HOW REVERSIBLE IS METHAMPHETAMINE-RELATED BRAIN DAMAGE?

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I. INTRODUCTION

A large body of medical evidence has accumulated that documents the significant brain damage associated with use of methamphetamine.[1] Dramatic pictures of brain scans show significant brain injury and have been widely published.[2]

Tragic stories of suicide, child abuse, violent crime, and personal disintegration are widespread and graphic. The limited success rate of traditional treatment programs has solidified a perception among judicial and law enforcement personnel that methamphetamine addiction is untreatable. Moreover, another common perception is that attempts to rehabilitate methamphetamine users are futile.

The brain damage associated with methamphetamine cannot be minimized. It is real and devastating to the users and families involved. But a realistic examination of the stages of addiction and time course of the recovery process is essential to forming a realistic prognosis for the methamphetamine addicted individual, taking into account pre-morbid characteristics, current mental status, family and social environment, and available resources. Once-a-meth-addict does not have to be always-a-meth-addict.

This article will first examine the brain structures and biochemical systems affected by methamphetamine, particularly as they relate to

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1. J.C. Maxwell, *Emerging Research on Methamphetamine*, 18 CURRENT OPINION IN PSYCHIATRY 235, 235-42 (2005).

2. *See, e.g.*, Pave M. Thompson et al., *Structural Abnormalities in the Brains of Human Subjects Who Use Methamphetamine*, 24 J. NEUROSCIENCE 6031, 6033 (2004); Nora D. Volkow et al., *Higher Cortical and Lower Subcortical Metabolism in Detoxified Methamphetamine Abusers*, 158 AM. J. PSYCHIATRY 383, 385-86 (2001) [hereinafter Volkow et al., *Higher Cortical*]; Nora D. Volkow et al., *Low Levels of Brain Dopamine D2 Receptors in Methamphetamine Abusers: Association with Metabolism in the Orbitofrontal Cortex*, 158 AM. J. PSYCHIATRY 2015, 2016 (2001); Nora D. Volkow et al., *Association of Dopamine Transporter Reduction with Psychomotor Impairment in Meth Abusers*, 157 AM. J. PSYCHIATRY 377, 378 (2001).

criminal activity and child abuse. Second, the article will evaluate the impact of pre-morbid psychiatric problems as they impact behavior during addiction and recovery from addiction. Third, the article will differentiate between those deficiencies that are not reversible and those that slowly, but surely, heal in most cases. Finally, the article will offer a guarded prognosis for the recovery of the addict and the society he lives in.

II. BRAIN STRUCTURES AFFECTED BY METHAMPHETAMINE

The personality centers in the midbrain are vulnerable to the toxic effects of all addictive drugs, particularly methamphetamine. These structures include the self-control tract (fasciculus retroflexus, ventral tegmental area),[3] the pleasure center (nucleus accumbens),[4] motivational and motor centers (striatum),[5] centers for emotional control (amygdala),[6] appetite and sleep cycle (reticular activating system),[7] judgment and cognitive processes (frontal lobes),[8] and memory (hippocampus).[9]

There are three major mechanisms for brain injury related to methamphetamine addiction.[10] The first is the acute neurotransmitter changes caused by repeated intoxication. Cellular transporters are damaged, and receptors are destroyed.[11] These biochemical changes are reversible after a detoxification process, which lasts from weeks to months. The neurons are otherwise intact and replace their neurotransmission transporters and

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3. Gaylord Ellison, *Continuous Amphetamine and Cocaine Have Similar Neurotoxic Effects in Lateral Habenular Nucleus and Fasciculus Retroflexus*, 598 BRAIN RES. 353, 355 (1992).

4. Sara L. Broom & Bryan K. Yamamoto, *Effects of Subchronic Methamphetamine Exposure on Basal Dopamine and Stress Induced Dopamine Release in the Nucleus Accumbens Shell of Rats*, 181 PSYCHOPHARMACOLOGY 467, 470 (2005).

5. Osman Acikgoz, *The Effects of a Single Dose of Methamphetamine on Lipid Peroxidation Levels in the Rat Striatum and Prefrontal Cortex*, 10 EUROPEAN NEUROPSYCHOPHARMACOLOGY 415, 416 (2000).

6. Susanne Brummelte et al., *Long-Term Effects of a Single Methamphetamine Challenge: Minor Impact on Dopamine Fibre Density in Limbic Brain Areas of Gerbils*, 28 BEHAV. BRAIN FUNCTIONS 2, 6-9 (2006).

7. Stephen Bittner et al., *Effects of a High-Dose Treatment of Methamphetamine on Caudate Dopamine and Anorexia in Rats*, 14 PHARMACOLOGY BIOCHEMISTRY BEHAV. 481, 482-85 (1981).

8. Rita Z. Goldstein et al., *The Orbitofrontal Cortex in Methamphetamine Addiction: Involvement in Fear*, 13 NEUROREPORT 2253, 2253-54 (2002).

9. Makoto Fukumoto et al., *Effects of Acute Administration of Methamphetamine on NARP mRNA in Rat Brain*, 10 ADDICTION BIOLOGY 257, 258-59 (2005).

10. Una McCann & George A. Ricuarte, *Amphetamine Neurotoxicity: Accomplishments and Remaining Challenges*, 27 NEUROSCIENCE & BEHAV. REV. 821, 821-24 (2004).

11. Brian D. Armstrong & Kevin K. Noguchi, *The Neurotoxic Effects of MDMA and Methamphetamine on Serotonin, Dopamine, and GABA-ergic Terminals: An In-Vitro Autoradiographic Study in Rats*, 25 NEUROTOXICOLOGY 905, 912 (2004); Taizo Kita, George Wagner & Tushikatsu Nakashima, *Current Research of Methamphetamine Induced Neurotoxicity: Animal Models Monoamine Disruption*, 92 PHARMACOLOGY SCI. 178, 190 (2003).

receptors over a period of twelve to eighteen months.[12] Anxiety, irritability, rage, apathy, depression, and insomnia are typical meth related personality changes that are reversible when neurotransmitter levels are restored to normal in the weeks and months after detoxification.[13]

The second mechanism of brain injury involves changes in the wiring of the reward center of the brain, ventral tegmental area, nucleus accumbens, and frontal lobe.[14] These structures mediate the sensation of pleasure and the desire or craving for pleasure. Chronic meth use increases the levels of a class of chemicals called cytokines, which direct the cells to produce new synapses and projections from existing neurons. By way of analogy, neural circuits add lanes to the old highways and build entirely new expressways to accommodate increased traffic. These are permanent changes in the way the brain is structured and are thought to underlie the lifelong propensity to relapse in affected individuals, even after years of sobriety.[15]

The third mechanism of brain injury is a function of brain cell death,[16] sometimes involving areas of the brain that are not "redundant," which means other brain centers cannot take over the functions of these areas. The addiction itself is related to cell death in the self-control tract and reward centers of the brain.[17] Other permanently affected areas include the frontal lobes, caudate nucleus, and hippocampus, which are areas implicated in the development of schizophrenia and dementia.[18] Some of the dementia and psychosis seen in advanced addicts is considered irreversible, though not necessarily untreatable.[19]

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12. Volkow et al., *Higher Cortical*, *supra* note 2, at 383-89.

13. Thomas F. Newton et al., *Methamphetamine Abstinence Syndrome: Preliminary Findings*, 13 AM. J. ADDICTION 248, 252 (2004).

14. Hui-Dong Wang et al., *A Shift in Information Flow Between Prefrontal Cortex and the Ventral Tegmental Area in Methamphetamine Sensitized Rats*, 44 INT'L J. PSYCHOPHYSIOLOGY 251, 257-58 (2002) [hereinafter Wang et al., *A Shift*].

15. Kiyofumi Yamada & Toshitaka Nabeshima, *Pro- and Anti-Addictive Neurotrophic Factors and Cytokines in Psychostimulant Addiction: Mini Review*, 1025 ANNALS N.Y. ACAD. SCI. 198 , 198-99 (2004).

16. *See generally* Joseph F. Cubells et al., *Meth Neurotoxicity Involves Vacuolization of Endocytic Organelles and Dopamine Dependent Intracellular Oxidative Stress*, 14 J. NEUROSCIENCE 2260 (1994).

17. Brummelte et al., *supra* note 6, at 2-12; Gene-Jack Wang et al., *Partial Recovery of Brain Metabolism in Methamphetamine Abusers After Protracted Abstinence*, 16 AM. J. PSYCHIATRY 242, 247 (2004) [hereinafter Wang et al., *Partial Brain Recovery*].

18. Yoshimoto Sekine et al., *Association of Dopamine Transporter Loss in the Orbitofrontal and Dorsolateral Prefrontal Cortices with Methamphetamine Related Psychiatric Symptoms*, 160 AM. J. PSYCHIATRY 1699, 1699, 1701 (2003).

19. Hiroshi Ujike & Mitsumoto Sato, *Clinical Features of Sensitization to Methamphetamine Observed in Patients with Methamphetamine Dependence and Psychosis*, 1025 ANNALS N.Y. ACAD. SCI. 279, 284 (2004).

Damage to the cells of the self-control tract is immediate upon the first dose of methamphetamine.[20] Where alcohol abuse may require months, or even years of chronic exposure to produce damage to this degree, methamphetamine differs because it can induce significant cell death within hours of exposure to a single high dose. This is the reason so many addicts say, "Man, I used it once and I was hooked." Damage to this tract results in severe cravings and poor impulse control.[21] The addict is unable to resist temptation when offered methamphetamine. He acts on impulse, unable to suppress his craving for another high.

The pleasure center, or nucleus accumbens, is also damaged by methamphetamine. However, because the pleasure center is a larger structure, it takes longer for the effects to be seen. In the early stages of addiction, the pleasure center is over-stimulated by methamphetamine, and responds to that by increasing the number of receptors and connections.[22] Again, by analogy, the pleasure circuit builds more lanes to accommodate increased traffic. Each high seems better than before. Each high is easier to reach and lasts longer. The user feels confident, powerful, intelligent, bullet proof, and invincible. At high doses of smoked or injected methamphetamine, he gets sexually aroused, has almost unlimited endurance and performance. The high has been described as "ten orgasms all at the same time" followed by many hours of intense arousal.[23] In the early stages of addiction, the crash after the high is just a mild depression, not nearly as bad as a hangover after getting drunk. There is no apparent downside, no adverse consequence to discourage future use. The sensation of power and control contributes to the developing addiction. The meth user feels like he is in control of his drug use.

As the pleasure center is damaged by the over-stimulation, the situation changes in several ways. As the drug destroys the dopamine and serotonin receptors on midbrain neurons, it takes more to get high—more meth, more often.[24] The user "chases the high," switches dealers and recipes, combines

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20. Feng C. Zhou & Sharon Bledsoe, *Methamphetamine Causes Rapid Varicosis Perforation and Definitive Degeneration of Serotonin Fibers: An Immunocytochemical Study of Serotonin Transporter*, 1 NEUROSCIENCE-NET 1, 7 (1996), *available at* http://www.neuroscience.com/manuscripts-1996-009-zhou.html.

21. Akira Nakajima et al., *Anatomic Substrates for the Discriminative Effects of Methamphetamine in Rats*, 91 J. NEUROCHEMISTRY 308, 313-14 (2004).

22. *See* Bow Tong Lett, *Repeated Exposures Intensify Rather than Diminish the Rewarding Effects of Amphetamine, Morphine, and Cocaine*, 98 PSYCHOPHARMACOLOGY 357, 357-62 (1989) (indicating that repeated exposure to meth produced a rewarding effect in rats).

23. FRANK SANELLO, TWEAKERS: HOW CRYSTAL METH IS RAVAGING GAY AMERICA 126 (2005).

24. Ann Marie Brady, *Selective Disruption of Nucleus Accumbens Gating Mechanisms in Rats Behaviorally Sensitized to Methamphetamine*, 25 J. NEUROSCIENCE 6687, 6692 (2005).

the use of meth with the use of heroin or cocaine, trying to reach that elusive "virgin experience." When the meth user is high, he is expansive and talkative, energetic and motivated. At the top, he is jittery and agitated. In withdrawal, he is irritable and depressed.[25]

With continued use, the crash gets worse and lasts longer, usually seven to fourteen days.[26] Acutely, (during the first twenty-four hours of withdrawal) the addict is exhausted. He may sleep for up to three days. He may wake up on the floor and look out the window to see if it is night or day. Subacutely, (up to fourteen days after taking meth into the body) the addict is grouchy and hungry. He may have a splitting headache. He commonly feels that he needs another hit to get back to "normal." With continued use the crash comes to control the addict's life. He starts "tweaking" or using continuously, shooting up as often as every two hours. He stays awake for days at a time, dreading the inevitable crash, and becoming more and more irrational as a result of sleep deprivation.

Loss of neurotransmission in the emotional centers results in anxiety, depression, wild mood swings, fits of rage, and violence.[27] Prolonged periods of insomnia result in hallucinations that are resolved with adequate sleep.[28] As neurotransmission is disabled, symptoms generalize into a true psychosis, indistinguishable from paranoid schizophrenia, and persistent even in recovery in ten percent of addicts.[29]

The violence associated with these mood disturbances is significant.[30] Binge users and "tweakers," those who use high doses repeatedly to maintain intoxication, are often delusional and extremely violent. Addicts lash out at those closest to them, resulting in high rates of domestic violence and child abuse.

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25. Edythe D. London, *Mood Disturbances and Regional Cerebral Metabolic Abnormalities in Recently Abstinent Methamphetamine Users*, 61 ARCH. GEN. PSYCHIATRY 73, 79-80 (2004); Mariana E. Wolf, *Making the Connection between Behavioral Changes and Neuronal Plasticity in Specific Pathways Molecular Interventions*, 2 ADDICTION 146, 146-57 (2002).

26. *See* Catherine McGregor, *The Nature, Time Course and Severity of Methamphetamine Withdrawal*, 100 ADDICTION 1320, 1327-28 (2005) (discussing a "crash period," which is characterized by excessive sleep); *see generally* Wolf, *supra* note 25, at 146-57.

27. Sekine et al., *supra* note 18, at 1700-01 ("[A] longer period of methamphetamine use may cause a relatively greater reduction in dopamine transporter density in the brain, a result that may also be closely associated with the severity of psychiatric symptoms.").

28. Kunio Yui, *Studies of Amphetamine or Methamphetamine Psychosis in Japan: Relation of Methamphetamine to Schizophrenia*, 914 ANNALS N.Y. ACAD. SCI. 1, 1 (2000).

29. *See* Ujike & Sato, *supra* note 19, at 286 ("[T]wo other distinct conditions, chronic schizophrenia in humans and behavioral sensitization in rodents, [have shown] temporal profile is quite similar to that of meth dependence and psychosis.").

30. *See* Joan E. Zweben et al., *Psychiatric Symptoms in Methamphetamine Users*, 12 AM. J. ADDICTIONS 181, 184-85 (2004) (discussing the high levels of violence and aggression that have been studied and are related to meth use).

The addict may fly into a rage and act aggressively and violently toward those with whom he lives. Impaired dopamine and serotonin transmission produce increasing irritability, impatience, and impulsiveness, impairing the addicts relationships with his family and his employer.[31] Additionally, the addict has profound insomnia, but is not distressed by it; he does not feel a need for sleep. He feels highly productive, important, and intelligent. He commonly does not realize he is impatient. Instead, he places the blame on others, thinking other people drive too slowly, or that his children make too much noise.

Meth causes an incredible appetite for sex.[32] The addict cannot perform or climax without using meth, so he uses compulsively and is sexually aggressive.[33] If his wife balks at his demands, he may become violent. This violence is not a result of structural changes, but is rather a neurochemical change in his brain, in which the meth addict experiences the loss of dopamine and serotonin neurotransmission.[34]

Loss of dopamine transmission in motivational centers causes profound apathy, inattention to schedules, work obligations, child care, and personal hygiene. At first the addicted mother is just "too busy" to care for her child. Her mind is racing; she is fidgeting and restless. When the child cries, she responds to him, but she does not notice his needs until he cries out. As she progresses in her addiction, she becomes more irritable and lashes out at him for crying. She is never hungry and so has no food in the house.[35] If the child complains that he is hungry, she throws something at him. If he makes a fuss, she might break his arm.[36] Again, this is the result of a biochemical change, not structural brain damage.

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31. *See* London *supra* note 25, at 73-84 (discussing a study of meth users, which focused on brain activity and the occurrence of depressive and anxiety related symptoms).

32. SANELLO, *supra* note 23, at 126-27.

33. *See* Adam I. Green & Perry N. Halkitis, *Crystal Methamphetamine and Sexual Sociality in an Urban Gay Subculture: An Elective Affinity*, 8 CULTURE, HEALTH, & SEXUALITY 317, 327 (2006) (explaining that meth users tend to be more sexually aggressive and compulsive).

34. See Yoshimoto Sekine et al., *Brain Serotonin Transporter Density and Aggression in Abstinent Methamphetamine Users*, 63 ARCH. GEN. PSYCHIATRY 90, 95-98 (2006) (discussing the significant increase in the magnitude of aggression in meth users when levels of serotonin in the brain are lower).

35. *See* Bittner et al., *supra* note 7, at 484-85 (reporting the results of a study surrounding the anorexigenic effects of meth on rats).

36. *See generally* NAT'L CTR. ON ADDICTION & SUBSTANCE ABUSE, NO SAFE HAVEN: CHILDREN OF SUBSTANCE ABUSING PARENTS (1999) [hereinafter C.A.S.A., NO SAFE HAVEN] (reporting the complicated relationship between substance addition and child abuse).

III. REHABILITATION

Eventually, the addict hits bottom and makes a decision to quit using methamphetamine. Methamphetamine withdrawal is prolonged and miserable; characterized by severe depression, anxiety, fatigue, and nausea. No addict will voluntarily subject himself to meth withdrawal. Many times, addicts are not forced to withdraw from the drug until they are arrested and incarcerated in the county jail. If no one bails him out, the meth addict will "wake-up" to his dependence on meth, and may decide to get clean. He will be motivated to tolerate the prolonged withdrawal period of depression and anxiety, lasting a year or more, in order to stay clean after spending some time behind bars.

Over the first six to twelve months of meth abstinence, the biochemical changes in the brain reverse.[37] New receptors are produced.[38] New transporters are made.[39] Neurotransmission is reestablished in the personality centers of the brain.[40] The depression lifts, the anxiety fades, and the mood swings stabilize. The nightmares go away. The meth addict starts sleeping better and can focus his attention during the day. Drug induced rages may continue for several weeks or months into recovery as neurotransmission is reestablished.[41]

The hallucinations related to meth abuse may be transient, resolving with adequate sleep and a brief course of neuroleptic medications.[42] Nearly ninety percent of meth addicts experience at least occasional hallucinations while intoxicated.[43] Long standing cases are more refractory to treatment. As drug use continues and the psychosis progresses, biochemical and structural changes indistinguishable from paranoid schizophrenia are seen and the prognosis worsens.[44] Hallucinations persist even in the absence of drug use. There is no insight, no understanding for the addict's frequent muse of: "Man, I am seeing things!" Delusions become more encompassing, involving ideas of reference (e.g., people are talking about me), persecution (e.g., people are out to get me), and paranoia. These changes are related to structural damage to the cingulate gyrus and frontal lobes in

37. McGregor, *supra* note 26, at 1328.

38. *See generally* Wang et al., *Partial Brain Recovery*, *supra* note 17, at 247.

39. *Id*.

40. *See generally* Volkow et al., *Higher Cortical*, *supra* note 2, at 383-89.

41. *See* Sekine, *supra* note 18, at 95 ("[M]ethamphetamine induced serotonergic disturbances are responsible for the elevated aggressiveness that is frequently observed . . . in abstinent methamphetamine users.").

42. Yui, *supra* note 28, at 6.

43. Ujike & Sato, *supra* note 19, at 283.

44. *Id*. at 284.

general—the same areas affected in endogenous paranoid schizophrenia. These patients respond, or fail to respond, to the same neuroleptic medications used for endogenous psychosis.[45]

The severity of methamphetamine addiction is related to the significant cellular damage to the self control tract (fasciculus retroflexus and ventral tegmental area).[46] These cells are damaged by even minimal exposure to methamphetamine. The cravings for meth are induced by simple stimuli—an old girlfriend, a piece of tin foil—and the normal method of extinguishing those cravings is no longer operative. Drug use is compulsive, even when the adverse social and physical effects are obvious. The meth addict no longer has the necessary neurons to be able to control his behavior.[47]

But even structural damage to specific neural pathways is not irreversible.[48] The typical perception is that brain damage is irreversible and neurons do not grow back, but that is not entirely true. The self-control tract in particular suffers significant cell injury and death, but over time function can be recovered. Let me explain. Two years ago, I had an injury to the radial and median nerves in my right arm. I was a surgeon. My right hand was nearly paralyzed. My doctor put me in rehabilitation. What did they do in rehabilitation? The therapist made me move my hand. I could not do it. He did it for me. Gradually, over the space of twelve to eighteen months, I regained some function in my hand. The remaining neurons grew new connections, new dendrites, new synapses, and I recovered approximately fifty percent normal function in my right hand. (This personal example makes a great visual aid when teaching this principle to addicts.)

When an addict's self-control tract is damaged, he goes to rehabilitation. What do rehabilitation professionals do? They make him move his self-control tract. He cannot do it himself; so the program does it for him. They make him get up at six in the morning, make the bed and fix breakfast, clean the bathrooms, and mop the floors. Nobody wants to do all those things, but if the addict really wants to get clean, he will do them anyway. By way of analogy, every time he *does* something he did not *want*

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45. Yui, *supra* note 28, at 6.

46. Taku Amano et al., *Repeated Administration of Methamphetamine Causes Hypersensitivity of D2 Receptor in Rat Ventral Tegmental Area*, 347 NEUROSCIENCE LETTERS 89, 91 (2003); Wang et al., *A Shift*, *supra* note 14, at 257-58.

47. Gene-Jack Wang et al., *Regional Brain Metabolic Activation During Craving Elicited by Recall of Previous Drug Experiences*, 64 LIFE SCI. 775 (1999).

48. Hideo Matsuzaki et al., *Brain Derived Neurotrophic Factor Rescues Neuronal Death Induced by Methamphetamine*, 55 BIOLOGICAL PSYCHIATRY 52, 58 (2004); Ralph J. Nudo et al., *Role of Adaptive Plasticity in Recovery of Function After Damage to Motor Cortex*, 24 MUSCLE NERVE 1000, 1013 (2001).

to do, he is doing a bench press with his self-control tract. It grows new connections, new dendrites, new synapses, and he regains self-control.[49]

This process is called "neurogenesis," and it is a more profound change than the recovery of normal neurotransmission between healthy brain cells. Neurogenesis involves regeneration of neural structural elements and it takes longer than just replacing the receptors on an otherwise intact neuron. It is also more dependent on patient participation. Neurotransmitters and receptors will recover with time, even without any active participation by the patient, but neurons will only regenerate when they are repeatedly stimulated to do so.[50]

This prolonged period of neural repair requires significant psychiatric care, counseling, nutrition, and spiritual support. No single method of rehabilitation is universally successful. A coherent, comprehensive approach including psychiatric medications, cognitive behavioral therapy, discipline, nutritional support, and spiritual awakening are necessary and time consuming. Aftercare and supervision are needed for many months, even years, after completion of a competent drug treatment program.

Some of the effects of methamphetamine use persist for many years into recovery. The residual effects of meth are related to duration and intensity of drug use, and include memory loss, hesitancy in speech, irritability, and short attention span.[51] Impairment of the pleasure center results in persistent depression and loss of motivation.[52] With successful drug rehabilitation, the addict can recover the majority of his pre-morbid personality, or characteristics of his personality before he used meth. He might never get his old sense of humor back, but he can learn to control his temper, meet his children's needs, and participate in meaningful relationships.

Those with severe structural damage (cell death) related to their meth use do not make such a rosy recovery. Psychotic features persist in approximately ten percent of recovered addicts, requiring lifelong medication.[53] Many recovered addicts have persistent memory loss, poor judgment and reasoning skills, halting speech, and delayed verbal response related to

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49. Wang et al., *Partial Brain Recovery*, *supra* note 17, at 245-47; Nudo et al., *supra* note 48, at 1006-09.

50. Nudo et al., *supra* note 48, at 1011-12.

51. Kristi S. Rau et al., *Methamphetamine Administration Reduces Hippocampal Vesicular Monoamine Transporter-2 Uptake*, 318 J. PHARMACEUTICAL EXPOSURE THERAPY 676-82 (2006).

52. Thomas R. Kosten et al., *Depression and Stimulant Dependence Neurobiology and Pharmacotherapy*, 186 J. NERVOUS & MENTAL DISEASE 737, 737-45 (1998); Wang et al., *Partial Brain Recovery*, *supra* note 17, at 242-48.

53. *See generally* Yui, *supra* note 28.

prolonged high dose methamphetamine use.[54] Persistent cognitive defects affect the occupational opportunities and communication skills of the recovered addict.[55]

IV. PRE-EXISTING MENTAL ILLNESS

A major consideration in evaluating an individual's probability for attaining success in treatment is the addict's pre-morbid personality traits which may have, in fact, precipitated the addiction.[56] It is well known that certain very common psycho-behavioral problems are significant risk factors for addiction to stimulants—cocaine and methamphetamine. The three most common are Attention Deficit Hyperactivity Disorder (ADHD), Bipolar Disorder, and Personality Disorder. Of meth addicts in treatment, almost thirty percent have a documented history of ADHD or Bipolar Disorder diagnosed and/or treated prior to their addiction.[57]

True ADHD is related to a dopamine deficiency in the mid brain areas responsible for attention and regulation of motor activity levels.[58] Inadequate dopamine release in the motivational areas leads to distractibility—the feeling that someone is always tapping on one's shoulder. It is a scatter-brained feeling, causing academic and vocational failure, impulsivity, and failure to complete assigned tasks (ADD). It is usually associated with dopamine deficiency in the stratum, an area in the midbrain charged with control of motor activity level. To explain by analogy, with inadequate dopamine stimulation, the addict's engine idles fast. He cannot hold still, he wiggles and squirms in the classroom (ADHD). He drives too fast and cannot keep a job as an adult.

The standard treatment for true ADHD is a low dose amphetamine (e.g., Ritalin™/(methylphenidate)), which increases dopamine levels in the midbrain just enough to enhance focus and attention, but not enough to

54. Thompson et al., *supra* note 2, at 6033; J.L. Obert et al., *Incorporating Brain Research Findings into Standard Treatment*, 23 J. SUBSTANCE ABUSE TREATMENT 107, 107-113 (2002).

55. Fukumoto et al., *supra* note 9, at 258-59.

56. C.K. Chen et al., *Premorbid Characteristics and Co-Morbidity of Methamphetamine Users With and Without Psychosis*, 33 J. PSYCHOL. MED. 1407, 1407-14 (2003).

57. *See generally* Russell A. Barkley et al., *Young Adult Follow-up of Hyperactive Children: Antisocial Activities and Drug Use*, 45 J. CHILD PSYCHOL. PSYCHIATRY 195 (2004); Howard Schubiner et al., *Prevalence of Attention Deficit Hyperactivity Disorder and Conduct Disorder Among Substance Abusers*, 61 J. CLINICAL PSYCHIATRY 244 (2000).

58. Gamze Capa Kaya et al., *Technicium 99m HMPAO Brain SPECT in Children With Attention Deficit Hyperactivity Disorder*, ANNALS NUCLEAR MED. 527, 527-31 (2002); Brong-Nyan Kim et al., *Regional Cerebral Perfusion Abnormalities in Attention Deficit Hyperactivity Disorder Statistical Parametric Mapping Analysis*, 252 EUR. ARCH. PSYCHIATRY CLINICAL NEUROSCIENCE 219, 223-24 (2002).

cause intoxication or brain injury at therapeutic doses.[59] As an adult, the ADHD patient has learned to manage his impulses and distractions, and so is not as severely impaired as he was as a child. He is no longer under the care of his pediatrician, but he has learned from his experience with Ritalin™ that stimulants work. He is comfortable with the side effects and views stimulants as "medicine"—not "drugs."

Ritalin™ does not cause sensitization to methamphetamine and so does not directly increase risk of meth addiction.[60] However, the true ADHD patient is at high risk to self-medicate, or administer medication to oneself without consulting a qualified doctor, with methamphetamine as an adult. The addict considers himself to be an intelligent user. He thinks his drug use is "under control," and he resents any challenge to his use of methamphetamine. Meth is about one hundred times more potent than the Ritalin™ he used as a child.[61] Even at low doses, it damages the dopamine neurons in the midbrain, resulting in worsening of his symptoms of impulsivity and fidgetiness. He feels like his problem must be getting worse, so he starts taking more meth to keep things under control. His addiction becomes noticeable to his wife and children a long time before *he* realizes he has a problem.

Some of the children diagnosed with ADHD do not have demonstrable dopamine deficiencies in their brains. They are normal, but immature little boys whose parents have failed to discipline them and whose teachers are eager to medicate them. Stimulants given to normal children will have "beneficial" effects, including improved attention, confidence, and participation in class. Low dose stimulants will make *anyone* more attentive and focused—that is why truck drivers like them so much. The diagnosis of ADHD is thus established by the response to medication, not by any careful diagnostic evaluation.

The pseudo-ADHD adult has carried a diagnosis of ADHD for most of his life. He has learned to avoid responsibility for his actions by blaming his ADHD for behavior that would otherwise be clearly unacceptable. He is also experienced with the use of stimulant medications, and enjoys the feeling of confidence and focus that they induce. He is likely to abuse drugs because he has learned to cope with life by medicating himself.

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59. Nat'l Inst. of Health Consensus Dev. Program, Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder, Consensus Dev. Conference Statement (Nov. 16-18, 1998).

60. Ronald Kuczenski & David S. Segal, *Exposure of Adolescent Rats to Oral Methylphenidate: Preferential Effects on Norepinephrine and Absence of Sensitization and Cross-Sensitization to Methamphetamine*, 22 J. NEUROSCIENCE 7264, 7264-71 (2002); Dorothy E. Willens et al., *Does Stimulant Therapy of Attention Deficit Hyperactivity Disorder Beget Later Substance Abuse? A Meta Analytic Review of the Literature*, 111 PEDIATRICS 179, 182-84 (2003).

61. Kuczenski & Segal, *supra* note 60, at 7264-71.

A similar process occurs with older adolescent children, but this time the diagnosis is Bipolar Disorder. Some of these children have a serious and lifelong mental illness that will require treatment with strong medications.[62] Others are poorly disciplined, moody teenagers whose parents will not take responsibility for controlling their children's behavior. The parents' thought process appears to be that if they can get the doctor to find something wrong with the child, they will not have to admit that maybe they have a spoiled teenager on their hands.

In true Bipolar Disorder, affecting approximately one percent of the population, mood swings are extreme, long lasting, irrational, and often self-destructive. They are not simple temper tantrums in response to a responsibility or restriction. The mood swings alternate between irritable mania with exaggerated self-esteem, and profound depression often culminating in suicide. Patients soon learn to prefer the manic phase of the illness, a high energy state with little need for sleep, rapid thoughts—a flood of great ideas, and a euphoric self-confidence. The Bipolar quickly finds that he can keep himself in a manic phase by using methamphetamine, thus avoiding the crushing depression that inevitably takes him over. As the methamphetamine-related brain damage occurs, his manic phase becomes more pronounced with psychotic features.[63]

The moody teenager who was not taught to control his emotions at age thirteen has also been diagnosed with "Bipolar Disorder." He has been told that he has a biochemical problem and he is not held accountable for his outbursts of anger and rage. He is given medications that are potentially dangerous, again based on their ability to sedate a normal person, not on an extensive diagnostic evaluation. He is likely to engage in drug abuse, not because of any inherent biochemical problem, but because he has learned that he will not be held responsible for his actions.

Obviously, a methamphetamine addict who is also a card carrying Manic Depressive Bipolar Disorder patient has a much worse prognosis for recovery than the spoiled teenager.[64] The spoiled teenager will grow up in the county jail—if his parents are mature enough to leave him there. The Bipolar will not. The true Bipolar will need significant mental healthcare

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62. Hilary P. Blumberg et al., *Significance of Adolescent Neurodevelopment for the Neural Circuitry of Bipolar Disorder*, 1021 ANNALS N.Y. ACAD. SCI. 376, 376 (2004); Kaya et al., *supra* note 58, at 527-31.

63. *See* Alvaro Camacho & Hagop S. Akiskal, *Proposal for a Bipolar Stimulant Spectrum: Temperament, Diagnostic Validation and Therapeutic Outcomes With Mood Stabilizers*, 85 J. AFFECTIVE DISORDERS 217, 221-22 (2003) (discussing the relationship between amphetamine abuse and psychosis).

64. *Id*. at 226-28.

for the rest of his life, and the nature of his illness predicts that he will not get it. The Bipolar thinks he is fine and everybody else needs a shrink.

Pre-existing personality disorders also contribute to risk of addiction and persist after professional rehabilitation. Anti-Social Personality Disorder and Conduct Disorder are usually symptomatic from early adolescence, with cruelty to animals, disregard for feelings of others, and lack of remorse. These individuals have no conscience, no empathy, and no capacity to learn these things. They are at high risk for meth addiction which exacerbates their anti-social tendencies.[65]

Successful drug rehabilitation does not completely heal the mentally ill methamphetamine addict. He will have at least minimal residual personality changes related to his meth use—namely, depression and irritability. He will also have the significant mental illness (e.g., Bipolar, ADHD, or Personality Disorder) he had before he became addicted. Rehabilitation consists of a progressive restoration of personality functions to a new baseline, which cannot be any higher than his pre-morbid baseline. This is the best hope for a depressed, but compensated psychiatric patient.[66] A fully compensated patient has achieved optimal adjustment to his mental illness, usually with a combination of medications, learned coping mechanisms, and a supportive living environment.

V. PROGNOSIS FOR RECOVERY

A realistic estimation of the probability and time course for recovery from addiction is essential for proper sentencing, child custody decisions, death penalty mitigation, and conduct of the drug court. Restoration of the otherwise healthy methamphetamine addict as a responsible citizen is possible. However, this will require significant time, resources, and commitment.[67]

Judicial and law enforcement personnel must recognize the limited cognitive ability, short attention span, and emotional instability of the addict with less than twelve months clean time. Explanations and requirements have to be repeated, written down, and repeated again. The need for prolonged periods of supervision, drug testing, and counseling is exaggerated in the case of meth addiction far beyond what was common practice in the past, especially if children are being placed back in the home.

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65. Chen et al., *supra* note 56, at 1412.

66. Joseph F. Goldberg et al., *A History of Substance Abuse Complicates Remission From Acute Mania in Bipolar Disorder*, 60 J. CLINICAL PSYCHIATRY 733, 738 (1999).

67. *See* Tracy D. Gunter et al., *Drug and Alcohol Treatment Services Effective for Methamphetamine Abuse*, 16 ANNALS CLINICAL PSYCHIATRY 195, 198 (2004) (indicating that patients addicted to meth benefited from a multidisciplinary approach to treatment).

Drug testing should be continued for at least a year after children are replaced in a meth abusing home, and the test should be a truly random unanticipated urine test, or a hair test which is impossible to adulterate.[68] Urine drug testing done only on court dates should not be considered reliable. Anticipated urine tests are far too easily adulterated to be trusted in a court of law. Numerous products designed to provide clean urine at body temperature are widely available on the Internet. Examples include artificial urine kept in a pouch with a miniature heating element to maintain normal body temperature, available from multiple internet vendors. A variety of diluents, additives, and chelating agents are also available.

Mental health care must be provided to those with concurrent psychiatric problems, and compliance with treatment must be encouraged. Placement of children with mentally ill addicts should be done only under the closest supervision, and termination of parental rights should be seriously considered in these cases. Compliance with treatment and proximity of reliable sober relatives can be taken into consideration, but the co-occurrence of serious mental illness argues against placement of a child in such a home.

In death penalty states, a mental health evaluation is essential. Violent crime is very common under the influence of methamphetamine, and drug use is not usually a mitigating factor in death penalty cases. Some states permit the execution of mentally ill persons, but judges and juries should be made fully aware of the exact nature of the mental illness involved and its prognosis for treatment. Since our society no longer houses our mentally ill in hospitals, it is compelled to house them in prisons.

Prison drug rehabilitation should be a national priority. Most people assume that drug treatment is part of the program in our prisons. A few prison systems do make an effort, but most county jails do not. Simply warehousing people will not solve their addiction problems. The cost of implementing real drug rehabilitation in prisons is exceeded only by the cost of *not* providing drug rehabilitation in prisons.[69]

The nationwide experience with drug courts has proven that coerced drug treatment is effective when backed up by the credible threat of incarceration. Long-term inpatient treatment with outpatient follow-up, twelve-step participation, case management, and random drug testing

68. Michael J. Welch et.al., *Two New Standard Reference Materials for the Determination of Drugs of Abuse in Human Hair*, 376 ANALYTICAL & BIOANALYTICAL CHEMISTRY 1205, 1205 (2003).

69. NAT'L CTR. ON ADDICTION & SUBSTANCE ABUSE, CROSSING THE BRIDGE: AN EVALUATION OF THE DRUG TREATMENT ALTERNATIVE TO PRISON (DTAP) PROGRAM 11 (2003).

accountability are successful in seventy to eighty percent of cases.[70] Inpatient treatment is needed when there are serious mental health issues related to meth addiction. However, most addicts can be treated effectively on an outpatient basis as long as there is a credible threat of incarceration—coercion—to maintain patient participation.

Unfortunately, drug courts are limited to addicts facing felony charges. Those arrested for misdemeanor possession are not eligible. The effectiveness of these programs argues for their expansion to the non-felony population, again with the credible threat of incarceration to ensure compliance. Interestingly, there is a recent trend toward Family Drug Courts, which award custody of children to parents based on drug court criteria for compliance with treatment and continued sobriety.[71]

In summary, methamphetamine is far more addictive than cocaine, and causes much more brain damage than cocaine does. Methamphetamine use is escalating and in many areas exceeds cocaine usage. The methamphetamine problem in the United States is not going to just go away. A clear, coherent, well-funded response to this problem is essential to the survival of this nation.

70. *Id*.
71. C.A.S.A., NO SAFE HAVEN, *supra* note 36, at 62.