

Methamphetamine motivated murder: Forensic psychological/psychiatric & legal applications in criminal contexts

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This article examines the clinical and forensic (psycho-legal) aspects of methamphetamine use. The author will describe the clinical and psychiatric effects of the drug on an individual's functioning. Forensic psychological/psychiatric issues including substance-induced psychosis relevant to a not guilty by reason of insanity defense, diminished capacity, and mitigation at capital sentencing will be addressed. Case law pursuant to forensic aspects of methamphetamine use will also be thoroughly explored.

KEY WORDS: *Methamphetamine, substance induced psychosis, forensic psychology, forensic psychiatry, insanity, mitigation.*

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In recent years there has been an increase in violent offenses, often homicides that are committed while an offender is under the influence of methamphetamine. Methamphetamine use has various psychophysiological effects and can lead to paranoid thoughts, acute substance induced psychotic states, and ultimately intense aggressive and violent behavior (Hunt, Kuck & Truitt, 2005).

An offender who commits a criminal offense while under the influence of methamphetamine, like other offenders, may be evaluated at various stages of the legal proceedings. Such examinations may include competency to waive Miranda rights, competency to stand trial, mental state at the time of the offense, diminished capacity, mitigation at sentencing, and competency to waive mitigation and appeals. The time between one's Miranda warnings and an evaluation regarding competency to waive appeals may be many years. Similarly, an individual's physiological effects from the drug methamphetamine may linger for long periods of time, disrupting his cognitive and affective states, and impairing his ability to function in his legal proceedings.

The forensic mental health professional who is requested to examine an offender with a chronic history of methamphetamine use should have a knowledge base about the biopsychosocial effects of this drug on the brain, and subsequently the consequences on an individual's functioning and behavior. The drug's properties and interaction within the brain can lead to acute psychotic states and subsequent violence (Subcommittee on Crime, House of Representatives, 1995). It may cause symptoms similar to schizophrenia, paranoid type (Caton, Samet & Hasin, 2000; Sato, Numachi & Hamamura, 1992). The chronic methamphetamine user may likely have a history of mental illness and dual diagnosis status (Lin, Ball, Hsiao, Chiang, Ree & Chen, 2004). Consequently, it may be difficult for the forensic clinician to determine the etiology of the violent acts from a mental health standpoint.

This article explores how a forensic mental health expert witness can assist the court, jury, and legal counsel in applying his/her knowledge of the drug to various legal referral issues. For purposes of this article, this author will specifically consider methamphetamine-induced psychosis relevant to diminished capacity, insanity, and mitigation at sentencing.

What is methamphetamine?

Methamphetamine is a stimulant drug that came into vogue in Hawaii in the 1980's and found its way eastward to California. Lately it has become very popular in Midwest areas (Yudko, Murray-Bridges & Watson-Hauanio, 2003). Methamphetamine is classified as a Schedule II substance by the Drug Enforcement Agency under the Convention on Psychotropic Substances (United States Drug enforcement Agency, 2007). The drug is illegally manufactured in various forms including: "Crank," (methamphetamine sulfate); "Crystal," (methamphetamine hydrochloride); and "Ice" which is a pure form of D-MA-hydrochloride (Ray & Ksir, 2002). Ice is produced from chemicals that until recently could be purchased over the counter at drug stores but are currently under stricter scrutiny and not as readily available. The drug is rarely sold in pure form; rather, it is diluted with other chemicals. It is commonly produced by the reduction of ephedrine or pseudoephedrine. In the U.S., the manufacturing often includes the use of red phosphorus and iodine, which forms hydroiodic acid and results in a dangerous process as the chemicals are highly inflammable, corrosive, and toxic.

Methamphetamine is a central nervous system stimulant that is injected, smoked, snorted, or ingested orally. It is occasionally prescribed for Attention-Deficit/Hyperactivity Disorder and narcolepsy under the brand name Desoxyn. Methamphetamine is highly psychologically addictive.

Physiological effects of methamphetamine

Methamphetamine has various psychophysiological effects (Murray, 1998). The drug affects neurochemical systems that regulate heart rate, body temperature, blood pressure, appetite, mood, attention, and cognitive functioning. When smoked, crystal methamphetamine enters the body and brain rapidly. It has an onset time of between 5 and 20 minutes and a subjective euphoria of intoxication for up to 8 hours, with a half-life of about 12 to 36 hours (Yukdo, Hall, McPherson & Twemlow, 2003). It has various negative psychosomatic side effects and can cause serious long-term health impairments. Some of the health problems include stroke, cardiac arrhythmia, and stomach cramps.

The drug can cause psychiatric symptoms including anxiety, insomnia, paranoia, hallucinations, and structural changes to the brain (Anglin, Burke, Perrochet, Stamper & Dawud-Noursi, 2000). In fact, there is an increasing prevalence of methamphetamine induced psychosis especially in chronic use (Hartel-Petri, Rodler, Schmeisser, Steinmann & Wolfersdorf, 2005).

Methamphetamine rapidly enters the brain and causes an efficient and powerful release of norepinephrine and dopamine and, to a lesser extent, serotonin. These effects result in a fight-or-flight response including increased heart rate and blood pressure, constriction of arterial walls, an increase in blood sugar, feelings of euphoria and increased energy. In particular, methamphetamine causes the release of dopamine from presynaptic neurons in the brain. The drug will have its most profound impact on dopaminergic neurons commencing from the ventral tegmental area to the cerebral cortex and limbic system (Kaplan, Sadock & Grebb, 1994; Kobayashi, Hasegawa, Ujike, Sekine, Ozaki, Inada, Harano, Komiyama, Yamada, Iyo, Shen, Ikeda & Sora, 2004).

The chronic methamphetamine user will experience a reduced density of dopamine transporter, abnormal cerebral blood

flow patterns (Iyo, Sekine & Mori, 2004) and tolerance (diminished physiological response to a drug after continuous use). In the case of methamphetamine tolerance, the individual will likely experience hypothermic cardiovascular effects (Perez-Reyes, White, McDonald, Hicks, Jeffcoat, Hill & Cook, 1991). There is a highly addictive quality to methamphetamine reflected in a rapid tissue tolerance in chronic users.

In addition to methamphetamine's role in causing psychotic features, the drug is likely to precipitate brain deterioration that continues for months after abstinence (Ernst, Chang, Leonido-Yee & Speck, 2000). Long-term use will likely result in neuro-injury to the dopamine system, in particular, axonic degeneration of the dopamine axon terminals in the striatum, frontal cortex, nucleus accumbens, and amygdala (Hall, McPherson, Tremlow & Yudko, 2003). Single Photon Emission Computerized Tomography (SPECT) scanning data has indicated that brain deterioration continues for months after cessation (Buffenstein, Coel & Combs, 1997; Iyo, Sekine & Mori, 2004). Prolonged use of methamphetamine may cause metabolite alterations in the basal ganglia (Sekine, Minabe, Kawai, Suzuki, Iyo, Isoda, Sakahara, Ashby, Takei & Mori, 2002).

Suggested positive effects of the drug include increased alertness, energy, euphoria, self-confidence, talkativeness, hypersexuality, and loss of appetite when using small doses (Hall, McPherson, Twemlow & Yudko, 2003).

Methamphetamine induced psychosis

Before describing etiological factors and symptoms of methamphetamine induced psychosis, it is helpful to understand the potential methamphetamine user classifications. Chronic methamphetamine users are more likely than recreational users to be at greater risk to experience symptoms that mimic psychotic states.

Methamphetamine user classifications include but are not limited to:

1. The chronic user who may experience substance induced mood and psychotic disorders and who has no comorbid psychiatric diagnoses or premorbid psychiatric history prior to the onset of use.
2. The chronic user who has a history of mental illness.
3. The recreational user who has a history of mental illness.
4. The recreational user without a history of mental illness.

Caton and colleagues (2000) distinguishes between methamphetamine users who simply abuse and become dependent on the drug but who are otherwise free of significant mental illness, and those who experience mental illness including depression, bipolar disorder and schizophrenia. Unfortunately, not infrequently, these two types of users are misdiagnosed which causes faulty treatment planning (Caton, et al., 2000). Some individuals who commit violent acts may have been misdiagnosed and it is crucial for the treating psychiatrist to establish the temporal relationship between the substance use and the onset and continuation of psychotic symptoms when considering differential diagnoses. In addition to addictive qualities of methamphetamine, many users experience psychiatric comorbidity (Lin, et al., 2004).

Critical to the forensic psychological/psychiatric issues detailed in this article, such as mental state at the time of the offense and mitigation at sentencing, methamphetamine can elicit a psychotic reaction in response to chronic use, or even after only one dose (Ando, Hironaka & Yanagita, 1986; Hartel-Petri, et al., 2005).

Methamphetamine use can cause acute or chronic psychosis and one episode of methamphetamine use can leave a person vulnerable to future episodes of psychosis, with or without subsequent methamphetamine use (Sato, Chen, Akiyama &

Otsuki, 1983; Wolkoff, 1997). About a third of chronic users will experience bizarre/psychotic symptoms and hallucinations (Griffith, Cavanaugh & Oats, 1969). The intensity of stimulant-induced psychotic symptoms appears to be in part dose related (Batki & Harris, 2004). Paranoid psychoses, including paranoid hallucinatory states and persecutory delusions, are common among methamphetamine induced psychotic states (Hartel-Petri, et al., 2005; Yui, Goto, Ikemoto, & Ishiguro, 1997).

Persecutory delusions are the most common lifetime psychotic symptoms experienced by chronic methamphetamine users, followed by auditory hallucinations, strange or unusual beliefs, thought reading, visual hallucinations, and negative symptoms such as negative affect, poverty of speech, and psychomotor retardation (Srisurapanont, Ali, Mardsen, Sunga, Wada & Monteiro, 2003). Psychotic flashbacks to subsequent spontaneous recurrences, which can be triggered by psychosocial stress, may also be the result of a methamphetamine induced psychosis (Hartel-Petri, et al., 2005; Yui, Goto & Ikemoto, 2004; Yui, Goto, Ikemoto, Nishijima, Yoshino & Ishiguro, 2001).

Three types of clinical courses of methamphetamine induced psychosis include transient type, prolonged type, and persistent type (Sato, 1992). The two latter types indicate a lasting change in the brain that continues to produce schizophrenia type paranoid psychotic states without methamphetamine use. This lasting vulnerability of the brain to psychotic like symptoms may be caused by a prolonged sensitization of the brain to the psychotogenic action of methamphetamine due to chronic use and can be termed a "residual methamphetamine psychosis."

It is difficult to distinguish the hallucinations of schizophrenia from those due to drug use. According to MacKenzie and Heischouer (1997), the hallucinations of schizophrenia usually are auditory, whereas those for chronic

methamphetamine use are more commonly visual or tactile. Kaplan and Sadock (1994) discuss the difference between paranoid schizophrenia and an amphetamine-induced psychotic disorder. An amphetamine-induced psychotic disorder typically includes a predominance of visual hallucinations, generally appropriate affect, hyperactivity, hypersexuality, confusion, incoherence, and disordered thinking. The affective flattening and alogia (inability to speak) of schizophrenia are generally absent in amphetamine-induced psychotic disorder.

Data suggest that increased noradrenergic hyperactivity resulting in elevations in dopamine release in response to mild stress may predict subsequent psychotic flashbacks (Yui, Ikemoto & Goto, 2002; Yui & et al. 2001). In addition, chronic methamphetamine users are susceptible to psychotic episode recurrences (Yui, Ikemoto, Goto, Nishijima & Kato, 2003). Interestingly, methamphetamine can cause a progressive sensitivity to relapse and a person may revert into states resembling methamphetamine intoxication which are triggered by environmental stress. The person then may even seek out other substances to mimic the effect of methamphetamine when the latter is not available (Ando, et al., 1986). A pathological sensitization of neuronal systems may be an important element for relapse or the onset of stimulant induced psychosis.

Heavier doses of methamphetamine often lead to intoxicated states and features including perceptions of increased competence, self-esteem, extreme impulsiveness, unstable emotions, manic like symptoms including grandiosity, hyper-talkativeness, hypersexuality, disorganized thought, and psychotic features. One may experience other psychological symptoms including panic attacks with fear of impending death, induced delirium, delusions that last for several days, compulsive and repetitive actions, agitation, and rage (Gabbard, 1995). Heavier doses of methamphetamine may lead to heightened hallucinations and intense persecutory

thoughts and delusions during and after intoxicated states. Due to these intense and fear inducing psychotic features, individuals are much more at risk for committing violent acts.

The period following the intoxication state, sometimes referred to as “crashing,” includes symptoms such as restlessness, depression, irritability, craving for the drug, fatigue, confusion, disorientation, increased thirst and appetite, and long periods of sleep (even days). Neurological/neuropsychological impairments include memory problems, poor attention and concentration, and visual-spatial deficits. At times a person may experience other psychotic like symptoms such as disorganized lifestyle, poor hygiene, poor judgment, and irresponsibility.

Data suggests that those individuals who experience methamphetamine induced psychosis improve significantly in six months after inpatient treatment (Yeh, Lee, Sun & Wan, 2001). However, many individuals experience psychosis after the discontinuation of methamphetamine. Some individuals who experience long-term abstinence from methamphetamine relapse, are prone to an acute exacerbation of psychotic states which are almost identical to the initial psychotic episode while using an amount less than initially used (Sato, et al., 1983). In one case at the forensic psychiatric hospital at which the author was formerly employed, a patient who experienced a methamphetamine induced psychosis upon admission continued to display residual psychotic features of the drug even after two years of abstinence while residing in the hospital.

Although methamphetamine induced psychosis is a psychotic disorder that is different than chronic schizophrenia, its effects can cause similar results including serious violence. When this is the case, a methamphetamine induced psychosis can be relevant when considering affirmative defenses, mitigation at sentencing, and providing the trier of fact an understanding of the derivation of the violent behavior.

Methamphetamine, diminished capacity and legal insanity

Diminished capacity For purposes of this article, we will consider mens rea in the context of specific intent homicide cases due to the fact that many methamphetamine induced psychotic related offenses include excessive violence and are homicidal in nature. From a legal/forensic standpoint, methamphetamine induced psychosis can be relevant to the legal issues of voluntary intoxication and both diminished capacity and criminal responsibility.

Many forensic mental health professionals confuse diminished capacity and legal insanity. In North Carolina for example, the North Carolina Supreme Court held in *State vs. Shank*, that a person was insane if he or she was incapable of knowing the nature and quality of his or her actions or whether those actions were wrong. While diminished capacity included whether the defendant lacked the capacity to form the state of mind necessary for conviction. Despite their differences, diminished capacity and insanity may be initiated in the same case along with other defenses related to the defendant's state of mind. In fact, the North Carolina Supreme Court has held in *State vs. Silvers*, that state of mind defenses such as insanity and involuntary intoxication are not mutually exclusive and they may coexist in the same case and be considered jointly or severally by the jury. In fact, the same high court in *State vs. Baldwin*, allowed diminished capacity, involuntary intoxication, and insanity to be considered by the jury in the same case.

While courts are unlikely to question strategy of not presenting a criminal defense with methamphetamine intoxication as in *Commonwealth vs. Davenport*, the failure to consider its worthiness may lead to ineffective assistance of counsel. When considering the examination and admissibility of voluntary intoxication to negate mens rea, courts may consider the following points:

1. Bar the use of evidence in all criminal offenses to negate an element of the offense.
2. Allow the evidence of voluntary intoxication when relevant to negate an element of the offense in general intent crimes (those precipitating with intentional misconduct).
3. Limit evidence of voluntary intoxication to negate specific intent offenses (those crimes requiring purpose, premeditation, and deliberation).
4. Voluntary intoxication can be used to negate mens rea only in first degree murder specific intent cases involving a requisite of deliberation and premeditation.

Methamphetamine induced psychosis may cause an individual to have a diminished capacity in forming the requisite intent of an element or elements of an offense. A substance induced psychosis due to methamphetamine intoxication can impair one's premeditation, deliberation, planning, and prior calculation and design necessary for first degree murder. Voluntary intoxication can diminish one's liability in a homicide case from first degree murder to voluntary manslaughter (*State vs. Keeton*). When considering voluntary manslaughter, a person must have committed the act in response to extreme emotional distress and arousal caused by the provocation of the victim. In some jurisdictions, the extreme emotional disturbance defense can lessen murder to manslaughter by negating the defendant's specific intent necessary for the crimes of murder. Further, voluntary manslaughter is based on an unreasonable belief in self-defense and whether a reasonable person would have believed he was in imminent danger of death or serious bodily injury. A methamphetamine induced psychosis may lead a defendant to mistakenly believe his mother was going to destroy the world. As a result, he may have acted in self-defense to protect himself and the universe, ultimately resulting in charges being lowered to voluntary manslaughter (Model Penal Code, 1962). This same fact pattern may also lead a defense attorney to initiate an evaluation and potential plea of not guilty by reason of insanity.

Insanity

Methamphetamine induced psychosis can play a role in an insanity defense. The specific language of the insanity defense statute, depending on the jurisdiction, is important when considering the efficacy of using methamphetamine induced psychosis in this context. Most states incorporate into their insanity statutory language a mental disease/defect component and a cognitive emphasis on knowing/appreciating right from wrong at the time of the offense. Case law will help dictate whether substance induced psychosis qualifies as a mental disease or defect. A defendant may argue that a substance induced psychosis caused him from not knowing the wrongfulness of his acts or impaired his ability to appreciate the nature and/or quality of their acts. In states that utilize a volitional impairment prong (Model Penal Code Test, 1962) in their insanity statute, one may argue that a substance induced psychosis impaired the individual's ability to conform his or her behavior to the requirements of the law during the time of the offense.

About one-third of the states allow for the consideration of an insanity defense based upon the effects of substance abuse and intoxication. The question may be asked to what extent should society excuse and hold a defendant not responsible for his criminal behavior due to his voluntary use of psychoactive substances (Carter-Yamauchi, 1998). In response, many jurisdictions rely on the idea of "settled insanity" which is based on the premise that some chronic users of substances experience a substance induced psychosis and/or dementia which both occurs before and continues after the intoxicated state during the time of the offense (*People vs. Free*). This may especially be relevant in methamphetamine cases due to the drug's residual and long lasting psychotic effects. In fact, many courts have considered this issue and held that a mental disease/defect caused by the long-term effects of substance use constitutes a mental state that necessitates an insanity defense rather than an intoxication defense (*State vs. Harden*).

Courts have struggled with distinguishing at what juncture the mental abnormality caused by the voluntary use of substances becomes insanity. Some courts have declared that the use of substances must cause a permanent or settled state that is distinct from the compulsion to use substances (*People vs. McCarthy*). Other courts have held that psychosis triggered by the voluntary drug use met the test for insanity and the cause behind the insanity (voluntary intoxication) was irrelevant (*State vs. Maik*).

In *State v. Hartfield*, the court held that a defendant may plead insanity when his voluntary use of alcohol or drugs have caused a permanent mental condition that has impeded his ability to know right from wrong. Essentially, the individual has used substances in such a chronic fashion that they experience a long-lasting or permanent organic brain condition. In some cases, they will have experienced substance related psychotic features for years. The law considers this “settled insanity” as more related to a “mental condition,” “disease,” or “defect” relevant to statutory Not Guilty by Reason of Insanity (NGRI) criteria than a one time substance intoxicated state. Simply, prolonged and chronic use has created a condition, syndrome or disorder. While the law has focused on permanent conditions, some case law such as *Porreca vs. State*, has accepted *fixed* rather than permanent conditions. In this case, a Maryland court of appeals held that an offender was allowed to raise an insanity defense relevant to his chronic use of Phencyclidine (PCP) and its causation of a substance induced persecutory delusional psychotic state. The defendant’s drug induced psychotic state permeated temporally beyond the intoxication-crime period.

In *State vs. Scales*, a North Carolina appellate court relied on a test to determine whether a condition of a “fixed or permanent” character commenced by the use of intoxicants may negate a defendant’s criminal responsibility. The court questioned whether the effects of a substance are great

enough to overcome the defendant's mental processes where he can no longer possess the capacity to think or plan. In *Watson vs. United States*, a district court held that such an intoxicated state should constitute an abnormal condition that substantially affects the person's mental or emotional processes and substantially impairs his behavioral controls.

Two California Supreme Court cases are noteworthy relevant to fixed and settled insanity issues. In *People vs. Kelly*, a court held that a defendant who had taken Lysergic acide diethylamide(LSD) and other drugs during a two month period had made the defendant psychotic before and after the offense in which he stabbed his mother. The California Supreme Court held that it did not matter whether a defendant's period of insanity lasted several hours or several months, the defendant was still entitled to utilize an insanity defense.

However, in *People vs. Skinner*, the California Supreme Court heard a case relevant to a defendant free-basing cocaine immediately prior to the offense and he claimed a "toxic psychosis" caused him to lack the mental capacity required by law for criminal responsibility. While experts believed that cocaine psychosis could last between two and eight days after the cessation of its use, the court cited *Kelly* and held that a temporary insanity due to cocaine psychosis was not a fixed or settled insanity applicable to long-continued intoxication effects. The court outlined criteria relevant to the "settled" nature of an insanity defense including:

1. The condition must be fixed and stable.
2. The condition must last for a reasonable duration of time.
3. The condition must not be solely dependent on the ingestion and duration of the drug.
4. The condition must meet the legal definition of insanity.

Other courts in various jurisdictions have offered inconsistent opinions relevant to settled insanity. In *Commonwealth vs.*

Tate, a court held that evidence of temporary rather than settled insanity caused by voluntary intoxication should be excluded. However, despite these notions, a defendant may have success pleading temporary insanity based on voluntary ingestion if the effects of the drug continue when the period of intoxication ends, causing a latent mental condition that remains dormant most of the time but can be triggered by the continued use of drugs (Hassman, 1977).

Conversely, in *Bieber vs. People*, the Colorado Supreme Court considered a settled insanity case in which the defendant experienced an amphetamine induced delusional disorder. While Bieber argued that the drug caused him temporary insanity, the court held that this was not a recognized defense in Colorado and was contrary to public policy. Specifically, the doctrine of settled insanity did not align with their statutory scheme relevant to insanity. The court held that a person voluntarily ingesting substances is aware of the potential consequences on their behavior and is morally culpable for both ingesting the substances and the end result of such ingestion.

The United States Supreme Court in *Montana vs. Egelhoff* supported a state statute that prohibited voluntary intoxication from being considered in determining the existence of a mental state which was an element of a criminal offense. The Court believed that voluntary intoxication was historically an aggravating factor and a defendant did not have a fundamental right to introduce this evidence.

Other courts have rejected the notion of voluntary intoxication as a mental disease relevant to a defense of insanity. In *United States vs. Knott*, the court held that voluntary intoxication coupled with a mental disease will not support an insanity defense under the Insanity Defense Reform Act (IDRA). In *United States vs. Burnium*, the court considered an insanity defense and disregarded whatever

incapacitating effects on one's behavior were attributable to the voluntary use of intoxicants. In addition, in *United States vs. Garcia*, the court held that voluntary intoxication may not be considered when determining whether a severe mental disease or defect exists. The fact that insanity statutes usually incorporate terms such as "mental disorder," "mental disease," and "mental defect," complicates matters when considering substance induced psychotic conditions. Although scholars and practitioners argue what constitutes a "mental disorder," the case law relevant to each jurisdiction may determine these definitions.

In jurisdictions that recognize substance induced states relevant to insanity, diagnostic considerations would include any relevant premorbid/baseline psychiatric disorders that the defendant suffered from before the onset of any methamphetamine use. Any comorbid diagnoses in addition to methamphetamine abuse and dependence would be relevant. For example, a defendant may have a history of schizophrenia and methamphetamine dependence. Differentiating psychotic features emanating from the psychotic disorder versus the methamphetamine induced psychosis may be difficult for the forensic clinician to assess. Relevant to the specific mental state at the time of the offense(s), substance induced psychosis (methamphetamine induced psychosis), methamphetamine intoxication, and chronic methamphetamine dependence would be most relevant diagnostic considerations.

Addiction and mitigation at sentencing

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| Substance use and its link to violence | Jurors often have a negative perspective about substance abuse and abusers of drugs and some argue that these issues should not be presented as mitigating factors at sentencing. In fact, it is asserted by some and it is the common law view, that intoxication is not an excuse, but an aggravating factor of a wrong committed (Am. Jur. 2d <i>Criminal Law</i> § 155). Not |
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infrequently, jurors have used substances themselves and had no problems controlling their use. Others have a history of substance abuse recovery and have overcome addictions. In either condition, jurors may have recognized that they have made a free choice over their decisions and behaviors. Despite this notion of free will, there still remains a strong association of violence with substance use and this nexus should be described in its context to the trier of fact.

Data associated with mock juror beliefs as to whether substance abuse is mitigating at capital sentencing is somewhat conflicted. Barnett, Brodsky & Davis (2004) found that when studying mock juror college students' attitudes of mitigating evidence, a defendant's status as an illegal drug addict and condition of being high on drugs at the time of the homicide was considered mitigating evidence more so than no evidence being presented at all. However, Barnett, Brodsky & Price (2007) studied the effectiveness of presenting drug dependence and alcohol/drug intoxication with undergraduate students. They found that a history of alcohol and drug dependence and alcohol/drug intoxication at the time of the homicide to be aggravating circumstances.

Defense counsel will argue that substance abuse disorders should be considered mitigating by jurors in capital litigation proceedings. How is it mitigating? Critically, substance abuse disorders are the most prevalent diagnoses in forensic and correctional populations and are the most common comorbid condition in cases of violence and criminality (Monahan, Steadman, Silver, Applebaum, Robbins, Mulvey & et al., 2001; Swanson, 1994). In fact, the National Institute on Drug Abuse suggests that half of all violent episodes in the United States occur when the victim and/or offender are under the influence of acute intoxication. Substance abuse likely plays a role in two-thirds of violent crimes, 62% assault; 68% manslaughter; 54% murder or attempted murder; and 52% rape or sexual assault (National Institute on Alcohol Abuse and Alcoholism (NIAAA), 1990). Specific to methamphetamine

use, research has indicated that use among parolees is correlated with a return to custody for any criminal recidivism and self-reported violent acts (Cartier, Farabee & Prendergast, 2006). Further, methamphetamine use has been linked to systemic violence with feuds between gangs trafficking in drugs (Miczek & Tidey, 1989).

While there is data linking substance use and violence, how is the nexus explained? Neurobiological factors associated with aggression include changes in the levels of monoamine transmitters. Specific to amphetamine/methamphetamine use, the drugs release norepinephrine, dopamine, and serotonin (Boles & Miotto, 2003). A decrease in serotonin levels has been implicated in causing psychiatric disorders and aggression.

The chronic amphetamine user is prone to experiencing behavioral pathology and mood altering affects (Fischman & Haney, 1999). As previously mentioned, methamphetamine use can lead to irritability, physical violence, hypervigilance, psychomotor agitation, psychotic and paranoid states, delusions and hallucinations that cause the individual stress. This stress may lead to aggression, delirium, confusion, and intense fear (Kosten & Singha, 1999). Importantly, psychosis is more commonly caused by methamphetamine use than other stimulants such as crack cocaine and the longer duration of half-life action versus other drugs may lead to this result. Further, withdrawal symptoms are significant and often lead to depression, anxiety, agitation, impulsivity, drug craving and may depend on the user's prior psychiatric history (Reiss & Roth, 1993). Those with preexisting histories of psychotic features may be more likely to experience violent outbursts after using large doses of methamphetamine (Roth, 1994). Others with a coexisting major depressive disorders and/or bipolar disorders may be at heightened risk to engage in suicidal and homicidal behaviors while under the influence of methamphetamine.

Likely the strongest risk factor of future violence for mentally ill and non-mentally offenders is a comorbid substance use disorder. This fact can work for and against the defendant. Simply, substance abuse may be presented as a psychiatric addiction impairing one's affective/cognitive functioning and ultimately his volitional control, or an aggravating factor leading the jury to believe chronic substance abuse may cause future violent acts. Whether methamphetamine will serve as a mitigating or aggravating factor will be in the hands of the trier of fact and will be a relevant issue at sentencing. In the case of a bench trial in a non-capital case, a judge may wish to consider treatment and intervention for methamphetamine addiction at sentencing.

Physiological addiction as mitigating

Case law substantiates substance addiction as mitigating in criminal proceedings. In *Pranther vs. Commonwealth*, the court held that a person who is addicted to drugs and is unable to resist the physiological craving for it, cannot be acting voluntarily with respect to its use. In *Commonwealth vs. Tate*, a court held that drug addiction by itself does not constitute a disease leading to psychiatric illness relevant to insanity. However, this decision does not preclude a presentation of this evidence as mitigation at sentencing. Given these holdings, counsel representing the defendant should perhaps be dissuaded from not presenting evidence of addiction and its association to behavior.

When taken in heavy and chronic dosages, methamphetamine has a very addictive quality, and this physiological response is linked to agitation, psychosis, paranoia, and sudden outbursts of aggression (National Institute of Drug Abuse (NIDA), 1998). Physiological effects of methamphetamine include the fact that short-term tolerance creates depleted levels of neurotransmitters and may last two to three days until the neurotransmitter levels are depleted. Continued

over-stimulation of dopamine receptors may cause the receptors to slow down to compensate for increases in levels of dopamine within the synaptic cleft (Bennet, Hollingworth, Martin & Harp, 1998). The individual will require more of the drug to achieve the same levels. Long-term users will often develop dependence, including tolerance and physiological withdrawal symptoms. Many chronic users continue to use despite dramatic negative effects on their bodies and a history of florid psychoses as well as interrupted and disorganized cognitive functioning.

Chronic methamphetamine users experience intense withdrawal symptoms, and relapse is common after abstinence. Once an individual terminates long-term use, he or she is more likely to experience slower thinking and symptoms of depression. Withdrawal symptoms include increase in sleeping (even for days), depressive symptoms, and drug craving (MacKenzie & Heischouer, 1997). Importantly to the forensic mental health professional, a thorough psychological/psychiatric evaluation of mood disorder and psychotic disorder features is relevant in a forensic methamphetamine case as this drug's features of intoxication and dependence include a wide array of psychiatric symptoms.

While free will and voluntary choice/behavior related to the use of a substance cannot be ignored, the addictive qualities of methamphetamine include both physiological tolerance and withdrawal. The disease concept of substance use is described by the tolerance and withdrawal phenomenon, coupled with a "loss of control" or an "inability to abstain" (Jellinek, 1960). The terms "loss of control" and "inability to abstain," suggest a compulsive nature to the addiction process. In fact, The American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revised (DSM-IV-TR), acknowledges at least three of the seven diagnostic criteria being related to volitional impairment relevant to substance use including:

“The substance is often taken in larger amounts over a longer period of time than was intended;”

There is a persistent desire or unsuccessful efforts to cut down or control substance use;”

“The substance use is continued despite knowledge of having a persistent or recurrent physiological or psychological problem that is likely to have been caused or exacerbated the substance (American Psychiatric Association, 2000, p. 197).

The biopsychosocial effects of chronic methamphetamine use cannot be understated. For example, data suggest that susceptibility of drug dependence and methamphetamine induced psychosis is influenced not only by the pharmacological and physiological effects of the drug but also by the genetic elements of the individual (Ohgake, Hashimoto, Shimizu, Koizumi, Okamura, Koike & et al., 2005; Ujike, Harano, Inada, Yamada, Komiyama, Sekine, Sora & et al., 2003).

When a forensic psychologist/psychiatrist is evaluating a methamphetamine related forensic criminal case, the mitigating factors of drug intoxication, abuse, and dependency related to methamphetamine use can be communicated by emphasizing the following factors:

1. Physiological addiction/dependency and its effects on behavior.
2. Settled psychosis/insanity—chronic and long-term use may lead to an organic brain condition, i.e., psychotic features and dementia that occur(ed) before, during, and after the offending behaviors.
3. Substance induced psychosis—even without qualifying for a settled psychosis condition, an acute intoxication state that mimics a genuine psychotic condition may be mitigating.
4. A defendant may be a novice methamphetamine user and not be aware of the specific effects of the drug on behavior.
5. Comorbidity of other psychiatric disorders—many methamphetamine users have other psychiatric conditions, i.e., mood disorders that may be relevant to the onset of the addiction and/or the initiation of violent behavior. The mood disordered defendant may use methamphetamine in part to stabilize his/her mood.

It is commonsensical to propose the argument relevant to the decision in *Bieber vs. People*, that the individual is aware of the potential consequences of his actions and is morally culpable for making a choice to ingest a drug and thus creating the resulting condition. However, addiction medicine may assist in explaining to the trier of fact that the individual may not have had complete control over his substance use or his behavior.

Mitigation at capital sentencing

When considering mitigation at capital sentencing proceedings, the above enumerated mitigating factors are obviously relevant. The trier of fact in capital proceedings must be concerned about the moral culpability, blameworthiness, and deathworthiness of the defendant (Crocker, 1997). In capital cases requiring mitigation at sentencing (*Lockett vs. Ohio*; *Eddings vs. Oklahoma*), any element can be presented as mitigating evidence. Methamphetamine induced psychosis causing either a psychotic condition or a “settled insanity” can be relevant to insanity-like statutory mitigating factors such as “whether at the time of committing the offense, the offender, because of a mental disease or defect, lacked substantial capacity to appreciate the criminality of his conduct or to conform his conduct to the requirements of the law, or the defendant acted under extreme mental or emotional disturbance” (Ohio Revised Code; *United States vs. Lyons*). As it was outlined above, some jurisdictions will consider such pathological intoxication and psychotic states as mental diseases or mental defects relevant to insanity. However, in capital cases, the jury may consider any factor mitigating as they see fit. The condition of “settled insanity,” due to chronic substance use causing physiological damage to the defendant’s brain, may be utilized when associating the defendant’s condition with causation of violence. For mitigation to have meaning at sentencing, it is ideal to associate the substance abuse/dependence/intoxication and

substance induced psychotic state to the murder since juries wish to understand the etiology of the homicide.

When undertaking this descriptive task, the expert witness should consider the following elements:

1. Understand the chronicity of the offender's use, i.e., the frequency, severity, intensity, and duration of his use. Did the offender relapse after multiple treatment attempts.
2. Whether the defendant had a rational motive for the offense or if it was based on a loss of contact from reality, i.e., psychosis or substance induced psychosis.
3. If he/she had preexisting psychiatric symptoms before his methamphetamine intoxicated state. Was he/she involved in psychiatric psychotropic medication treatment and/or relapse prevention treatment at the time of the homicide.
4. If he/she had a pattern of violent offending prior to the homicide, and was this pattern drug related.
5. If he/she could consider alternative courses of actions instead of the offending behaviors, or if the addictive qualities of the drug impaired such a course.
6. The expert should also consider whether the defendant attempted to conceal the offense, avoid detection, and was perceived by others as being intoxicated (Marlowe, Lambert & Thompson, 1999).
7. Characteristics of the offense including its premeditated and planned nature versus a spontaneous and impulsive irrational and unplanned sequence of events should be considered. The drug intoxication's effect on impulse control, planning and sequencing abilities, and motor coordination may also be relevant.
8. Violent acts by the chronic methamphetamine user may be more affective rather than instrumental in nature.

Further roles of the forensic mental health professional

When testifying as an expert witness, the forensic psychologist and psychiatrist ideally will be able to assist the trier of fact in explaining the role of methamphetamine on violent

behavior, assess other premorbid and/or comorbid psychiatric disorders, and differentiate such symptoms of substance induced mood and psychotic disorders from alternative diagnostic possibilities. It is critical for the expert witness to consult with other neuropsychological and neurological professionals. For example, positive emission tomography (PET) assessment may indicate an imbalance in the activity of dopamine D2 receptors in the striatum and serotonin S2 receptors in the frontal cortex leading to the susceptibility of methamphetamine-induced psychosis (Iyo, Nishio, Itoh, Fukuda, Suzuki, Yamaski & et al., 1993). Magnetic resonance imaging (MRI) studies have determined the pattern of structural brain alterations associated with chronic methamphetamine abuse (Thompson, Hayashi, Simon, Geaga, Hong, Sui & et al., 2004). Such imaging has indicated abnormalities in the cortex, hippocampus, white matter, and ventricles. Such abuse has caused cerebral deterioration that leads to impaired memory performance. Other research by (London, Simon, Berman, Mandelke, Lichman, Bramen & et al., 2004), has indicated that chronic methamphetamine users experience lower glucose metabolism in the anterior cingulate and higher levels in the lateral orbitofrontal area, middle and posterior cingulate, amygdala, and ventral striatum cerebellum when assessed with PET scans based on methamphetamine use. These results show a neurobiological basis for mood disorders and anxiety. Neuropsychological testing may assist in assessing the diminished functional aspects of the defendant's current abilities after prolonged methamphetamine use causing potential organic impairment on the brain.

Conclusion

Methamphetamine use can lead to the onset of profound mood and psychotic symptoms. The drug may also exacerbate preexisting symptoms of genuine mental illness. The drug has been associated with violent behavior and has a strong addictive quality. When considering this drug in legal proceedings,

evidence of voluntary intoxication and methamphetamine substance induced psychosis is admissible in some jurisdictions relevant to pretrial affirmative defenses such as diminished capacity and insanity. Additionally, it is admissible in sentencing proceedings, including capital litigation.

When considering the psycho-legal aspects in court-related proceedings, both defense and prosecution should consider their advantageous perspectives. Defense counsel should consider presenting an argument/theory that chronic methamphetamine use is a disease with highly compulsive and addictive properties in which some individuals suffer from a volitional impairment over their behaviors. When considering pretrial and presentence investigations, the diagnosis of a methamphetamine related disorder may be presented as a psychiatric disorder in which the effects of methamphetamine may cause a person to suffer from symptoms that mimic non-substance related psychiatric disorders. Defense counsel must describe the nexus between the effects of methamphetamine and the violent act itself. When considering the effects of the drug on volitional aspects of human behavior, expert testimony may shed light on the addictive quality of the drug on the brain and on one's behavior and relate this to a disorder of the will from a neuroscience perspective (Burns & Bechara, 2007). Substance dependence is associated with impairment of the neural processes allocated to decision making and ability to decide and exercise willpower.

On the other hand, prosecutors should consider emphasizing the voluntary nature of the defendant's drug use and link this loss of control over his use to both the violent act in question, and risk of future violent acts if the individual is not incapacitated in an institution. They should attempt to coordinate an association between the defendant's choice to use methamphetamine, his awareness of the consequences of such use, and the nature of the violent act and its relationship to the drug use.

Finally, the forensic mental health expert witness must remain objective as a neutral fact-finder to the trier of fact and continue to operate within his/her areas of expertise. The expert witness should consider consulting with appropriate professionals such as neurologists and neuropsychologists when appropriate. The expert should be aware of the relevant research regarding methamphetamine use, abuse, dependency/addiction, and drug effects. Additionally, the expert should have an understanding of the legal standards relevant to affirmative defenses and mitigation at sentencing. Finally, the expert should have a functional knowledge of the case law associated with substance induced psychotic states, violent conduct, and legal defenses and sentencing issues.

References

- American Psychiatric Association (APA) (2000). *Diagnostic and Statistical Manual, 4th Ed., Text Revision*. Washington, DC.
- 21 Am. Jur. 2d *Criminal law* §155.
- Ando, K., Hironaka, N. & Yanagita, T. (1986). Psychotic manifestations in amphetamine abuse-experimental study on the mechanism of psychotic recurrence. *Psychopharmacological Bulletin*, 22(3), 763-767.
- Anglin, M., Burke, C., Perrochet, B., Stamper, E. & Dawud-Noursi, S. (2000). History of the methamphetamine problem. *Journal of Psychoactive Drugs*, 32(2), 137-141.
- Barnett, M.E., Brodsky, S.L. & Davis, C.M. (2004). When mitigation evidence makes a difference: Effects of psychological mitigating evidence on sentencing decision in capital trials. *Behavioral Sciences and the Law*, 22(6), 751-770.
- Barnett, M.E., Brodsky, S.L. & Price, R.J. (2007). Differential Impact of Mitigating Evidence in Capital Case Sentencing. *Journal of Forensic Psychology Practice*, 7(1), 39-45.
- Batki, S. & Harris, D. (2004). Quantitative drug levels in stimulant psychosis: Relationship to symptom severity, catecholamines and hyperkinesias. *American Journal of Addictions*, 13(5), 461-470.
- Bennett, B., Hollingsworth, C., Martin, R. & Harp, J. (1998). Methamphetamine-induced alterations in dopamine transporter function. *Brain Research*, 782(1-2), 219-227.

- Bieber vs. People, 856 P. 2d 811 (1993).
- Boles, S. & Miotto, K. (2003). Substance abuse and violence. A review of the literature. *Aggression and Violent Behavior*, 8, 155-174.
- Buffenstein, A., Coel, M. & Combs, B. (1997). *Functional Neuroimaging of Chronic Crystal Methamphetamine Users*. Unpublished grant application, University of Hawaii, John A. Burns School of Medicine.
- Burns, K. & Bechara, A. (2007). Decision Making and Free Will: A Neuroscience Perspective. *Behavioral Sciences and the Law*, 25, 263-280.
- Caton, C.L., Samet, S. & Hasin, D.S. (2000). When acute-stage psychosis and substance co-occur: Differentiating substance-induced and primary psychotic disorders. *Journal of Psychiatric Practice*, 6(5), 256-266.
- Carter-Yamauchi, C. (1998). *Drugs, Alcohol And The Insanity Defense: The Debate Over "Settled" Insanity*. Legislative Reference Bureau.
- Cartier, J., Farabee, D. & Prendergast, M. (2006). Methamphetamine use, self-reported violent crime, and Recidivism among offenders in California who abuse substances. *Journal of interpersonal violence* (21)4, 435-445.
- Commonwealth vs. Davenport, 494 Pa 532, 431 A. 2d 982 (1981).
- Commonwealth vs. Tate, 893 S.W. 2d 368 (Ky. 1995).
- Crocker, P. (1997). Deathworthiness, Differentiating Between Guilt and Punishment in Death Penalty Cases. *66 Fordham Law Review*, 21.
- Eddings vs. Oklahoma, 455 U.S. 104 (1982).
- Ernst, T., Chang, L., Leonido-Yee, M. & Speck, O. (2000). Evidence for long-term neurotoxicity associated with methamphetamine abuse: A 1H MRS study. *Neurology*, 54(6), 1344-1349.
- Fischman, M. & Haney, M. (1999). Neurobiology of stimulants. In Galanter, M. & Kleber, H.D. (Eds.), *Textbook of substance abuse treatment, 2nd ed.*, pp. 21-31. Washington, DC: American Psychiatric Press.
- Gabbard, G.O. (1995). *Treatment of Psychiatric Disorders*, 2nd ed. Vol. 1, pp. 708-720, American Psychiatric Press, Washington, DC.
- Griffith, J.D., Cavanaugh, J. & Oats, J. (1969). Schizophrenic psychosis induced by large dose administration of diamphetaime. *Journal of Psychadelic Drugs*, 2, 42-48.

- Hall, H., McPherson, S., Twemlow, S. & Yudko, E. (2003). Boca Raton, FL: CRC Press.
- Hartel-Petri, R., Rodler, R., Schmeisser, U., Steinmann, J. & Wolfersdorf, M. (2005). Increasing prevalence of amphetamine- and methamphetamine-induced psychosis. *Psychiatric Practice*, 32(1), 13-17.
- Hassman, P.E. (1977). *Drug Addiction or Related Mental State as Defense to Criminal Charge*, 73 A.L.R.3d 16.
- Hunt, D., Kuck, S. & Truitt, L. (2005). *Methamphetamine Use: Lessons Learned*. Analytic Support Program Contract. Cambridge, MA: Abt Associates Inc.
- Iyo, M., Nishio, M., Itoh, T., Fukuda, H., Suzuki, K., Yamasaki, T., Fukui, S. & Tateno Y. (1993). Dopamine D2 and serotonin S2 receptors in susceptibility to methamphetamine psychosis detected by positron emission tomography. *Psychiatry Research*, 50(4), 217-231.
- Iyo, M., Sekine, Y. & Mori, N. (2004). Neuromechanism of developing methamphetamine psychosis: A neuroimaging study. *Annals New York Academy of Science*, 1025, 288-295.
- Jellinek, E. (1960). *The Disease Concept of Alcoholism*. New Haven, CT: College & University Press.
- Kaplan, H. & Sadock, B. (1998). *Kaplan & Sadock's Synopsis of Psychiatry Behavioral Sciences/Clinical Psychiatry* 8th ed. Philadelphia, PA: Lippincott, Williams & Wilkins.
- Kobayashi, H., Ide, S., Hasegawa, J., Ujike, H., Sekine, Y., Ozaki, N., Inada, T., Harano, M., Komiyama, T., Yamada, M., Iyo, M., Shen, H., Ikeda, K. & Sora, I. (2004). Study of association between alpha-synuclein gene polymorphism and methamphetamine psychosis/dependence. *Annals of New York Academy of Science*, 1025, 325-334.
- Kosten, T. & Singha, A. (1999). Stimulants. In Galanter, M., Kleber, H.D. (Eds.), *Textbook of substance abuse treatment*, 2nd ed., pp. 183-193. Washington, DC: American Psychiatric Press.
- Lin, S., Ball, D., Hsiao, C., Chiang, Y., Ree, S. & Chen, C. (2004). Psychiatric comorbidity and gender differences of persons incarcerated for methamphetamine abuse in Taiwan. *Psychiatry Clinical Neuroscience*, 58(2), 206-212.
- Lockett vs. Ohio, 438 U.S. 586 (1978).
- London, E.D., Simon, S.L., Berman, S.M., Mandelkern, M.A., Lichman, A.M., Bramen, J., Shinn, A.K., Miotto, K., Learn, J., Dong, Y., Matochik, J.A., Varughese, K., Newton, T., Woods, R., Rawson, R.

- & Ling, W. (2004). Mood Disturbances and Regional Cerebral Metabolic Abnormalities in Recently Abstinent Methamphetamine Abusers. *Archives of General Psychiatry*, *61*(1).
- MacKenzie, R. & Heischouer, B. (1997). Methamphetamine. *Pediatric Review*, *18*(9), 305-309.
- Marlowe, D.B., Lambert, J.B. & Thompson, R.G. (1999). Voluntary Intoxication and Criminal Responsibility. *Behavioral Sciences and the Law*, *17*, 195-217.
- Miczek, K. & Tidey, J. (1989). Amphetamines: Aggressive and social behavior. *NIDA Research Monograph*, *94*, 68-100.
- Model Penal Code 4.01(1) (Official Draft 1962).
- Monahan, J., Steadman, H.J., Silver, E., Applebaum, P.S., Robbins, P.C., Mulvey, E.P., Roth, L., Grisso, T. & Banks, S. (2001). *Rethinking risk assessment: The MacArthur study of mental disorder and violence*. New York: Oxford University Press.
- Montana vs. Egelhoff, 518 U.S. 37 (1997).
- Murray, J.B. (1998). Psychophysiological aspects of amphetamine-methamphetamine abuse. *Journal of Psychology*, *132*(2), 227-237.
- National Institute on Alcohol Abuse and Alcoholism (NIAAA) (1990). *Alcohol and health: Seventh special report to Congress*. Rockville, MD:Author.
- National Institute on Drug Abuse (1998). Methamphetamine abuse alert. *NIDA Notes*, *13*(6).
- Ohgake, S., Hashimoto, K., Shimizu, E., Koizumi, H., Okamura, N., Koike, K., Matsuzawa, D., Sekine, Y., Inada, T., Ozaki, N., Iwata, N., Harano, M., Komiyama, T., Yamada, M., Sora, I., Ujike, H., Shirayama, Y. & Iyo, M. (2005). Functional polymorphism of the NQO2 gene is associated with Methamphetamine psychosis. *Addiction Biology*, *10*(2), 145-148.
- Ohio Revised Code O.R.C. 2929.04 (B).
- People vs. Free, 447 N.E. 2d 218 (1983).
- People vs. Kelly, 10 Cal. 3d 565, 516 P.2d 875 (1973).
- People vs. McCarthy, 110 Cal. App. 3d 296 (1980).
- People vs. Skinner, 39 Cal. 3d 765 (1985).
- Perez-Reyes, M., White, W., McDonald, S., Hicks, R., Jeffcoat, Hill, J. & Cook, C. (1991). Clinical effects of daily methamphetamine administration. *Clinical Neuropharmacology*, *14*, 352-358.

- Porreca vs. State, 49 Md. App. 522, 433 A.2d 1204 (1981).
- Pranther vs. Commonwealth, 215 Ky. 714 (1926).
- Ray, O. & Ksir, C. (2002). *Drugs Society and Human Behavior*, 9th ed. New York, NY: McGraw-Hill.
- Reiss, A., & Roth, J. (1993). Alcohol, other psychoactive drugs and violence. In Reiss, A.J. & Roth, J.A. (Eds.), *Understanding and preventing violence* (pp. 182-220). Washington, DC: National Academy Press.
- Roth, J. (1994). *Psychoactive substance and violence*. Washington, DC: National Institute of Justice, Office of Justice Programs (February).
- Sato, M., Chen, C.C., Akiyama, K. & Otsuki, S. (1983). Acute exacerbation of paranoid psychotic state after long-term abstinence in patients with previous methamphetamine psychosis. *Journal of Biological Psychiatry*, 18(4): 429-440.
- Sato M. (1992). A lasting vulnerability to psychosis in patients with previous methamphetamine psychosis. *Annals New York Academy of Science*, 28: 160-170.
- Sato, M., Numachi Y. & Hamamura, T. (1992). Relapse of paranoid psychotic state in methamphetamine model of Schizophrenia. *Schizophrenia Bulletin*, 18(1): 115-122.
- Sekine, Y., Minabe, Y., Kawai, M., Suzuki, K., Iyo, M., Isoda, H., Sakahara, H., Ashby, C., Takei, N. & Mori, N. (2002). Metabolite alterations in basal ganglia associated with methamphetamine-related psychiatric symptoms. A proton MRA study. *Neuropsychopharmacology*, 27(30): 453-461.
- Srisurapanont, M., Ali, R., Marsden, J., Sunga, A., Wada, K. & Monteiro, M. (2003). Psychotic symptoms in m psychotic inpatients. *International Journal of Neuropsychopharmacology*, 6(4), 347-352.
- State vs. Baldwin, 330 N.C. 446 (1992).
- State vs. Harden, 260 Kan. 365 (1971).
- State vs. Hartfield, 300 S.C. 469, 388 S.E. 2d 802 (1989).
- State vs. Hartfield, 300 S.C. 469; 388 S.E. 2d 802 (1990).
- State vs. Keeton, 166 W.Va. 77, 272 S.E. 2d 817 (1980).
- State vs. Maik, 287 A. 2d 715 (1972).
- State vs. Scales, N.C. App. 509 (1976).
- State vs. Shank, 322 N.C. 243 (1988).

- State vs. Silvers, 323 N.C. 646 (1989).
- Subcommittee on Crime of the Committee of the Judiciary, House of Representatives, 104th Congress, First Session, October 26, 1995 (Ser. No. 49), *Rising Scourge of Methamphetamine in America*.
- Swanson, J.W. (1994). Mental disorder, substance abuse, and community violence: An epidemiological approach. In Monahan. J. & Steadman, H.J. (Eds.), *Violence and Mental Disorder: Developments in Risk Assessment* (pp. 101-136). Chicago, IL: University of Chicago Press.
- Thompson, P.M., Hayashi, K.M., Simon, S.L., Geaga, J.A., Hong, M.S., Sui, Y., Lee, J.Y., Toga, A.W., Ling, W. & London, E.D. (2004). Structural Abnormalities in the Brains of Human Subjects Who Use Methamphetamine. *The Journal of Neuroscience*, 24(26), 6028-6036.
- Ujike, H., Harano, M., Inada, T., Yamada, M., Komiyama, T., Sekine, Y., Sora, I., Iyo, M., Katsu, T., Nomura, A., Nakata, K. & Ozaki, N. (2003). Nine-or fewer repeat alleles in VNTR polymorphism of the dopamine transporter gene is a strong risk factor for prolonged m psychosis. *Pharmacogenomics Journal*, 3(4), 242-247.
- United States Drug Enforcement Agency. Retrieved March 18, 2007 from <http://www.usdoj.gov/dea/pubs/>
- United States vs. Burnium, 576 F. 2d 236 (1978).
- United States vs. Garcia, 94 F. 3d 57 (1996).
- United States vs. Knott, 894 F. 2d 1119 (1990).
- U.S. vs. Lyons, 731 F.2d 243 (5th Cir. 1984).
- Watson vs. United States, 141 App. D.C. 335 (1970).
- Wolkoff, D.A. (1997). Methamphetamine abuse: an overview for health care professionals. *Journal of Medicine*, 56(2): 34-6, 44.
- Woodson vs. North Carolina, 428 U.S. 280 (1976).
- Yeh, H., Lee, Y., Sun, H. & Wan, S. (2001). Six months follow-up of patients with methamphetamine psychosis. *Zhonghua Yi Xue Za Zhi (Taipei)*, 64(7), 388-394.
- Yudko, E., Hall, H., McPherson, S. & Twemlow, S. (2003). Physiology. In Yudko, E., Hall, H. & McPherson, S. Eds. *Methamphetamine Use Clinical and Forensic Aspects*. Boca Raton, FL: CRC Press.
- Yudko, E., Murray-Bridges, S. & Watson-Hauanio, S. (2003). History of Methamphetamine. In Yudko, E., Hall, H. & McPherson, S. Eds. *Methamphetamine Use Clinical and Forensic Aspects*. Boca Raton, FL: CRC Press.

- Yui, K., Goto, K., Ikemoto, S., Nishijima, K., Yoshino, T. & Ishiguro, T. (2001). Susceptibility to subsequent episodes of spontaneous recurrence of m psychosis. *Drug Alcohol Dependence*, 64(2), 133-142.
- Yui K., Goto K., Ikemoto, S. & Ishiguro, T. (1997). Methamphetamine psychosis: Spontaneous Recurrence of paranoid-hallucinatory states and monoamine neurotransmitter function. *Journal of Clinical Psychopharmacology*, 17(1): 34-43.
- Yui, K., Goto, K. & Ikemoto, S. (2004). The role of noradrenergic and dopaminergic hyperactivity in the development of spontaneous recurrence of methamphetamine psychosis and susceptibility to episode recurrence. *Annals of New York Academy of Science*, 1025, 296-306.
- Yui, K., Ikemoto, S. & Goto, K. (2002). Factors for susceptibility to episode recurrence in spontaneous recurrence of m psychosis. *Annals of New York Academy of Science*, 965, 292-304.
- Yui, K., Ikemoto, S., Goto, K., Nishijima, K. & Kato, S. (2003). Susceptibility to episode recurrence in spontaneous recurrence of methamphetamine psychosis. *Journal of Clinical Psychopharmacology*, 23(5), 525-528.