, sleep disturbance. Then we'll often see, particularly for those individuals that have a diagnosis of schizophrenia will convert over into something more chronic. We'll start to see prodromal symptoms, pre-morbid symptoms that are sort of sub-threshold that begin potentially.

I remember also in graduate school, there was a view that this schizotypal personality disorder was a way to conceptualize some of the phenomenological or observational pieces that you would potentially encounter clinically. Just a sort of oddness, less interest in connecting to others.

What about cannabis-induced psychosis? One way to help with differential diagnosis is that if a person is able to abstain, the symptoms will typically have rapid decline. Now, rapid is relative. I mean, by that, in a couple of days to a couple of weeks, they may have lingering symptoms of anxiety, some different sort of functional challenges, but you'll typically see it go down pretty quick. That was why we were calculating an instance of that case study, how many days since last use were sort of taken to see how it impacted his reported psychosis.

They typically have more mood symptoms than you might see with a primary psychotic disorder with anxiety, depression. The thought processes typically are more organized and sequential. If you remember from my case study, he actually was fairly aware of what was happening, had a real sense that something was quite wrong. You typically see less family history.

To go back to that case study too, there are lots of diagnoses that you can give somebody that are this heartbreaking and you don't want to give. I think it's hard to argue that schizophrenia is not up there in the first tier of ones. It's because of the impact. I just remember as well with the mother, I wanted her prepared for what I thought was going to happen, which eventually was a full diagnosis of schizophrenia.

You were really talking about something really horrifying, particularly when they're young people, and families are observing and are trying to understand what's happening here. You'll typically see less mental health history than you'll see with schizophrenia, which has a genetic load component. You'll often see other associations to mental health in the family history.

How often does this happen? In addition to sort of not having a moral perspective on the use of cannabis or whether somebody should use or shouldn't use, I also try not to be alarmist, especially when the epidemiological data supports it, in terms of the risk component here. If you look here, it's estimated, and this is a fairly large sample, that about 1 in 200 people who use cannabis will end up with a cannabis-induced psychosis, which if you go with the World Health Organization's estimate of the global percentage of individuals with a schizophrenia diagnosis is also 0.5%. It still is uncommon.

Now, obviously, if it's that mother or that young man, it's devastating. You are just profoundly impacted. From an epidemiological perspective, when we start to consider risk components and how we message this to others, if there is a messaging component, it is still fairly uncommon. Nearly half of those that have a cannabis-induced psychosis episode will convert to a full bipolar or schizophrenia diagnosis, typically about half of those within the first three years, and then half of those over many years.

Now, this is where, when the question is posed, where should we stand? Really, are there populations that are specifically at risk where we want to have more of a preventative care approach? This is where we start to get into that. McDonald and

colleagues came out with a study this year, and a lot of our data that did age associations to cannabis and to psychosis was using that older cannabis-induced cannabis sampling, so the 4% to 16% to 20%.

They were looking at the impact of the concentrates, and they found an even stronger age relationship so that younger people were particularly at risk. Then, obviously, it's not terribly surprising that we would find personality disorders and substance use disorders. Somewhat strangely, though, again, not really for those of you that work in the substance use disorder treatment world, you'll often notice that you'll find eating disorders clustered within families that have histories of substance use disorders.

This is why I think it's like Wegovy. When you use substances like stimulants, it suppresses appetite. You use cannabis, it increases appetite. They're all typically connected to each other, and why-- I think it's Wegovy, I think it's the name of it, the diabetes medication that's used for weight loss. That's why there are currently clinical trials underway to see because there is evidence that it does help reduce cravings for substances. There are associations on there.

Again, when we start to think about our patients that are coming through, are they young, do they have history of substance use, are there personality elements in here, do they have history of eating disorders, now we really start to consider those people that are really at risk.

Here's the debate. People who use cannabis are considered two to four more times more likely to develop a psychotic episode. One of the rebuttals to that, and we should expect to see a similar increase in the general population. If cannabis itself is taking individuals that were not going to end up with a schizophrenia diagnosis or psychosis and resulting in psychosis, we would expect to see an increase in psychotic episodes in the general population. Again, **[unintelligible 00:29:59]**, I don't know if I pronounced that correctly in colleagues, did find that, they took a look at two samples, I think, from 2001 to 2011, 2012. Very large sample, did find a 2.5-fold increase.

Then the other rebuttal to that, and we see that when there are arguments around the massive increases in ADHD diagnoses or autism spectrum disorders, that part of the explanation is we're just more cognizant of it. Maybe we've lowered the diagnostic threshold for making a diagnosis. Obviously, we know that when we pay attention to things, we're more likely to see it, but it doesn't really seem to be an explanation for this when we control for that. It doesn't appear, though, any change in diagnostic practice leads to an explanation increase in cannabis psychosis numbers.

Then when we really start to build this argument here, we also know, like for example, in this Ganesh and colleagues study, where it was a placebo-controlled environment with IVTHC, there was a very clear dose-response relationship to producing schizophrenia-like symptoms in the participants. It included both positive and negative symptoms, but very strong data there to support the relationship. At least we know there's biological plausibility from a study like that.

Then we look at this, the Forte and colleagues, where they really, again, take a look, okay, what are the strongest independent variables? What are we looking at here? They were very clear that the two strongest variables are frequency of use, particularly daily use and use of concentrates. As we start to build this clinical picture of the candidate of individual that is really at risk, we start to think about a young person with a substance use, personality, eating component that is using daily and using concentrates. That's really where you start to say, "Okay, I'm pretty concerned about that population in particular."

Now, this is not to distract from anybody else that may be using it and their vulnerability to it. In my mind, if I start to go through differential diagnosis, I'm really going to sort of have that as the example of which clients to be most concerned about. Then if we are to take away, or at least when I look at the data, look at the research, the one thing I can say with confidence, I'll get to the sort of conclusion of where we are with this.

The one thing I think we can say with confidence is that cannabis use, particularly daily use or concentrates use, if there is a vulnerability to something like schizophrenia, again, going back to graduate school with something like the diathesis-stress model, which explains schizophrenia as a genetic vulnerability or epigenetic component that is impacted by a stress, so that certain genes are turned off or turned on as a result that allows for the expression of schizophrenia. If someone doesn't have that particular stress, it doesn't fully express and conversely. The one thing that we think we can say fairly clearly is if there is that vulnerability, it's going to accelerate the age of onset, and it's going to worsen the prognosis and course of the illness.

I want to say something going back to the case study as well. The issue, or going back to cannabis-induced psychosis, I hold space for harm reduction models and want to talk to people about what's most appropriate, and I try to be as honest as I can be about my clinical opinion about things. If people are not interested in abstinence, I explore that with them.

The one thing that's clear is this young man or anybody else that has that cannabis-induced psychosis, and this is why the hospital referred them to what they largely viewed as the primary SUD program. Once it happens, it's going to happen again, and it's not going to go away. It's the same thing, those of you that have worked with people with stimulant use disorders or amphetamine use disorders. Once they start to have psychotic responses to the substance, that does not go away. It's not like later on, someone has a PSA, "I used to use methamphetamine. I got psychotic and then I stopped for two years and now I can use safely." That's not going to be the case.

I was very clear with him. Fortunately, he did have an absent in school, but it's the same. I remember in some of my trainings in psych hospitals, I remember the one attending psychiatrist that would come in with the people with bipolar diagnoses, particularly with bad sleep hygiene, and like a parent, "You will go to bed and get eight hours of sleep," because we would see repeat individuals that would come back in every three months. It's the same thing. You're done.

I sometimes use the pickle example, it's never going to go back to being a cucumber. Whatever it is, you're not going to go back to being if it was safely used. Again, going back to the debate, we have biological markers that are similar to psychotic disorders, again, expanding the biological plausibility explanation.

Then when we look at genomics, which I think a lot of us have a great deal of confidence in, it helps provide a great deal of clarity in terms of the causality component. Again, think about bipolar with its tremendous genetic load component. We certainly see the family histories there. There's evidence clearly of a bidirectional relationship, but I do want to say it's typically, it's more weighted toward a reverse causal mechanism, meaning that if the authors had a sort of-- if they were in Vegas or they had a bet at the table, they would argue ultimately that it's weighted more towards individuals with genetic vulnerability to schizophrenia that leads them to use cannabis.

Maybe a different explanation is genetic confounding, that the underlying vulnerabilities for schizophrenia are the same as the-- or there's a great deal of overlap to those with cannabis use. They're confounded. It's not really causal. They're sort of similarly present. If someone has access to cannabis and if vulnerable to schizophrenia, they're more likely to use. It doesn't really have a causal element.

The causal relationship continue to be debated, but again, I'll go back to that piece. I feel fairly confident in saying just that particular population that I described in terms of vulnerability in the earlier age of onset and worst course of treatment, I feel pretty comfortable as we sit here today to sort of make that claim as to where we're at.

The treatment. Unfortunately, we're sort of in, I guess, in a similar way to use of social media, particularly with preteen and early teen cisgender females, where we're just like, "Oh my goodness," or really any teens with social media, but we're doing this large experiment. I think there's something similar here, and typically, it takes a while, treatments tend to lag behind.

There's no real treatment strategy for cannabis-induced or first-episode psychosis that resulted from cannabis use other than atypical antipsychotics. That's what this young man came to treatment with. I think it was like 2 milligrams of Risperidone or Risperdal, which again, atypical antipsychotics come with involuntary movement risk, metabolic risk. It's not inconsequential, it's not a large dose, but how does the person respond to the medication? It could rise higher.

Again, going back to the abstinence piece, it's important. It's not going to work. Again, I feel like I can fairly confidently claim that. I like SAMHSA's approach to co-occurring disorders, which is the idea of no wrong door in here. Regardless if you're a college counselor or you're a primary care physician or you're a private practice psychologist, there's really no wrong door here. You want them engaged in treatment. You should have training in co-occurring disorders, obviously, relationships with appropriate reference in the community.

Really, anybody can be that first point of contact in treating. Like I mentioned, I still supervise psych associates. A lot of times, especially if they're early career or early training, they want to know what to do. How do I do acceptance and commitment

therapy? How do I do dialectical behavioral therapy interventions, informed interventions?

The thing I focus on, and often to their dismay, is like, "Hey, how about you just learn how to be with a patient, give some consideration to the impact you're having and how you guys are working together?" Then, if they do that, if they feel like you are respecting them, and you're willing to talk about goals, you may not agree with the goals, but you're willing to talk about them, you respect their position, and the various elements of therapeutic alliance, that's really going to work. It's the same thing with individuals with cannabis-induced psychosis, or ultimately convert to schizophrenia.

We see really that it has a really impact on functioning, engagement, satisfaction, quality of life. Again, not new information, but just wanted to make it explicit here, that that really is the most important thing. Often, too, I remember in some of my trainings, that an explanation for psychosis was often a really underlying terror. We think about when a person with a stimulant use disorder, cocaine, ends up with psychosis and the paranoia, it's the amygdala that's lit up.

We can think about psychosis in a similar way, the brain's trying to fill in the empty spaces, something's wrong, something's wrong, something's wrong. Again, this young man with the paranoia. I like that idea, that if we think about it just empathetically as underlying terror, that helps us sort of approach, even if they are bizarre, even if it is difficult to engage.

I mentioned here, too, I remember I did two years of clinical training in inpatient psych hospitals. My peers and myself found out very quickly, whether or not this was a population you were comfortable with. There were some people that were like, "This is not me, it freaks me out." I kind of liked working with the severely mental ill or gravely disabled. There was something very-- a soul empathy eliciting for me. Again, I'm not necessarily a whisperer on this, but it definitely was something I was comfortable with. You want to invite those feelings of discomfort with it.

Again, perhaps this is most evident when we think about severe mental illness with psychosis, just because often, we don't realize how neurotic we are. I'll speak for myself. I have two children. Until you're with a kid or you're with someone with psychotic disorder, and then you're like, "I'm so structured and I expect these things. My kids are absolutely bananas and don't hold their stories together. He might take off his clothes or run around the house." It's the same thing with an adult patient. You're like, "Ah, no, you got to go wear clothes."

Generally speaking, you don't want to dismiss, minimize, or argue, try to empathize. I find, particularly with young clinicians, young training psychologists, there's often a desire, and not even expressed, but to challenge delusions, "Hey, let's think through this. Do you really think it's possible that Bill Clinton is-- the government has-- let's get a CAT scan. Do you ever think about getting a CAT scan and seeing if there's a chip in your head?"

Paranoid delusions are probably the most common delusions that come with CIP. You typically don't want to do that. Again, just try to empathize with it. Again, I don't think I made that clinical interpretation when I was with patients, but I definitely made a lot of errors, so to speak, when I was working with certain populations. Again, just

trying to dismiss, "Oh, boy, it must be really terrifying. It must be really difficult. Let's see if we can get you some help."

I talked about the FEP program that we sent. If you're not familiar with them, again, I don't want to speak for all of them. I have my anecdotal information. I have my clinical experience with it. I have my experience in working with colleagues. I have my experience in working with supervisees. We recently got a young man into a FEP in New Jersey. My experiences, these are absolutely wonderful resources, first episode psychosis. The limitation is they're typically for 15 to 25-year-olds. Some places go up to 30, but they can provide a tremendous amount of support, including in-home services.

Really, what the goal is to have the least impactful experience with the disease so that people can continue to function and do well, and pursue their goals, whether educational or employment goals. They provide a lot of education and support, medication management. It's simple as you just Google local FEP or FEP in Connecticut, and you can see, and the first one in New Jersey that I found and they said, "No, we're not the right place, but there's another one in Southern New Jersey that I think pretty close." Got them connected. They connected with the family, and it really was a very good door-to-door transition between. Again, my experiences, these are great programs.

Now, this is 2019, there were 260. I couldn't find more recent data. It could be more, it could be less. I don't necessarily know that there's been a tremendous-- I think, if anything, there's probably greater funding available. I might expect that to have increased, but I don't know for sure. A couple other things, intersectionality, as you know, is that there are layers related to different demographic or socioeconomic, cultural, religious elements of the individual that create an increasingly discriminatory environment in which they lived.

Historically, non-white cannabis users has been characterized as dangerous and unstable versus if, again, like myself, if I was in college and I was smoking a lot of cannabis and acting strange, someone who looks like me wouldn't draw a lot of attention necessarily. If you're in a different environment and you're in dark skin, there's a much different connotation.

The other one, this is really unfortunate when you think about it. Also, someone that looks like me, if I go into a primary care environment, I'm going to have a much shorter time to first contact with the coordinated specialty care or FEP program than somebody that doesn't look like me. Then just in general, diverse COD populations, they're just underassessed, underdiagnosed, underreferred. A lot of reasons for that, including distrust of Western medicine. In general, though, obviously, I think we all would agree we could do a better job of having similar care opportunities for all people. I'm cognizant of the time here.

We could obviously stop here. Again, if it was a live environment, we could talk about it. Going back to this piece, I try not to have-- particularly around political views on this, but it sort of begs certain questions. What we know, is there a societal responsibility? For example, we do put limits on the percent of alcohol in things, what's called a spirit.

I remember, again, going back to my high school times, there was a thing called Everclear or green alcohol. It had 190 proof. It's only legal in 36 states. Should there be limits on potency? We know that there were tremendous-- again, I'm not overly familiar with the literature on what led to reduction in cannabis, but I know enough to know that the campaigns, alerting people to the dangers were helpful, including warning labels.

Now, again, that obviously wasn't the only element in there, but particularly for young people, should there be warning labels on here? Obviously, the other piece there, how do we improve access to care? If this is happening, and again, going back to that vulnerable population, if you're non-white, really, how do we be more responsive as a society to make sure that they have access to these programs like FEP?

In summary, the importance of causality, which we don't really have yet, but we know that if we're able to make that clear connection, then we have much better grounds with which to, one, sort of answer the societal questions, but two, then, from a treatment perspective. You can see here that I don't think that the movement toward legalization and acceptability is going to change.

Again, this is where I'm trying not to be too political here, but with the revenue dollars that come in from the tax base around the states that have legalized, typically, those states get used to use spending that amount, and it's hard to roll back those. Then it's like gambling, like what happened with gambling.

Then with implications for treatment, the other piece I didn't say here is early intervention, like a lot of things, is very, very important. Not only is it no wrong door, but early intervention is really, really key here. Then there are these societal implications about exactly what we're doing with the cannabis. I do think that we can conceptualize this as a really large-scale social experiment in the same way that we are seeing it with smartphones and social media and things. Maybe not as clearly direct, but clearly, there's something going on, particularly with the concentrations, which I also didn't say. They're legal in all the states in which cannabis is legal.

In summary, we're sort of left with this unknown factor, but we clearly have some sense of where we need to go, what kind of future in research we need to proceed with. I really do expect in the next couple of years, we're going to have a better sense of how to treat. Thank you.

Moderator: Thank you, Dr. Roeske. Looks like we have some questions from attendees. Our first question being, is there any research on combination cannabis and tobacco use in psychosis?

Michael: That's a good question. I have not seen it. I have not seen that in any of the studies, but having said that, there weren't exactly keywords I was putting in, so it may exist. I'm just not familiar with it.

Moderator: A different question asks, does the chances of psychosis or schizophrenia increase when someone is using cannabis and alcohol?

Michael: Again, I haven't seen a lot of additional-- because I did this talk recently, and someone else asked similar questions about that. The challenge is that the

research really just takes a look at if the data elements that I see are around-- it's sort of a binary, cannabis use and no cannabis use.

We know, obviously, a lot of times with clinical trials, comorbidities are exclusionary criteria. I haven't necessarily seen the data on that. What I can say in general, going back to nicotine and alcohol, there typically is not, with the exception of delirium tremens with alcohol, that's more of a withdrawal-related symptom. I have not seen any evidence with the exceptions of stimulants and amphetamines being really predominant with respect to other substances leading to psychotic disorders.

Moderator: Brilliant. Another question asks, what predisposes a cannabis user to schizophrenia or psychosis?

Michael: I appreciate the question. That really is the crux of it here. If I go back to some of the bait slides, I think a lot of people have the opinion that there are underlying vulnerabilities to both, that the effects of cannabis that people are treating some of maybe the prodromal symptoms of schizophrenia, or certainly when someone has a psychotic disorder or schizophrenia, that there's a medicinal component, that in the same way people with schizophrenia like to drink coffee and smoke cigarettes, that there's a way in which it helps reduce anxiety.

I imagine there's some element of that, going back to the question of, who's vulnerable to cannabis use? I think we could obviously go off and talk about vulnerability to substances, which you can sort of take a lot of different paths along that way. Certainly, I think that one of the things people like about cannabis is it is anxiolytic. Typically, for a lot of people, they report a reduction in anxiety, and life is anxiety-producing right now. I think that's sort of inherent in there.

Moderator: Brilliant. Someone else asks, how addictive is cannabis? Is there a safe level of use?

Michael: I guess it goes to your question of what safe is. My general feeling is, and this includes-- and not everyone agrees with this. This includes even opioids, because I think people are obviously scared about opioids, and there's important uses of opioids. Generally speaking, the substances are not overwhelmingly dangerous.

I feel similarly about cannabis. I think that the majority of cannabis users would report little symptoms or a few symptoms or tolerable symptoms that come with it. Having said that, there is a subpopulation of individuals that end up with a substance use disorder. Within that, there's a subset that end up with severe substance use disorders. Obviously, that group is very concerning.

Moderator: Another question asks, is cannabis-related psychosis reversible?

Michael: Yes. I think the data I showed you was about 50% of people will not end up with a primary psychotic disorder diagnosis later on. A lot of people, the symptoms will dissipate, will go away. If they stop using cannabis, they're not going to end up with psychotic disorders. It is reversible.

Obviously, to me, the extension of the question is, if someone ends up with a schizophrenia diagnosis that we think is associated with the cannabis use, what's their prognosis? Again, going back to graduate school, I don't have to teach this anymore, but I was taught the rule of thirds. One-third will not have additional episodes, one-third will be intermittent, and one-third will be chronic. I suspect it's probably something like that in terms of the full-on impact.

Moderator: Another question asks, what are the benefits, if any, of using cannabis to treat anxiety and depression?

Michael: That's a good question. I have looked at some of the literature that has examined the question of its purported medicinal benefits. I'm not a TikTok user, but I know that there was certainly a period where there were a lot of influencers who were proclaiming the benefits. Certainly, we see celebrities, Joe Rogan, Snoop Dogg, individuals who really sort of point to it as a lifestyle, as something that's really beneficial.

I think, in general, I think the evidence is fairly weak as to an anxiolytic, for people with anxiety disorders, actually can have-- again, some of you in the audience may know more than me, but it actually can increase anxiety in some individuals. I go with the reports of people, though. There are a lot of people who reported benefits them, and so I don't discount that.

Moderator: Brilliant. Someone else asks, does starting age play a significant factor?

Michael: That does seem to be the case. There's a great deal of evidence of age association in this. Now, we could argue that people with vulnerability to schizophrenia and cannabis use disorders are more likely to start earlier because they're drawn to the substance. It's not entirely clear that if we delayed initiation, they wouldn't have experienced it.

Generally speaking, it does appear to be the case. Now, in part, one of the partial explanation is the brain is still developing, the nervous system is still developing. It's hard to imagine, particularly with the concentrates, that there's not some impact on development. I think. like McDonald and colleagues that came out this year, they were pretty emphatic that there's a very robust relationship with the age association.

Moderator: I think we have time for one last question. Someone asks, what frequency is considered use? Cannabis use.

Michael: That's a really good question. When we do on our outcomes, on our intake and outcomes approach, we do a 30-day back, last 30 days, typically, how many days? I don't know. A lot of the literature does, again, have a sort of binary approach to it, daily versus non-daily. Again, that's maybe a little bit cruder than it is.

I don't have the data. I haven't looked at the data sets, obviously, to know how they clustered the information, but that seems to be the case. If you're a daily user, that's one thing versus less than daily, which is maybe once a week or multiple times per week or once per month. That's a great question, though, right? That's where you start to get in complexity around the research and why we need additional research.

Moderator: All right. Thank you so much for joining us, Dr. Roeske. Thank you to all of our listeners for your participation. We'd also like to thank Newport Healthcare for making this webinar possible. A recording of this presentation will be emailed to everyone in two weeks' time. The recording will include the presentation slides. As soon as the webinar has ended, a short survey will appear on your screen. Please take the survey and give us your feedback. We thank you for your attention and hope you have a great day.

[01:00:34] [END OF AUDIO]