

Commentary: Intoxication and Settled Insanity—Unsettled Matters

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The role of exogenous substances in the genesis of mental symptoms has found relevance in some jurisdictions when a defense of settled insanity is raised. However, the current nosology and knowledge base reveal ambiguity and unresolved questions about the present science related to settled insanity.

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Feix and Wolber¹ present a case in which the defendant successfully argued an insanity defense with a claim of settled insanity under Virginia law. In their case, in which a conventional assessment approach was used, the forensic opinion in support of settled insanity was derived from the hypothesis that the defendant's marijuana and alcohol use resulted in a psychotic disorder (schizophreniform disorder, psychotic disorder not otherwise specified, or possible schizophrenia). In this commentary, we use the index case of settled insanity to highlight the unresolved clinical aspects of this concept and their potential to cause confusion in the legal system.

In clinical terms, the central debate in a case of settled insanity revolves around the origins of the mental disorder at the time of the charged crime, particularly whether a defendant's voluntary use of one or more psychoactive substances led to a transient mental state such as acute intoxication, to a more stable mental state such as a substance-induced psychotic disorder, or to a permanent condition, such as psychotic disorder not otherwise specified or a schizophrenia spectrum disorder. We briefly explore the current diagnostic system and recent re-

search to highlight the continuing conundrums that forensic psychiatry, the legal system, and society may encounter when considering the concept of settled insanity.

Nosological Uncertainty

Jurisdictions have differing mental criteria definitions and thresholds where settled insanity is permissible. Although the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*² is not statutorily mandated, it serves as a starting point for our discussion, as it standardizes the clinical description of mental disorders.

The diagnoses of substance intoxication, substance intoxication delirium, and substance-induced psychotic disorder require that an exogenous substance be identified as the etiologic agent. The central feature of substance intoxication lies in its reversibility and the direct and temporal linkage of substance use as the etiologic agent to the behavioral or psychological changes. The cardinal feature of substance intoxication delirium involves disturbances in consciousness and cognition of greater severity than that exhibited in substance intoxication. The central feature of a substance-induced psychotic disorder is the presence of hallucinations or delusions that cannot be better explained by a preexisting or independent mental disorder.

Although the immediate impulse would be to view these three diagnostic entities as lying along a continuum, they do not appear to do so. Our current nosology adds to the confusion when one attempts to extend clinical descriptions to psycholegal analysis. The diagnoses of substance intoxication and sub-

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stance intoxication delirium do not exclude the possibility of psychotic symptoms. The diagnosis of substance intoxication delirium appears to be a more cognitively toxic response to the substance use. In this sense, the substance intoxication delirium appears to branch off from the line between substance intoxication and substance-induced psychotic disorder. No time frames are given to assist in differentiating among these three diagnoses. For each diagnosis, the diagnostic criteria force a dichotomous choice between assigning causation to either the exogenous substance or a preexisting or independent mental disorder, when in clinical practice the contributions may be derived from a variety of factors, including acute or recent consumption of a substance and the individual's preexisting neurobiological matrix.

Beyond the ambiguity of the substance-related diagnosis, we reach the boundary between a substance-induced and a non-substance-induced psychotic disorder. In particular, the boundary between a substance-induced psychotic disorder and a psychotic disorder not otherwise specified may not be as well demarcated as we would like to believe. Again, forcing a dichotomous decision point regarding causation may not be in synchrony with clinical reality. We have performed only a cursory surface analysis of the diagnostic confusion that may arise when approaching diagnostic decisions in actual practice.

To highlight this diagnostic uncertainty, we present the following example extracted from a recent Washington Court of Appeals case. None of us had any professional involvement in this case. Our knowledge of it comes directly from the appellate court's ruling, though the case generated considerable media attention because a law enforcement officer died in the incident. We give only the clinically pertinent parts of *State v. Matthews*,^{3,4} of which some derives from the unpublished part of the opinion.

State v. Matthews

On June 22, 2002, Ronald Matthews ingested cocaine and ran naked into traffic. He was shouting racial epithets and banging on cars and a bus. A sheriff's deputy, Richard Herzog, arrived at the scene and ordered Matthews to get out of the street. Matthews charged at Herzog and was unaffected by Herzog's pepper spraying him. A struggle ensued. As Matthews tried to remove Herzog's firearm from its holster, the gun fell to the ground and the clip fell from the gun. Matthews picked up the gun, inserted the

clip, and fired at Herzog, who fell to the ground after being struck by a bullet. Matthews then stood over Herzog and fired four shots into the deputy's head. When a person came to assist Herzog, Matthews pointed the gun at him and said, "I'll shoot you, you hero." Matthews then walked back to a nearby apartment building and climbed up a balcony into his own apartment. He placed the gun under the mattress of his bed and then stood on his balcony ranting and holding a Bible. He called 911, admitted he had killed the deputy, and told the operator that the deputy deserved the death penalty. The local police subsequently took Matthews into custody. The police found the gun, crack cocaine residue, and drug paraphernalia in the apartment.

The state charged Matthews with first-degree aggravated murder. He entered an insanity plea and the trial began on July 15, 2004. Three expert witnesses testified for the defense. These three agreed on the diagnosis of paranoid schizophrenia and that the patient had been completely out of touch with reality. The prosecution's expert witness testified to the following: Matthews had cocaine metabolites in his blood that indicated that he had used cocaine within three days of the shooting; he may have disrobed because of an increase in body temperature due to cocaine use; his use of cocaine could have resulted in incoherence, agitation, and aggression toward others; and his presentation on the day of the shooting was consistent with "cocaine-excited delirium." The prosecution's expert conceded, however, that the literature about cocaine-excited delirium generally includes four components—hyperthermia, delirium, respiratory arrest, and death—and that Matthews did not undergo respiratory arrest or die. A defense expert witness opined that Matthews had bipolar disorder and cocaine intoxication at the time of the shooting. The expert stated that Matthews did not have cocaine-excited delirium and opined that Matthews was insane at the time of the shooting. Before trial, another prosecution expert witness was planning to testify that Matthews had cocaine-induced psychosis on the day of the shooting, but that Matthews was not insane. However, by the time he testified, the examiner had re-evaluated his diagnosis after reviewing testimony and literature provided by the first prosecution expert. The second prosecution expert then opined that Matthews had bipolar disorder, was not insane, and had had cocaine-excited delirium. That latter expert described cocaine-ex-

cited delirium as a kind of cocaine-induced psychosis, or what the DSM would call cocaine intoxication. During trial, the judge said on August 12, 2004:

I'm also cognizant of the fact that all these experts say that this is a theory that is at least somewhat new. That it does not appear in the DSM and that this is a theory that to some extent has been developed during the cross-examination and examinations of experts in this case . . . [Ref. 3, p 6].

After the second prosecution expert testified, the defense presented two more expert witnesses who contradicted the prosecution expert's opinions with respect to cocaine-excited delirium. One of the experts testified that the condition would affect the ability of a person to understand the nature and quality of his acts; the other expert testified that Matthews did not fit the symptoms of a person with this condition.

On August 19, 2004, after a brief deliberation, the jury found Matthews sane and guilty as charged. The court sentenced Matthews to life imprisonment without parole, and he appealed on due process grounds. The court of appeals affirmed the trial court's ruling.

Sampling of Recent Biological Research

A quick detour into the research world finds substantial exploration at the microscopic level. There has been recent robust research activity involving the psychotogenic role of the major psychoactive drugs of abuse, including alcohol, amphetamines, cannabis, and cocaine, and to a lesser extent methylenedioxymethamphetamine (MDMA, also known as Ecstasy), phencyclidine (PCP), ketamine, and inhaled hydrocarbons.⁵

In a review of the literature by Thirhalli and Benegal,⁵ relevant findings were that: psychosis and substance abuse co-occur more frequently than can be explained by chance alone; and there is a twofold higher risk that psychotic symptoms will manifest than for those with alcohol (but not other drug) dependence. Thirhalli and Benegal also reported on some previously unpublished data by Thirhalli and colleagues from their study of out-of-treatment drug abusers in St. Louis (intravenous drug, crack cocaine, and heroin users). Most of the participants had a history of multiple-substance dependence. The prevalence of psychotic symptoms in the context of specific substances was 83 percent for hallucinogens, 82 percent for PCP, 80 percent for cocaine, 64 percent for cannabis, 56 percent for amphetamine, 54 percent for opioids, 41 percent for alcohol, and 32

percent for sedatives. The prevalence of psychotic symptoms increased with the increasing activity or dependence, reaching up to 100 percent among those deeply dependent on cocaine.⁵

Researchers have used neuroimaging tools, including single proton emission computed tomography (SPECT), magnetic resonance spectroscopy (MRS), and positron emission tomography (PET) to examine changes in activity by brain location and by specific neurotransmitters.^{6,7} For example, long-term methamphetamine use has been associated with abnormal cerebral blood flow patterns, reduction of brain dopamine transporter density, and metabolite alteration, which may be closely related to a susceptibility to methamphetamine psychosis.⁶ The brain areas affected in users of alcohol, opioids, marijuana, cocaine, MDMA, and methamphetamine include, but are not limited to, the striatum, the orbitofrontal region, prefrontal cortices, and frontal white matter.⁷ The disturbance of the dopaminergic system has been hypothesized as the cause of methamphetamine-induced psychosis. PET studies have found reductions in D2 receptor density and in dopamine transporter in the striatum and nucleus accumbens, the orbitofrontal and dorsolateral prefrontal cortices, and the basal ganglia in abstinent methamphetamine users. Perturbations in the dopaminergic system appeared to be linked to longer methamphetamine use.⁵

Cannabis appears to be the one commonly used psychoactive substance that has a direct relationship to the onset of psychosis. Cannabis-induced psychosis in most cases can be regarded as the first manifestations of a long-term psychotic illness.⁸ Brain neuroimaging studies of cannabis users have found regions of differential brain activity in the frontal, limbic, and cerebellar regions, suggesting involvement of the extended dopamine reward pathways and possibly of the frontocerebellar network.⁹ Prevailing current theoretical models of induction of schizophrenia by cannabis use appear to involve disturbances in the dopamine pathways and effects on the brain's cannabinoid system.^{10,11} Thirhalli and Benegal⁵ noted that there seems to be overwhelming evidence that cannabis use is associated with subsequent development of schizophrenia or psychotic symptoms. However, their review also indicates that cases of psychosis can develop following heavy cannabis use, though they appear skeptical that only a toxic, but not psychotic reaction had taken place in the literature that they had reviewed. In contrast, in a

recent study of eight healthy male volunteers conducted to assess the effects of cannabis use on driving ability, two cases of acute psychosis developed, which argued for the position that psychosis can arise with cannabis use.¹²

Discussion

The expert witnesses in *State v. Matthews*³ had differences of opinion as to both Matthews' mental disorder and his substance-related disorder. They differed on whether Matthews had paranoid schizophrenia or bipolar disorder. We have three experts whose views are mentioned regarding the role of cocaine in Matthews' case. We have a prosecution witness who explained that Matthews presented with "cocaine-excited delirium," which is not a DSM-IV-TR diagnosis but could be considered to fall under cocaine intoxication delirium. A second prosecution witness who initially planned to testify that Matthews presented with "cocaine-induced psychosis," changed his diagnosis to cocaine-excited delirium after the first prosecution witness testified. In addition, according to the appellate court opinion, this witness described "cocaine excited-delirium as a kind of cocaine-induced psychosis, or what the DSM would call cocaine intoxication" (Ref. 3, p 6). If this transcription of the testimony is accurate, it indicates that this prosecution witness appeared to confuse the three different DSM-IV-TR diagnoses of substance intoxication, substance intoxication delirium, and substance-induced psychotic disorder. A defense witness opined that Matthews did not have cocaine-excited delirium. In summary, we have three expert witnesses who did not arrive at the same substance-related diagnosis.

Although we have not reviewed the court transcripts, from what was written in the appellate court ruling, each expert witness provided a clear dichotomous choice for the causation of Matthews' mental condition and behavior at the time of the shooting (i.e., whether it was related to the defendant's mental disorder or to cocaine use).³ In Washington, voluntary intoxication excludes a defendant from raising the insanity defense, and so the distinction between the various substance-related diagnoses may not be critical. However, given the distinction between a substance-induced psychotic disorder and a psychotic disorder not otherwise specified, eschewing the contribution of an exogenous substance could alter the legal outcome of a case.

A retrospective review affords us the opportunity to second-guess the experts in *State v. Matthews*. Nonetheless, the appellate court ruling did not mention the current clinical complexities concerning the use of psychoactive substances by individuals who have serious mental disorders, which is commonplace clinically. Practicing psychiatrists often face diagnostic uncertainty, even when dealing with dual-diagnosis patients whom they know well. Attributing manifested behavior solely to the acute effects of psychoactive substances may be overly simplistic in many cases.

From our sampling of the literature, we note that the prevalence of psychosis with substance abuse is substantial and that neurotransmitter and brain activity changes may persist long after abstinence. The latter finding may have particular relevance to the contentiousness surrounding the role of substance abuse in insanity evaluations. Longer and/or greater use of psychoactive substances appears to increase the likelihood of significant neurobiological changes that do not remit for substantial periods that can last beyond the time of trial. In other words, should these drug users commit crimes, they appear to have a greater likelihood of manifesting a stable psychosis and qualifying for settled insanity (or insanity), due to the higher likelihood of longer periods of molecular-level brain changes. This possibility could create a disparity among the different types of substance users and in particular "reward" those who have longer and/or greater drug abuse histories.

Another potential inequity could arise when considering the origins of acute psychosis. In the case of individuals who have a serious mental disorder (such as a schizophrenia spectrum or bipolar spectrum disorder), adherence to a treatment plan plays a crucial role in reducing the likelihood of recurrence of or minimizing the symptoms of the mental disorder. Frequently, these individuals unilaterally discontinue their medications and commit crimes. Many of these criminal defendants then qualify for the insanity defense upon manifestation of acute mental symptoms of their ongoing serious mental disorder. But should an act of refusing to take prescribed medications differ from ingesting a psychoactive substance, since a psychotic state results from either? The situation becomes more complex in cases of individuals with serious mental disorders who use psychoactive substances and commit crimes. The attribution of causation solely to the mental disorder or

to drug use may be based more on the persuasive talents of the expert witnesses than any science.

The current nosology and knowledge base indicate that the science related to settled insanity raises more questions than answers. Although the lack of uniformity across state lines regarding the viability of settled insanity reflects differing social policies, it also reflects the clinical uncertainty in the contentious world of psychosis, drugs, and insanity. We certainly need more research into the area of substance abuse and psychosis, both in those without preexisting serious mental disorders and those with these disorders. Whether the law and society will soon be able to incorporate and/or appreciate the complexities of the interaction between drug abuse and psychosis remains uncertain, but we should nevertheless endeavor to find more answers.

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