Methamphetamine-Associated Psychosis and Criminal Responsibility

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Stimulants are among the most widely used substances in the world after cannabis, with a rapid rise in methamphetamine use in the last 15 years. Methamphetamine has a high propensity to cause psychosis ranging from transient psychosis during acute intoxication to persisting psychosis with similarities to schizophrenia. Although the former condition may not abrogate criminal responsibility, the latter is recognized as a basis for an exculpatory mental state in a majority of jurisdictions across the United States. Methamphetamine use can therefore complicate criminal responsibility evaluations. We present the literature on methamphetamine-induced psychosis, underscoring the shortfalls in existing classificatory schemes for methamphetamine-associated psychosis that can complicate forensic mental health evaluators' opinions in criminal responsibility evaluations. We offer practical considerations for forensic mental health professionals performing criminal responsibility evaluations where methamphetamine use is a concern.

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It is estimated that, in 2021, 36 million people had used amphetamines, 22 million had used cocaine, and 20 million had used ecstasy-like substances in the past year, placing stimulants as among the most widely used chemicals in the world after cannabis.[1] Amphetamines are a class of chemically related compounds that are used extensively in both recreational and medical settings. Amphetamine-like stimulants are often prescribed to treat conditions including attention deficit and hyperactivity disorder and narcolepsy. They are also frequently sought after for nonmedical use.

Methamphetamine is distinguishable from amphetamine by an additional methyl group, which makes methamphetamine highly lipophilic and consequently more able to cross the blood-brain barrier.[2] Methamphetamine is primarily abused for its euphoriant effects and to a lesser extent for increased wakefulness, decreased fatigue, and weight loss. In methamphetamine-naive humans, low doses produce a sense of heightened alertness, attentiveness, and energy. Higher dose intoxication produces a sense of well being, euphoria, and enhanced self-esteem that can approach hypomania and grandiosity. Adverse effects include restlessness, insomnia, bruxism, excessive weight loss, and suspiciousness that can develop into psychosis. Compulsive repetitive behaviors, such as skin picking, are common and are at times accompanied by tactile hallucinations and delusions of parasitosis.[3] About 0.9 percent of Americans aged 12 or older (2.5 million people) used methamphetamine and approximately 0.6 percent (1.6 million) experienced a methamphetamine use disorder in 2021.[4]

In a comprehensive meta-analysis of 149 studies of mental health outcomes with amphetamine use, McKetin and colleagues[5] found that the most compelling evidence for a causal association was between the use of amphetamines and increased risk of psychosis, with consistent moderate to large effects across various populations, including in well-controlled, population-level studies and longitudinal studies. In a 2020 review of methamphetamine-associated psychosis, Arunogiri *et al.*[6] found that the prevalence of people who develop psychosis after using methamphetamine ranges

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from 15 to 23 percent in recreational or community settings and up to 60 percent in treatment settings.

McKetin and colleagues[5] reviewed 12 studies that examined violence as an outcome of amphetamine use. They reported that any use of amphetamines was associated with 2.2 times the odds of violence. Studies that adjusted for other substance use, demographics, and premorbid risk factors yielded a pooled odds ratio of 1.4, which was nonsignificant. The authors noted that the evidence mainly came from observational cross-sectional studies, most of which were of low quality and based on relatively small idiosyncratic samples. Further, there were differences in the strength of the association, depending on the measure of violence used. The authors found that longitudinal studies provide evidence for increased violent behavior during periods when amphetamines were being used (which persisted after adjustment for contemporaneous changes in other substance use) and higher rates of recidivism for violent offenses among forensic inmates with an amphetamine use disorder postrelease.[5] They concluded that amphetamine use may be related to interpersonal violence, but further research is needed to confirm this relationship and to capture its complexities (e.g., the extent to which it is modified by antisocial personality, polysubstance use, or other contextual factors) (Ref. 5, p 93). In an attempt to control for confounding factors noted in previous studies and with the aim to elucidate the dose-response relationship between methamphetamine use and violence outcomes, Foulds *et al.*[7] conducted a longitudinal birth cohort study comprising 1,265 children in New Zealand in 1977. Members of the cohort were studied frequently between birth and age 35. The authors reported that 28 percent of participants reported using methamphetamine at least once between ages 18 and 35. Compared with no use, a history of any methamphetamine use in each age period was associated with an increased adjusted risk of violence perpetration, intimate partner violence perpetration, and violence victimization. There was a dose-response relationship between frequency of methamphetamine use and elevated adjusted odds for violence involvement when compared with people who used methamphetamine less often or had never used. The authors concluded that methamphetamine use is an independent risk factor for violence perpetration and victimization in the general population.[7]

Forensic mental health professionals may be asked to perform criminal responsibility assessments in cases where the defendant has a history of methamphetamine use and has been charged with a violent crime. The central question to be answered is if, in the evaluator's opinion, the accused satisfies the criteria for not guilty by reason of insanity in that particular jurisdiction. In cases complicated by methamphetamine use, this often requires distillation of whether the defendant's altered state of mind at the time of commission of the offense resulted from methamphetamine use or a primary psychosis. Methamphetamine use can lead to several different patterns and duration of psychoses that can frequently complicate such evaluations. Further, existing diagnostic schemata fail to capture the complexity of psychoses associated with methamphetamine use.

In this article, we discuss the pharmacological properties of methamphetamine and their implications for forensic psychiatrists. We present an update on the state of existing literature on methamphetamine-associated psychosis and its variegated presentation. We underscore the shortfalls in existing classificatory schemes for methamphetamine-associated psychosis that can complicate forensic mental health evaluators' opinions in criminal responsibility evaluations. We offer practical considerations for forensic mental health professionals performing criminal responsibility evaluations where methamphetamine use is a consideration.

We chose methamphetamine as the subject of our study for several reasons: amphetamine-type stimulants are some of the most widely used substances in the world after cannabis, with a rapid rise in methamphetamine use in the last five to 15 years[2]; methamphetamine has a high propensity to cause psychosis; and, as our review indicates, symptoms of psychosis induced by methamphetamine have a high degree of overlap with primary psychoses, including schizophrenia, making methamphetamine use an important confounder in criminal responsibility evaluations.

Pharmacology of Methamphetamine

Methamphetamine is an indirect agonist at dopamine, noradrenaline, and serotonin receptors.[8] It also causes norepinephrine, dopamine, and serotonin transporters to reverse their direction of flow, resulting in increased stimulation of postsynaptic receptors.[9] Further, methamphetamine attenuates monoamine metabolism by inhibiting monoamine oxidase.[8] Through these mechanisms, methamphetamine increases dopamine levels in the central nervous system by as much as 2,600 percent.[9]

It is believed that a significant proportion of the dopamine-producing cells in the brain can be damaged by prolonged exposure to even low levels of methamphetamine.[9] Repeated exposure to amphetamines damages serotonergic axons as well.[8] It has been postulated that dopamine plays a role in methamphetamine-induced neurotoxicity and the consequent reduction in dopamine ultimately affects memory, attention, and decision-making.[9]

Effects of methamphetamine last for days in the body, and some degree of neurological impairment may last for two or more years after cessation of drug use.[9] Primate experiments demonstrate that methamphetamine use, within a dose range consistent with human illicit use, can lead to prolonged neurotoxicity that may require more than a year for complete recovery.[8] Brain abnormalities persist beyond the period of methamphetamine consumption.[8] Striatal abnormalities can persist for years after cessation of habitual methamphetamine use but may recover partially after six to 12 months of abstinence.[8] These studies offer a plausible biological mechanism of lasting deficits because of habitual methamphetamine use that persists after complete cessation.

Methamphetamine is commonly smoked, injected, ingested, insufflated, dissolved sublingually, taken rectally, or solubilized and consumed as a liquid. Different routes of administration also have different rates of metabolism. A study measuring plasma levels of methamphetamine found that, after oral administration, plasma levels of methamphetamine began to rise 30 minutes after ingestion and reached peak levels at about three hours.[10] Plateau levels were maintained for three to four hours and slowly declined over the next four hours.[10] After smoking a roughly equivalent dose, plasma levels of methamphetamine reached approximately 80 percent of peak levels within minutes and peaked about two hours after administration.[10] Plateau levels were maintained for another two hours and then slowly declined over the next four hours.[10]

The mean elimination half-life for methamphetamine is approximately 10 hours, and small, single intravenous doses are detectable in plasma for 36 to 48 hours. Methamphetamine accumulates in the urine with repeated dosing and has been detected in urine seven days after completing a regimen of four daily 10 mg doses or a single large oral dose.[8] Self-reported illicit doses are typically 50 to 500 mg (and up to four grams per day), indicating that the drug could be detected in the urine for even longer.

Methamphetamine withdrawal is characterized by disturbed sleep, depressed mood, anxiety, and drug-craving.[8,9] Acute withdrawal typically lasts seven to 10 days.[8,11,12] Residual symptoms associated with neurotoxicity may persist for several months.[8]

Methamphetamine-Associated Psychosis

The risk of developing psychosis from methamphetamine use varies, with some people not developing any psychotic symptoms despite habitual and excessive methamphetamine use and others developing psychosis with minimal use. There is increased risk of psychosis with earlier age of use, larger amounts consumed, or more frequent use.[2] In their prospective longitudinal study, McKetin *et al.*[13] found a dose-response effect between frequency of methamphetamine use and psychotic symptoms. They reported that the likelihood of psychotic symptoms peaked at 48 percent after 16 or more days of chronic use. People who develop psychosis from methamphetamine use are more likely to have a diagnosis of methamphetamine dependence and a family history of a psychotic disorder.[6] In fact, in a study of 309 participants in Australia, dependent methamphetamine users were three times more likely than non-dependent methamphetamine users to have had psychotic symptoms in the past year.[14] Studies have found several genes, including DTNBP1, that are implicated in the dopamine and glutamate signaling pathways and are associated with both schizophrenia and methamphetamine-associated psychosis.[6] There is evidence that, if a person has developed psychotic symptoms from methamphetamine use once, that person is more vulnerable to developing psychotic symptoms with subsequent use.[6]

Wearne and Cornish's[2] 2018 review concluded that 10 to 28 percent of people with methamphetamine-associated psychosis continued to have psychosis for more than six months after cessation. This persistent psychotic syndrome has been found to have similarities to schizophrenia. When psychotic symptoms last longer than six months, a person with methamphetamine-associated psychosis may be diagnosed with schizophrenia. In 2020, Arunogiri *et al.*[6] estimated that up to a third of people who develop methamphetamine-associated psychosis are later diagnosed with a primary psychotic disorder. This estimate is based on population-based linkage studies, such as

rates of admission for psychotic disorders, instead of prospective studies of people who use methamphetamine. The relationship between methamphetamine use and schizophrenia is complex, with debate in the literature about whether or not methamphetamine-associated psychosis is an entity distinct from schizophrenia.

Early efforts to differentiate the symptoms of methamphetamine-associated psychosis from schizophrenia, as cited by Arunogiri and colleagues,[6] noted a striking lack of negative symptoms in methamphetamine cases. Recent studies paint a more nuanced picture. A 2024 meta-analysis found that negative symptoms are significantly less common in methamphetamine-associated psychoses than in schizophrenia, but they are still frequently observed.[15] Ali and colleagues[16] estimated that negative symptoms were present in 26 percent of methamphetamine-associated psychosis cases. A 2019 systematic review found that only six to 19 percent of studies surveyed reported any negative symptoms in methamphetamine-associated psychosis.[17] The studies that observed negative symptoms tended to be longitudinal, suggesting that the "profile of negative symptoms may change over the course of [the disorder] to more closely resemble schizophrenia" (Ref. 17, p 555). Relatedly, individuals with chronic methamphetamine-associated psychosis have cognitive deficits similar to individuals with schizophrenia.[2] These include deficits in tasks of memory, sustained attention, selective attention, and executive function that are mediated by the frontal and temporal lobes.

Auditory hallucinations and paranoid delusions commonly seen in methamphetamine-induced psychosis are often indistinguishable from those in schizophrenia with comparable frequency and severity.[2] Thought disorder and disorganized speech, however, occur more commonly in schizophrenia than methamphetamine-associated psychosis.[2] Bousman *et al.*[18] studied 40 people dependent on methamphetamine and concluded that delusions occurred in most participants, but only some experienced hallucinations. In contrast, a study of 102 individuals by Shelly and colleagues[19] found that delusions of thought broadcasting were more common in schizophrenia, whereas auditory hallucinations were more common in methamphetamine-associated psychosis. Wang *et al.*[20] found that visual and tactile hallucinations, especially the sensation of insects crawling under the skin, are more common in methamphetamine-induced psychosis compared with schizophrenia.

Substance Use and Criminal Responsibility

Intoxication with substances can affect a person's mental state and induce symptoms of psychosis identical to that of a primary mental illness. The law recognizes that involuntary intoxication (or the unknowing ingestion of an intoxicating substance) may be the basis for a complete defense. In the United States, voluntary intoxication cannot be used as a complete defense to a crime or as a basis for a plea of not guilty by reason of insanity.[21] Statutory law and case law defining voluntary intoxication could potentially negate the *mens rea* necessary for certain crimes, although the exact mechanism and availability vary by jurisdiction.[21,22] Notably, the law does not make a distinction between whether the altered state of mind was because of a singular use of an intoxicant or if the defendant experienced an addiction, commonly defined as persistent drug use in the face of negative consequences.[21]

In 2022, Glancy and colleagues[23] reviewed the Canadian case *R v. Sullivan* (2020) on automatisms, unconscious involuntary behaviors, secondary to intoxication. In *R v. Sullivan*, the Ontario Court of Appeal found that an Ontario law infringed on the presumption of innocence by substituting the intention to become intoxicated with the intention to commit violence. In 2023, Glancy and colleagues[24] reviewed Canadian, British, and American case law on automatisms. They highlighted inconsistencies in how various courts have tried to deal with the complex legal question of self-induced intoxication causing automatism.

Since the mid-19th century, courts in the United States have recognized one circumstance in which behavior resulting from the consumption of intoxicating substances can be the predicate for an insanity verdict. When consumption of a substance results in an exculpatory mental state that persists beyond the period of acute intoxication, courts have allowed insanity verdicts, deeming such cases instances of settled insanity.[25] To use this defense, the defendant must demonstrate insanity at the time of the offense because of the continued effects of the prior intoxication.

A majority of states and the District of Columbia have accepted settled insanity, i.e., persistent psychosis beyond intoxication, as the basis for an insanity defense, and only one state, Colorado, has explicitly

rejected it.[26] These cases exemplify that the type of substance, duration and frequency of use, or permanency of the insanity are not factors precluding a defense of settled insanity.[27,28] Instead, the common thread in most cases of settled insanity is the occurrence of an altered mental state because of prior substance use that meets statutory criteria for insanity.

Criminal Responsibility

In recent years, state courts have had occasion to apply their settled insanity jurisprudence to cases involving methamphetamine intoxication and methamphetamine-associated psychosis. Despite the complex relationship between methamphetamine use and mental impairment, cases involving methamphetamine use serve to underscore the heterogeneous application of settled insanity law both because of the highly fact-intensive nature of this legal analysis as well as state-specific differences in common law and statutory law. Current case law illustrates the need for a clear nosological entity describing psychosis caused by methamphetamine use but persisting beyond the period of acute intoxication by methamphetamine.

Hawaii v. Tome[29] provides an early example of the complexity of assessing criminal responsibility where underlying primary mental illness is complicated by methamphetamine use. In that case, the defendant was charged with drug- and weapon-related offenses. She relied upon the insanity defense. The court concluded that the defendant proved by a preponderance of the evidence that, at the time of the alleged offenses, she experienced schizophrenia, exacerbated by her chronic use of methamphetamine, or that she experienced a methamphetamine-induced psychotic disorder, both of which are mental diseases, disorders, or defects that caused her to lack substantial capacity to either appreciate the wrongfulness of her conduct or conform her conduct to the requirements of the law.[30] The court also concluded that the state had failed to prove beyond a reasonable doubt that, at the time of the alleged offense, the defendant was in fact intoxicated or otherwise substantially impaired as a direct result of being intoxicated, thereby negating voluntary intoxication as the precursor of her state of mind at the time of the offense.

In *State v. Abion*,[31] the defendant was convicted of assault in the second degree. During the trial, the defendant attempted to admit the testimony of a medical examiner that suggested he had a genetic predisposition to psychosis. The prosecution asked the trial court to exclude this testimony as inadmissible because, during the alleged offense, the defendant was impaired because of methamphetamine intoxication and self-induced intoxication precluded a "lack of penal responsibility defense" under Hawaii law (Ref. 31, p 271). In this case, the defendant had used methamphetamine several days prior to the offense. The defendant appealed this decision to the Supreme Court of Hawaii, which held that the trial court erred in excluding the medical examiner's testimony. The court found that "self-induced intoxication" does not include "permanent mental impairment caused by the ingestion of. . . methamphetamine" (Ref. 31, p 282). The defendant had the right to present evidence that he was experiencing long-term effects of methamphetamine at the time of the offense and that he was not using methamphetamine in the several days preceding.

In *State v. Brennauer*,[32] the defendant was convicted of four felony charges stemming from the stabbing of two police officers during an arrest. Relying on the insanity defense at trial, the defendant educed testimony that: he had a long history of mental illness in the absence of intoxication; he had used methamphetamine heavily during the four years prior to the alleged offense; he had last consumed methamphetamine two days prior to the offense; and he was not intoxicated with methamphetamine at the time of the offense.

After conviction, the defendant appealed to the Nebraska Supreme Court, which assessed whether the trial court committed a plain error in its instructions to the jury. The court found that delirium or psychosis that is "immediately produced" by intoxication with methamphetamine is an incomplete defense, because this form of impairment is not a mental disease or defect as a matter of law. The court noted that mere intoxication is not a bar to raising an insanity defense because "one may be both intoxicated and insane" (Ref. 32, p 321). Further, the court held that the defendant was entitled to jury instructions on "settled insanity produced by intoxication," which "affects criminal responsibility in the same way as insanity produced by any other cause" (Ref. 32, p 321–22).

A careful reading of case law surrounding methamphetamine and settled insanity reveals that appellate courts are generally not sympathetic to defendants who appeal convictions where evidence of acute

intoxication with methamphetamine and an underlying primary psychiatric disorder are present. Indeed, of state courts that have directly addressed psychosis in the setting of recent methamphetamine use, Nebraska is an exception in its narrow interpretation of what circumstances qualify as self-induced intoxication and are therefore excepted from the insanity defense. By contrast, in *Lickliter v. Commonwealth*,[33] the defendant was convicted of murder and tampering with evidence after the trial judge failed to instruct the jury on the insanity defense. At trial, the defendant presented evidence that chronic use of methamphetamine caused him to experience paranoia and delusional thinking at the time of the alleged offense; however, no testimony was given as to whether the defendant experienced a mental illness that could qualify for Kentucky's insanity defense. Further, although the defendant "did present evidence that he killed the victim based on delusional thoughts, [these] manifested. . . because of [his] chronic abuse of methamphetamines" (Ref. 33, p 68). The court ruled against the defendant, writing, "[t]he legislature of this state has not expressed any intention that drug addiction arising from the voluntary ingestion of drugs, by itself, affords a defense to a criminal charge based on mental illness" (Ref. 33, p 68). The court made no distinction between methamphetamine dependence and methamphetamine-associated psychosis.

Similarly, the Court of Appeals of Oregon reviewed a trial court's decision to instruct a jury that substance-induced psychosis was not a mental disease or defect in *State v. Folks.*[34] Trial evidence suggested that the defendant had been continuously using large doses of methamphetamine for the weeks leading up to the alleged murder; however, his mental impairment did not persist when he was no longer intoxicated with methamphetamine. The appellate court interpreted a state law defining "mental disease or defect" for the purposes of the insanity defense to exclude any "abnormality manifested [by]. . . antisocial conduct" or "consisting solely of a personality disorder" (Ref. 34, p 472). The court found that, in this context, the term "personality disorder" was a legal term of art and read it broadly to include methamphetamine dependence as well as "drug-induced psychosis" that existed only when the defendant was under the influence of methamphetamine. Of note, the court stated that the defendant did not qualify for a settled insanity defense, as this requires "a chronic mental impairment" and a disability that "continues to manifest independently of the use of or withdrawal from drugs" (Ref. 34, p 476).

Appellate case law directly addressing settled insanity in the setting of methamphetamine use is quite sparse, and the first time this topic was addressed by a state appellate court was in 2003 in *People v. Curran*.[35] Accordingly, legal precedent in this area is absent in most jurisdictions and may rapidly change as courts address cases involving methamphetamine-associated psychosis as a matter of first impression. It would appear that the relationship between settled insanity and methamphetamine use is far from settled. Accordingly, forensic examiners performing criminal responsibility assessments must exercise care as to the local cases and statutes addressing settled insanity generally and methamphetamine-associated psychosis specifically.

Notably, the case law has been complicated, in part, by the lack of a clear diagnostic entity that describes psychosis caused by acute methamphetamine intoxication but persisting chronically when the defendant is no longer intoxicated with methamphetamine. For example, in *Gorman v. State*,[36] one expert witness diagnosed the defendant with "[u]nspecified psychotic disorder" and another with "substance-induced psychosis and/or the residual effects of the substance." The trial court explicitly contemplated the difficulty of ruling in a case with this kind of diagnostic uncertainty, stating, "[w]e have multiple conclusions. . . [o]ne doctor says that this was the effect of methamphetamine. . . [t]he other doctor says it was an unspecified psychosis" (Ref. 36, p 9). Ultimately, the trial court concluded that, because of the possibility that unspecified psychosis could be because of methamphetamine, the defendant did not meet the burden of showing insanity at the time of the offense by a preponderance of evidence. The appellate court affirmed this ruling. Relatedly, in *Folks* (Ref. 34, p 471), one expert testified that the defendant had experienced "drug-induced psychosis. . . caused by his long-term methamphetamine use," whereas another testified, lacking a diagnostic specifier, that the defendant may have experienced "some type of psychotic phenomenon that doesn't appear to fully resolve. . . [and] can last a long time" (Ref. 34, p 471). These examples serve to highlight the manner in which the lack of a nosological entity describing prolonged psychosis caused by methamphetamine use in the absence of acute methamphetamine intoxication has led to difficulties in providing expert testimony and

confusion in the case law regarding settled insanity in cases involving methamphetamine use.

Discussion

Methamphetamine can cause a range of psychotic symptoms, from transient psychosis during acute intoxication to chronic, persisting psychosis with similarities to schizophrenia. The duration of methamphetamine-associated psychosis depends on numerous factors, such as earlier age of use, larger amounts of methamphetamine consumed, genetic predisposition, and more frequent methamphetamine use.[2] Methamphetamine use can have prolonged biochemical and neuroanatomical effects.

When psychosis persists longer than 30 days after cessation of methamphetamine use, it no longer meets the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) criteria for a "substance-induced psychotic disorder."[37] To resolve this diagnostic dilemma, some researchers have proposed nomenclature of acute and chronic methamphetamine-associated psychosis with a 30-day cutoff separating the variants.[2] Research has highlighted differences between chronic methamphetamine-associated psychosis and schizophrenia, suggesting that it may be a separate clinical entity not captured by the DSM. Tomiyama[38] proposed a new nomenclature for this chronic psychosis: "residual methamphetamine psychosis." "Methamphetamine-associated psychotic disorder" has since been adopted to describe this condition, as suggested by Mathias *et al.*[39]

Although the potential for methamphetamine to lead to acute psychosis has been well recognized, its potential to lead to persistent schizophrenia-like psychosis is poorly understood. Wearne and Cornish[2] summarize the spectrum of hypotheses that have been proposed to explain the complex relationship between methamphetamine-associated psychosis and schizophrenia. Some researchers have hypothesized that methamphetamine use may be a factor in the stress-vulnerability paradigm leading to schizophrenia along with genetic and environmental vulnerabilities.[30–42] Others have proposed that methamphetamine may directly cause schizophrenia.[43–45] Both of these explanations suggest that methamphetamine-associated psychosis and schizophrenia may be the same disorder on a continuum. Alternatively, other researchers have theorized that methamphetamine-associated psychosis and schizophrenia are entirely distinct entities.[46,47] In sum, there continues to be a lack of consensus about whether methamphetamine-associated psychosis and schizophrenia are distinct disorders or the same disorder on a continuum.

The similarities and complex relationship between chronic methamphetamine-associated psychosis and schizophrenia make them, in many instances, nearly impossible to distinguish, especially if a person's symptoms remit substantially with antipsychotic treatment before 30 days. Yet this distinction is often asked, if not required, by courts when determining an individual's criminal responsibility.

Separating these diagnoses in cases of criminal responsibility is further complicated by the DSM-5-TR criteria for primary psychotic disorders. Under current diagnostic schemata, psychosis that lasts for more than six months after abstinence would best fit diagnostic criteria for schizophrenia.[37] Some researchers have hypothesized that methamphetamine-associated psychosis lasting longer than six months represents a distinct disorder that may be misdiagnosed as schizophrenia.[2] Diagnosing schizophrenia because of methamphetamine use may be in adherence to current diagnostic criteria but may not be a true reflection of the cause of the psychosis. As things stand, there are no specifiers available to indicate that methamphetamine use was in fact a precursor of this persisting psychosis. Whether a person is diagnosed with chronic methamphetamine-associated psychosis or schizophrenia can eventually have significant implications on a determination of criminal responsibility.

We present two hypothetical clinical scenarios where reliance on DSM-5-TR diagnostic criteria complicates criminal responsibility evaluations for defendants with both methamphetamine use and symptoms of psychosis. In Scenario A, an individual with habitual methamphetamine use commits an offense while in a state of psychosis but has not used methamphetamine in the two weeks prior to commission of the offense. According to diagnostic criteria stated in the DSM-5-TR, the most appropriate diagnosis for this defendant is "methamphetamine-induced psychosis," because the most obvious etiology for symptoms of psychosis would be habitual use of methamphetamine. It could be argued that the defendant was not intoxicated with methamphetamine at the time of the offense, and the body had metabolized any residual methamphetamine from the last use. Moreover, for the forensic psychiatrist who determines that the defendant was acting under the influence of delusions at the time of commission of

the crime, teasing out the extent to which there is a correlation between voluntary intoxication and the defendant's acts is a vexing task with no simple answers. For example, Bourget[48] has argued that it may not be fair to compare people who know what will happen to them if they drink or take drugs with those who cannot foresee the consequences because the consequences occur rarely (i.e., psychosis or development of a mental disorder).

In Scenario B, an individual who uses methamphetamine daily and has experienced psychosis for several months commits a crime and the symptoms of psychosis persist for over six months after the offense despite abstinence. In this scenario, the most appropriate initial DSM-5-TR diagnoses would be "methamphetamine-induced psychosis" or "unspecified schizophrenia spectrum and other psychotic disorder." After six months have lapsed, however, the most appropriate diagnosis would be schizophrenia or another primary psychotic illness, indicating that this may have been a person whose primary psychosis was precipitated by methamphetamine use. Forensic examiners who evaluate the defendant early on may be tempted to attribute symptoms of psychosis entirely to methamphetamine use, especially because the defendant was engaged in active use in the days leading up to the offense. Evaluators who assess the defendant after six months may attribute the symptoms to either a primary psychosis or a settled psychosis, thereby minimizing the role of intoxication with methamphetamine at the time of the offense.

The ability to evaluate whether defendants meet a jurisdiction's test for a finding of not criminally responsible is a core skill in forensic psychiatry. Forensic evaluators assigned to perform criminal responsibility evaluations should familiarize themselves with the approaches and methods set forth in the American Academy of Psychiatry and the Law (AAPL) Practice Resource for Forensic Psychiatric Evaluation of Defendants.[49]

The importance of gathering a detailed and accurate history cannot be overstated in cases raising the insanity defense.[48] With regards to methamphetamine use, it is not only important to obtain information about history of onset, duration, frequency, pattern, and severity of use, it is also equally important to ascertain the date of last use, its temporal relationship to the offense, and the pattern and severity of symptoms in the period surrounding the offense. With respect to symptoms of psychosis, evaluators must carefully trace the chronology of symptoms since onset, the nature and severity of symptoms, and their temporal relationship to substance use. Further, independent corroboration of all information should be done using objective sources of collateral information. These may include police reports; psychiatric, substance abuse, and medical records; personal, school, and employment records; other expert evaluations; results of brain imaging; psychometric tests; and photographic or audiotape evidence available in the case.

Toxicology reports, where available, should be correlated with the clinical picture. Evaluators should be familiar with the strengths or limitations of the particular toxicological method used, including the type of sample taken, the time of sampling in relationship to the time of the offense, which drugs the particular toxicological method screens for, and whether the toxicological method was designed as a screen or as a definitive test (Ref. 48, p S23). A toxicologist should be consulted when deemed necessary. This is especially true in jurisdictions that have held that the settled insanity defense may apply in circumstances where psychosis persists immediately after the acute effects of methamphetamine intoxication have abated. In these jurisdictions, courts have allowed evidence to be presented supporting a settled insanity defense in instances where the defendant had consumed methamphetamine "several days preceding" the offense[31] and even two days prior to the offense.[32] In these jurisdictions, toxicology reports estimating whether methamphetamine was present in a defendant's system at the time of the offense may prove just as important to the resolution of the case as the particular diagnoses assigned to the defendant.

When providing their opinion in written or oral format, forensic evaluators must provide a detailed account of methamphetamine use and its temporal relationship (or lack thereof) to the symptoms of psychosis in each case. The final opinion should be guided by the specific laws in the jurisdiction of practice and the acknowledgment that the arbiter of fact may choose not to accept the examiner's opinion as it pertains to the impact of methamphetamine use on the symptoms of psychosis or actions that constituted the offense. Evaluators must understand that the scope of the settled insanity doctrine could also be shaped by state legislation and social conditions, which may oppose the broadening of the doctrine to cases where self-induced intoxication with methamphetamine leads to persistent psychosis.

Conclusion

Our review indicates that there is a great deal of overlap between symptoms of schizophrenia and persisting psychosis because of methamphetamine use that can complicate criminal responsibility evaluations. Forensic examiners must grapple with the limitations of the current diagnostic scheme that is silent on substance-induced psychosis that persists beyond one month after abstinence.

We have witnessed an increase in the utilization of the diagnosis "unspecified schizophrenia spectrum and other psychotic disorder" in many such instances, where the onset of psychosis occurs in the course of substance use but residual symptoms of psychosis persist beyond 30 days. In these scenarios, clinicians are no longer able to diagnose the evaluee with "substance-induced psychotic disorder" but do not have sufficient information to offer another definitive diagnosis (e.g., schizophrenia). Unlike primary psychotic disorders, there are no diagnostic or duration criteria specified in the DSM-5-TR for making this diagnosis, and the DSM-5-TR specifically notes that this diagnosis is to be made only tentatively while waiting for more information (e.g., in emergency room settings) (Ref. 37, p 138). This indicates that the diagnosis, "unspecified schizophrenia spectrum and other psychotic disorder," was not intended in the DSM-5-TR to be a syndromal diagnosis, but its increasing use as a syndromal diagnosis in forensic evaluations underscores this diagnostic conundrum.

Given these diagnostic complexities, we suggest an evolution in classification of psychosis similar to the way diagnoses have evolved in other areas of medicine. Within psychiatry, diagnostic schemata have moved away from named disorders to instead supplant them with syndromal diagnoses. For example, the diagnosis of "dementia" in Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)[50] has since evolved into "major neurocognitive disorder" beginning with the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).[51] In describing the proposal for this diagnostic change, the workgroup on major neurocognitive disorder noted that, first, a syndromal diagnosis is made and then potential causative factors are examined to determine the etiology.[52] Under the current scheme, the underlying etiology can subsequently be incorporated into the diagnosis with a specifier.

Applying the same principle to psychosis, a new diagnostic scheme could be conceived that first describes syndromal psychosis and then by way of specifiers denotes whether it is primary (as in the case of schizophrenia) or secondary (because of a general medical condition or substance use). Ours is by no means a novel proposition, and evidence for this line of thinking comes from the framers of the DSM themselves. Beginning in DSM-5, "substance/medication-induced psychotic disorders" were removed from the section on "substance-related and addictive disorders" and placed in the chapter on "schizophrenia spectrum and other psychotic disorders"[51] to signify a shift in conceptualization of these disorders as primarily being psychoses, albeit secondary to substance or medication use. Although we realize that our specialty may never be able to give up on the diagnosis of schizophrenia because of its iconic identity, our improved understanding of chemically induced psychosis and its overlap with idiopathic psychoses already compels us to view the two categories in one basket.

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